UNIVERSITI PUTRA MALAYSIA

CADMIUM CONCENTRATION IN ANCHOVY SAUCE (BUDU)ANDHEALTH RISK ASSESSMENT AMONG RESIDENTS IN TUMPAT, KELANTAN

BY
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ABSTRACT

CADMIUM CONCENTRATION IN *BUDU* AND HEALTH RISK ASSESSMENT AMONG RESIDENT IN TUMPAT, KELANTAN

NOR HUSNA HASSAN

Introduction: *Budu* is fish product which obtained by salt fermentation of anchovies or mixture of anchovies with other small fishes. **Objectives:** A cross-sectional study was conducted to determine cadmium concentration in *budu,* estimate the health risk among respondents and to determine relationship between frequency intake of *budu* with cadmium poisoning and health risk encountered by respondents. **Methods:** A total of 160 respondents were selected from the study areas based on a few inclusive criteria. Three *budu* samples were purchased from local mini market. The extraction of *budu* was performed by dry ashing method and cadmium concentration in *budu* was analyzed using Inductively Coupled Plasma Mass Spectrometer (ICP-MS). **Results:** Cadmium was detected in all *budu* samples at range level of 0.0317 to 0.0467 mg/kg which these concentrations not exceed the permissible limit (1mg/kg) by Malaysia Food Regulation 1985. Health risk assessment indicated by average daily dose (ADD), lifetime average daily dose (LADD), hazard quotient (HQ) and lifetime excess cancer risk (LCR). The ADD and LADD value not exceed cadmium oral reference dose (5.0 x 10-4 mg/kg/day) and Provisional Tolerable Weekly Intake (PTWI) (0.007 mg/kg/weekly or 0.001 mg/kg/day). All the respondents had HQ (<1) and LCR ($>1x10^{-6}$) which this results showed acceptable risk encountered by the respondents. The result of this study did not show any significant relationship between frequency intakes of *budu* and prevalence of cadmium poisoning signs. However, significant relationship was found between frequency intake with HQ (r=0.681, p <0.0001) and LCR (r=0.626, p <0.0001). **Conclusion:** cadmium was detected in *budu* samples and its concentration was not exceed the limit permitted by Malaysia Food Regulation 1985 and ADD, LADD, HQ and LCR values also not exceed acceptable limit. Recommended the respondents to monitor their cadmium level by perform biological sample check-up for cadmium exposure.

Key word: Cadmium Concentration, *Budu*, Health Risk Assessment, ADD, LADD, HQ, LCR

ABSTRAK

KEPEKATAN KADMIUM DI DALAM BUDU DAN PENILAIAN RISIKO KESIHATAN DI KALANGAN PENDUDUK TUMPAT, KELANTAN

NOR HUSNA HASSAN

Pengenalan: Budu adalah produk ikan yang diperolehi oleh penapaian garam ikan bilis atau campuran ikan bilis dengan ikan kecil yang lain.**Objektif**: Satu kajian keratan rentas telah dijalankan untuk menentukan kepekatan kadmium dalam budu, menganggarkan risiko kesihatan di kalangan responden dan untuk menentukan hubungan antara kekerapan pengambilan budu dengan keracunan kadmium dan risiko kesihatan yang dihadapi oleh responden. **Kaedah**: Sebanyak 160 responden telah dipilih dari kawasan kajian berdasarkan beberapa kriteria yang telah ditetapkan. Tiga budu sampel telah dibeli dari pasar mini tempatan. Pengesktrakan budu dilakukan dengan menggunakan kaedah pengabuan kering dan kepekatan kadmium dalam budu dianalisis menggunakan "Inductively Coupled Plasma Mass Spectrometer (ICP-MS)". **Keputusan**: Kadmium telah dikesan dalam semua sampel budu dengan purata antara 0.0317 hingga 0.0467 mg / kg yang mana kepekatan ini tidak melebihi had yang dibenarkan oleh Peraturan Makanan Malaysia, 1985 iaitu 1mg/kg. Penilaian risiko kesihatan ditunjukkan oleh dos purata harian (ADD), dos sepanjang hayat purata harian (LADD), HQ dan LCR. Nilai ADD dan LADD tidak melebihi kadmium dos (5.0×10^{-4} mg / kg / hari) dan PTWI (0.007 mg / kg / mingguan atau 0.001 mg / kg / hari). Semua responden mempunyai HQ kurang daripada 1 dan LCR lebih daripada 1×10^{-6} yang mana keputusan ini menunjukkan risiko yang boleh diterima oleh responden. Hasil kajian ini tidak menunjukkan apa-apa hubungan yang signifikan antara kekerapan pengambilan budu dan kelaziman tanda-tanda keracunan kadmium. Walau bagaimanapun, hubungan yang signifikan antara kekerapan pengambilan dengan HQ ($r = 0.681$, $p < 0.0001$) dan LCR ($r = 0.626$, $p < 0.0001$). **Kesimpulan**: kadmium telah dikesan dalam sampel budu dan kepekatan tidak melebihi had yang dibenarkan oleh Peraturan Makanan Malaysia, 1985 dan ADD, LADD, HQ dan LCR juga tidak melebihi had yang boleh diterima. Responden disyorkan untuk memantau tahap kadmium dalam badan mereka dengan menjalani pemeriksaan sampel biologi untuk pendedahan kadmium.

Key word: Kepekatan Kadmium, *Budu*, Penilaian Risiko Kesihatan, ADD, LADD, HQ, LCR

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WHO	World Health Organization
USEPA	United State Environmental Protection Agency
EFSA	European Food Safety Authority
FAO	Food and Agriculture Organization
ASTDR	Agency for Toxic Substances and Disease Registry
ADD	Average daily dose
LADD	Lifetime average daily dose
HQ	Hazard quotient
LCR	Lifetime excess cancer risk
PTWI	Provisional Tolerable Weekly Intake

CHAPTER 1

INTRODUCTION

1.1 Background of Study

According to Malaysia Food Regulation, 1985 (Section 164; Fish Sauce), anchovy sauce (*budu)* is fish product which obtained by salt fermentation of anchovies of *Stolephorus*species or mixture of anchovies with other small fish. *Budu* is a solution fermentated in certain duration together with pickled anchovies. In an addition, *budu* is also one of the traditional food for people in the east coast of Peninsular Malaysia especially among Kelantanese people. It is believed that, each family in Kelantan has at least a bottle of *budu* in their kitchen and they commonly consumed as a condiment or flavoring agent in certain dishes. *Budu* is served together with onion, chilies and lime that have been squeeze out and more delicious if it is mixed with '*tempoyak*'.

Cadmium is one of the pollutants in the environment. Cadmium is a soft metal and electropositive silver-white color and has an atomic number of 28, the molecular weight of 112.41 and melting point of 321 °C (Klaassen *et al*., 1989) while its boiling point is 767 °C. Metal cadmium cannot be break down from its original form in water, soil and air for a long time (Jamal and Zailina, 1995). Most of the cadmium present in the environment is from the mining process, combustion, industrial and disposal.

Cadmium is used in electroplating, in nickel-cadmium storage batteries, as a coating for other metals, in bearing and low melting alloys and as control rods in nuclear reactors. Besides that, cadmium compounds have numerous applications, including dyeing and printing textiles, astelevisionphosphors, as pigments and enamels and semiconductors and solar cells. Inhalation of cadmium may cause lung damage and death. Ingestion of food and drink contaminated with cadmium can cause diarrhea, vomiting and irritation of abdominal (ATSDR, 2009).

1.2 Problem Statement

In an addition to physiological and/or nutritional safety, chemical safety in terms of food contaminants has drawn attention as more media reports appear on heavy metals and pesticides in foods. Serious heavy metal contamination does not only because of the toxicity effect to human but also because of its bioaccumulation and biomagnifications in the food chain (Begum *et al*., 1999). Concept of Hazard Analysis Critical Control Point (HACCP) has been know and accepted by the food industry and scientific organization as well as the current control. This concept introduced for the awareness among consumer about the food safety.

There are variety of incident that involve of heavy metal contamination occur in the ocean such asmercury contamination in Minamata, Japan in 1956 (Langford and Ferner, 2002; Tomiyasu *et al*., 2006) and pollution that occur in Amazon, Brazil (Barbosa *et al*., 2009). The impact from the pollution causes the variety of health effect to hand and leg, vision problem, hearing damage and speech problem. This situation shows that, the threat of heavy metal is serious to human health.

It is well known that ingestion of contaminants in excessive amounts can have detrimental effects on health (Lowik, 1996). Furthermore, an increased risk of lung cancer has been reported following inhalation exposure in occupational settings. Acute pulmonary symptoms of cadmium exposure are usually caused by the inhalation of cadmium oxide dusts and fumes, which results in cadmium pneumonitis characterized by edema and pulmonary necrosis. Chronic exposure sometimes produces emphysema severe enough to be disabling (ATSDR, 2008).

Besides that, the kidney is generally regarded as the organ most sensitive to chronic cadmium poisoning. The function of renal tubules is impaired by cadmium as manifested by excretion of both high molecular mass protein (such as albumin) and low molecular mass proteins. Maternal exposure to cadmium is associated with low birth weight and an increase of spontaneous abortion (ATSDR, 2008).

Food and Agriculture Organization (FAO), United Nations Environmental Programme (UNEP) and Graduate Employability Management Scheme (GEMS) shown that the concentration of cadmium that has been study in a day in the range 0.006 mg/day until 0.0096 mg/day with average 0.031 mg/day (Tukimat *et al*., 2006) conducted previous study.

The aim of this study is to determine whether there is cadmium present or not in *budu.* According to previous study, there are a lot of aquatic life especially fish contaminated with heavy metal. Heavy metals accumulate in fish from water, food, sediment and some suspended particulate materials (Agusa *et al*., 2005). Varied levels of heavy metals in fish samples in Malaysia were reported from various collection sites (Hajeb*et al*., 2009; Irwandi *et al*., 2009; Kamaruzzaman *et al*., 2011). Findings of these studies are consistently important to indicate the safety of local fish products in terms of the heavy metals concentration. The most of fishes are contaminated with mercury, cadmium and lead. Contamination statusof organochlorines (Monirith *et al*., 2003), tributyltin(Tong *et al*., 1996; Sudaryanto *et al*., 2002, 2004)andheavy meals (Ismail *et al*., 1993;Yap *et al*., 2002, 2004) have been reported in coastalarea of Malaysia.

1.3 Study Justification

Fish is considered as significant indicator to assess the pollution level of heavy metal and risk to human (Begum *et al*. 2005, Papagiannis *et al*., 2004). Research on heavy metal in fish is important to ensure the food safety and human health. This is because, dangerous substance like cadmium that polluted the ocean can accumulation in sea life through food chain. Today, there are lot of food are contaminated with heavy metal and most probably the *budu* is not an exception.

Furthermore, there are many of news about water pollution occurred in our country especially in the river and sea where the rapid economical growth has resulted in increasingproduction and usage of toxic chemicals such astrace elements in Malaysia. The Straits of Malacca issubjected to a great variety of pollutants due to its strategiclocation as a major international shipping lane andthe concentration of agriculture, industry and urbanizationon the west coast of Peninsular Malaysia (Abdullah *et al*., 1999). Moreover, the Strait of Malacca is one ofthe most vulnerable areas to contamination by oil spills but srait of Malaccais the most important fishing ground in Malaysia where approximately 70% of total fish landingsof the country (Eng *et al*., 1989).

Because of this situation, the aquatic life maybe contaminated with the polluted substances where the polluted substances most probably is heavy metal. Then, the contaminated anchovies will be used to process the *budu*. Therefore, this study is going to be done because in our country there is lack of research about the contamination of cadmium in food especially *budu*. Thus, through this research, control measure will be taken to reduce cadmium exposure level from consumption of *budu* and to reduce health risk caused by cadmium.

This study depends on contamination of cadmium in fish because fish is main ingredient to make *budu*. Although, there are person had be done the research about the benefit intake the *budu* but we do not know the *budu* that we take is free from heavy metal contamination or not. The contamination of heavy metal in *budu* maybe occurs naturally in anchovies and small fish or it is contaminant during fermentation process. *Budu* shall be clean and wholesome and shall not contain other extraneous matter (Malaysia Food Regulation, 1985). The limit of cadmium are allow in *budu* is 1 mg/kg(Malaysia Food Regulation, 1985). Hopefully the outcome of this study will answer the question about the level of cadmium in *budu* and the exposure that faced by who consumed *budu* in their daily menu.

1.4 Conceptual Framework

Figure 1 shows the model design to explain the problems that to be studied in this research.

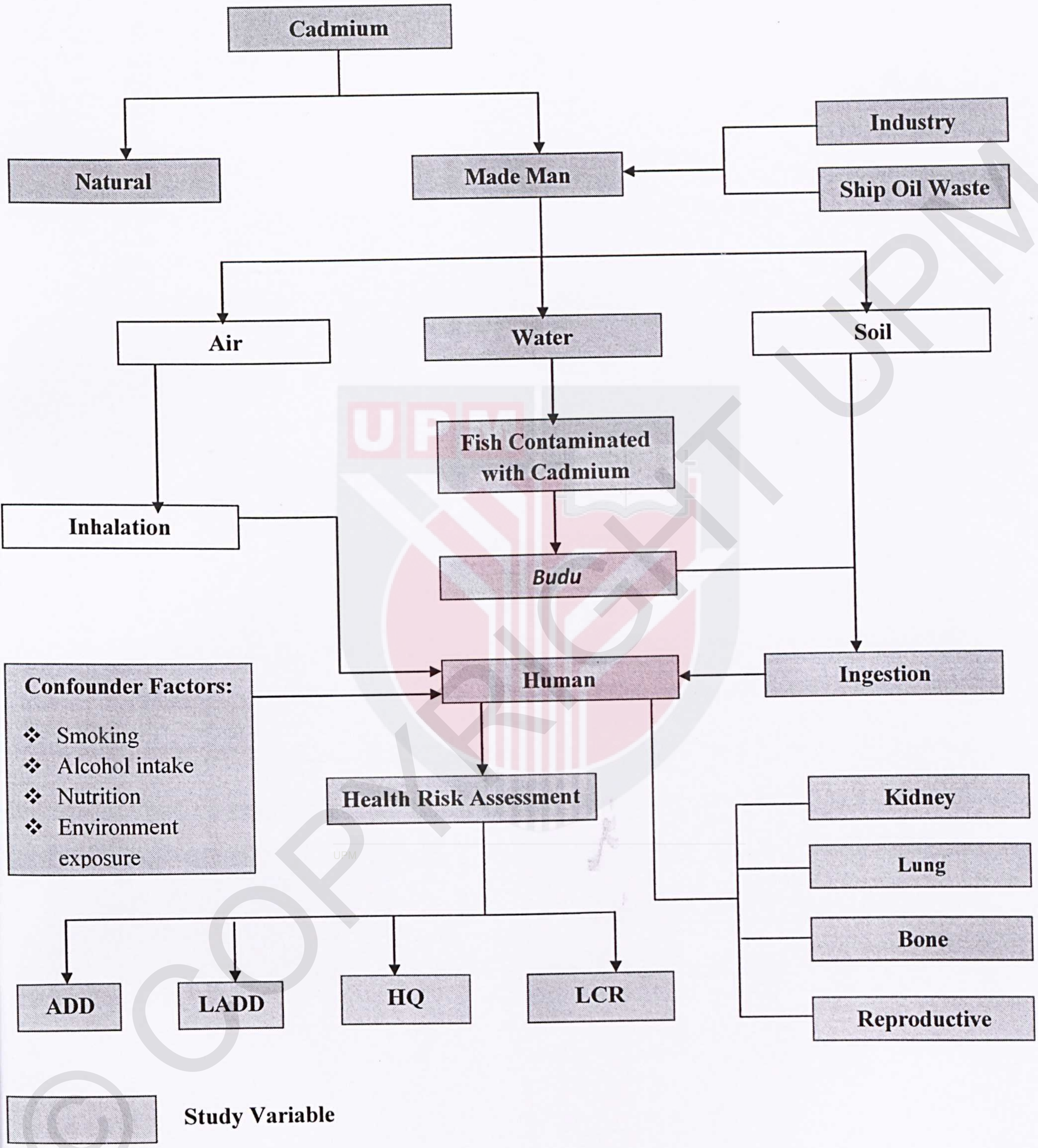

Figure 1: Conceptual Framework

1.5 Definition

1.5.1 Conceptual Definition

Cadmium Concentration in Food

People will be exposed to cadmium through their diet, since cadmium is absorbed into plant and animal foods that they eat. People will be exposed to higher amounts of cadmium by breathing cigarette smoke. In the workplace, people will be exposed where cadmium is used or generated, such as in battery manufacturing, metal soldering, or welding. When people take the food, over dose of cadmium can severely irritate the stomach and cause vomiting and diarrhea. Inhalation to high levels of cadmium will damages people's lungs and can cause death. Exposure to low levels of cadmium in air, food, water, and particularly in tobacco smoke over time may build up cadmium in the kidneys and cause kidney disease and fragile bones (Jarup *et al*, 2009).

Health Risk Assessment

In recent years, the public has become increasingly aware of the presence of harmful chemicals in our environment. Many people express concerns about pesticides and other foreign substances in food, contaminants in drinking water, and toxic pollutants in the air. Health risk assessment is a scientific tool designed to help answer these questions. There are four steps involve in health risk assessment; hazard identification, exposure assessment, dose response assessment and risk characterization. Risk assessments can also guide regulators in abating environmental hazards. Furthermore, health risk assessments can help risk managers weigh the benefits and costs of various alternatives for reducing exposure to chemicals (USEPA ,2005)

1.5.2 Operational Definition

Cadmium in *Budu*

The samples of *budu* were obtained at the mini market at Tumpat. The determination of cadmium concentration in *budu* was conducted by using Inductively

Coupled Plasma Mass Spectrometer (ICP-MS) model Perkin Elmer ElanDre-e. These systems generate superior results and provide faster, more accurate analyses. The unit for the ICP-MS is μg/L.

Health Risk Assessment

The health risk Assessment was evaluated through the calculating of lifetime excess cancer risk (LCR) and hazard quotient (HQ) and the Food Frequency Questionnaire to estimate the health risk of the respondents who exposed to cadmium through *budu* intake in their daily diet. Because of cadmium is carcinogenic and non-carcinogenic agents to human, the hazard value of carcinogenic and non-carcinogenic effect were calculated. The lifetime excess cancer risk (LCR) and hazard quotient (HQ) calculation was shown in the methodology part.

1.6 Objective

1.6.1 General objective

To determine cadmium concentration in anchovy sauce (*budu*) and health risk assessment among residents in Tumpat, Kelantan.

1.6.2 Specific objectives

1) To determine the socio-demographic of the respondents.
2) To determine the frequency intake of *budu* by the respondents.
3) To determine the concentration of cadmium in the *budu.*
4) To determine the prevalence of signs associated with cadmium poisoning among respondents
5) To determine the health risk of respondents from the consumption of *budu* indicates by lifetime average daily dose (LADD), average daily dose (ADD), hazard quotient (HQ) and lifetime excess cancer risk (LCR).

6) To determine the relationship between frequency intake of *budu*and prevalence of signs associated with cadmium poisoning among respondents
7) To determine relationship between the frequency intake of *budu* and health risk encountered among respondents.

1.7 Hypothesis

1) There is significant relationship between the frequency intake of *budu* and health risk encountered by respondents.
2) There is significant relationship between frequency intake of *budu* and prevalence of signs associated with cadmium poisoning among respondents.

1.8 Limitation

The limitation in this study is did not take the biological sample of respondents. Besides that, duration of study is tooshort and only one village was selected in this study. In this study, we took only one village because there was not enough of manpower to conduct this study. Furthermore, budget was provided in this study was also limited.

CHAPTER 2

LITERITURE REVIEWS

2.1 Heavy Metal

Heavy metals are thus commonly defined as those having a specific density of more than 5 g/cm3. Heavy metals occur naturally within the environment and have been utilized in a wide variety of ways including building materials, electronics, medicines, and even to sweeten wine (Jarup, 2003). Heavy metals can travel within the environment via the air, water, and soil and they are not biodegradable. Depending on the specific heavy metal, any number of adverse health effects can be observed. Some of the aforementioned metals can cause health problems including but not limited to kidney and skeletal damage, neurological and psychological symptoms, gastrointestinal problems, liver damage, disturbances of the cardiovascular system, and skin lesions (Jarup, 2003 ; USEPA, 2009).

2.2 Cadmium

Cadmium is a metallic element of Group IIB (Zn, Cd, Hg) of the Periodic Table whichdictates its prevalent valence state of +2. This metal is soft, ductile and silvery-white or bluish-white. Cadmium chloride and cadmium sulfate are soluble in water. Cadmium exists as the hydrated ion or as ionic complexes with other inorganic or organic substances . This metal is rarely found in the pure state in nature. Elemental cadmium has a relatively high vapour pressure, but the vapour is rapidly oxidised to produce cadmium oxide in the air. Gases like carbon dioxide, water vapour, sulfur dioxide, sulfur trioxide, and hydrogen chloride react with elemental cadmium. For example, smoke stacks to form salts that are emitted to the environment (ATSDR, 2009).

Cadmium also is an inorganic toxicant of great environmental and occupational concern and its exposure is associated with lung cancer in humans (Paschal *et al.*, 2000; Sorahan and Esmen, 2004). Many epidemiological observations suggest that mortality from lung cancer has been increased in cadmium workers who had experienced the very high levels of cadmium, mainly as oxide dust or fumes (Lamm *et al.*, 1991; James *et al.*, 2004).

2.3 Cadmium in Food

Cadmium is a contaminant found in most human foodstuffs, which renders diet a primary source of exposure among nonsmoking, no occupationally exposed populations (Clemens 2006; McLaughlin *et al.* 2006; Franz *et al.* 2008) , but concentrations vary greatly, and individual intake also varies considerably due to differences in dietary habits. Besides that, people who regularly consume shellfish and organ meats will have higher exposures. In general, leafy vegetables such as lettuce and spinach, potatoes and grains, peanuts, soybeans, and sunflower seeds contain high levels of cadmium.

In an addition tobacco leaves accumulate high levels of cadmium from the soil. Cadmium absorption after dietary exposure in humans is relatively low (3–5 %) but cadmium is efficiently retained in the kidney and liver in the human body, with a very long biological half-life ranging from 10 to 30 year. Based on estimation of cadmium intake, more than 80% of the food-cadmium comes from cereals, vegetables and potato (Olsson *et al.*, 2002). The average cadmium intake from food generally varies between 8 and 25 µg per day (Larsen et al., 2002;Olsson *et al.*, 2002; Llobet *et al.*, 2003; Egan *et al.*, 2007).

2.4 Cadmium in Marine Environment

Cadmium can remain present in lake and river suspended particles for 1-3 years. In coastal sediments its estimated half-live is 2 years, and in oceanic water it is estimated to be about15,000 years (Cabrera *et al.*, 1998). Soluble forms of cadmium migrate in water. Insoluble forms of cadmium are immobile and will deposit and absorb to sediments. Reported bioconcentration factors range from 113 to 18,000 for invertebrates from 3 to 4,190 for fresh water aquatic organisms and from 5 to 3,160 for saltwater aquatic organisms. The crabs and lobsters are able to accumulate, mainly in the digestive gland, between 10 % to 40 % of the cadmium present in their food (Neff, 2002).

Today, fish has become the main supply of protein besides meat and poultry products and contributed to a large percentage of dietary protein globally. In most Asian countries, especially in Southeast Asia like Thailand, Indonesia and Malaysia fish is taken as the main dish of their diet. However, fish that we take maybe contain mercury, arsenic, and cadmium that can cause negative effects to health. Basically, the marine organisms accumulate contaminants such as metals from the environment and have been extensively used in marine pollution monitoring programmes (Mora *et al*., 2004).

Dietand food of animals origin are the most predominant sources (>90%) of heavy metals and other chemical contaminants to human (Svensson, 1991). Although, fish and shellfish only represent small percentage (10%) of these contaminants, these foods are one of the major routes of the contaminants in the body (Harisson *et.al.*, 1998). About 50% of the cadmium that reaches the sea comes from human activities such as industrial waste, fertilizers containing phosphate of animal origin.

Study on Molluscs shown that it tend to accumulate cadmium in a quantity moderately higher than other organisms, since the element is essential for the formation of haemocyanin, a copper-containing protein (UNEP/WHO, 1989). High concentration of cadmium are present in molluscs and crustaceans such as oysters and other bivalve molluscs, cephalopods and crabs especially the parts with brown meat.

Sadiq (1992) summarized cadmium concentrations in marine sediments ofseveral countries in Europe, USA and Canada, notingthat levels ranged from 0.04 to 31.80 µg/ g and he also suggested that cadmium concentrations in relatively pristine marine sediments are usually below 1.00 µg/g. Based on this suggestion, about 40% and 20% of the total sampling sites of the intertidal and offshore sediments, respectively, had higher cadmium levels than pristine marine sediments. Another study reported 116.4

mg/kgcadmiumin SVM from CishanFisheries, Shandong Province, China(Mai *et al.*, 2006).

Among the studies done in Malaysia, Ismail (1993) reported 1.00–5.00 µg/g for cadmium in theBintulu, Sarawak coastal sediment. Besides, the cadmium concentrationsin sediments sampled from offshore Terengganu,Pahang, Sarawak and Sabah were as high as 5.60 µg/g(Shazili *et al.*, 1988) while for the samples collected inthe South China Sea the cadmium levels ranged from 0.41 to2.39 µg/g (Shazili *et al.*, 1988).

2.5 Sources of Cadmium

Cadmium (Cd) is a heavy metal found as an environmental contaminant, both through natural occurrence and from industrial and agricultural sources. Two major sources of the environmental toxic heavy metal cadmium for humans are cigarette smoke and food because of its high rate of soil-to-plant transfer (Satarug and Moore, 2004; Satarug *et al.*, 2010).

2.5.1 Air

Industrial activities are the main sources of cadmium release to the air and emissions from anthropogenic sources have been found to exceed those of natural origin by an order of magnitude (ATSDR, 1999). Cadmium and cadmium compounds have negligible vapour pressures but may exist in air as suspended particulate matter derived from sea spray, industrial emissions, combustion of fossil fuels, or the erosion of soils. In processes that involve extremely high temperatures such as the iron and steel industries, cadmium can volatilize and be emitted as a vapour (Wilber *et al.*, 1992).

Total emission to air from natural sources mainly volcanoes is estimated at about 150-2,600 tonnes per year. These figures may be compared to an estimate of total anthropogenic air emission in 1995 of approximately 3,000 tonnes. The largest source of atmospheric cadmium is non-ferrous metal production, which contributes about 75 % of total anthropogenic cadmium emissions (Pacyna and Pacyna, 2001).

In urban areas of the Eropean Union, cadmium concentrations in air are in the range between 1 and 10 ng/m3. The principal chemical species in air is cadmium oxide, although some cadmium salts, such as cadmium chloride, can enter the air, especially during incineration (IARC, 1993). These are stable compounds that do not undergo significant chemical transformation. Cadmium pollutants present in the air may be transported from a hundred to a few thousand kilometres and have a typical atmospheric residence time of about l-10 days before deposition occurs by wet or dry processes (ATSDR, 1999).

2.5.2 Soil

Cadmium in soil is derived from both natural and anthropogenic sources. Atmosphericpollution, phosphate fertilisers and sewage sludge appear to be the major contributors tocadmium deposition in agricultural soils. Atmospheric cadmium deposition onto soil hasgenerally decreased over the last 20 years in Europe. Recent studies have documented thatatmospheric emissions do not presently have a significant impact upon the cadmium contentof soils (Bak *et al.*, 1997).

Cadmium in soil may exist in soluble form in soil water, or in insoluble complexes withinorganic and organic soil constituents. Cadmium in soil tends to be more available when thensoil pH is low. Background cadmium levels in surface soils range from 0.01 to 2.7 mg/kg, though values up to 1,781 mg /kg soil have been reported from very contaminated sites (Kabata-Pendias,2001). In Europe, the mean cadmium concentration in cultivated soils is 0.5 mg/kg (Davister, 1996; Underwood and Suttle, 1999). A more general picture of common ranges of cadmium in soil is given by the latest report of United Nation Environment Programme (UNEP, 2006) with maximum values of about 50 mg/kg.

Elevated concentrations of cadmium in soils (compared to background values) have also been reported following the application of sewage sludge and farmyard manure, which containvariable and occasionally excessive cadmium concentrations (Eriksson,2000; Bergkvist *et al.*, 2003). Since cadmium is taken up by plants, an increased soil concentration can result in increased levels in food and feeds (UNEP, 2006). However, the concentration of cadmium in soils is not the primary determinant of cadmium in plants. Cadmium is much less mobile in soils than in air and water. The major factors governing cadmium mobility in soils are speciation, pH, soluble organic matter content, hydrous metal oxide content, clay content and type, presence of organic and inorganic ligands, and competition from other metal ions.

2.5.3 Water

In surface water and groundwater, cadmium can exist as free ion, or as ionic complexes with other inorganic or organic substances. While soluble forms may migrate in water, cadmium is relatively non-mobile in insoluble complexes or adsorbed to sediments. In seawater, the most common forms are chlorine ion complexes, and in freshwater the free hydrated or carbonated ions (depending on the pH) are the most frequent forms. With regard to aquatic systems, rivers transport large quantities of cadmium from weatheringand erosion processes to the world's oceans.

An annual gross input of about 15,000 tonnes of cadmium has been estimated. Moreover, between 900 and 3,600 tonnes of cadmium are estimated to be deposited into aquatic environments throughout the world by atmospheric deposition of emissions originating from anthropogenic and natural sources (UNEP, 2008). For example, the contribution of cadmium via rivers into the marine environment of the North Sea is of the same order of magnitude as the atmospheric deposition as the other main regional dispersion pathway of cadmium. The oceanic residence time of cadmium has been estimated to be about 15,000 years. This indicates that cadmium may be accumulated and transported in significant amounts over long distances in the ocean. There are large natural reservoirs of cadmium in the oceans.

2.5.4 Wastes

Global cadmium releases to the terrestrial environment is in total between 2,500-15,500tonnes per year, with atmospheric deposition being the dominant source. An additional 7,500-29,500 tonnes per year are assumed to be directed to landfills and various deposits in the formof discarded products and production waste (Nordic Council of Ministers, 2003). The longterm fate of cadmium accumulated in landfills is uncertain and may represent a future source of releases. The handling of wastes may lead to elevated local and regional releases, especially in developing countries (UNEP, 2002).

2.5.5 Tobacco smoke

Tobacco smoking is another important source of cadmium exposure. The tobacco leaves accumulate cadmium in a manner similar to certain food from plants. One cigarette may roughly contain 1–2 μg cadmium depending on the type and brand (Olsson *et al*., 2002). Roughly 10% of the cadmium content is inhaled with an approximate 50% absorption in the lung. It is estimated that a person smoking 20 cigarettes per day will absorb about 1 μg cadmium daily.

2.6 Health Effects of Cadmium

About 25–60% of the cadmium you breathe will enter our body through the lungs. A small amount of the cadmium in food and water (about 5–10%) will enter the body through the digestive tract. If human body do not have enough iron or other nutrients in diet, the human are likely to take up more cadmium from food than usually.Because of the long half-life of cadmium(~30 years), it has been implicated in several human diseases. It hasalso been demonstrated to cause pathological changes in organssuch as liver, brain, kidney, heart, bone, and lung (Johnson, 2001; Mannino *et al.*, 2004 ; Kim etal., 2005;Arisawa et al.,2007; Houston, 2007; Gallagher *et al.*, 2008). Cadmium is also a toxic environmental contaminant that is carcinogenic in humans and laboratory animals (IARC, 1993; Waisberg *et al.*, 2003).

2.6.1 Renal Effects

Cadmium is primarily toxic to the kidney, especially to the proximal tubular cells where it accumulates over time and may cause renal dysfunction. After prolonged and/or high exposure the tubular damage may progress to decreased glomerular filtration rate, and eventually to renal failure.A safe intake limit of 7 µg cadmium/week/kg body weight was set based on the critical renal cadmium concentration of between 100 and 200 µg/gwet weight that corresponds to a urinary threshold limit of 5–10 µg/g creatinine (WHO, 1993).

Examination on provisional tolerable weekly intake (PTWI) model by studying cadmium accumulation in kidneys and livers of environmentally exposed subjects suggested that the safe intake level for an adult should be < 30 µg/day (Satarug*et al*., 2000, 2003). They also showed that cadmium accumulation in the kidney cortex increased with age, reaching a plateau by 50 years of age (Satarug *et al*. 2002).

Long-term exposure to high-dose cadmium causes Itai-itai disease. This disease affects mainly women and is characterized by severely impaired tubular and glomerular function and generalized osteomalacia and osteoporosis that result in multiple bone fractures (Inaba *et al*. 2005). An estimate of cadmium intake, based on historic rice cadmium content, in the Itai-itai disease endemic area during the 1960s was 600 µg/day, and the threshold lifetime intake was estimated to be between 1,580 and 2,000 mg of cadmium (Kobayashi *et al*. 2002, 2006).

2.6.2 Cancer

The International Agency for Research on Cancer has classified cadmium as a human carcinogen (Group 1) on the basis of occupational studies. Lung cancer has been found in some studies of workers exposed to cadmium in the air and studies of rats that breathed in cadmium. United State Department of Health and Human Services has determined that cadmium and cadmium compounds are known human carcinogens. International Agency for Research on Cancer has determined that cadmium is carcinogenic to humans. The EPA has determined that cadmium is a probable human carcinogen. There is sufficient evidence that cadmium metal and a number of cadmium compounds, such as cadmium chloride, oxide, sulfate, and sulfide, are carcinogenic in animals. Increased rates of testicular, prostate, and lung cancer in animals have been described (ATSDR,1999; Sahmoun *et al*., 2005).

Occupational cohort studies have suggested possible associations between chronic exposure to cadmium, particularly cadmium oxide, and cancers of the lung, prostate, and genitourinary system such as renal carcinoma. These studies conclude that, to date the epidemiological evidence shows a small increase in the relative risk of lung cancer in workers exposed to cadmium and cadmium compounds (Verougstraete *et al*.2003).

2.6.3 Reproductive Effect

Testicular effects have been observed in animals exposed to cadmium for acute or intermediate durations; the testicular effects included necrosis and atrophy of seminferous tubule epithelium, increased testes weight, and decreased sperm count and motility (ATSDR, 1999). Chronic oral studies have not tested the reproductive toxicity of cadmium. The oral studies suggest that the testicular effects occur at doses of 5.8 mg/kg/day. No observe adverse effect level (NOAEL) and low observe adverse effect level (LOAEL) values of 2.9 and 5.8 mg cadmium/kg/day, respectively, for increased relative testes weight were identified in a study in which rats were exposed to cadmium chloride in the drinking water for 14 weeks (Pleasants *et al*. 1992).

2.6.4 Respiratory Effects

Severe, often fatal, pulmonary disease can result from brief inhalation exposure to high concentrations of cadmium compounds; however, such exposures are now very unusual. These types of exposures can occur in occupational settings such ascadmium alloy production,welding involving cadmium coated steel, and cadmium smelting and refining (Newman, 1998), onset of symptoms is usually delayed for 4 to 10 hours.

Newman also said, initial symptoms resemble the onset of a flu-like illness-chills, fever, and myalgias. Later symptoms include chest pain, cough, and dyspnea. Bronchospasm and hemoptysis may also occur. Histiologic findings in the lungs after such exposures include hyperemia of the trachea and bronchi,pulmonary edema,intra-alveolar hemorrhage,fibroblastic proliferation, hyperplasia of alveolar lining cells, and thrombosis of small blood vessels (Newman, 1998)

2.6.5 Bone effects

Although cadmium accumulates in bone, the bone disease that results from excessive cadmium exposure is believed to be secondary to changes in calcium metabolism due to cadmium-induced renal damage (ATSDR, 1999). Clinically significant bone lesions usually occur late in severe chronic cadmium poisoning and include pseudofractures and other effects of osteomalacia and osteoporosis. Pseudofractures are spontaneous fractures that follow the distribution of stress in normal skeleton or occur at sites where major arteries cross the bone and cause mechanical stress through pulsation.

The OSCAR study in Sweden examined whether environmental cadmium exposure could be a risk factor for reduced mineral density in bone (Jarup et al. 2000; Alfven et al. 2002, 2004). The study authors found a negative correlation between urinary cadmium and bone density. Skeletal effects appear to be secondary to increased urinary calcium and phosphorus losses due to cadmium-induced renal effects (Jarup *et al.* 2000). These effects are compounded by inhibition of renal hydroxylation of vitamin D, which eventually leads to a deficiency of its active form (Nogawa *et al.*2004). Some investigators believe cadmium also exerts an inhibitory effect on calcium absorption from the gastrointestinal tract. Enhanced secretion of prostaglandin E2 may also contribute to bone resorption

2.7 Health Risk Assessment

Risk assessment is a scientific process by which quantification of potential environmental hazards to human health is achieved. This process utilizes the tools of science, engineering, and statistics to identify and measure a hazard, determine possible routes of exposure, and finally use that information to calculate a numerical value to represent the potential risk. Risk assessment has played a significant role in efforts to protect the environment and public health and its use has been applied to a number of technical fields such as toxicology, industrial hygiene, occupational safety, environmental impact assessment, engineering-reliability studies, weather prediction, epidemiology, and the social and behavioral sciences (Cohrssen and Covello, 1989).

A human health risk assessment consists of four steps. These steps include hazardidentification, dose-response assessment, exposure assessment, and risk characterization. The first step, hazard identification, focuses on whether exposure to a particular chemical iscapable of causing adverse health effects. A qualitative literature evaluation can show ifexposure to a chemical causes an increase in the incidence of particular adverse health effectsand whether those health effects occur in humans. Once adverse health effects are identifiedfor a chemical, the dose-response assessment is used to quantitatively determine arelationship between the dose of the chemical and the incidence of adverse health effects inthe exposed population. This step involves the evaluation of toxicity information in animalsand humans and there are different models used to quantify dose-response based on whetherthe chemical is carcinogenic or noncarcinogenic (USEPA, 1989)

The third step in the risk assessmentprocess is exposure assessment and this step estimates the amount and route by which aperson is exposed to a chemical. Finally, the last step is risk characterization. This step bringstogether all the information gathered during the previous three steps to provide a quantitativeestimate of the risk associated with exposure to the chemical of concern.An essential part of the dose-response portion of a risk assessment includes the use of areference dose (RfD). The RfD is based upon toxicological data from animal studies andmarks the highest average daily exposure over a lifetime that would not be expected to causeadverse human health effects (USEPA, 2000). The RfD is expressed in mg/kg-day.Carcinogens are expressed by their cancer slope factor (CSF). This measure is derived from animal studies and uses the 95% confidence limit of the slope of the chemical's dose-response curve.

CHAPTER 3

METHODOLOGY

3.1 Study Location

The study location of this study was Kampung Pengkalan Kubor, Tumpat, Kelantan. This location was being chosen because it was the home of the *budu* factory in Kelantan, which is 'Budu Ketereh'. Therefore, the researcher would have opportunity to observe the process of *budu* making. This factory was located at Kampung Pengkalan Kubor, Tumpat which is near the border of Thailand. Besides that, the residents of this area have easy access to the stock of *budu*. Furthermore, this location also was being chosen because it can represent all residents in Kelantan because from observation most Kelantanese people took *budu* in their daily menu. 'Budu Ketereh' got certificate from Intellectual Property Corporation of Malaysia (PHIM) and the first type of *budu* in Malaysia that be accept in Intellectual Property.

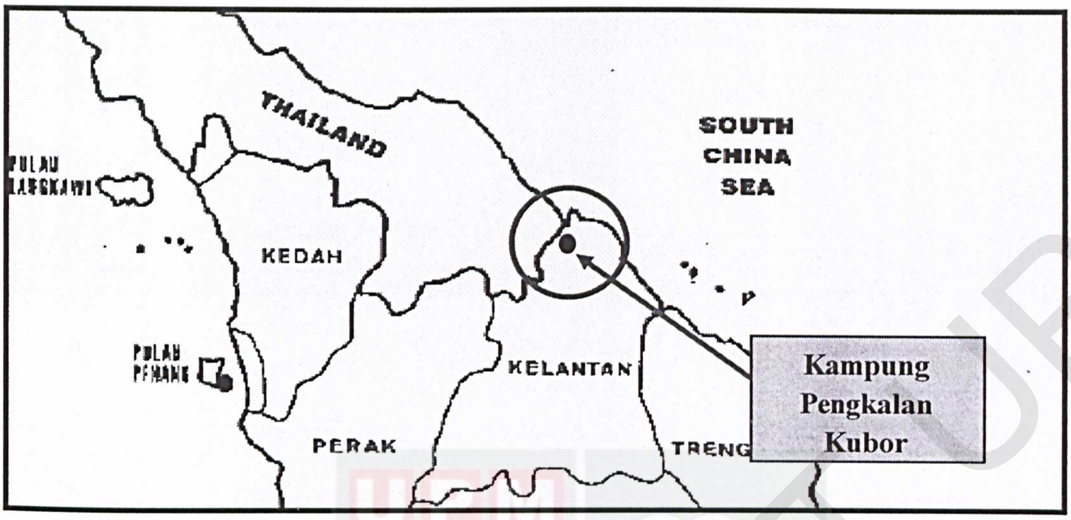

Figure 2: Sampling Location

3.2 Study Design

Cross sectional study was conducted to determine the concentration of cadmium in *budu* and chronic daily intake of *budu* among residents in Tumpat. This cross sectional study was done in nine months which from September 2011 until June 2012.

3.3 Study Population

3.3.1 Study Sample

In this study, the samples were selected using random sampling method. Firstly, the list of people who took *budu* in their daily menu was obtained. After that, the respondents were randomly chosen based on inclusion criteria which were aged 18-59 years old (Norimah *et al*, 2008).

3.3.2 Sample size

Based on District Health Office of Tumpat data, the total of populations in Kampung Pengkalan Kubur is 1584 people. According to sample size proportion, if the the total population is between 1001 until 149 999 people, only 10% from that population will be selected (Neuman, 1997). The total sample size for this study was calculated as below:

i) Kg. Pengkalan Kubor

10/100 x 1584 = 158.4 respondents

Estimate total sample size =158.4 respondents

≈159

Thus, at least 159 respondents should be selected to represent the study population. In this study, a total of 160 respondents were selected.

3.4 Data Collection and Instrumentation

3.4.1 Potential Exposure to Cadmium

A set of pre-tested modified questionnaires from Malaysia Adults Nutrition Survey 2000-2008 (Norimah *et al*, 2008) were used to gather information on background information and potential exposure to cadmium from respondents **(Appendix 1)**. This questionnaire consist five parts.

The first part contained questionnaires regarding respondents' background including age, gender, education level, marital status, employment, income per household and numbers of household. The second part asked questions of acute and chronic cadmium poisoning signs. The third part contained questions regarding anthropometry which height and weight of respondents were measured and the values of height and weight were used to calculate the body mass index (BMI) while value of weight was used to calculate average daily dose (ADD) and lifetime average daily dose (LADD). The fourth part consist of FFQ questions regarding *budu* frequency intake, duration and quantity of *budu* as well as the most consumed food to respondents would be the sources of cadmium exposure. The information regarding to *budu* frequency intake, duration and quantity of *budu* were used to calculate ADD and LADD. The last part of the questionnaires asked questions regarding the household living environment like smoking habit and sources of water. This part provided information regarding other possible sources of cadmium exposure to respondents. **Figure 3** showed the data collection flow chart of the study.

3.4.2 Cadmium Concentration in *Budu*

***Budu* sample**

Samples of *budu* were selected based on the finding of Food Frequency Questionnaire (FFQ) regarding the most popular brand of *budu* consumed by the respondents. The study samples were purchased from local mini market **(Appendix 2).**

Extraction of *budu* sample

Dry ashing method (modified method from Issac and Kerber, 1971) was usedto extract the *budu* samples prior to analysis by ICP-MS to determine the cadmium concentration level **(Appendix 3).**All the *budu* samples were duplicated before extraction process was performed. Firstly, ten gram of sample was placed in a crucible and preheated in a muffle furnace **(Appendix 4)** at 200–250 °C for 30 minutes and then ashed for 4 hours at 480 °C. The sample was removed from the furnace and brought to cool. Twenty ml of 5 M HNO_3 was added and evaporated to dryness on a water bath.

The sample was left to cooland heated to 400 ^{o}C for 15 minutes. Then it was removed from the furnace, brought to cool and then moistened with forty drops of distilled water.

Twenty ml of concentrated hydrochloric acid (HCl) was added and the sample was evaporated to dryness, removed, and then fifty ml of 2 M HCl was added and the tube was again swirled. The solution was filtered through Whatman No. 42 filter paper and <0.45 lm Millipore filter paper, and then transferred quantitatively to a 250 ml volumetric flask by adding deionized water.

Cadmium concentration determination

After ashing was completed, the *budu* samples were analyzed by using Inductively Coupled Plasma Mass Spectrometer (ICP-MS) model Perkin Elmer ElanDre-e in the unit of part per μg/mL **(Appendix 4)**.The concentration of cadmium in *budu* was calculated by using the following equation:

$$\text{Concentration of cadmium in } \textit{budu} \text{ (mg/kg)} = (A \times B) \times \frac{C}{D}$$

Where:

A = the concentration of cadmium in the extract (µg/mL)

B = dilution factor (4)

C = the volume of extract (60mL)

D = weight of sample (10g)

3.4.3 Health Risk Assessment

In estimating risk levels, the mean and maximum total cadmium concentrations, represented by C, were determined from the data from the *budu* samples. The Exposure frequency (EF) represents the average per-capita number of meals by the population based on a long-term average contact rate (USEPA, 1992). For all calculations, an average adult body weight (BW) of 59 kg, standard exposure duration (ED) of 70 years, and averaging time (AT) of 365 days was assumed.

The health risk associated with cadmium in *budu* the average daily dose (ADD) and lifetime average daily dose (LADD) were calculated by using the following equation (Lushenko, 2010):

$$\textbf{Average daily dose (ADD) (mg/kg-day)} = \frac{(C \times IR \times EF \times ED)}{(BW \times AT)}$$

Where:

C = Total cadmium concentration in *budu* (mg/kg)

IR = Ingestion rate of *budu* (kg/day)

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time period of exposure (ED x 365 days)

$$\textbf{Average daily dose (ADD) (mg/kg-day)} = \frac{(C \times IR \times EF \times ED)}{(BW \times AT)}$$

Where:

C = Total cadmium concentration in *budu* (mg/kg)

IR = Ingestion rate of *budu* (kg/day)

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time period of exposure (70 years x 365 days)

Risk for both non-carcinogenic and carcinogenic heavy metals calculated for this study and the equation listed above is the most commonly used intake equation. For non-carcinogens the average daily dose, or ADD, calculated which expresses the average daily intake of a specific chemical over a certain period. In contrast, the lifetime average daily dose, or LADD, is generally an estimate of the daily intake of a carcinogenic agent throughout the entire life of an individual.

Calculation of Significant Exposure for Carcinogen and Non-carcinogen

Non-carcinogenic risks quantified by the calculation of a hazard quotient (HQ) while carcinogen risks were quantified by the calculating of lifetime excess of cancer risk (LCR). The risk was determined according to the following equation (Lushenko, 2010):

$$\textbf{Hazard Quotient (HQ)} = \frac{ADD}{\text{Oral RfD}}$$

Where:

Hazard Quotient (HQ) = Non-cancer hazard of a health effect from intake of cadmium

ADD = Average daily dose (mg/kg-day)

Oral RfD = Oral Reference dose of cadmium (mg/kg-day)

=5.0 x 10^{-4}(USEPA, 2009) IRIS database

Lifetime excess cancer risk (LCR) = LADD x q*

Where:

LCR = Upper limit cancer risk

LADD = Lifetime average daily dose (mg/kg-day)

q* = Cancer potency factor, also known as slope factor of cadmium (mg/kg-day)

= **6.1 mg/kg/day** (USEPA, 2005) IRIS data base

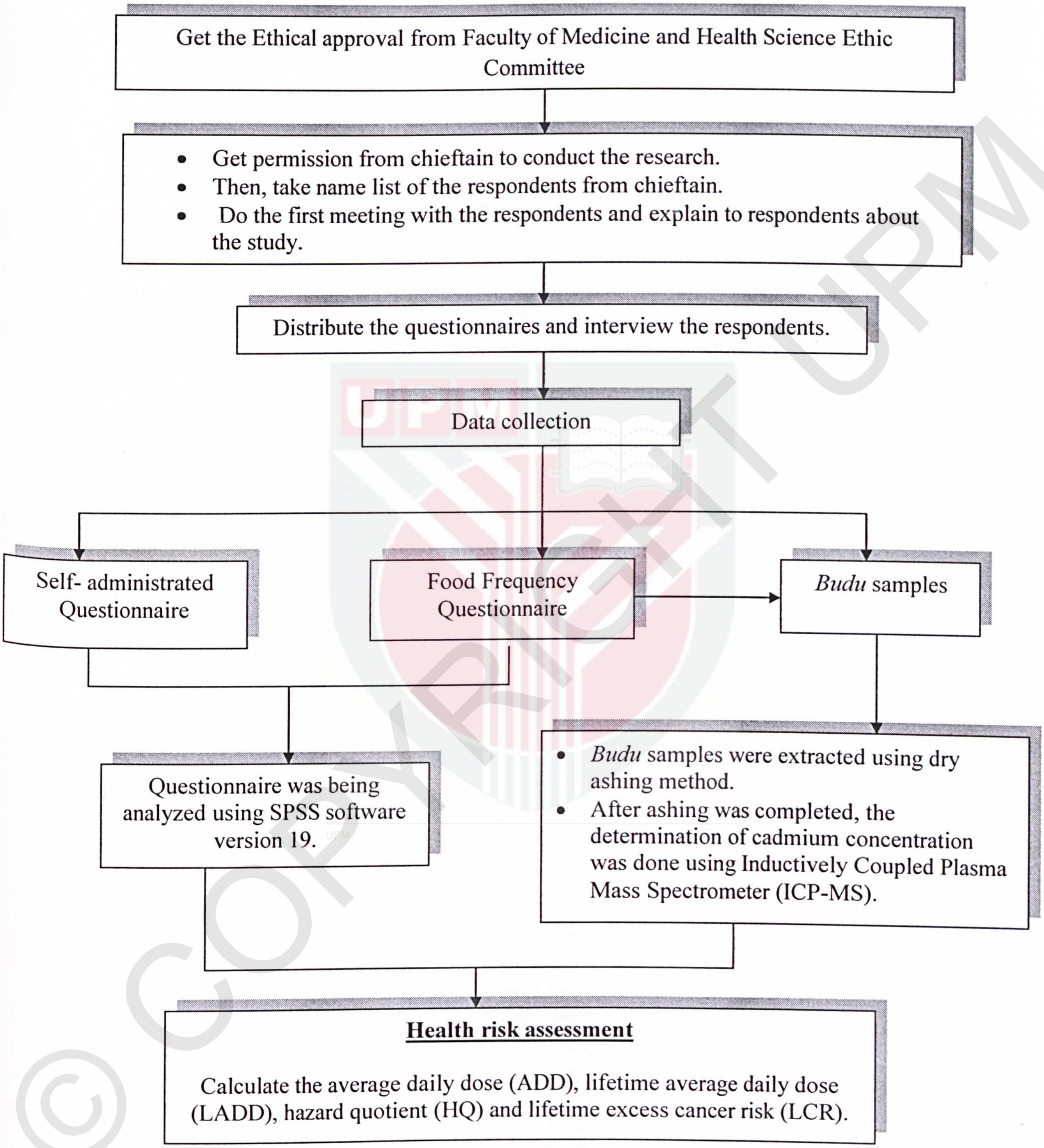

Figure 3: Data Collection Flow Chart

3.5 Quality Assurance and Quality Control

The pre-testing of the questionnaire was performed on twenty respondents in Kampung Raja, Besut, Terangganu prior to the data collection to ensure appropriateness of the questions with Cronbach's Alpha 0.79. Allglassware used in this study was washed with detergent, soaked in nitric acid and rinsed in deionizer water to minimize and prohibit external metal contamination. In an addition, the long sample treatment would be necessary for a slow dry ashing to prevent volatilization losses.

The analysis of *budu* samples using Inductively Coupled Plasma Mass Spectrometer (ICP-MS) model Perkin Elmer ElanDre-e was following the Standard Operation Procedure (SOP) as given by the manufacturer in order to minimize analytical errors. Besides, a blank and three standard solutions were prepared and run together with study samples to check for interference and cross contamination.

3.6 Statistical Analysis

All data were analyzed using Statistical Package for Social Science (SPSS) version 19 with the significant value was set at $p < 0.05$. Kolmogorov Smirnov tests were used to determine the normality distribution of the variables. The descriptive analysis was used to determine percentage, mean, median, standard deviation and range of variables such as age, gender, education level, signs of cadmium poisoning, cadmium concentration and other study variables. Chi square test and correlation coefficient Spearmen test were used to determine significant relationship between two tested variables. Chi square test was used to determine relationship between frequency intake of *budu* and prevalence of cadmium poisoning signs. Correlation coefficient Spearmen test was used to determine relationship between frequency intake of *budu* with HQ and LCR.

3.7 Ethical Concern

Approval from Medical Research Ethics Committee, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia (UPM) was obtained. Written consent was signed by each respondents involved before data collection commenced. Ethical concern was to ensure that respondent's information should be kept confidante, to protect the respondents from allegation of wrongdoing, malpractice, individual harm and misconduct. The respondents were informed regarding the aim, methods, and sources about the research that has been studied before the questionnaire was distributed. The respondents'involvements were volunteered. Research application letter was also sent to the chieftain of the village. All respondents' survey information was kept confidential and not presented to the public. Respondents had been informed on the result of the study through their village chief.

CHAPTER 4

RESULTS

4.1 Socio Demographic Information

This study was conducted among 160 residents at Kampung Pengkalan Kubor, Tumpat, Kelantan. All of them were Muslim, aged 18 to 59 years old and majority of them were female (61.9%) and had normal body mass index (62.5%). More than 50% of respondents were married and small percentages (0.6%) of them were either divorced or widow. Majority of respondents studied until SPM level (48.8%) and employment scope was other employments (39%) like student and housewife. Most of respondents (56.9%)earned income more than RM720 per month and the higher numbers of household were range between 5-7 persons (48.1%). The data were showed in **Table 4.1 and 4.1.1**

Table 4.1: Background Information of the Respondents

Variable	N	%	Mean ±S.d	Median (IQR)	Range
Age (year)					
13-19	11	6.9			
20-39	84	52.5		34(40)	18-58
40-59	65	40.6			
Gender					
Male	61	38.1			
Female	99	61.9			
Marital Status					
Single	68	42.5			
Married	90	56.3			
Divorced	1	0.6			
Widow	1	0.6			
Education level					
Primary school and below	7	4.4			
PMR	21	13			
SPM	78	48.8			
STPM/Matriculation/ diploma	22	13.8			
High education	32	20			
Employment					
Government	8	5			
Private	19	11.9			
Self-employed	36	22.5			
Retired/unemployed	34	21.3			
Others	63	39			
BMI					
<18.5 (underweight)	16	10			
18.5 – 24.9 (normal)	100	62.5		22.6(21.7)	16-37.7
25.0– 29.9 (overweight)	33	20.6			
≥30.0 (obesity)	11	6.9			

N=16

4.1.1 Socio economy Information

Table 4.1.1: Total Household and Number of Household

Variable	N	%	Median (IQR)	Range
Total household income(RM)				
<720	69	43.1	800(11800)	200-12000
>720	91	56.9		
Number of household				
2-4	47	29.4		
5-7	77	48.1	5(11)	2-13
8-10	33	20.6		
11 and above	3	1.9		

N= 160

4.2 *Budu* FrequencyIntakes

Majority of respondents frequently consumed *budu* in their diet as shown in **Figure 4.** The mean of quantity and duration intake of *budu* were 2 tea spoons and 26 years respectively.

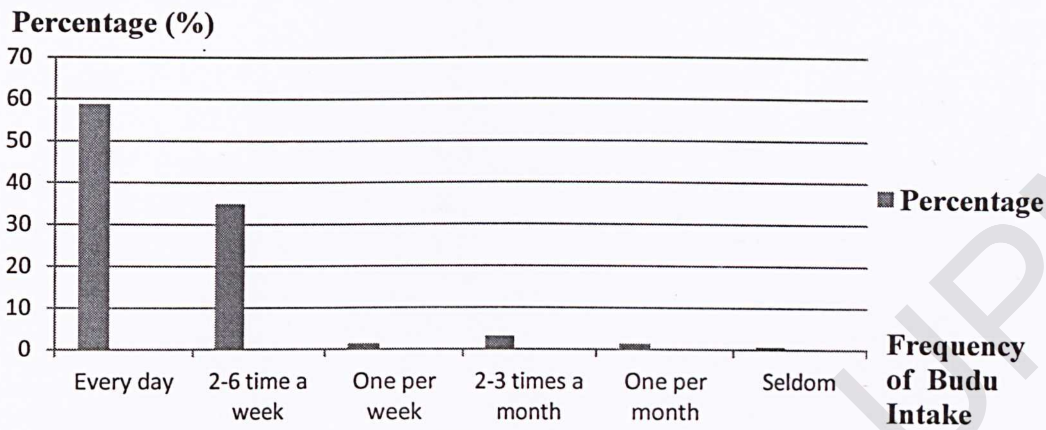

Figure 4: ***Budu*** **Frequency Intake by Respondents**

4.3 Other Food Consumption by Respondents

Table 4.3.1showed the frequency of top 10 most consumed daily while **Table 4.3.2** was top 10 weekly food consumed by respondents. The most consumed daily food was rice (100%) while chili/tomato sauce (70.6%) was the most consumed weekly food.

Table 4.3.1: Frequency of Top 10 Daily Consumed Food by Respondents

Type of food	N	%	Mean frequency per day	Total amount consumed daily
Rice	160	100	2	1 plate
Sugar	153	95.6	3	2 teaspoons
Marine fish	135	84.4	2	1 pieces
Leafy vegetable	128	80	1	1 cup
Bitter /pumpkin/cucumber	81	50.6	1	1 cup
Salads	81	50.6	1	1 cup
Cabbages	79	49.4	1	1 cup
Shrimp paste	38	23.8	2	2 teaspoons
Chili/tomato sauce	36	22.5	1	2 tablespoons
Soy sauce	34	21.3	1	1tablespoons

N=160

Table 4.3.2: Frequency of top 10 weekly consumedfood by respondents

Types of food	N	%	Mean frequency per weekly	Total amount consumed weekly
Chili/tomato sauce	113	70.6	2	2 tablespoons
Anchovies	112	70	1	2 tablespoons
Soy sauce	106	66.3	2	2 tablespoons
Fish crackers	105	65.6	2	5 pieces
Prawn	96	60	1	3 medium pieces
Dried fish	90	56.3	3	1 slice
Cuttlefish	84	52.5	1	3 medium pieces
Bean sprouts	81	50.6	2	1 cup
Dark soy sauce	79	49.4	1	2 teaspoons
Fish ball/fish cake	78	48.8	1	3 balls

N=160

4.4 Concentration of Cadmium in Anchovy Sauce *(budu)*

Table 4.4.1 showed the concentration of cadmium in three types of anchovy sauce (*budu*). The higher concentration of cadmium was in sample 2 (0.0467 mg/kg) followed by concentration in sample 3 (0.0427 mg/kg) and the low concentration of cadmium was in sample 1 (0.0317mg/kg).

Table 4.4.1: Concentration of cadmium in three types of anchovy sauce *(budu)*

Type of anchovy sauce *(budu)*	First concentration (mg/kg)	Second concentration (mg/kg)	Mean concentration (mg/kg)
Sample 1	0.0317	0.0316	0.0317
Sample 2	0.0468	0.0466	0.0467
Sample 3	0.0421	0.0432	0.0427
Total	**0.0402**	**0.0405**	**0.0404**

N=3

4.5 Cadmium Poisoning's Signs of Respondents

Figure 5 showed the prevalence of acute cadmium poisoning signs.The higher acute sign experienced by respondents was dizziness (6.9%)while the small percentage signs were stomach ache, diarrhea and difficult to breath which only 0.6% respectively. Thehigher chronic sign experienced by respondents was numbness (6.9%) while the small percentage signs were pale and swollen which 0.6% respectively. The chronic cadmium poisoning signs of respondents were shown in **Figured 6**. These results showed that, only small % of respondents reported with cadmium poisoning signs.

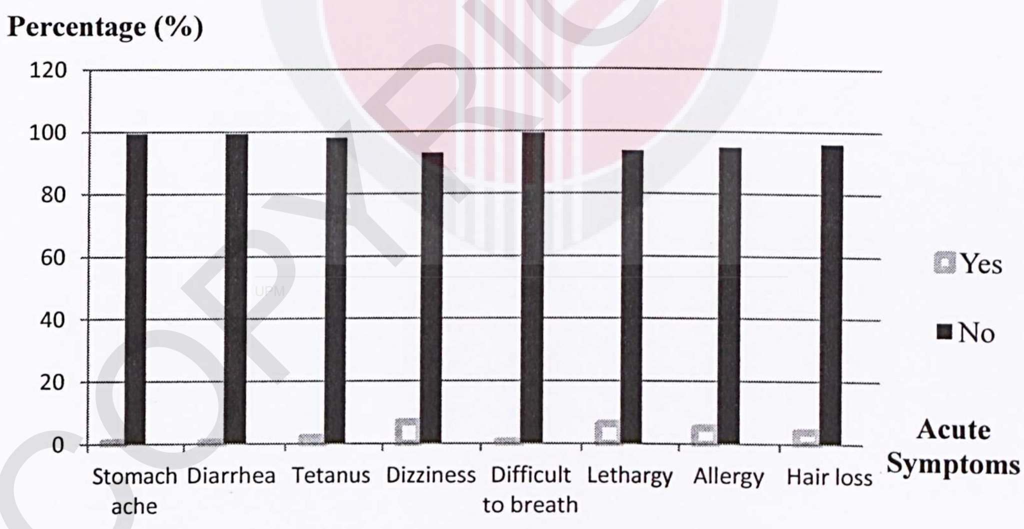

Figure 5: Prevalence of Acute Cadmium Poisoning Signs

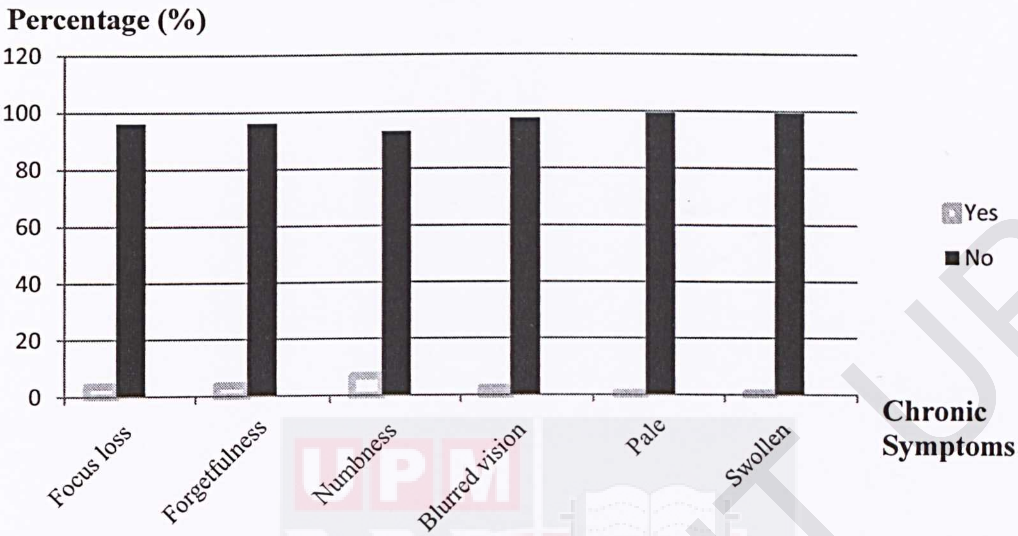

Figure6: Prevalence of Chronic Cadmium Poisoning Signs

4.6Cadmium Exposure from Other Sources

Table 4.61 showed the sources of water while **Table 4.6.2** showed smoking habit of respondents. Majority of them used well water and did not smoke.

Table 4.6.1: Smoking habit of respondents

Smoking Habit	N	%
Yes	36	22.5
No	119	74.4
Stop smoking	5	3.1

N=160

Table 4.6.2: Sources of water were consumed by respondents

Water Source	N	%
Tap water	18	11.3
Well water	133	83.1
Tap water & well water	9	5.6

N=160

4.7 Health Risk Assessment from Consumption of *Budu*

Table 4.7.1 showed the average daily dose (ADD) and lifetime average daily dose (LADD) of respondents. The median(IQR) values of ADD and LADD of respondents were [0.4(0.5)] x10^{-5}mg/kg and[0.1(0.2)] x10^{-5} mg/kg respectively. These results showed that the ADD and LADD values did not exceed cadmium oral reference dose (RfD) (5.0 x 10^{-4} mg/kg) and PTWI. **Table 4.7.2** showed the hazard quotient (HQ) and lifetime excess cancer risk (LCR) of respondents. All of respondents had hazard quotient value less than 1and about 87.5% of respondents have lifetime excess cancer risk value more than 1 x 10^{-6}. These results indicate that respondents did not experience any cadmium poisoning health risk.

Table 4.7.1: Average Daily Dose (ADD) and Lifetime Average Daily Dose (LADD)

Variable	Median(IQR)	Range
Average daily dose (ADD)	[0.4(0.5)] x10^{-5}	(0.002-2.1) x10^{-5}
Lifetime daily dose (LADD)	[0.1(0.2)] x10^{-5}	(0.001 – 1.1) x10^{-5}

N=160

Table 4.7.2: Hazard Quotient (HQ) and Lifetime Excess Cancer Risk (LCR)

Variable	N	%	Median(IQR)
Hazard Quotient (HQ)			
(Acceptable)	160	100	0.0076(0.01)
(Unacceptable)	0	0	
Lifetime Excess Cancer Risk (LCR)			
(Clearly acceptable)	140	87.5	
(Acceptable)	20	12.5	[0.7(1.4)]x10^{-5}
(Unacceptable)	0	0	

N=160

4.8 Relationship between *Budu* Frequency Intake and Prevalence of Cadmium Poisoning Signs

Chi-square statistical analysis had been done to determine relationship between *budu* frequency intake with prevalence of acute and chronic cadmium poisoning signs of respondents. Results showed there were any significant relationships between *budu* frequency intake and prevalence of acute and chronic cadmium poisoning signs of respondents.

Table 4.8.1: Relationship between *Budu* Frequency Intake and Prevalence of Acute Signs Associated with Cadmium Poisoning.

Acute Symptoms		*Budu* Frequency Intake		Total	X^2	p
		Frequently	Not Frequently			
Stomach ache	Yes	1	0	1	0.689	0.407
	No	94	65	159		
Diarrhea	Yes	0	1	1	1.471	0.225
	No	95	64	159		
Tetanus	Yes	2	2	4	0.149	0.699
	No	93	63	156		
Dizziness	Yes	7	4	11	0.890	0.766
	No	88	61	149		
Difficult to breath	Yes	1	0	1	0.684	0.407
	No	94	65	159		
Lethargy	Yes	4	5	9	0.881	0.348
	No	91	60	151		
Allergy	Yes	4	4	8	0.307	0.580
	No	91	61	152		
Hair loss	Yes	2	5	7	2.880	0.090
	No	93	60	153		

N=160
***p<0.05=There was significant relationship**

Table 4.8.2: Relationship between BuduFrequency Intake and Prevalence of Chronic Signs Associated with Cadmium Poisoning

Chronic Symptoms		BuduFrequency Intake		Total	X^2	p
		Frequently	Not frequently			
Focus loss	Yes	5	1	6	1.483	0.223
	No	90	64	154		
Forgetfulness	Yes	6	1	7	2.105	0.177
	No	89	64	153		
Numbness	Yes	7	4	11	0.890	0.766
	No	88	61	149		
Blurred vision	Yes	2	3	5	0.803	0.370
	No	93	62	155		
Pale	Yes	1	1	2	0.740	0.786
	No	94	64	158		
Swollen	Yes	1	0	1	0.689	0.407
	No	9	65	159		

N=160
$^{*}p<0.05$ = There was significant relationship

4.9 Relationship between *Budu* Frequency Intakes with Hazard Quotient (HQ) and Lifetime Excess Cancer Risk (LCR)

As shown in **Figure 7** a moderate relationship ($r= 0.681$, $p<0.0001$) between *budu* frequency intake and hazard quotient (HQ) were found. **Figure 8** also showed a moderate relationship ($r=0.626$, $p<0.0001$) between frequency intake of *budu* and lifetime excess cancer risk.

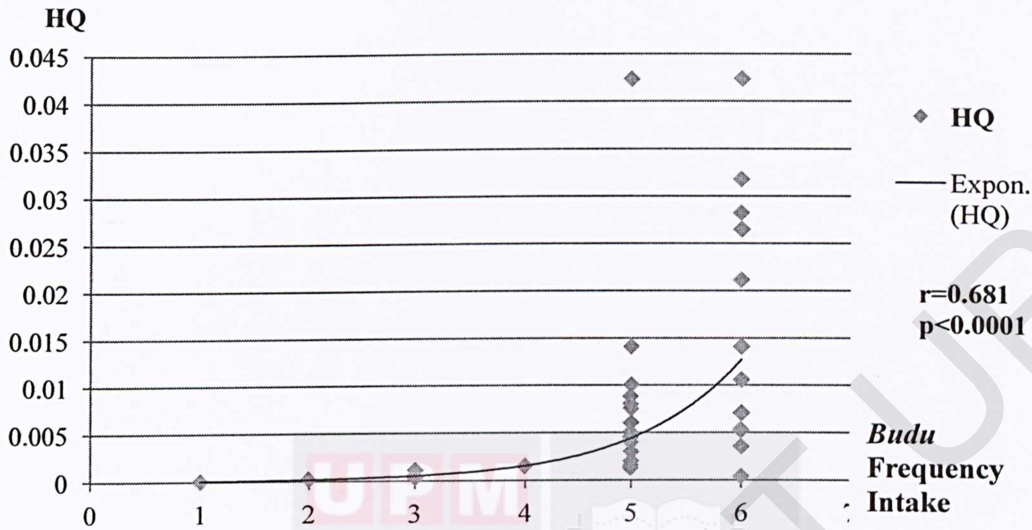

Figure 7: HQ versus *Budu* frequency intake scatter plot graph

NOTE:

Budu **Frequency Intake**	
1=Seldom	2=one per month
3=2-3 per month	4=one per week
5=2-6 per week	6=every day

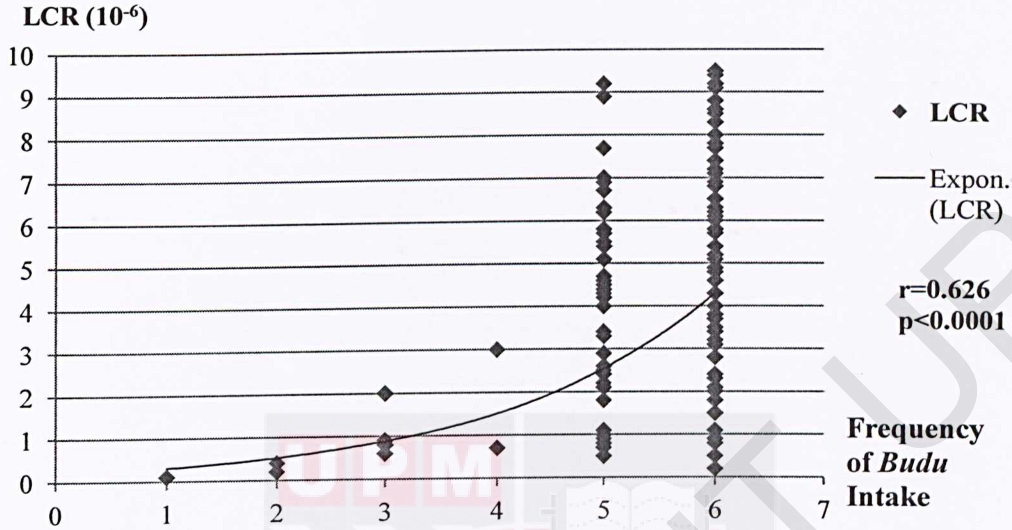

Figure 8: LCR versus *Budu* frequency intake scatter plot graph

NOTE:

***Budu*Frequency Intake**	
1=Seldom	2=one per month
3=2-3 per month	4=one per week
5=2-6 per week	6=every day

CHAPTER 5

DISCUSSION, CONCLUSION AND RECOMMENDATION

5.1Discussion

5.1.1 Socio-demographic Information

Respondents were chosen from Kampung Pengkalan Kubor, Tumpat, Kelantan. All the respondents were Muslim. Most respondents were in the range of 18 to 59 years old. The higher number of respondents was aged between 20-39 years old. This was because during data collection period it was Chinese New Year holiday, many Kelantanese people who worked and studied outside Kelantan came back to their hometown especially among that age group. With regards to gender, 61 (38.1%) were males and the 99 (61.9%) females. Females exceeded males because housewives were at home during data collection and their husband went to work and back home in the late evening. Thus, chances to meet the female respondents were higher than the male respondents especially when involving village people. Majority of respondents were married. The married status was higher compare than other marital statuses because most respondents had been interviewed were adults group. Besides, they also said, after finished their SPM level they would get married.

Majority of respondents had normal body mass index (BMI) which about 62.5%, overweight (20.6%) and obese (6.9%). The median BMI of respondents was 22.6. BMI was generally related to body fat and provided a good measure of obesity. Beside that, BMI can be indicator for nutritional status to certain groups of people. Higher BMIs usually mean higher body fat. As body fat or BMI increased, especially from values equal to or greater than 30, health risks increase (NIH/NHLBI, 1998). Being overweight (BMI of 25 to 29.9) or being obese (BMI greater than 30) increases the risk of having high blood pressure, heart disease, stroke, diabetes, certain types of cancer, arthritis, and breathing problems. Besides, if the body fat increased, it encourages absorption of cadmium in the body (NIH/NHLBI, 1998). The accumulation of cadmium at long exposure and high dose in body fat may cause cadmium adverse health effect like kidney disease, cancer and bone disease.

5.1.2 *Budu* Frequency Intake

Most of study respondents consumed *budu* every day. Frequent consumption of *budu* contaminated with cadmium can increased the accumulation of cadmium in the body because cadmium was detected in all of the *budu* samples in this study. Based on previous study, although cadmium absorption after dietary exposure in human is relatively low (3-5%) but cadmium was efficiently retained in the kidney and liver of human body with a very long biological half-life ranging 10 to 30 years (EFSA , 2009).

A study done by Goyer in 1990 found that, in workers with only short-term exposures to low levels of cadmium, the cadmium will be bound again in the kidney to the locally produced metallothionein providing a protective effect from cadmium. However, after prolonged exposure, the binding process in the kidney becomes saturated leading to an increase in unbound cadmium which can result in toxic effects.

5.1.3 Cadmium Concentration in *Budu*

This study found that, the range of cadmium concentration detected in the *budu* samples were between 0.0317 to 0.0467 mg/kg. The total mean of cadmium concentration in three samples of *budu* was 0.0404 mg/kg. The cadmium concentration in *budu* samples not exceed limit allow (1mg/kg) in Malaysia food law (Malaysia Food Regulation, 1985).

Cadmium which enters the human body can affect human health in short term and long term exposure. Besides, based on information from respondents during data collection, *budu* contained coloring substance from Thailand and the status of that coloring was not clear either it's safe or not. Unfortunately, the coloring was not mentioned in the list of ingredients of *budu* which displayed at the *budu* bottle.

5.1.4 Prevalence of Signs Associated with Cadmium Poisoning

This study used a list of acute and chronic signs of cadmium poisoning as an indicator of health effect of exposure to cadmium. Relatively, small numbers of respondents admitted that they experienced signs which might relate to cadmium such as dizziness, numbness and lethargy. The result may indicate that, using the list of acute and chronic signs was not a suitable method to measure health effect of small dose intake, although for continuous exposure. Besides, only one type of food was studied in this research which is *budu*. Bettermethods should be used as the indicator of cadmium poisoning such as cadmium in urine, blood or hair. Urine and blood sample were taken can determine the recent cadmium exposure while hair sample can determine the previous exposure.

5.1.5 Health Risk Assessment from Consumption of *Budu*

Health risk assessment was determined by calculating average daily dose (ADD), lifetime average daily dose (LADD), hazard quotient (HQ) and lifetime excess of cancer risk (LCR). The mean values of ADD and LADD of respondents were 0.1×10^{-5} mg/kg/day and0.04×10^{-5} mg/kg/day respectively. It's showed that the value of ADD and LADD did not exceed oral reference dose (5.0x10-4mg/kg/day) and Provisional Tolerable Weekly Intake (0.007mg/kg/weekly or 0.001 mg/kg/day). This means that it was safe level on dose-response curve. Besides, it was assumed that no chronic risks are likely occurred to respondents.

Hazard quotient was calculated to determine non-carcinogenic health effects while lifetime excess cancer risk for carcinogenic health effect. If HQ value less than 1 its meant that the risk was acceptable while HQ value more than 1 the risk was unacceptable. All of the respondents had hazard quotient value less than 1 and about 87.5% of respondents had lifetime excess of cancer risk(LCR) value more than 1×10^{-6}.If LCR value more than 1x10-6 the risk was clearly acceptable while LCR value between 1x10-6-1x10-4 the risk is acceptable and if LCR value less than 1x10-4 the risk is unacceptable. This showed that, the respondents did not experience any non-carcinogenic and carcinogenic cadmium exposure effects (USEPA, 2005).

5.1.6 Relationship between Budu Frequency Intake and Prevalence of Signs Associated with Cadmium Poisoning

The finding of the study did not show any significant relationship between *budu* frequency intakes with cadmium poisoning signs because this study calculated the health risk only based on one food type which is *budu* sample. This finding was in contrast with previous studies. Previous study showed that, people who regularly consume shellfish and organ meats will have higher exposures of cadmium(Alina *et al*., 2012).Futhermore, there were other possible sources of cadmium exposure other than *budu* such as smoking habit, water sources, pesticide used and other food consumed by repondents. Majorityof respondents consumed rice in their daily menu because rice was a staple food for Malaysian people especially for Malay. From observation, majority of Kelatanese people consumed rice from Thailand. A previous study showed that, rice from Thailand contain cadmium from trace to 5 mg cadmium/kg rice with the average of 1.33 mg cadmium/Kg rice where it was exceed Codex Committee on Food Additives and Contaminants (CCFAC) of 0.2 mg cadmium/kg rice (Simmons *et al*.,2005).

Beside that, other study found that, cadmium levels slightly higher than the standard limits were detected in rice produced in a specific area of a town in Japan at rice inspections in 1998 and 1999 (Niigata Prefecture Office, 2000). The concentrations were 0.41 and 1.58 ppm, respectively. The former marginally exceeded the standard limit for food use (0.40 ppm) and the latter was higher than the allowable concentration for use of any kind (1.0 ppm). Other previous study done in Straits of Malacca found that cadmium was detected in fish (0.6-2 to 3.5-2 μg/g wet sample) and shellfish (0.9-2 to 47-2 μg/g wet sample) (Alina *et al*., 2012). In general, leafy vegetables such as lettuce and spinach, potatoes and grains, peanuts, soybeans, and sunflower seeds contain high levels of cadmium (Clemens, 2006; McLaughlin *et al*,. 2006 ; Franze *et al*., 2008).

In this study also, about 23% of respondents were smoker. The respondents smoked 5 to 24 of ciggerates daily. Previous study discussed that, the tobacco leaves accumulate cadmium in a manner similar to certain food from plants. One cigarette may roughly contain 1–2 μg cadmium (varies depending on the type and brand). It is estimated that a person smoking 20 cigarettes per day will absorb about 1 μg cadmium daily (Olsson *et al*., 2002).Besides,a significant amount of cadmium has also been detected in the lenses of chronic smokers (Ramakrishnan *et al*., 1995).

Majority of respondents used well water for daily use such as washed clothes, cooking, bathing and water source for drinking.Themajor sources of cadmium in the drinking water supply are corrosion of galvanized pipes, erosion of natural deposits, discharge from metal refineries, runoff from waste batteries and paints (USEPA, 2009). Natural deposits of cadmium in rock and soil are more likely to be eroded when water is soft or acidic.Cadmium concentrations in municipal water supplies are regulated in the US and other developed countries but traces are inevitable and consumption of cadmium in drinking water over time may be a health concern. Median concentrations of dissolved cadmium measured at 110 stations around the world were <1 μg/l, the maximum value recorded being 100 μg/l in the Rio Rimao in Peru (WHO/UNEP, 1989).

Regardless of the route of absorption or the type of cadmium compound, approximately one half to one third of the body burden of cadmium was found in the kidneys after chronic low-level exposure, with the highest concentrations found in the renal cortex. As exposure level increases, a greater proportion of the body burden of cadmium will be found in the liver relative to the kidney. Also, upon the onset of renal dysfunction, the level of cadmium in the kidney will increase (Friberg, 1990).From 160 respondents, 6.9% of them experienced with dizziness and numbness while 6.3% experienced with lethargy and 2.5% experienced with blurred vision. These signs were the kidney failure signs. They would be advised to checkup their signs as soon as possible before these become more severe.

5.1.7 Relationship between the Frequency Intakes of *Budu* with Hazard Quotient (HQ) and Lifetime Excess of Cancer Risk (LCR)

This study revealed that, there were significant relationships between frequency intake of *budu* with hazard quotient (HQ) (r=0.681, p= <0.0001) and lifetime excess cancer risk (LCR) (r=0.626, p=<0.0001). The results showed significant relationship because accumulation of cadmium in the body through continuously small quantity intake may cause health effect such as kidney disease.

This statement was supportedby previous study where cadmium is primarily toxic to the kidney, especially to the proximal tubular cells where it accumulates over time and may cause renal dysfunction. After prolonged or high exposure the tubular damage may progress to decreased glomerular filtration rate, and eventually to renal failure (ATSDR, 2008). Besides that, long-term exposure to high-dose cadmium causes Itai-itai disease. Its syndrome principally consists of a painful skeletal condition resulting from weak and deformed bones. The disease is characterized initially by complaints of spinal and leg bone pain, and an increasingly waddling gait due to bone deformities. These symptoms can persist and typically progress for several years, until the patient is eventually unable to walk and becomes bedridden. The clinical symptoms then progress rapidly, with eventually severe debilitating pain, multiple bone fractures from even mild traumas such as coughing, severe skeletal deformities, anemia, and severe kidney problems such as impaired tubular and glomerular function and generalized osteomalacia and osteoporosis that result in multiple bone fractures and leading to death (Inaba *et al*. 2005) . This disease affects mainly women.

5.2 Conclusion

Cadmium was detected in *budu* and the concentration did not exceed the permissible limit (1 mg/kg) by Malaysia Food Regulation 1985. The ADD and LADD values did not exceed cadmium oral reference dose and PTWI while HQ and LCR values also not exceed acceptable limit. The respondents did not experience any non-carcinogenic and carcinogenic health risk. There was no significant relationship between frequency intake of *budu* and cadmium poisoning signs but had significant relationship with HQ and LCR.

5.3 Recommendation

As the recommendation, the respondents should monitor their cadmium level by perform biological sample check-up for cadmium exposure. Beside that, people can consume *budu* frequently but must be alert with its hygiene aspect and consumed the food with high calcium and iron. There was a study shown, there are numerous other factors that affect the intestinal absorption and organ retention of cadmium which most important one is the interaction between cadmium and other minerals nutrients that affect its absorption. It has been known for some time that feeding high concentration of zinc (Zn), iron (Fe) calcium (Ca), its can reduces the rate of absorption of cadmium from various food sources (ATSDR, 2008). Besides, anotherprevious studied found that humans with low ironand calcium intake will have an enhanced absorption of cadmium (Akesson *et al*., 2002). Manufacturer must follow the guideline of safe hygiene practice for food processing by Ministry of Health. People also must be aware of what risk they might encountered from consuming food. In addition, during study performed we can cooperate with local agency and Ministry of Healthto encourage people to aware about the food safety.Lastly, if the similar study will be carried out in the future, the same method can be use to analyze the samples and all Kelantanese people can become as respondents.

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A P P E N D I X 1

ID Responden: | | |

JABATAN KESIHATAN KOMUNITI
FAKULTI PERUBATAN DAN SAINS KESIHATAN
UNIVERSITI PUTRA MALAYSIA

Kajian kepekatan logam berat kadmium dan risiko kesihatan kepada penduduk di Tumpat,Kelantan

Adalah dimaklumkan bahawa satu kajian tentang kesihatan akibat pendedahan kepada kepekatan arsenik dan kadmium dalam budu sedang dijalankan di tempat kerja anda. Sehubungan dengan itu, sukacita dimaklumkan bahawa anda telah terpilih untuk menjadi salah seorang responden kajian ini. Oleh demikian, anda diminta menjawab semua soalan yang dikemukakan dengan mengikut arahan yang telah diberikan. Segala maklumat berkenaan responden akan dirahsiakan dan hanya akan digunakan untuk kajian ini.

ID Responden: | | |

Tarikh kajian: ....

No Kad Pengenalan: | | | | | | | | | | | |

No Telefon: (R) | | | | - | | | | | | | |

(Hp) | | | | - | | | | | | | |

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SELANGOR, MALAYSIA

BAHAGIAN 1	KETERANGAN DIRI	RUANGAN KOD

Tandakan (√) dalam kotak berkenaan.

Nama Penuh: ....

Alamat Semasa: ....

....

1. Umur: .... Tahun

2. Jantina: ☐ Lelaki ☐ Perempuan

3. Bangsa: ☐ Melayu ☐ Cina ☐ India
   ☐ Lain-lain (Sila nyatakan): ....

4. Agama:
   ☐ Islam
   ☐ Kristian
   ☐ Buddha
   ☐ Hindu
   ☐ Lain-lain (Sila nyatakan): ....

5. Status:
   ☐ Bujang
   ☐ Berkahwin
   ☐ Bercerai/berpisah
   ☐ Balu/Duda

6. Taraf pendidikan:
   ☐ Tidak Bersekolah
   ☐ UPSR
   ☐ PMR
   ☐ SPM
   ☐ Sijil/STPM/Matrikulasi
   ☐ Diploma
   ☐ Ijazah Sarjana Muda
   ☐ Sarjana Muda
   ☐ Doktor Falsafah

7. Jenis pekerjaan:
   ☐ Kerajaan
   ☐ Swasta
   ☐ Bekerja Sendiri
   ☐ Pencen/Tidak Bekerja
   ☐ Lain-lain

8. Pendapatan sebulan (RM) : _______________

9. Pendapatan isi rumah sebulan (RM) : ______________

10. Bilangan ahli isirumah : ___________ orang

BAHAGIAN 2	MAKLUMAT KESIHATAN	RUANGAN KOD

11. Adakah anda mengalami masalah kesihatan berikut?

- [ ] Kanser, nyatakan : ____________________
- [ ] Diabetes/kencing manis
- [ ] Darah tinggi
- [ ] Penyakit buah pinggang
- [ ] Penyakit jantung
- [ ] Pekak
- [ ] Bisu
- [ ] Terlantar sakit teruk
- [ ] Penyakit lain, nyatakan : ____________________

12. Adakah anda mengalami simptom-simptom seperti berikut?

- [ ] Sakit perut
- [ ] Muntah
- [ ] Cirit-birit
- [ ] Kekejangan otot
- [ ] Pening
- [ ] Kesukaran bernafas
- [ ] Kelesuan
- [ ] Alergi

13. Adakah anda telah mendapatkan rawatan untuk simptom-simptom di atas?

- [ ] Ya, sila nyatakan kali terakhir anda mendapatkan rawatan : ……………………………
- [ ] Tidak

BAHAGIAN 3:	ANTHROPOMETRI	RUANGAN KOD
1. Nama peserta :	1.	
2. Berat (kg) :	2. **i.** [ ][ ][ ] . [ ] kg ii. [ ][ ][ ] . [ ] kg **iii.** [ ][ ][ ] . [ ] kg	
3. Tinggi (cm) :	3. **i.** [ ][ ][ ] . [ ] **cm** **ii.** [ ][ ][ ] . [ ] **cm** **iii.** [ ][ ][ ] . [ ] **cm**	

Pengukuran Indeks Jisim Tubuh (IJT) / *Body Mass Index (BMI)*

BMI = <u>Berat (kg)</u>
Tiggi x Tinggi(m2)

Klasifikasi ⇨

IJT / BMI < 18.5 = Kurang Berat Badan
IJT / BMI 18.5 - 24.9 = Normal
IJT / BMI 25.0 - 29.9 = Berlebihan berat badan
IJT / BMI ≥ 30.0 = Obes

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BAHAGIAN 4: BORANG KEKERAPAN PENGAMBILAN MAKANAN

Kod	**Jenis makanan**	**Kekerapan pengambilan**					**Ukuran sajian(pilih satu jenis ukuran sahaja)**	**Berapa banyak sajian setiap kali makan**
	(A)Bijiran dan hasil bijiran	**Berapa kali sehari**	**Berapa kali seminggu**	**Berapa kali sebulan**	**Berapa kali setahun**	**Tidak makan**		
A1	Nasi						Pinggan	
							Mangkuk cina	
							Cawan	
							senduk	
A2	Mee kuning/mee segera						Pinggan	
							Mangkuk cina	
							Cawan	
							senduk	
A3	Mihun/kuew teow/laksa /laksam						Pinggan	
							Mangkuk cina	
							Cawan	
							senduk	
A4	Roti						Pinggan	
							Mangkuk cina	
							Cawan	
							senduk	
A5	Bijirin sarapan pagi						Pinggan	
							Mangkuk cina	
							Cawan	
							senduk	

Kod	**Jenis makanan**	**Kekerapan pengambilan**					**Ukuran sajian(pilih satu jenis ukuran sahaja)**	**Berapa banyak sajian setiap kali makan**
	(C)Kekacang dan hasilnya	**Berapa kali sehari**	**Berapa kali seminggu**	**Berapa kali sebulan**	**Berapa kali setahun**	**Tidak makan**		
C1	Kekacang						Sudu makan	
C2	Tauhu						Keping	
C3	Tempe						Keping	
							Sudu makan	
C4	Kacang Tanah						Sudu makan	

Kod	Jenis makanan	Kekerapan pengambilan					Ukuran sajian (pilih satu jenis ukuran sahaja)	Berapa banyak sajian setiap kali makan
	(D) Sayuran	Berapa kali sehari	Berapa kali seminggu	Berapa kali sebulan	Berapa kali setahun	Tidak makan		
D1	Sayuran berdaun						Cawan	
D2	Sayuran kacang						Cawan	
D3	Sayuran berubi						Cawan	
D4	Sayuran kobis						Cawan	
D5	Petola/labu /timun						Cawan	
D6	Ulam-ulaman						Cawan	
D7	Putik jagung						Sudu makan	
D8	Cendawan basah /kering						Cawan	
D9	Taugeh						Cawan	

Kod	Jenis makanan	Kekerapan pengambilan					Ukuran sajian(pilih satu jenis ukuran sahaja)	Berapa banyak sajian setiap kali makan
	(E)Makanan perencah /perasa	Berapa kali sehari	Berapa kali seminggu	Berapa kali sebulan	Berapa kali setahun	Tidak makan		
E1	Gula						Sudu teh	
E2	Madu						Sudu teh	
E3	Belacan						Sudu makan	
E4	Cencaluk						Sudu teh	
E5	Budu						Sudu teh	
E6	Kicap pekat						Sudu teh	
E7	Kicap cair						Sudu makan	
E8	Sos cili/tomato						Sudu makan	
E9	Sos tiram						Sudu teh	
E10	Sos ikan						Sudu teh	
E11	Otak udang						Sudu teh	

Kod	Jenis makanan	Kekerapan pengambilan					Ukuran sajian(pilih satu jenis ukuran sahaja)	Berapa banyak sajian setiap kali makan
	(D)Makanan yang dikaji	Berapa kali sehari	Berapa kali seminggu	Berapa kali sebulan	Berapa kali setahun	Tidak makan		
F1	Budu							

Sumber bekalan budu:

☐ Buatan sendiri

☐ Perusahaan kecil (IKS)

Senaraikan 5 jenis jenama budu yang paling kerap anda makan?

1. ______________________
2. ______________________
3. ______________________

Sudah berapa lamakah anda mengambil budu sebagai salah satu menu harian?

Sila nyatakan : ______________________________

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BAHAGIAN 5:	**FAKTOR-FAKTOR PENDEDAHAN LAIN**	**RUANGAN KOD**

Tandakan (√) dalam kotak berkenaan.

Sumber bekalan air

1. Dari manakah anda mendapat sumber bekalan air minuman?

☐ Air paip
☐ Air perigi
☐ Lain-lain :____________

Pendedahan pekerjaan

2. Pernahkah pekerjaan anda melibatkan penggunaan pestisid ?

☐ Ya ; Sila nyatakan pekerjaan anda : ____________
☐ Tidak

Jika Ya, sila ke soalan seterusnya. (3) jika Tidak, sila terus ke soalan (4)

3. Berapa lama anda terlibat dengan pekerjaan ini?

____

4. Adakah anda menggunakan racun serangga selain daripada waktu bekerja?

☐ Ya
☐ Tidak

Amalan gaya hidup

a) Merokok

5. Adakah anda merokok?

☐ Ya
☐ Tidak
☐ Sudah berhenti

Jika Ya, sila nyatakan berapa tahun anda sudah merokok dan bilangan batang rokok dihisap dalam sehari:

Bil.tahun: ____________ Bil. Batang rokok sehari __________

6. Semasa anda merokok,adakah anda menyedut asap rokok?

☐ Tidak sama sekali
☐ Sedikit
☐ Sederhana
☐ Mendalam

Jika sudah berhenti, sila jawab soalan 7

7. Pada umur berapa anda berhenti merokok sepenuhnya?

____

b) Pengambilan alkohol

8. Adakah anda pernah mengambil minuman beralkohol?

☐ Ya
☐ Tidak

Jika ya, sila nyatakan berapa botol sehari anda minum?

__________________ botol

A

P

P

E

N

D

I

X

2

Budu Samples

APPENDIX 3

<u>**Extraction of Sample Flow Using Dry Ashing Method**</u>

1) Firstly, ten gram of sample was placed in a crucible.

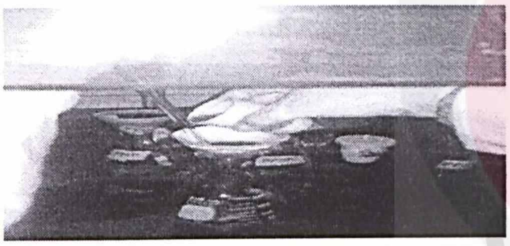
2) Samples were preheated in a muffle furnace at 200–250 °C for 30 minutes and then ashed for 4 hours at 480 °C.

3) Samples were removed from the furnace and brought to cool.

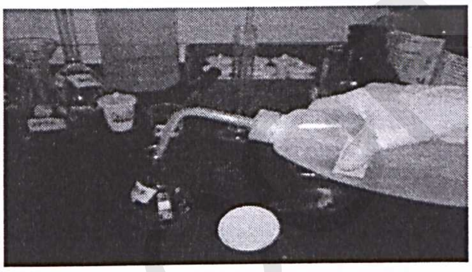
4) Twenty ml of 5 M HNO_3 was added into samples

5) The samples were evaporated to dryness on a water bath. The sample was left to cool and heated to 400 °C for 15 minutes.

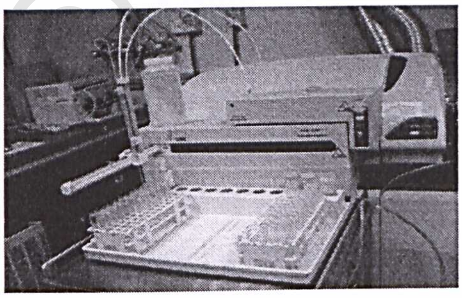
6) Twenty ml of concentrated hydrochloric acid (HCl) was added and the sample was evaporated to dryness, removed, and then fifty ml of 2 M HCl was added and the tube was again swirled.



7) The solution was filtered through Whatman No. 42 filter paper and <0.45 lm Millipore filter paper



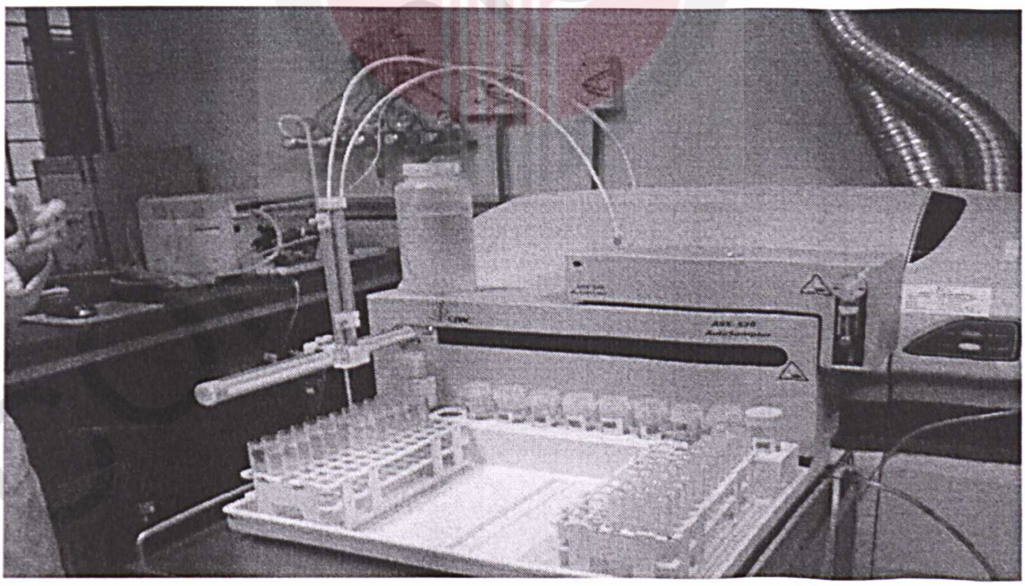
8) Then, the solution was transferred quantitatively to a 250 ml volumetric flask by adding deionized water.



9) *Budu* solutions were analyzed by using Inductively Coupled Plasma Mass Spectrometer (ICP-MS) model Perkin Elmer Elan Dre-e.

A P P E N D I X 4

Muffle furnace



Inductively Coupled Plasma Mass Spectrometer (ICP-MS)

model Perkin Elmer Elan Dre-e

A P P E N D I X 5

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PENERANGAN KEPADA PESERTA

TAJUK KAJIAN: PENCEMARAN LOGAM KADMIUM DI DALAM SOS IKAN BILIS (BUDU) DAN PENILAIAN RISIKO KESIHATAN DALAM KALANGAN PENDUDUK DI TUMPAT, KELANTAN.

RESEARCHER : NOR HUSNA BT HASSAN

Terima kasih kerana membantu saya di dalam kajian ini.

Apakah kajian ini?

Sejak kebelakangan ini, air semulajadi telah dicemari oleh bahan toksik daripada pelbagai sumber. spesies hidupan laut kini semakin terancam bukan sahaja kerana kewujudan logam secara semulajadi di dalam laut tetapi juga hasil daripada aktiviti manusia. spesies ikan terutamanya ikan bilis digunakan sebagai bahan utama dalam pemprosesan budu. Disebabkan ada pencemaran air dan kemungkinan ikan bilis yang digunakan dalam pemprosesan budu juga dicemari oleh logam Kadmium. Masyarakat yang mengambil Budu sebagai menu harian mereka mungkin akan dicemari oleh logam kadmium di dalam sistem badan mereka.

Apakah tujuan kajian ini?

Kajian ini dijalankan bertujuan untuk mengkaji pencemaran logam kadmium di dalam sos ikan bilis (budu) dan penilaian risiko kesihatan dalam kalangan penduduk di Tumpat, Kelantan.

Berapa ramai responden yang terpilih?

Responden akan dipilih daripada kalangan penduduk yang tinggal berdekatan dengan kawasan pemprosesan sos ikan bilis (budu) di sekitar Tumpat. Seramai 160 orang responden dari Kg. Pengkalan Kubur dipilih untuk kajian ini.

Apakah jenis ujian yang akan dijalankan?

Semua responden akan diberikan borang soal selidik untuk diisi sendiri oleh responden. Selain daripada itu, pengukuran berat dan tinggi diambil dan akan digunakan untuk menganggar risiko yang dihadapi oleh penduduk sekiranya sampel sos ikan bilis (budu) yang diambil mengandungi pencemaran logam kadmium. Di samping itu juga, sedikit temu ramah akan dibuat bagi mendapatkan maklumat mengenai kekerapan pengambilan makanan laut dan sos ikan bilis (budu) dalam menu harian.

Adakah bayaran dikenakan?

Pengkaji akan menanggung segala pembiayaan ujian yang akan dijalankan dan tiada sebarang bayaran dikenakan terhadap setiap responden.

Adakah maklumat dijamin sulit?

Semua maklumat yang diberikan oleh responden di dalam borang kaji selidik adalah dijamin sulit. Tiada huraian individu akan dibuat pada mana-mana bahagian di dalam kajian atau penerbitan.

Adakah hak anda?

Kajian ini melibatkan anda secara sukarela. Oleh itu, peserta mempunyai hak untuk menarik diri dari penyertaan dalam kajian ini pada bila-bila masa sekiranya peserta merasa tidak selesa untuk memberikan maklumat kepada pengkaji.

Apakah yang harus anda lakukan?

Anda dikehendaki menandatangani borang penyertaan responden yang menyatakan minat anda untuk menyertai kajian ini. Ianya boleh dilakukan setelah anda membaca dan memahami isi kandungan penerangan ini. Borang penyertaan responden haruslah dikembalikan kepada penyelidik sebelum ujian dijalankan. Sekiranya anda mempunyai sebarang kemusykilan, penyelidik akan membantu untuk memberi maklumat yang selanjutnya.

Terima kasih atas kerjasama dan bantuan anda.

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BORANG PERSETUJUAN RESPONDEN

TAJUK KAJIAN: PENCEMARAN LOGAM KADMIUM DI DALAM SOS IKAN BILIS (BUDU) DAN PENILAIAN RISIKO KESIHATAN DALAM KALANGAN PENDUDUK DI TUMPAT, KELANTAN

RESEARCHER : NOR HUSNA BT HASSAN

Saya ………………………………………………………… No.K/P: ………………………………………
alamat………
……………………………………………………………dengan ini secara sukarela bersetuju untuk mengambil bahagian dalam penyelidikan klinikal (kajian klinikal, kajian soal selidik/dadah percubaan) yang dinyatakan di atas. Saya telah dimaklumkan mengenai latar belakang penyelidikan ini dari segi kaedah, kemungkinan kesan buruk dan komplikasi(rujuk kepada risalah maklumat). Saya faham bahawa saya mempunyai hak untuk menarik diri dari kajian ini pada bila-bila masa tanpa memberikan apa jua sebab. Saya juga faham bahawa kajian ini adalah sulit dan semua maklumat yang diberikan mengenai identiti saya adalah sulit dan persendirian.

Saya ingin *tahu/tidak ingin mengetahui keputusan ujian yang dijalankan ke atas sampel saya.

* potong mana yang tidak berkaitany

Tandatangan ……………………………………… (*Responden*)

Tandatangan……………………………………… (Saksi)

Tarikh :………………………………………………

Nama :………………………………………………

No. K/P: ………………………………………………

Saya mengesahkan bahawa saya telah menjelaskan kepada responden latar belakang dan tujuan penyelidikan di atas.

Tarikh ……………………………………

Tandatangan……………………………………… (Penyelidik)

APPENDIX 6

GANNT CHART –RESEARCH SCHEDULE TABLE

Table 1: Gannt chart table

ACTIVITY	**2011**							**2012**		
	9	**10**	**11**	**12**	**1**	**2**	**3**	**4**	**5**	**6**
Research proposal										
Proposal approval										
Data collection										
Data analysis										
Thesis writing										

APPENDIX 7

A

P

P

E

N

D

I

X

8

Daily Performance Report

Sample ID: Smart Tune Solution
Sample Date/Time: Wednesday, March 28, 2012 11:32:31
Sample Description: Performance check
Method File: C:\Elandata_LC-ICPMS\Method\daily performance.mth
Dataset File: C:\Elandata_LC-ICPMS\Dataset\daily performance\Smart Tune Solution.256
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Dual Detector Mode: Pulse
Acq. Dead Time(ns): 55
Current Dead Time (ns): 55

Summary

	Analyte	Mass	Meas. Intens. Mean	Net Intens. Mean	Net Intens. SD	Net Intens. RSD
	Mg	24.0	77734.2	77734.190	1189.049	1.5
	In	114.9	272861.7	272861.704	5849.314	2.1
	U	238.1	349530.7	349530.687	7137.074	2.0
[>	Ce	139.9	244612.1	244612.138	5946.389	2.4
[	CeO	155.9	4022.0	0.016	0.001	4.6
[>	Ba	137.9	196406.8	196406.801	3630.390	1.8
[	Ba++	69.0	4820.6	0.025	0.001	2.7
	Bkgd	220.0	7.9	7.900	0.693	8.8
	Bkgd	8.5	17.4	17.433	2.016	11.6

Current Optimization File Data

Current Value	Description
0.72	Nebulizer Gas Flow [NEB]
1.20	Auxiliary Gas Flow
17.00	Plasma Gas Flow
8.00	Lens Voltage
1100.00	ICP RF Power
-1700.00	Analog Stage Voltage
750.00	Pulse Stage Voltage
0.00	Quadrupole Rod Offset Std [QRO]
-12.00	Cell Rod Offset Std [CRO]
25.00	Discriminator Threshold
-26.00	Cell Path Voltage Std [CPV]
0.00	RPa
0.25	RPq
0.91	DRC Mode NEB
-5.50	DRC Mode QRO
-0.50	DRC Mode CRO
-16.00	DRC Mode CPV
0.00	Cell Gas A

Current Autolens Data

Analyte	Mass	Num of Pts	DAC Value	Maximum Intensity
Be	9	45	6.5	4441.1
Co	59	45	7.3	115569.0
In	115	45	8.3	241458.6

Quantitative Analysis - Summary Report

Sample ID: Blank

Sample Date/Time: Wednesday, March 28, 2012 12:04:15
Sample Description:
Solution Type: Blank
Blank File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File:
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	216	7.911		
Cd	111	113	14.264		
Pb	208	200	10.149		

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75					ppb
Cd	111					ppb
Pb	208					ppb

Quantitative Analysis - Summary Report

Sample ID: Std 1 (10 ppb)

Sample Date/Time: Wednesday, March 28, 2012 12:05:58
Sample Description:
Solution Type: Standard
Blank File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Std 1 (10 ppb).007
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File:
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	12348	3.975	216.003	7.911
Cd	111	16767	4.184	113.334	14.264
Pb	208	168746	5.967	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	12132.387	**10.000**	0.40	4.0	ppb
Cd	111	16653.465	**10.000**	0.42	4.2	ppb
Pb	208	168546.015	**10.000**	0.60	6.0	ppb

Quantitative Analysis - Summary Report

Sample ID: Std 2 (30 ppb)

Sample Date/Time: Wednesday, March 28, 2012 12:07:41
Sample Description:
Solution Type: Standard
Blank File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Std 2 (30 ppb).008
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File:
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	37071	2.159	216.003	7.911
Cd	111	51849	4.230	113.334	14.264
Pb	208	525358	1.457	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	36855.453	**30.037**	0.65	2.2	ppb
Cd	111	51735.611	**30.103**	1.28	4.2	ppb
Pb	208	525157.706	**30.112**	0.44	1.5	ppb

Quantitative Analysis - Summary Report

Sample ID: Std 3 (50 ppb)

Sample Date/Time: Wednesday, March 28, 2012 12:09:25
Sample Description:
Solution Type: Standard
Blank File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Std 3 (50 ppb).009
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File:
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	63909	0.885	216.003	7.911
Cd	111	86427	1.847	113.334	14.264
Pb	208	866919	6.218	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	63692.525	**50.531**	0.45	0.9	ppb
Cd	111	86313.644	**50.064**	0.93	1.8	ppb
Pb	208	866718.563	**49.913**	3.10	6.2	ppb

Quantitative Analysis - Summary Report

Sample ID: Std 4 (100 ppb)

Sample Date/Time: Wednesday, March 28, 2012 12:11:09
Sample Description:
Solution Type: Standard
Blank File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Blank.006
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\Std 4 (100 ppb).010
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File:
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	132397	3.491	216.003	7.911
Cd	111	174685	1.445	113.334	14.264
Pb	208	1665278	3.038	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	132180.547	**101.218**	3.54	3.5	ppb
Cd	111	174571.910	**100.322**	1.45	1.4	ppb
Pb	208	1665078.028	**98.901**	3.00	3.0	ppb

Calibration Report

Analyte	Mass	Curve Type	Slope	Intercept	Corr Coeff
As	74.922	Linear Thru Zero	1305.901369	0.000	0.999754
Cd	110.904	Linear Thru Zero	1740.109343	0.000	0.999981
Pb	207.977	Linear Thru Zero	16835.846092	0.000	0.999821

Quantitative Analysis - Summary Report

Sample ID: 11

Sample Date/Time: Wednesday, March 28, 2012 12:35:46
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\11.021
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	156289	2.527	216.003	7.911
Cd	111	2412	3.267	113.334	14.264
Pb	208	53793	1.561	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	Conc. Mean	Conc. SD	Conc. RSD	Sample Unit
As	75	156072.541	**119.513**	3.02	2.5	ppb
Cd	111	2298.986	**1.321**	0.05	3.4	ppb
Pb	208	53593.376	**3.183**	0.05	1.6	ppb

Quantitative Analysis - Summary Report

Sample ID: 12

Sample Date/Time: Wednesday, March 28, 2012 12:37:30
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\12.022
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	155972	1.994	216.003	7.911
Cd	111	2402	0.855	113.334	14.264
Pb	208	52799	5.616	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	Conc. Mean	Conc. SD	Conc. RSD	Sample Unit
As	75	155756.301	**119.271**	2.38	2.0	ppb
Cd	111	2288.316	**1.315**	0.01	0.9	ppb
Pb	208	52598.530	**3.124**	0.18	5.6	ppb

Quantitative Analysis - Summary Report

Sample ID: 13

Sample Date/Time: Wednesday, March 28, 2012 12:39:13
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\13.023
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	154728	1.070	216.003	7.911
Cd	111	3509	4.264	113.334	14.264
Pb	208	37189	4.554	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	154512.399	**118.319**	1.27	1.1	ppb
Cd	111	3396.011	**1.952**	0.09	4.4	ppb
Pb	208	36989.348	**2.197**	0.10	4.6	ppb

Quantitative Analysis - Summary Report

Sample ID: 14

Sample Date/Time: Wednesday, March 28, 2012 12:40:58
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\14.024
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	156723	0.242	216.003	7.911
Cd	111	3489	6.570	113.334	14.264
Pb	208	37036	4.167	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	Conc. Mean	Conc. SD	Conc. RSD	Sample Unit
As	75	156506.701	**119.846**	0.29	0.2	ppb
Cd	111	3375.337	**1.940**	0.13	6.8	ppb
Pb	208	36836.040	**2.188**	0.09	4.2	ppb

Quantitative Analysis - Summary Report

Sample ID: 15

Sample Date/Time: Wednesday, March 28, 2012 12:42:42
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\15.025
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	147485	1.814	216.003	7.911
Cd	111	3168	1.355	113.334	14.264
Pb	208	34888	4.209	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	147268.977	**112.772**	2.05	1.8	ppb
Cd	111	3054.551	**1.755**	0.02	1.4	ppb
Pb	208	34688.227	**2.060**	0.09	4.2	ppb

Quantitative Analysis - Summary Report

Sample ID: 16

Sample Date/Time: Wednesday, March 28, 2012 12:44:26
Sample Description: Food samples
Solution Type: Sample
Blank File:
Number of Replicates: 3
Peak Processing Mode: Average
Signal Profile Processing Mode: Average
Dual Detector Mode: Dual
Dead Time (ns): 55

Sample File: C:\Elandata_LC-ICPMS\Sample\Nor Aisyah (FPSK).sam
Method File: C:\Elandata_LC-ICPMS\Method\nor aisyah (fpsk).mth
Dataset File: C:\Elandata_LC-ICPMS\DataSet\Nor Aisyah (FPSK)\16.026
Tuning File: C:\Elandata_LC-ICPMS\Tuning\default.tun
Optimization File: C:\Elandata_LC-ICPMS\Optimize\default.dac
Calibration File: C:\Elandata_LC-ICPMS\System\Nor Aisyah (FPSK).cal
Calibration Type: External Calibration

Summary

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Intensities

Analyte	Mass	Meas. Intens. Mean	Meas. Intens. RSD	Blank Intensity	Blank Intens. RSD
As	75	146485	1.702	216.003	7.911
Cd	111	3248	1.623	113.334	14.264
Pb	208	37283	3.671	200.002	10.149

Concentration Results

Analyte	Mass	Net Intens. Mean	**Conc. Mean**	Conc. SD	Conc. RSD	Sample Unit
As	75	146268.969	**112.006**	1.91	1.7	ppb
Cd	111	3134.579	**1.801**	0.03	1.7	ppb
Pb	208	37083.028	**2.203**	0.08	3.7	ppb