ARSENIC SPECIATION IN BLUE CRAB (CALLINECTES SAPIDUS) AND SEDIMENT FROM A LOUISIANA FRESHWATER MARSH OILFIELD

by

Kathleen Webb

A Thesis Submitted in Partial Fulfillment

of the Requirements for the Degree

MASTER OF SCIENCE

Major Subject: Environmental Science

West Texas A&M University

Canyon, Texas

December 2015

ABSTRACT

Arsenic is ubiquitous in the environment occurring from both natural and anthropogenic sources. The major route of exposure to arsenic is via ingestion of tainted water and food—primarily rice and seafood. This is especially true for U.S. Gulf Coast populations where the seafood consumption rate is higher than the rest of the nation.

Oilfields in the U.S. Gulf of Mexico Basin are some of the top producers of hydrocarbons in the world. Produced water is a byproduct of the oil and gas brought to the surface; the U.S. Environmental Protection Agency (USEPA) and U.S. Geological Survey (USGS) have estimated that in the U.S. for every one barrel of oil, ten barrels of water are produced. Historically produced water was discharged directly into marshes and unlined pits, contaminating surface sediments and waters with arsenic and other contaminants.

Since arsenic toxicity is highly dependent upon species and physicochemical properties, there is a need to accurately determine the arsenic species present in a freshwater marsh oilfield ecosystem in a subsistence fishing community. The inorganic arsenites (As (III)) and arsenates (As (V)) are known to be highly toxic and carcinogenic. As (V) dominates in surface waters and sediments, while two organic forms arsenobetaine (AsB) and arsenocholine (AsC)—regarded as non–toxic—are the species primarily found in seafood. Previous research has shown that monomethylarsonic acid

(MMA) and dimethylarsinic acid (DMA) are metabolites of the inorganic arsenicals. It has been a long–held belief that this transformation was a mechanism of detoxification. However, MMA and DMA are classified by the World Health Organization's (WHO) International Agency for Research on Cancer (IARC) as Group 2B possible human carcinogens and they likely form toxic intermediates.

It is therefore important for human and ecological health risk assessments to determine how arsenicals behave and metabolize in an aquatic food chain. Arsenic bioaccumulation is dependent upon the organism, its trophic level, and ecosystem, (i.e., marine, freshwater, estuarine). The goal of this study was to determine the arsenic species present in the sediment and blue crab (*Callinectes sapidus*) from an oilfield situated in a freshwater marsh located in south central Louisiana and how metabolic processes impact arsenic speciation and the potential transfer of arsenic within the food chain.

Sediment samples and blue crabs were collected from 13 locations across the East White Lake Oilfield, including three locations on the easternmost edge of the lake. Composited sediment samples and three crabs from six locations—three lake and three oilfield—were analyzed. The arsenic species As (III), As (V), MMA, and DMA in the sediment and three blue crab tissue types (meat, shell, and hepatopancreas) were determined by ion chromatography coupled with inductively coupled plasma mass spectrometry (IC/ICP–MS).

Complex chemical reactions and interactions determine the mobility of arsenic in sediment, water, and biological systems. As (V) was the most stable and predominant

species found in the sediment (91.3%). There was no MMA detected in the sediment, while 5.5% was DMA and 3.1% was in the As (III) form. All four species were found in the crabs in the following order of predominance: 88.3% DMA, 6.7% As (V), 4.7% As (III), and 0.2% MMA. While arsenic was found in all three tissue types, the highest levels were found in the hepatopancreas.

The distribution of arsenic species in the sediment and crabs in the East White Lake oil and gas field illustrates the complexity of the arsenic cycle in this freshwater marsh ecosystem. It also illustrates the importance of speciating arsenic in multiple tissue types of the blue crab since local residents are potentially ingesting arsenic from the entire organism, not just the edible portions.

ACKNOWLEDGEMENTS

I would like to thank those without whom this thesis would not have been possible. I am grateful to my thesis committee: Dr. Gary Barbee, Dr. David Parker, and Dr. Jim Rogers (Doc) for their endless support, boundless encouragement, and patient guidance. They all deserve special thanks for their individual contributions over the last two years. Doc, I thank you for helping me out whenever I needed it and for the confidence you had in me as a student and a chemist. Dr. Parker, as the former Director of the West Texas A&M University (WTAMU) Palo Duro Research Facility (PDRF) Core Laboratory and my direct supervisor, I thank you for your patience, knowledge, and for always having my back when times got tough. I thank Dr. Barbee, my committee chairman, for pulling me out of my comfort zone and requiring me to hone my public speaking skills through seemingly endless presentations, entrusting this research to me, sweltering in the Louisiana summer heat to collect samples, and keeping me focused on my topic as I explored numerous tangents.

I would like to express appreciation to Dr. John Sweeten, Dr. Ken Casey, and Brandon Dukes of Texas A&M AgriLife Research and Extension for the Excellence Fund Scholarship that funded a portion of my expenses. I owe special recognition to two Louisianans: Dr. George Castille of Baton Rouge for his hospitality, assistance, and local knowledge and Mr. Mike Hebert, commercial fisherman, of Pecan Island for ferrying us

around White Lake and for maintaining the crab traps and collecting crabs in our absence. I would also like to thank Kara Mjølhus, Jason Hafliger, and Jamie Farren for their companionship, assistance sampling and processing, and levity when it all seemed so serious and overwhelming.

I am grateful to Ms. April Swindell, WTAMU Environmental Health and Safety Supervisor, and Dr. Angela Spaulding, V.P. of Research and Compliance and Dean of Graduate Studies, for their support of the Core Laboratory. This research and thesis would not have been possible had they not purchased the critical equipment and analytical instrumentation and been an enduring source of reassurance.

Lastly, I would like to extend my gratitude to family, friends, and loved ones for their patience, assistance, and unending love and support over the years as I endeavored to further my education. This has been a lifelong journey and I couldn't have done it without you. With all my love, Mom, this one's for you.

Approved:

_____ _____

Chairman, Thesis Committee Date

_____ _____

Member, Thesis Committee Date

_____ _____

Member, Thesis Committee Date

_____ _____

Head, Major Department Date

_____ _____

Dean, Graduate School Date

TABLE OF CONTENTS

LIST OF TABLES

TABLE Page

LIST OF FIGURES

FIGURE Page

LIST OF REPORTS

CHAPTER I

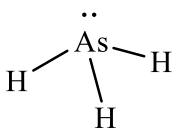
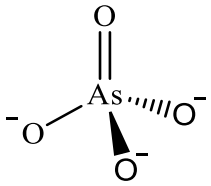
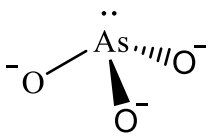
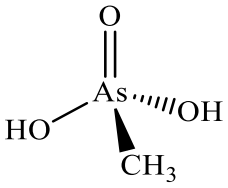
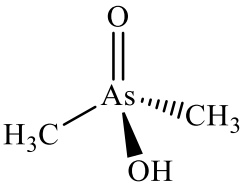
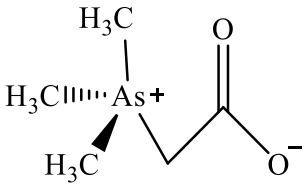
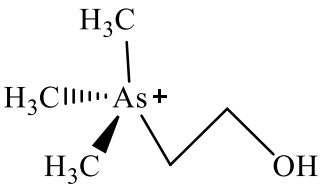
INTRODUCTION AND RESEARCH OBJECTIVES

Arsenic (As) is a naturally occurring metalloid that is pervasive in the environment stemming from both natural and anthropogenic sources. It occurs in numerous chemical forms; more than 50 different arsenic species have been identified in the marine environment alone (Francesconi, 2010). It is the 20th most abundant element in the Earth's upper continental crust (UC) with an average concentration of 2,000 µg/kg (Mohan and Pittman, 2007; Matschullat, 2000; Ruttens et al., 2012). The total crust, estimated to be between 30–50 km thick, comprises the upper crust (UC) and the lower crust (LC) (Pavão et al., 2013). Arsenic occurs in the UC, which is composed of sedimentary rocks, plutonic rocks and, granites (primarily silica and aluminum) (Dunn, 1993). The LC is composed of metamorphic rocks (Wedepohl, 1995). Arsenic is a component of >245 minerals, mostly iron sulfides (Bissen and Frimmel, 2003; Mandal and Suzuki, 2002; Shankar et al., 2014). Khorasanipour and Esmaeilzadeh (2015) reported geochemical association between arsenic and some native minerals including copper, molybdenum, selenium, iron, and manganese. Arsenic ranks 14th in seawater abundance (Sharma and Sohn, 2009) with relatively stable concentrations below 2 µg/L (Ng, 2005). In trace amounts, arsenic is an essential element (Mertz, 1981; Neff, 1997); it has been found to improve the appearance of hair and skin in mammals (Bowen,

1972). It ranks12th in abundance in the human body (Mandal and Suzuki, 2002), with concentrations between 14—21 mg in the "standard man" comparable to manganese and iodine (Schroeder and Balassa, 1966). When determining concentrations of trace elements in the human body, the "standard man" was defined in the 1960s as an average man weighing 70 kg, or 154 pounds (Schroeder et al., 1969), a number that is not representative today. Because it can be found as an environmental contaminant throughout the geosphere, the effects of arsenic toxicity have global implications on human health.

The toxicity of arsenic is dependent upon its chemical form. The inorganic arsenic (iAs) species—which contain no carbon—are considered to be approximately 100 times more toxic than the organic arsenicals that contain carbon (Feldman and Krupp, 2011; Geng et al., 2009; Jain and Ali, 2000; Zmolinski et al., 2015). Furthermore, As (III) is approximately 60 times more toxic than As (V) (Jain and Ali, 2000). Research has shown that toxic iAs converts to organic metabolites such as DMA and MMA in mammals (Hughes et al. 2011; Thompson, 1993; Vahter, 1999). In aquatic environments, phytoplankton and bacteria cycle the different arsenicals through the ecosystem via accumulation and biotransformation (Hong et al., 2014; Rahman et al., 2012). Researchers have expressed varying results concerning As biomagnification (Barwick and Maher, 2003; Huq et al., 2001; Maher et al., 2011), although some studies suggest the process exists in aquatic food webs (Ghaeni et al., 2015; Goessler et al., 1997; Umunnakwe et al. 2013). Some of the different inorganic and organic arsenic species of biological and environmental relevance are shown in Table 1.

Table 1: Some of the biologically and environmentally relevant arsenic species; structures created in ChemDraw Professional version 15.0.

Name	Abbreviation	Chemical Formula	Structure	Class
Arsine	As (−III)	AsH_3		IARC Group 1
Arsenates	As (V)	AsO_4^{3-}		IARC Group 1
Arsenites	As (III)	AsO_3^{3-}		IARC Group 1
Monomethyl–arsonic acid	MMA	$CH_3AsO(OH)_2$		IARC Group 2B
Dimethyl–arsinic acid	DMA	$(CH_3)_2AsO(OH)$		IARC Group 2B
Arsenobetaine	AsB	$(CH_3)_3As^+CH_2COO^-$		IARC Group 3
Arsenocholine	AsC	$(CH_3)_3As^+(CH_2)_2OH$		IARC Group 3

Arsenic in history, industry, and the marketplace

The toxic properties of arsenic were described by Pliny the Elder in the first century BCE (Frith, 2013). Although the discovery of arsenic as an element is credited to Albertus Magnus in the 13th century (Abbott, 1889). Arsenic has been used in potions, cosmetics, and embalming fluid by the ancient Egyptians, Greeks, and Romans (Doyle, 2009). Arsenic became known as the "Poison of Kings" and the "King of Poisons" due to its popularity as a homicidal agent, used to thin out the ruling classes from the Middle Ages through the Renaissance (Vahidnia et al., 2007). The Medici and Borgia families were well known for using arsenic to eliminate their rivals, and it has long been theorized that Napoleon Bonaparte died of arsenic poisoning (Hughes et al., 2011). Arsenic oxide was favored by poisoners due to its availability, the fact that it has no odor or taste, and could not be detected in food or drink (Bartrip, 1992; Nriagu, 2002). Poisoning with arsenic started to decline in 1832 when a British chemist named James Marsh established an analytical method to test for the presence of arsenic. It is now known as the Marsh test, and has been refined many times over. The Marsh test effectively ended the reign of the "King of Poisons" in the latter half of the 19th century (Hughes et al., 2011).

Despite its poisonous attributes, arsenic has also been used medicinally for a couple thousand years. Fowler's solution, a 1% solution of potassium arsenite, was widely used in the late 18th century to treat asthma, malaria, psoriasis, and syphilis (Scheindlin, 2005). Though Fowler's solution and most other arsenic compounsd have been replaced by other medications, there are some arsenicals still used in modern

medicine. For example, arsenic compounds are still used in Traditional Chinese Medicine (TCM) to treat tooth marrow disease. The arphenamine Salvarsan, introduced in the early 1910s, replaced mercury compounds to treat syphylis (Wang et al., 1998). Another antispirochetal, Melarsoprol, has been used to treat trypanosomiasis (African Sleeping Sickness) (Rust and Soignet, 2000). Consolidation treatment with all–trans–retinoic acid (ATRA) and arsenic trioxide has been successfully used to treat acute promyelocytic leukemia (APL) in the 2nd trimester of pregnancy (Agarwal et al., 2015). Shen et al. (1997) reported on fifteen case studies where nine relapsed APL patients were treated to complete remission (CR) with arsenic trioxide alone and five were treated to CR with arsenic trioxide and ATRA. The success of arsenic trioxide in treating APL has led researchers to investigate it as a possible treatment regimen for other cancers (Murgo, 2001; Sekeres, 2007).

In the past century, arsenic has been used in consumer products, agriculture, electronics and metallurgy (Mandal and Suzuki, 2002). As early as the 1800s, arsenic was used as a pigment (e.g., Paris Green or copper acetoarsenite), in toys, fabrics, and wallpaper (Hughes et al., 2011). Arsenic–based pigments are no longer used in consumer products. Paris Green is also an effective insecticide against mosquitoes, the codling moth, and the Colorado potato beetle, but its use ended in 1900 due to its toxicity to plants (Peryea, 1998). Paris Green was replaced by another arsenical pesticide—lead arsenate—which was less toxic to plants. It was used mostly in apple and cherry orchards until the 1960s, when a cohort study in Washington state linked cancers in orchard workers to lead arsenate pesticide use (Nelson et al., 1973). The U.S.

banned lead arsenate use in 1988 (Hughes et al., 2011), which was followed up by an EPA cancellation order to phase out organic arsenical pesticide use by 2013 (IARC, 2012).

One exception, monosodium methanearsonate (MSMA), is a broad spectrum herbicide registered by the EPA to control grasses and broadleaf weeds on sod farms, golf courses, highway rights–of–way, and cotton fields. Its use on cotton is limited to two post–emergent applications per year (EPA, 2009). Another exception, chromated copper arsenate (CCA) , is a common preservative that has been used since the 1940s to protect wood from mold, fungi, and dry rot (Baptist and Leslie, 2008). Such CCA–treated wood, also known as pressure–treated wood, has been used in patio decks, boat docks, playground equipment, picnic tables, and fences. In 2003, CCA manufacturers voluntarily halted production and recalled all pressure–treated wood that had been used for residential purposes. The EPA has listed chromated arsenicals intended for use in wood preservation as Restricted Use Pesticides (RUP) and limited their use to commercial, institutional, and some farming applications (EPA, 2008). This includes utility poles, boat dock pilings, farm fence rails, and highway noise barriers.

The U.S. is one of the major producers of copper ore, which is commonly extracted via open pit mining. Open pit mining produces vast quantities of waste rock in order to extract the metal of interest. When copper ore is mined, arsenic is one of the ore impurities that can leach out of the rock, especially in the acidic pH conditions created in mine drainage (Safe Drinking Water Foundation, 2009). Workers in the copper industry are considered to be at high risk of arsenic exposure via inhalation or ingestion from

hand–to–mouth activities. They are at risk during copper ore mining, smelting, electrolysis, grinding, and milling (Sun et al., 2015).

The global occurrence of arsenic and arsenic compounds

Arsenic enters surface and groundwater from natural sources such as rock weathering and geothermal activity, as well as anthropogenic sources such as mining and smelting processes, insecticide and pesticide use, and through the discharge of produced water (Barbee and Castille, 2010; Singh et al., 2015). Geochemical activity and iron oxide dissolution due to petroleum products in mining and landfill wastes are common sources of arsenic in groundwater (Jain and Ali, 2000; Welch et al., 2000). Arsenic–contaminated surface and groundwater has been reported in China, Taiwan, Japan, India, Bangladesh, Chile, Argentina, Poland, Hungary, Mexico, the United States, and Canada, among others (Jain and Ali, 2000). Shankar et al. (2014) estimated that more than 150 million people worldwide are affected by the high levels of arsenic in contaminated groundwater. Arsenic–contaminated drinking water in Bangladesh has caused what the World Health Organization (WHO) called the largest mass poisoning in history (Argos et al., 2010). Smith et al. (2000) reported that 35–77 million Bangladeshis live in regions with known contamination issues. Of those 35–77 million people, Anawar et al. (2002) estimated that around 9 million are drinking water containing arsenic at levels known to be hazardous. The WHO and U.S. EPA set the drinking water limit for arsenic at 10 μg/L, but arsenic concentrations in the groundwater reportedly varies from less than 0.5 μg/L to 5000 μg/L in over 70 countries worldwide (Singh et al., 2015). Arsenic concentrations in surface waters have a much

greater variance. Smedley and Kinniburgh (2002) reported arsenic concentrations ranging from <0.005 µg/L in the U.S. Mid–Atlantic coast up to 850,000 µg/L in mine tailings of the California Iron Mountains, with intermediate concentrations occurring worldwide.

Wenzel (2013) estimates that between 1.7–2.5 x 10^9 tons of arsenic is present in the global soil reservoir—approximately 5 orders of magnitude less than the total amount of arsenic in the Earth's UC and approximately 30% of the total amount of arsenic in the Earth's oceans. Arsenic concentrations in uncontaminated soils (determined largely by the concentration in parent materials) range from 0.1 to 55 mg/kg, but can also be found in single digit percent concentrations in soils contaminated by industrial activities (e.g., mining operations, metal smelters) (Wenzel, 2013). The main natural source of arsenic in soil is bedrock weathering. Elevated levels of arsenic can be found in shales, clays, and phosphate rocks, while sandstones, limestones, and igneous rocks contain arsenic at lower concentrations (Wenzel, 2013; Woolson, et al. 1977). Arsenic can occur in soil and sediment as As (III) and As (V), though As (V) dominates (Bolan et al., 2013; Wenzel, 2013). In aerobic soil, arsenic occurs mainly as arsenate (As (V) in the form AsO_4^{3-}) bound to iron and manganese oxyhydroxides and clay minerals (Bolan et al., 2013; Matschullat, 2000; Wenzel, 2013).

Arsenic–containing compounds are abundant in aquatic ecosystems. Bacteria and phytoplankton are pivotal in cycling arsenicals through water, sediment, and marine life. The so–called fish arsenicals, arsenobetaine (AsB) and arsenocholine (AsC), are the predominant species found in seafood matrices. After ingestion, they are rapidly excreted

in the urine so they are considered toxicologically insignificant (Leffers et al., 2013a; Sirot et al., 2009). Ritsema et al. (1998) reported near complete excretion of AsB within 48 hours after ingestion, with concentrations peaking at 12 hours.

While AsB is the primary arsenical in seafood, scientific literature reports that approximately 0.5%–5% of the arsenic present in seafood occurs in an inorganic form, that is, As (III) and/or As (V) (Moreda–Pineiro et al., 2012; Schintu et al., 2012; Sirot et al., 2009).

There are >15 structurally different arsenosugars—the main arsenical species in marine algae and seaweed (Feldmann and Krupp, 2011; Vahter, 1994). Arsenosugars are also present in animals that feed on algae and seaweed (e.g., mussels and oysters) (Leffers et al., 2013a). In contrast to AsB and AsC, arsenosugars metabolize in humans to numerous products, including DMA (V) and the toxicologically uncharacterized metabolites thio–dimethylarsinic acid (thio–DMA (V)), oxo– and thio–dimethylarsenoacetate (oxo–DMAA (V)/thio–DMAA (V)), and oxo– and thio–dimethylarsenoethanol (oxo–DMAE (V)/thio–DMAA (V)) (Leffers et al., 2013b). Thio–DMA (V) and thio–DMAE (V) have intestinal bioavailability similar to that of As (III), leading Leffers, et al., (2013b) to conclude that arsenosugar intake should be considered when assessing human health risk for consumers of food containing arsenosugars.

Navratilova et al., (2011) studied arsenosugar degradation in decomposing brown alga (*Ecklonia radiata*), concluding that arsenosugars cycle through a multi–step conversion to inorganic arsenic. Other studies have supported the findings that

arsenosugars metabolize to toxic and carcinogenic compounds (Ebert et al., 2014; Hughes et al., 2011; Leffers et al., 2013b).

Another form of arsenic has been found in aquatic ecosystems—lipid soluble arsenic compounds, or arsenolipids. Arsenolipids, which are analogues of neutral lipids, have been found in fish tissues, fish oil, marine mollusks, lichens, plants, and microorganisms (Amayo et al., 2014a; Dembitsky and Levitsky, 2004). Lipid soluble arsenic compounds reportedly account for 10%–30% of the total arsenic in marine organisms (Sele et al., 2012). Although the current belief is that the arsenic species found in seafood are non–toxic, studies have shown that arsenolipids metabolize in humans to produce potentially toxic intermediates along with the methylated metabolites MMA and DMA (Amayo et al., 2014a; Amayo et al., 2014b; Schmeisser et al., 2006). Therefore, as with arsenosugars, intake of arsenolipid–containing food and supplements should be considered when determining human health risk.

Arsenic chemistry

Arsenic (atomic number 33) is characterized as a metalloid. Most metalloids are lustrous solids at room temperature, exhibit brittle elasticity (the ability to stretch, but will break once the elastic limit is reached), and can form alloys. Arsenic exists in three allotropes, that is, elemental structures that differ in arrangement (McNaught and Wilkinson, 1997)—analogous to carbon that exists as coal, diamond, or graphite. The most common and stable allotrope of arsenic at standard temperature and pressure is gray arsenic (Eiduss et al., 1996; Karttunen et al., 2007), which is metallic, brittle, and nonductile (unable to be stretched into a thin wire under tensile stress) (Henke and

Hutchison, 2009). Amorphous yellow arsenic (As_4), the most toxic and unstable allotrope (Pichon, 2013), is also known as molecular arsenic because of its tetrahedral conformation (Rodionov et al., 1996; Spinney et al., 2009). Yellow arsenic can be synthesized by deposition on cooled substrates, existing in several forms, as evidenced by infrared (IR) spectroscopy, depending upon the condensation temperature (Kalendarev et al., 1984; Rodionov et al., 1995). Black arsenic, which is also unstable, is most often found stabilized by other atoms (Osters et al., 2012). One such compound is arsenolamprite, a rare metallic mineral that has been mined in South America, Africa, and Western Europe.

Three major arsenic biotransformations occur in the environment: 1) oxidation and reduction (redox) transformations between As (III) and As (V), 2) inorganic arsenic metabolism by biomethylation, and 3) the subsequent biosynthesis of organoarsenic compounds (Gomez–Caminero et al., 2001).

Arsenic can exist in four oxidation states (−III, 0, +III, +V) but is rarely encountered as the zero–valence free element (Mohan and Pittman, 2007). As (−III) (arsine—a highly flammable and toxic gas) is also rarely encountered but can exist in anoxic environments where sulfate reducers function (Ferguson and Gavis, 1972; Oremland and Stolz, 2003). The arsenic species found in natural waters are mostly the trivalent (III) and pentavalent (V) inorganic forms, though As (V) is thermodynamically favored (Shankar et al., 2014).

In aqueous solutions, redox potential (E_H) is an indication of the capacity of the system to accept or donate electrons when a new species is introduced and is expressed in volts. Redox potential is a measure of the electron (e^-) activity of a solution and is analagous to pH, which is a measure of the hydrogen ion (H^+) activity of a solution.The higher the redox potential, the more affinity the solution has for electrons and is therefore more likely to be reduced. Reduction potential is obtained by measuring the potential difference between an inert sensing, or working, electrode acting as the electron donor or acceptor and a reference electrode. Platinum (Pt) is a commonly used sensing electrode, while silver/silver chloride (Ag/AgCl) is a common reference electrode. The universal reference electrode that all redox potentials are determined against is the standard hydrogen electrode (SHE). The SHE has been assigned a standard electrode potential (E^0) of zero at all temperatures under standard conditions (McNaught and Wilkinson, 1997). Redox potential and pH are two factors that determine the form arsenic will take in aquatic environments (Ferguson and Gavis, 1972; Gorny et al., 2015; Mandal and Suzuki, 2002). One can easily measure pH using litmus paper or a pH meter that measures the voltage between two electrodes, much like the system used for measuring E_H. The equilibrium phases of an aqueous electrochemical system can be shown in an E_H–pH, or Pourbaix, diagram as shown in Figure 1.

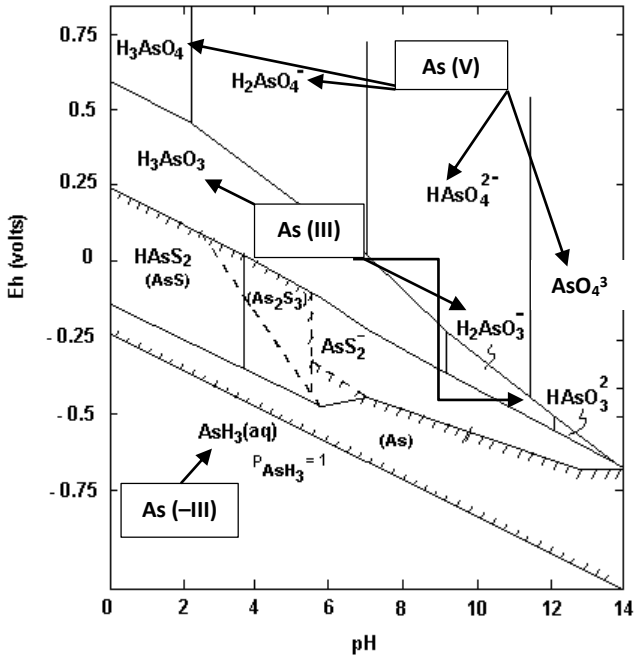


Figure 1: The E_H–pH diagram for As at 25°C and one atmosphere with total arsenic 10^{-5} mol/L and total sulfur 10^{-3} mol/L. Solid species are enclosed in parentheses in cross–hatched area, which indicates solubility less than $10^{-5.3}$ mol/L. Reproduced from Ferguson and Gavis (1972) with permission from Elsevier.

Arsenic has extremely high contamination potential in the environment when compared with other toxic trace elements due to its increased stability, solubility, and mobility over a wide range of pH and redox conditions (Barats et al., 2014). Other toxic metals such as chromium, selenium, vanadium, uranium, and molybdenum become insoluble in reducing environments due to stronger adsorption or reduction to their metallic forms. In the neutral conditions found in most groundwater (pH range of 6.5–8.5), metal cations such as Pb^{2+}, Cu^{2+} , Cd^{2+}, and Co^{2+} precipitate while anions, such as arsenates (i.e., As (V)), become more soluble as pH increases (Smedley and Kinniburgh, 2002). As shown in the E_H–pH diagram in Figure 1—in aqueous environments

containing oxygen, and sulfur—the arsenic sulfides realgar (AsS), also known as "ruby sulfur", and orpiment (As_2S_3) occur in low to moderate pH and moderate E_H conditions. These reducing environments include low temperature hydrothermal veins, volcanic fumaroles, and hot springs. Trivalent arsenous acid (H_3AsO_3) becomes mobile in moderately reducing conditions from 0.25 to ≈ 0.6 volts and a pH range of acidic < 2.5 to alkaline ≈ 9.5. Pentavalent arsenic acid (H_3AsO_4) is also stable in extremely acidic conditions (pH ≤ 2), but only in highly oxidizing conditions when redox potential is between 0.6–0.8 volts. Those conditions can be found during acid rain and in mining waters. The other pentavalent arsenates dominate in oxidizing conditions across the pH spectrum—dihydrogen arsenate ($H_2AsO_4^-$) domiates from pH 2to ≈7, monohydrogen arsenate ($HAsO_4^{2-}$) is mobile from pH ≈7 to 11 and arsenates of the form AsO_4^{3-} are stable from pH 11 to 14 (Sharma and Sohn, 2009).

Selective adsorption onto sediment and sediment minerals is another factor that determines the speciation and mobility of arsenic in aqueous environments (Feng et al., 2013; Goldberg et al., 2005). For example, it is widely accepted that iron oxides and oxyhydroxides are important arsenic adsorbants in aqueous systems (Dixit and Hering, 2003; Molinari et al., 2013; Sharma and Sohn, 2009). The iron oxides that arsenic strongly adsorbs onto include ferrihydrite (a widespread surface mineral with an incompletely understood structure), goethite (α–FeOOH), hematite (Fe_2O_3), limonite ($FeOOH \cdot nH2O$), magnetite (Fe_3O_4), and siderite ($FeCO_3$) (Gallegos–Garcia et al., 2012).

Competitive adsorption can also occur between arsenic and phosphorous. Arsenic and phosphorus are members of the nitrogen family (Group 15) of the periodic table and therefore have similar chemical properties (Rosen et al., 2011a). Their similarities allow arsenic to act as an analogue to phosphorus, which is an essential element (Wenzel 2013). There are multiple arsenic compounds analogous to phosphorus compounds: arsenic acid (H_3AsO_4) and phosphoric acid (H_3PO_4), yellow arsenic (As_4) and white phosphorus (P_4), and arsenate (As (V) in the form of AsO_4^{3-}) and phosphate (PO_4^{3-}). Arsenate and phosphate are both plentiful in nature, but arsenate is toxic while phosphate is an essential compound (Elias et al., 2012). The similarities between arsenate and phosphate and their ubiquity engender the question whether arsenic competes with and substitutes phosphorus in all biological processes involving phosphorus. Arsenate is known to enter cells via phosphate transporters; this has been observed in bacteria (*Escherichia coli* and *Streptococcus faecalis*), zebrafish, human erythrocytes, and rats (Beene et al., 2011; Harold and Baarda 1966).

The pH and E_H values in sediment drive the equilibrium of arsenic and its favored adsorption minerals. Arsenic partitions in soil in different physicochemical forms particular to clay content and organic matter, forming insoluble and immobile complexes with aluminum, magnesium, iron, and manganese (ATSDR, 2007; Goh and Lim, 2005). These complexes will solubilize in reducing soil and sediment conditions, potentially mobilizing to runoff into surface waters or leach into the groundwater. Masscheleyn et. al. (1991) reported that under these conditions (0–100 mV) As (V) coprecipitates with iron oxyhydroxides, releasing arsenic upon solubilization if the soil become oxidized.

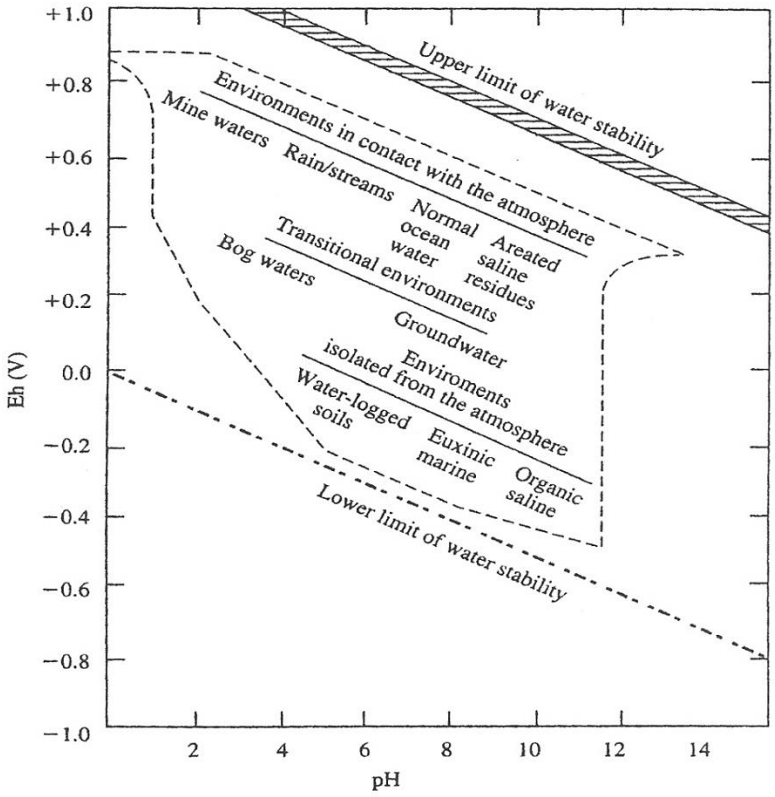


Figure 2: Natural environments in terms of E_H and pH (Langmuir et al., 2005).

Arsenic biotransformations

The classic metabolic pathway for arsenic transformations was proposed by Frederick Challenger in 1945 based upon 19th century studies on arsenic metabolism by microorganisms (Challenger, 1979; Drobna et al., 2009; Hughes et al., 2011). The pathway of arsenic metabolism in prokaryotes is a complex system of transporters, reductase enzymes, and protein–mediated chelation (Pandey et al., 2015). Microbial transformations of arsenic include redox, methylation and demethylation, and respiration reactions which are used as detoxification mechanisms (Cavalca et al., 2013; Lloyd and Oremland, 2006). Microalgae have been shown to reduce As (V) to As (III), methylate

inorganic arsenic to MMA and DMA, and form arsenosugars (Levy et al., 2005; Murray et al., 2003). Paul et al, (2015) reported that certain strains of As– and Fe– reducing bacteria (*Staphylococcus* BAS108i and *Arthrobacter* CAS4101i) catalyze the release of increased amounts of arsenic under anaerobic conditions while certain strains of arsenite–oxidizing bacteria (*Pseudomonas* CAS907i and BAS323i) hinder arsenic release under aerobic conditions.

Arsenic biotransformations in soil purportedly follow the Challenger pathway—alternating steps of reduction and oxidative methylation. These reversible transformations beginning with As (V) are shown in Figure 3. Challenger's scheme is depicted by solid arrows in Figure 4, which also includes alternative pathways proposed by Gao and Burau (1997) and Huang et al. (2007) that include demethylation to species occurring in soil.

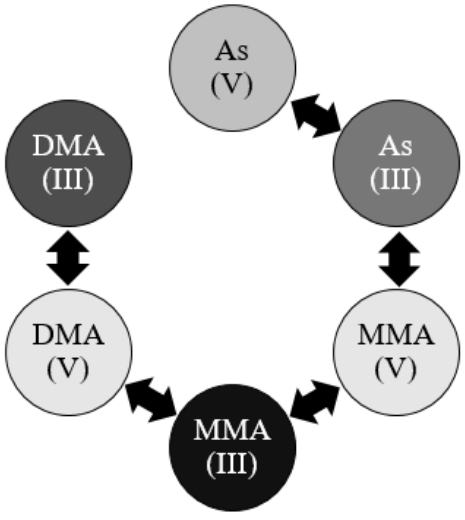


Figure 3: Arsenic biotransformations. Order of toxicity (Mass et al., 2001; Petrick et al., 2000) from most toxic to least toxic is depicted from dark (MMA (III)) to light (MMA and DMA (V)).

The steps of the Challenger pathway (Figure 4) are:

1) Reduction of arsenate (V) to arsenous acid (III)

2) Methylation of arsenous acid (III) to monomethylarsonic acid (MMA (V))

3) Reduction of MMA (V) to MMA (III)

4) Methylation of MMA (III) to dimethylarsinic acid (a.k.a. cacodylic acid (CA)) (DMA (V))

5) Reduction of DMA (V) to DMA (III)

6) Methylation of DMA (III) to trimethylarsine oxide (TMAO) (TMA (V)) and

7) Reduction of TMA (V) to trimethylarsine (TMA (III)).

Other proposed mechanisms of note pictured on Figure 4 but not considered a part of the classic Challenger pathway:

- Pathway labeled with ⑥: demethylation of MMA (V) to As (V)
- Pathway labeled with ⑦: demethylation of DMA (V) to As (V)

These two steps in alternative pathways of demethylation support the idea that arsenic biotransformations are not merely detoxification steps in a one–directional transformation scheme.

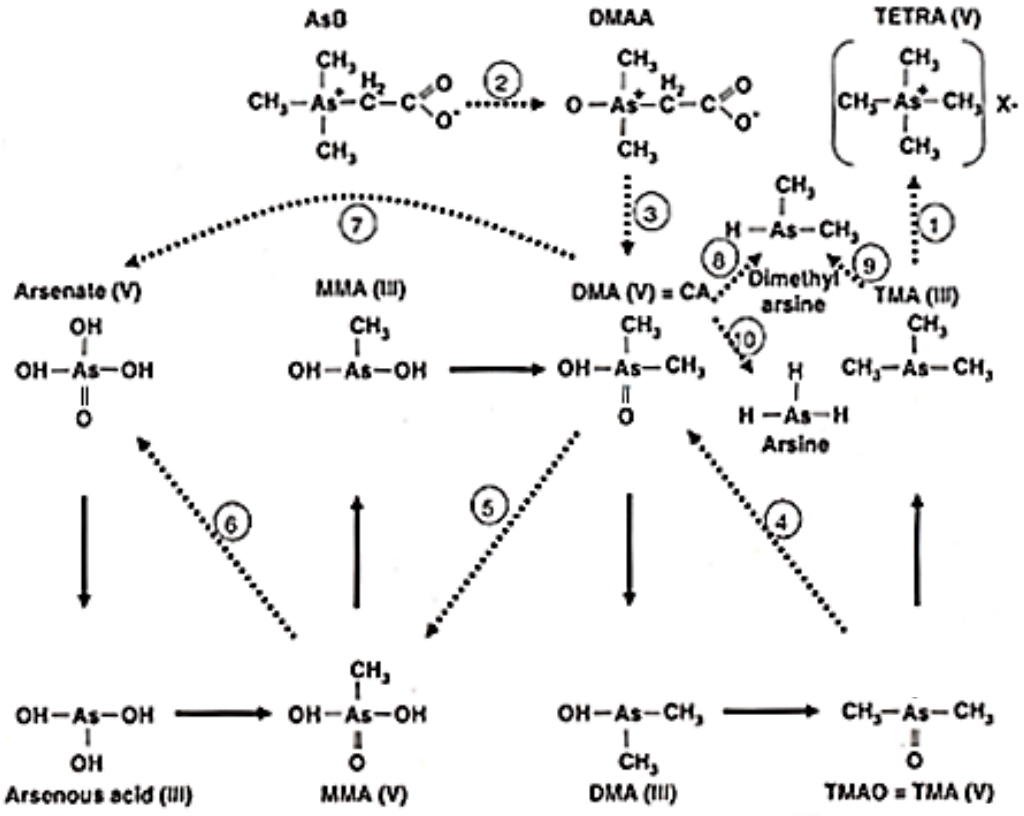


Figure 4: Biotransformation pathways of arsenic in soil (Wenzel, 2013).

Once arsenic has entered the environment as a contaminant, the question becomes one of bioavailability. There are numerous terms that tie into the concept of bioavailability. Once arsenic is absorbed, it can undergo excretion, metabolism, and induce toxic effects. Arsenic bioavailability is influenced by and dependent upon numerous site–specific variables including matrix composition, ecosystem processes, and the availability of human or ecological receptors (National Research Council, 2003). Moreda–Pineiro et al. (2012) assessed the bioavailability of six As species in raw seafood samples using an *in vitro* model that simulated gastric and digestion methods. They reported bioavailability percentages from 56%–114% in the non–toxic AsB and AsC

fractions, from 95%–114% for the metabolite DMA, from 90%–113% for the toxic As (V), and from 87%–106% for the most toxic species As (III). These high bioavailability percentages agree with values reported by Koch et al. (2007) and Laparra et al. (2007).

Table 2: Definitions related to bioavailability. Compiled from McGeer et al. (2004); Nowell et al. (1999).

Environmentally Available Fraction (EAF)	The total amount of metal in soil/sediment, water, or air that is available for processes such as fate, transport, or bioaccumulation
Bioaccessible Fraction (BF)	The fraction of environmentally available metal that is available for absorption or adsorption by an organism
Bioavailability	The bioaccessible metals that have been absorbed or adsorbed by an organism and have the potential for distribution, metabolism, elimination, or bioaccumulation
Relative Bioavailability (RBA)	The fraction of metal that has been absorbed or adsorbed by an organism under defined conditions as compared to a reference metal tested under standard conditions
Absolute Bioavailability (ABA)	The fraction of externally administered metal that has been absorbed and reaches systemic circulation or the central compartment of the receptor
Bioaccumulation	The net accumulation of metal in specific tissue or whole organism resulting from exposure
Bioconcentration Factor (BCF)	Ratio of the metal concentration in an organism to the metal concentration in water (in sediment and soil this is the net accumulation of metal in an organism from pore water only)
Bioaccumulation Factor (BAF)	Ratio of the metal concentration in an organism to the metal concentration in the surrounding medium
Biota–Sediment Accumulation Factor (BSAF)	Bioaccumulation of sediment–associated metals into tissues of ecological receptors
Biomagnification	Tissue concentrations of the metal increases as it moves up through at least two trophic levels of the food chain

Arsenic toxicity

Arsenic is both acutely and chronically toxic. It is the highest ranking substance on the Agency for Toxic Substances and Disease Registry (ATSDR) Priority List of Hazardous Substances. The list ranks hazardous substances commonly found on the National Priorities List (NPL) that have the potential to pose the greatest threat to human health based on toxicity, frequency of occurrence at NPL sites, and the potential of human exposure at those sites (ATSDR, 2013). Arsenic exposure can occur through dermal, oral, and inhalation routes and causes adverse effects to multiple organ systems. After ingestion of the organic "fish arsenicals" AsB and AsC from seafood, those relatively non–toxic compounds are rapidly excreted in the urine (Bhattacharya et al., 2007; Ratnaike, 2003). After chronic exposure and ingestion of inorganic arsenic, it accumulates in multiple sites including the heart, lungs, liver, kidneys, spleen, and the gastrointestinal tract (Ratnaike, 2003). Inorganic arsenic also deposits in the nails, skin, and hair after chronic exposure (Jiang et al., 2015). Approximately 70% of ingested arsenic (organic and inorganic) is excreted by the kidneys, though the inorganic arsenic species remain in the body longer and are not excreted as rapidly as the organic species (Abdul et al., 2015; Joseph et al., 2015; Ratnaike, 2003).

Many arsenic–related diseases have a long latency period, so people can fall victim to chronic exposure while remaining asymptomatic. Nevertheless, there are some classic symptoms associated with acute and chronic arsenic poisoning. According to the ATSDR, 600 μg/kg/day of ingested arsenic is a fatal dose for a human, causing death from hypovolemic shock and cardiovascular collapse, while the minimum risk level

(MRL) of acute oral exposure is 5 µg/kg/day for up to 14 days (ATSDR, 2007). Ingesting as little as 10 µg/L of arsenic via drinking water can cause adverse effects including skin lesions, diabetes, hepatic and renal dysfunction, and neurological complications (Abdul et al., 2015). Note: the EPA maximum contaminant level (MCL) for arsenic in drinking water is 10 µg/L (EPA, 2001). Not all acute arsenic poisonings are fatal, however. Some of the first signs of acute arsenic poisoning are common signs and symptoms of other illnesses, such as nausea and vomiting, headache, light headedness, lethargy, and abdominal pain. Other symptoms of acute arsenic poisoning include Mee's lines (transverse lines on the fingernails), garlic breath, dysphagia, dehydration, pulmonary edema, peripheral neuropathy, hematuria, and anemia, among others (ATSDR, 2009). The first and most recognizable manifestations of chronic arsenic poisoning, or arsenicosis, are skin blemishes characterized by hypo– and hyperpigmentation, keratosis, and Bowen's lesions. Hypo– and hyperpigmentation manifest as fine freckles distributed on the trunk and extremities, while keratosis appears primarily on the palms and soles of the feet. Bowen's disease (squamous cell carcinoma *in situ*) is characterized by lesions that are pigmented, well–delineated, rough, and may vary in size from 0.8 to 3.5 cm in diameter (Gahalaut et al., 2012; Tseng, 1977). Chronic arsenic exposure has been linked to an increased risk of diabetes, vascular disease, and cirrhosis (Drobna et al., 2009; Hughes, 2002).

Blackfoot disease (BFD), endemic to southwestern Taiwan, is a chronic peripheral vascular disease resulting from arsenic–contaminated artesian well water (Tseng, 1977). The use of artesian wells in that area ended when a tapwater system was

implemented in the 1970s (Chung et al., 2013). Blackfoot disease emerged in the 1930s and peaked in the 1950s, initially developing as hyperpigmented spots on the soles of the feet, graduating to blackened and hardened lesions that oftentimes resulted in gangrene and amputation (Chen, 1994; Tseng et al., 1996). Blackfoot disease is also associated with skin, kidney, bladder, and lung cancers—in fact, mortality rates of cancers have increased throughout Taiwanese populations exposed to arsenic (Chen and Wang, 1990; Cheng et al., 2015; Chung et al., 2013). Arsenic concentrations in contaminated wells across southwestern Taiwan have been reported from 10 µg/L to around 2,500 µg/L (Chen et al., 1985; Chung et al., 2013). The minimal level of arsenic to induce BFD has been reported as 350 µg/L (Chen and Wang, 1990).

Table 3 summarizes the four clinically recognized stages of chronic arsenic poisoning and Figure 4 displays some key effects of arsenic on major organ systems.

Table 3: Clinical stages of arsenicosis (Abdul et al., 2015; Choong et al., 2007).

Preclinical	Patient asymptomatic; arsenic metabolites can be detected in urine and/or tissues
Clinical	Dermal manifestations arise, including pigmentations and keratosis
Internal Complications	Internal organs may become enlarged (liver, kidneys, and spleen) with marked dysfunction
Malignancy	Tumors or carcinoma of skin and/or organs, Bowen's disease; gangrene may develop

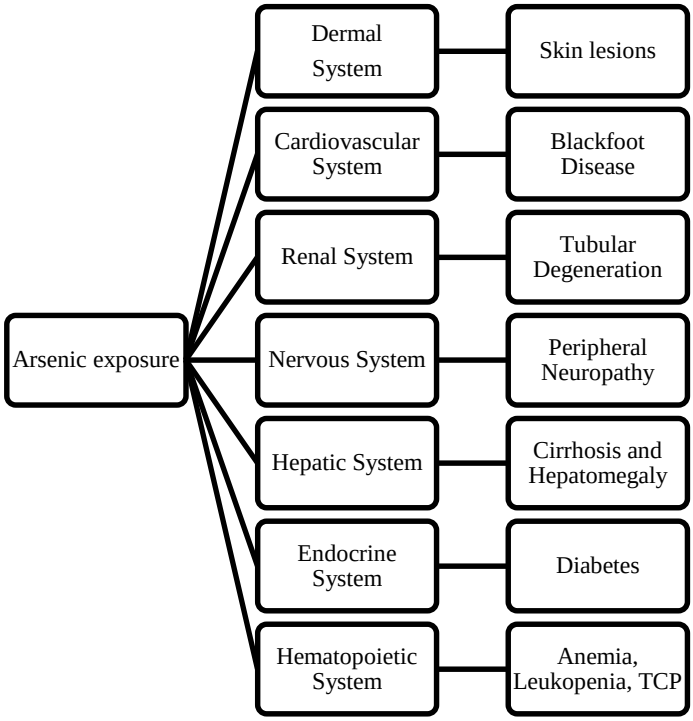


Figure 5: Key effects of arsenic on major systems. Modified from Abdul et al. (2015).

It has been a long held belief that arsenic methylation is a detoxification mechanism since inorganic arsenic is more toxic than its organic metabolites. This idea persisted because the primary metabolite, DMA (V), is less toxic than inorganic arsenic. However, Cullen et al., (1989) reported the increased toxicity of an MMA (III) derivative over As (III) to the yeast *Candida humicola*. Following the studies of the Cullen group, Petrick et al. (2000) performed *in vitro* cytotoxicity studies on human Chang hepatocytes based on cellular potassium and lactate dehydrogenase leakage and mitochondrial metabolism of a tetrazolium salt. From these cytotoxicity assays, they determined that MMA (III) was more toxic than As (III) and outlined an order of toxicity to human hepatocytes of MMA (III) > arsenite > arsenate > MMA (V) = DMA (V). Through *in vivo* toxicity studies based on kidney and purified porcine heart pyruvate dehydrogenase

inhibition, Petrick et al. (2001) reported that MMA (III) had a lower LD_{50} than As (III) in hamsters. These and other studies have brought around a new school of thought that arsenic methylation is not directly a detoxification process and some of the toxic and carcinogenic effects of arsenic are due in part to its methylated metabolites (Cullen, 2008; Hughes, 2002).

Mass et al. (2001) performed in vitro studies, determining an order of toxicity based on DNA damage to human peripheral lymphocytes of MMA (III) > DMA (III) > As (III) > As (V) > MMA (V) = DMA (V). They found that MMA (III) damaged DNA at a rate 77 times greater than As (III), and DMA (III) damaged DNA at a rate of 386 times greater than As (III) (Mass et al., 2001). Based on glutathione reductase inhibition and thiol affinity, Styblo et al. (2000; 2002) reported that MMA (III) was the most toxic form in all cell types tested, including liver, skin, bladder, and lung. They also observed that DMA (III) was at least as toxic as As (III) for most cell types. In addition, As (III) is known to be approximately 60 times more toxic than As (V) (Ferguson and Gavis, 1972; Ratnaike, 2003). Prior to these toxicity studies, inorganic arsenicals were considered approximately 100 times more toxic than all organic arsenic compounds (Jain and Ali, 2000).

The inorganic arsenic metabolites MMA (III) and DMA (III) work at the level of tumor promotion, inhibiting key enzymes, damaging DNA structure, and modulating AP–1–dependent gene transcription, inducing DNA binding that regulates cell proliferation and cell death (Simeonova and Luster, 2000; Styblo et al., 2002). Further

in–depth research needs to be conducted to determine the magnitude of increased toxic responses from methylated trivalent arsenicals over other arsenic species.

Arsenic (III) enters cells via membrane proteins and targets thiol enzymes, such as pyruvate dehydrogenase and 2–oxo–glutarate dehydrogenase, thus disrupting their ability to function in cellular respiration and the citric acid cycle—important biological processes that convert nutrients to energy and eliminate waste (Lloyd and Oremland, 2006).

Like other metal ions, arsenic ions are known inducers of oxidative stress, stimulating the production of reactive oxygen species (ROS) such as superoxides and hydrogen peroxide that can damage cellular tissue and induce carcinogenicity (Halliwell, 1992; Hughes et al., 2011; Lushchak, 2011; Singh et al., 2011). The genotoxic effects of ROS that contribute to carcinogenicity include deletion mutations, oxidative DNA damage and strand breakages, sister chromatid exchanges, and chromosomal aberrations (Klaunig et al., 2010; Shankar et al., 2014). Bin Sayeed et al., (2012) found that arsenosugars induced oxidative stress and DNA damage in the blood and hippocampus in swiss albino mice, causing motor and cognitive dysfunction.

Arsenic (V), known to metabolize to the more toxic As (III) species in humans, also interferes with phosphate–based energetic processes, inhibiting oxidative phosphorylation, thus resulting in the formation of an unstable arsenate–phosphate ester that reduces ATP production (Marlborough and Wilson, 2015).

Nemeti and Gregus (2013) termed the pentavalent methylated arsenicals MMA (V) and DMA (V), the major urinary metabolites of inorganic arsenic, as 'weakly genotoxic', noting the rapid reduction to the highly toxic trivalent methylated forms in rat livers. This has implications for human and ecological health and needs further investigation.

Human intake of arsenic occurs primarily through water and food ingestion, most notably seafood, thus there is a need to assess the human health risks associated with seafood consumption. This is especially true where subsistence fishing populations have their primary source of seafood, or have a seafood–based diet, from seafood caught in an oilfield produced water–contaminated ecosystem.

To assess human health risks associated with arsenic–contaminated seafood, previous studies have either: 1) assessed total arsenic concentrations in various organs or parts of an organism, or 2) speciated arsenic in specimens that have been homogenized or composited. However, human health risks are best assessed using matrices such as tissue screening levels (TSL), which measure contaminant levels in the edible portions of an organism (LDEQ, 2012), and sediments, which are contaminant sinks and act as a long–term source of pollution to aquatic life (EPA, 1985; Mukherjee et al., 2014).

In this research, the most toxic arsenic species present in sediments and blue crabs (*Callinectes sapidus*) from a Louisiana freshwater marsh oilfield were measured. Three tissue types—the meat, hepatopancreas, and shell—were chemically analyzed to assess arsenic species loads in edible tissues (i.e., meat) and in the whole organism (i.e., meat, hepatopancreas, shell), which is often consumed entirely. Such data will be used in

subsequent studies and risk assessments to evaluate potential health risks to humans, and likely ecological, populations that are ingesting such arsenic–contaminated crabs.

Study area

Louisiana is home to approximately three million acres of wetlands that are suffering losses of around 75 km^2 annually (Gomez, 2008; Williams, 1995). Wetland losses can be attributed to natural processes such as storm erosion as well as human activities including dredging and industrialization. Sediments undergo resuspension, transport, and redistribution from storm–induced erosion and scouring, affecting the lakes and canals in coastal landscapes (Freeman et al., 2015). Figure 6 illustrates the historical wetland loss of coastal Louisiana.

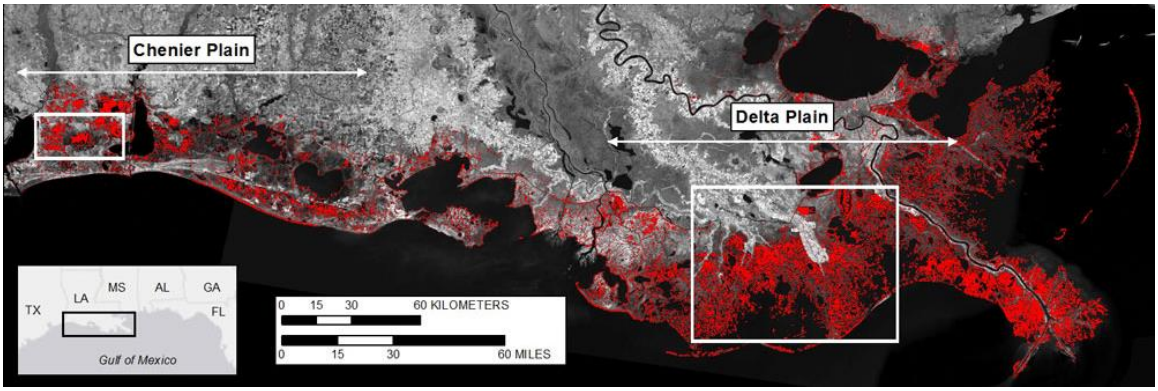


Figure 6: Satellite image showing the extent of historical wetland loss (in red) in coastal Louisiana. From USGS (2011).

Wetlands include swamps, peat bogs, and marshes, and they are some of the most productive and important natural systems on the planet, providing contaminant cycling, wildlife habitat, groundwater recharge, and food sources (Reddy et al., 2000). Wetlands are extremely biodiverse, acting as nurseries and refuges for many species, including the

threatened American alligator (*Alligator mississippiensis*) and endangered whooping crane (*Grus americana*), important members of Southwestern Louisiana's marshlands (Gomez, 1992; Williams and Dodd, 1978). Louisiana's wetlands extend approximately 300 km along the coast (Williams, 1995), which is divided into two regions: the Chenier Plain and the Mississippi River Deltaic Plain (Figure 7). The Chenier and Deltaic Plains are separated by Vermilion Bay (Visser et al., 2000).

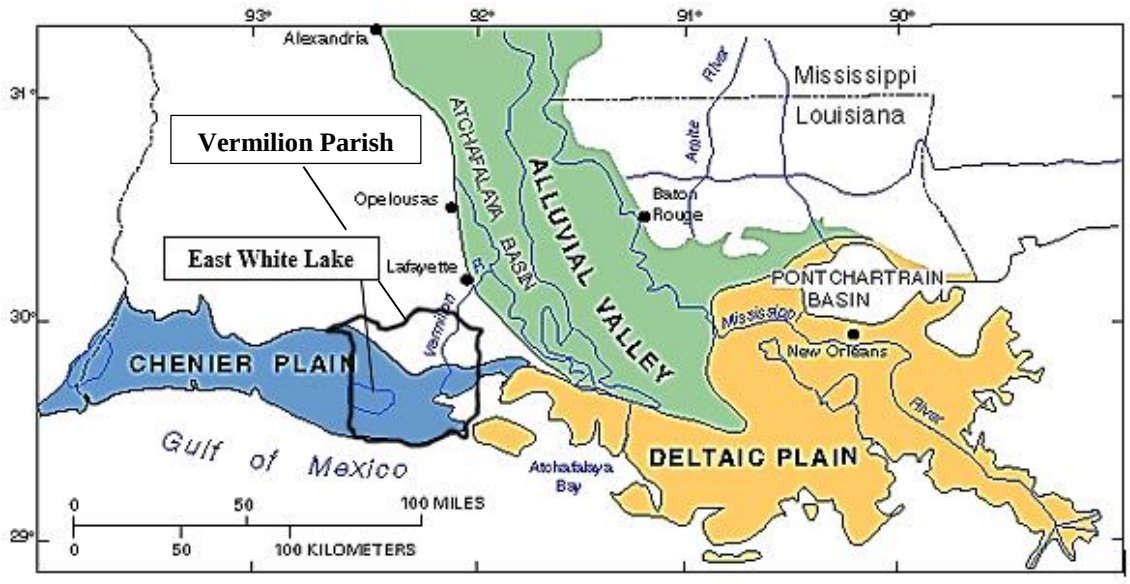


Figure 7: Map of Coastal Louisiana showing the boundaries of the Chenier and Deltaic Plains, Vermilion Parish, and East White Lake. Modified from Britsch et al. (1998).

The Chenier Plain is a 200 km long microtidal coast composed of cheniers (sand– and shell–rich beach ridges); tidal mudflats; and fresh, intermediate, and brackish marshlands (Gomez, 2008; McBride et al., 2007; Mo et al., 2015; Rosen and Xu, 2011). Through core analysis of the Chenier Plain, four faunal zones (*Streblus, Streblus–Elphidium, Quinqueloculina, and Trochammina*) and seven sedimentary facies

consisting of different ratios of sand, silt, and clay have been identified in the late Holocene deposits (Byrne et al., 1959; Henderson et al., 2002). The Holocene alluvium (formed primarily from the Mississippi, Red, and Ouachita Rivers) and coastal marsh deposits of the Chenier Plain occupy approximately 55% of Louisiana's surface exposures (Louisiana Geological Survey, 2010). Figure 8 illustrates the generalized geology of Louisiana.

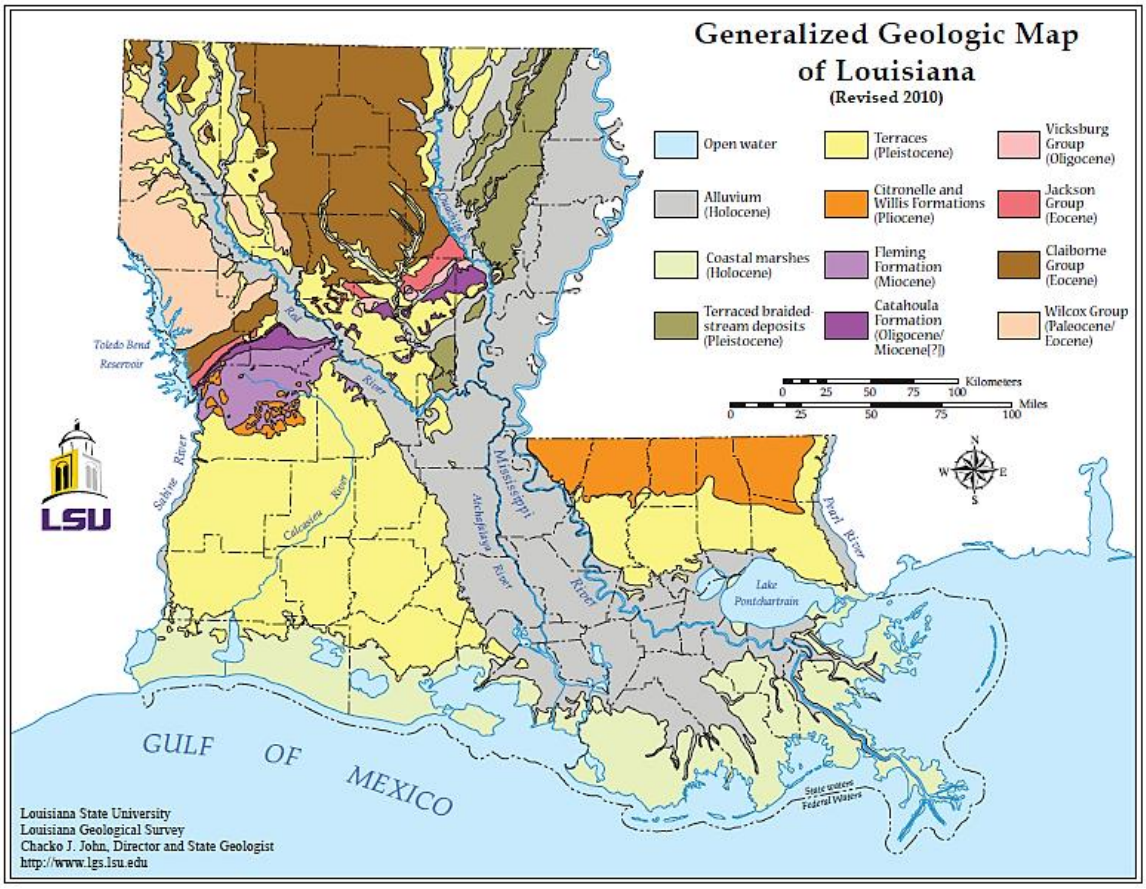


Figure 8: Generalized geologic map of Louisiana. (Louisiana Geological Survey, 2010).

The Chenier Plain is bisected north to south by the Sabine, Calcasieu, Mermentau, and Vermilion Rivers that drain into Sabine, Calcasieu, and Grand/White Lakes, and Vermilion Bay, respectively (Nichol et al., 1992; Phillips, 2003; Rosen and Xu, 2011). These rivers' drainage basins are connected by the Gulf Intracoastal Waterway (GIWW), an important shipping channel for numerous commercial industries.

Oil and gas in Louisiana

Figure 9 shows U.S. oil and natural gas production in 1998. Today, Louisiana is the number two producer of crude oil in the United States, providing over 300,000 jobs and billions of dollars in state revenue (Scott, 2014).

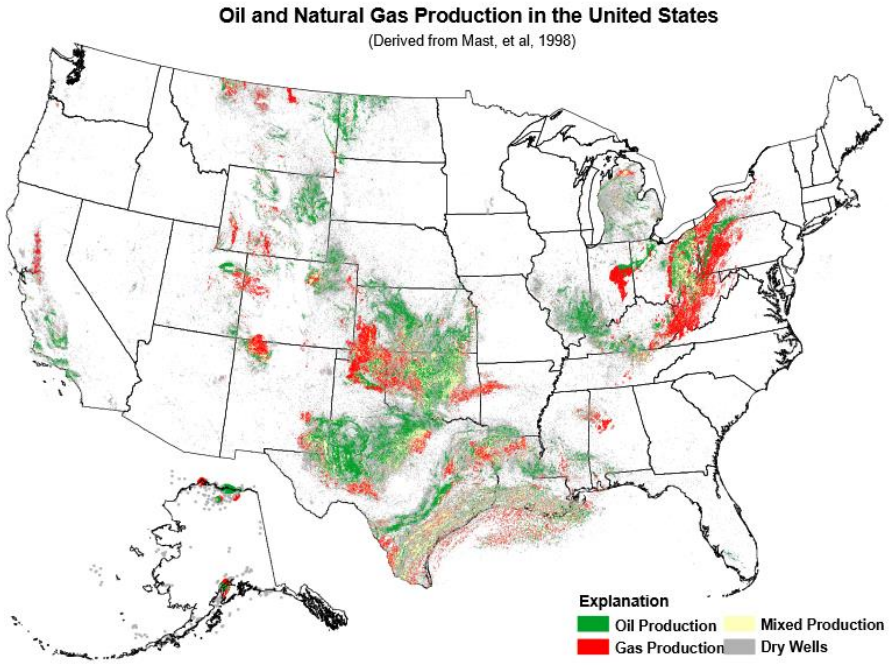


Figure 9: Oil and natural gas production in the United States. Source: Mast, et al. (1998).

Large volumes of water are stored in reservoirs tapped for oil and gas production. That water—termed produced water—is brought to the surface as a byproduct of oil and gas extraction; in fact, produced water is the largest volume waste associated with oil and gas production (USGS, 2013; Veil et al., 2004). The composition of produced water—also called oilfield water or brine—varies greatly depending upon the type of resource extracted, oilfield life stage and geographical location, and formation geochemistry (Guerra et al., 2011; OGP, 2005). Most produced water contains hydrocarbons, inorganic salts, chemicals used during oilfield operations, dissolved gases, radionuclides (unstable radioactive isotopes) including naturally occurring radioactive material (NORM; $^{226, 228}$Ra, ^{238}U daughter products, and ^{232}Th), and metals such as arsenic, barium, and lead (Barbee and Castille, 2010; De Felippo, 2014; Fisher, 1995). The water–to–oil ratio increases over the lifetime of a producing oil well. Current estimates are that U.S. wells produce 7–10 barrels (an oilfield barrel equals 42 U.S. gallons or ~ 159 liters) of water per barrel of oil—a number that adds up to 20–30 billion barrels of produced water annually (Otton and Mercier, nd; Veil, 2015; Veil et al., 2004).

Southern Louisiana oil and gas exploration and production (E&P) occurs in stratified Upper Cretaceous, Lower Eocene, and Quaternary horizons (Harris, 1910) of the Oligocene/Miocene Frio Sand Formation of the South Louisiana Salt Basin (Figure 10). Veil et al. (2004) reported that between 1985 and 2002, Louisiana annually generated approximately 1.3 billion barrels of onshore produced water along with 200 million barrels of oil. According to the U.S. Department of Energy, as of August 2015,

Louisiana ranked 9th out of 31 states in crude oil production (5,282,000 barrels) (EIA, 2015).

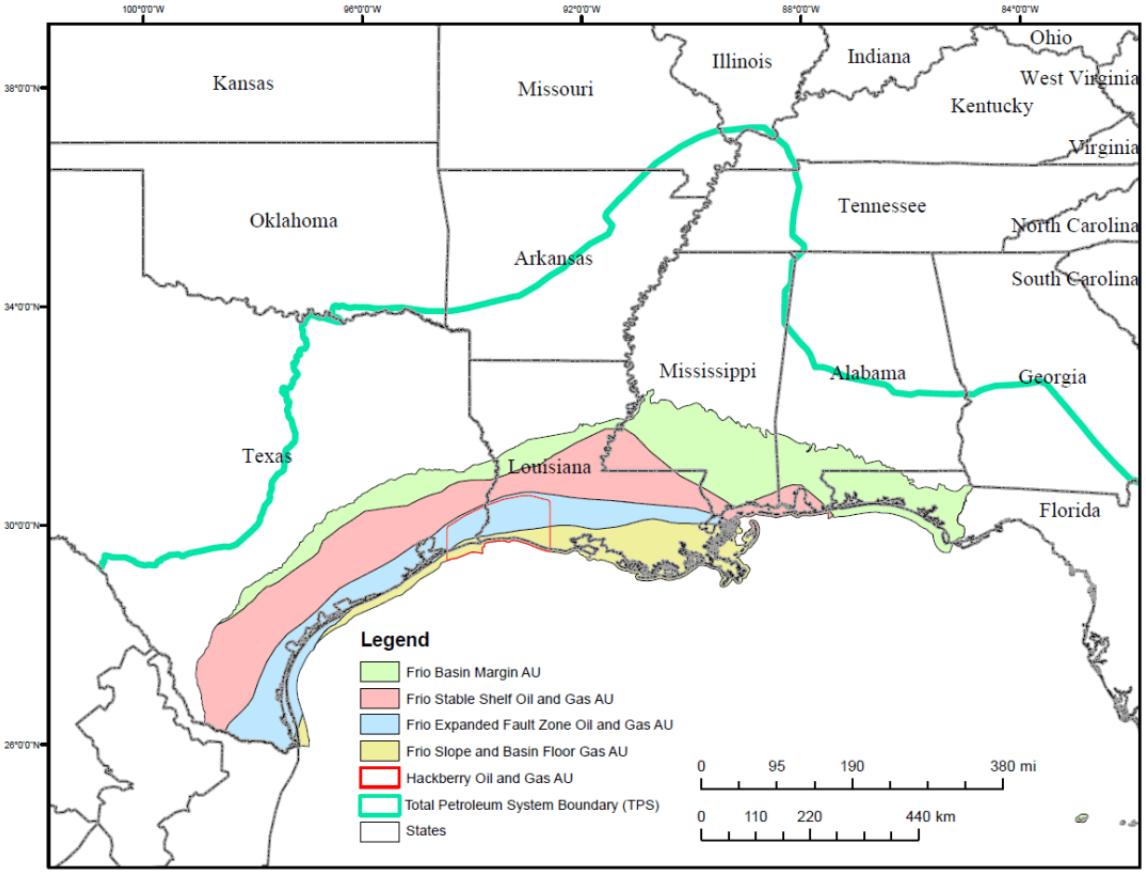


Figure 10: Assessment units of the Frio formation (Circle Star Energy, 2015).

The Jennings oilfield in Acadia Parish, fifty miles inland from the Gulf Coast, is home to the first oil well drilled in Louisiana. This well was drilled into the Port Hudson (Pleistocene) formation in 1901 (Fenneman, 1906). The location of the Jennings oilfield is marked with a red star in Figure 11.

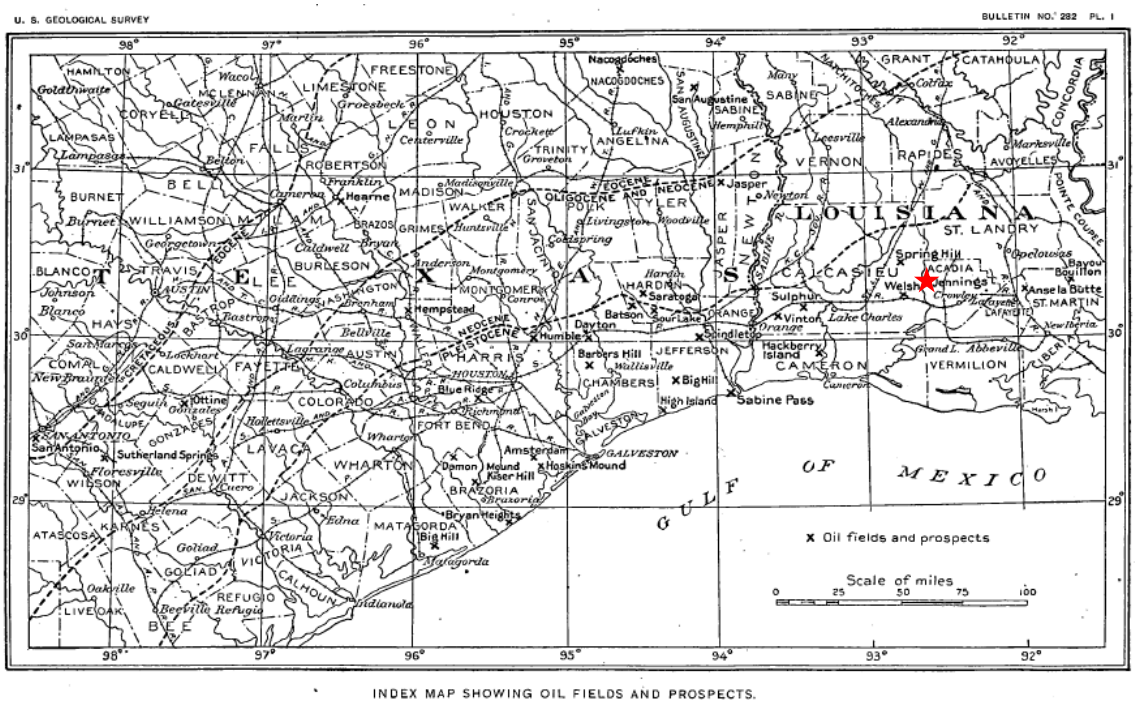


Figure 11: Index map showing oilfields and prospects in the Texas and Louisiana Gulf Coast, 1906. The Jennings oilfield is marked with a red star. Modified from Fenneman (1906).

The second oilfield to appear in Louisiana was the Caddo oilfield located in northwestern Louisiana's Caddo Parish. The center of the oilfield, north of Caddo Lake, came to be known as Oil City (Harris, 1910). Caddo Parish is also home to the first oil well drilled over water, which was drilled in 1911.

In 1909, the Perry Oil and Mineral Company drilled wells 1½ miles east of Abbeville, Louisiana in Vermilion Parish (Harris, 1910). During this E&P activity, well operators and geologists gathered data regarding oil and gas strata and developed a list of productive formations (Harris, 1910).

Oil and gas development activities began in 1935 in Schooner Bayou Canal (Barbee and Castille, 2010), a portion of which lies along the Northern border of the East White Lake oil and gas field (Figure 14).

By 2007, Vermilion Parish was labeled a medium–level oil producer, holding the following rankings in relation to the other southern Louisiana parishes: 9th in production wells (102 total) 13th in dry holes, and 38th in horizontal wells (BLM, 2008).

The East White Lake (EWL) oil and gas field is situated directly east of East White Lake in an isolated freshwater marsh in Vermilion Parish, Louisiana (Barbee and Castille, 2010) (Figures 13 and 14).

Figure 12: White Lake with the East White Lake oil and gas field to the east. Source: Google Earth, 29°42′49.66″ N 92°20′54.51″ W. Imagery date 10/30/2014.

Figure 13: East White Lake oil and gas field and Schooner Bayou Canal. Source: Google Earth, 29°43′35.80″ N 92°22′15.00″ W. Imagery date 10/30/2014.

The White Lake Wetlands Conservation Area (WLWCA) is comprised of more than 50,000 acres of marshland habitat for alligators, shorebirds, and waterfowl, including the whooping crane (LDWF, 2004). In 2011, the Louisiana Department of Wildlife and Fisheries (LDWF) worked in conjunction with the Louisiana Cooperative Fish and Wildlife Research Unit, the US Fish and Wildlife Service, the USGS, and the International Crane Foundation to reintroduce the endangered whooping crane into the WLWCA (Boehringer and MacKenzie, 2011). The blue crab (*Callinectes sapidus*) is an important component of the whooping crane diet (Pugesek et al., 2013). Figure 15 shows the location of the WLWCA on the northern edge of White Lake, in close proximity to the East White Lake oil and gas field.

The marshes surrounding the EWL oil and gas field, and other coastal zones of the Chenier Plain, are economically important in southwestern Louisiana for two disparate but important industries—fishing/crabbing and petrochemical exploration and refining (DeLaune et al., 2008). The communities surrounding White Lake rely on the oil and gas industry for work, and they rely on the catch that comes out of the lake and freshwater marshes for their diet and livelihood. Because these two industries typically take place on the same land, any release of oil and gas contaminants during E&P activities potentially can contaminate fish and crab products taken from these same lands.

Figure 14: Map of the White Lake Wetlands Conservation Area (WLWCA) north of White Lake. Source: Google Earth, imagery date 4/9/2013.

Regional wildlife include shrimp, channel catfish, blue crab, gar, largemouth bass, and crappie (Barbee and Castille, 2010). Blue crabs in the Gulf of Mexico mate year round in estuarine environments and the catadromous female migrates to spawn in high

salinity waters (Guillory et al., 2001). In general, juvenile blue crabs are widely distributed in the estuaries while the females tend to remain in high–saline waters and the males are commonly found in waters of low salinity (Guillory et al., 2001). The mean salinity of the East White Lake oil and gas field across four monitoring sites (S8, S9, S10, and S11) in Figure 16 from 6/23/2015 to 8/4/2015 was measured as 0.2 ppt (parts per thousand), which indicates the East White Lake oil and gas field resides in a freshwater system (USACE, 2009).

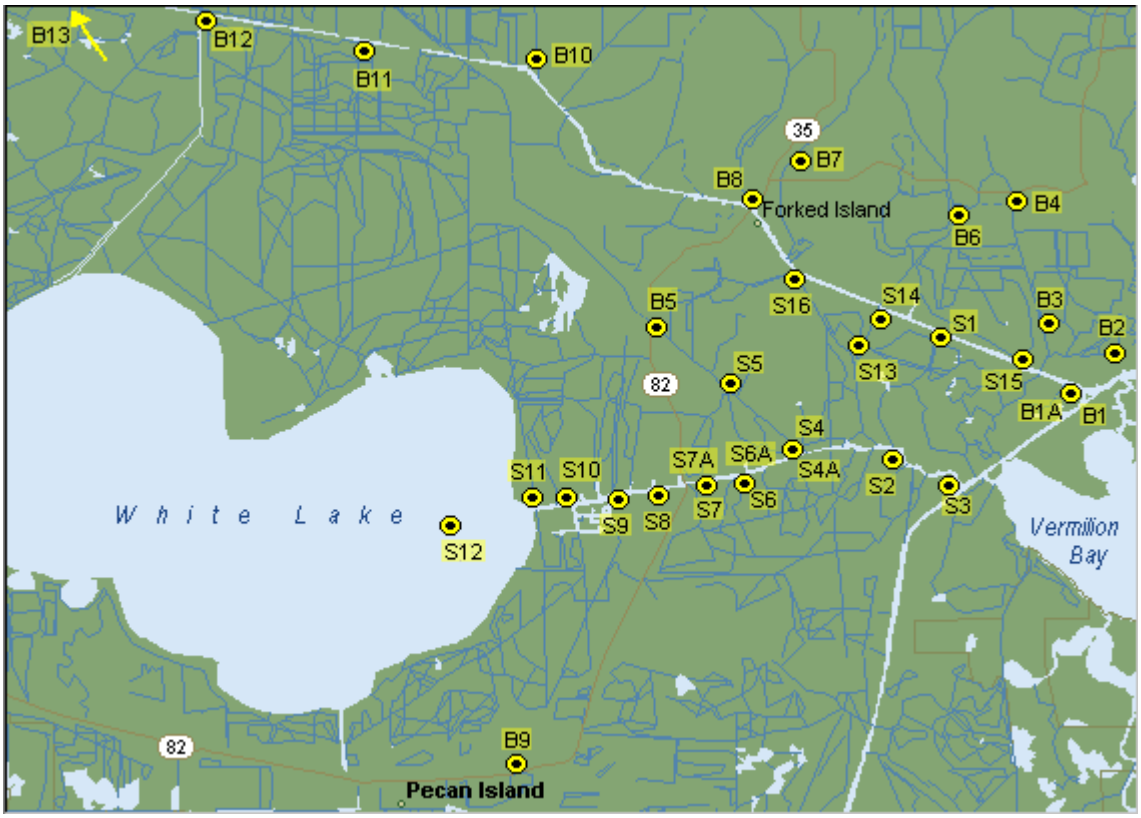


Figure 15: Schooner Bayou, Leland Bowman Lock, and Gulf Intracoastal Waterway (GIWW) Saline Monitoring Sites.

Sediments of White Lake and the coastal Chenier Plain that have been carried down by the Mississippi River are dense composed of organic marsh silt, clay, and peat (Byrne et al., 1959; Frazier and Osanik, 1969). Figure 16 and Table 4 describe the soil series in the East White Lake oil and gas field (USDA, 2015).

Figure 16: USDA soil series of the East White Lake oil and gas field (USDA, 2015).

Table 4: Soil series found at the East White Lake oil and gas field (USDA, 2015).

Map Unit Symbol	Map Unit Name	Description	Acres in AOI	Percent of AOI
AE	Allemands mucky peat, 0 to 0.5 % slopes, tidal	Mucky peat, clayey, smectitic, herbaceous organic material	1,954.5	55.3 %
LR	Larose mucky clay, 0 to 0.2 % slopes, tidal	Herbaceous organic material over fluid clayey alluvium	350.3	9.9 %
AN	Aquents, frequently flooded	Alluvium	125.7	3.6 %

CHAPTER II

MATERIALS AND METHODS

Sample collection

Sediment and crab samples were collected from 13 locations around the East White Lake oil and gas field: three from White Lake (L–1, L–2, and L–3) and ten directly from within the oil and gas field (S–1 through S–10) (Figure 17).

Figure 17: Locations of crab and sediment sampling sites around the East White Lake oil and gas field. Arsenic speciation was performed for locations marked with red pins. Source: Google Earth, imagery date 10/30/2014.

At each sampling location shown in Figure 17, five sediment grab samples from a 100 square foot area were composited and thoroughly mixed to generate one sediment sample. The surface sediment (i.e., 0–6” depth) was collected, which represents the greatest exposure concentrations to the crabs.

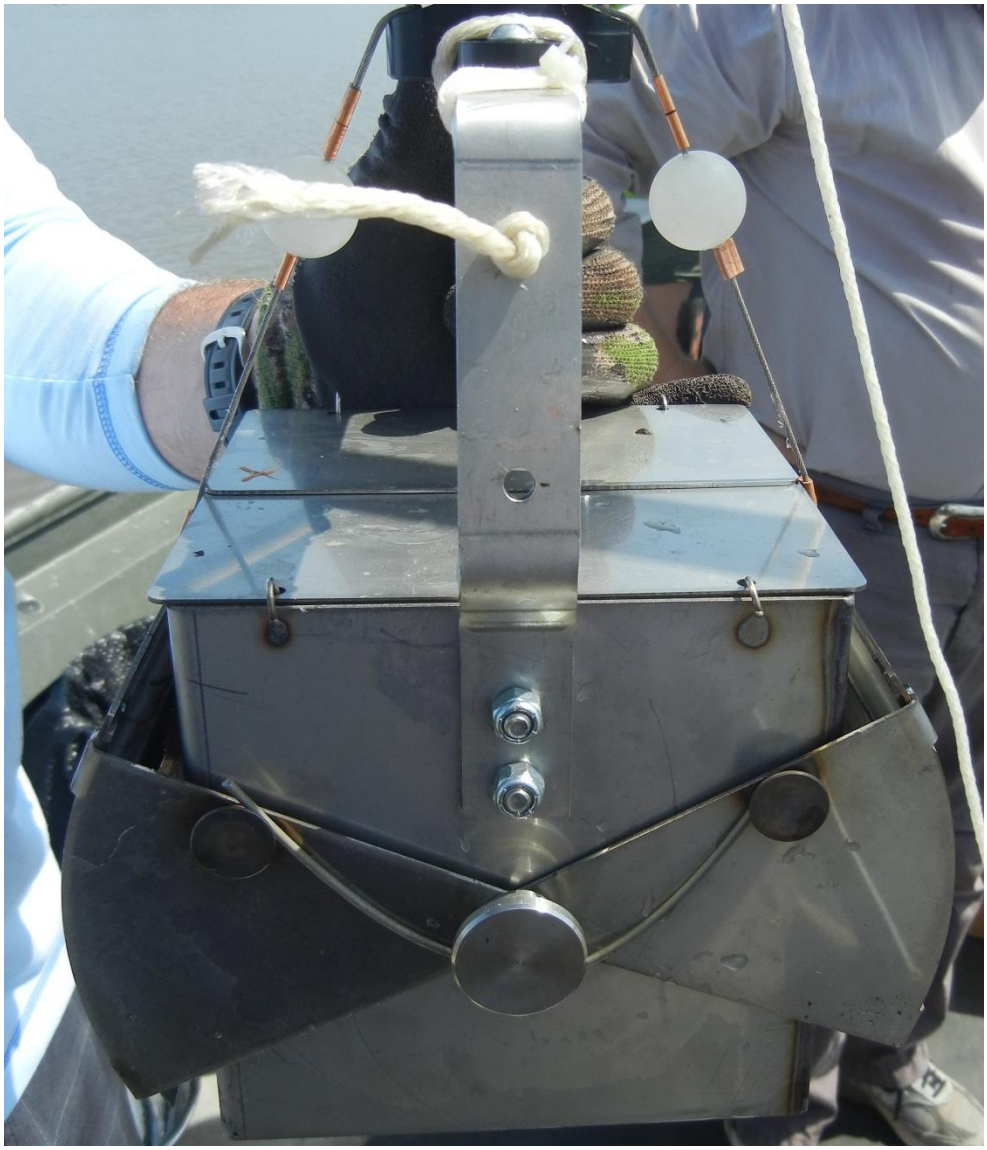


Figure 18: Ekman bottom grab sampler from Rickly Hydrological Corporation.

Figure 19: Sampling location L–1 in East White Lake. GPS coordinates 29°44′087″ N 92°23′114″ W.

Figure 20: Ekman sampler being pulled from location L–1 in East White Lake.

Figure 21: Emptying sediment from the Ekman sampler at location L–1 into a clean stainless steel bowl.

Figure 22: Sediment sample bottles.

Once the sediment was composited and homogenized at each sampling location, it was placed into a 500 mL Nalgene® high density polyethylene (HDPE) sample container (Figure 22) containing 3 mL of nitric acid preservative (catalog number 0500–15NA QC from Environmental Sampling Supply, San Leandro, CA). The containers were Quality Certified™ pre–cleaned with Teflon®–lined closures and came with a quality control (QC) certificate tracing to independent certification. The sample containers were filled to the top and immediately placed on wet ice to preserve the arsenic species present. The sediment samples were kept on wet ice and returned to the Core Analytical Laboratory (the Core Lab) at West Texas A&M University (WTAMU) where they were immediately put into a laboratory refrigerator and maintained at ~ 4 °C until sample extraction.

Male blue crabs were collected from 6/25/2015 to 8/4/2015 in crab traps deployed at locations marked in Figure 17. The total number of crabs collected is shown in Table 5. Male crabs were used in this study for two reasons: 1) the ratio of males to females in this freshwater marsh ecosystem during the sampling period was 9:1, so insufficient numbers of female crabs were collected, and 2) male crabs are preferred for assessing exposure and contaminant uptake because their home range and extent of migration is much smaller.

Table 5: Number of male crabs collected at each crab trap location.

Trap #	L–1	L–2	L–3	S–1	S–2	S–3	S–4	S–5	S–6	S–7/8	S–9	S–10
# Crabs	5	10	5	3	4	3	2	6	9	0	4	2

As soon as crabs were pulled out of the traps they were put on dry ice to preserve the arsenic species present in the different tissue types. The crabs were kept on dry ice and transported to the WTAMU Core Lab where they were immediately placed in an ultra–low laboratory freezer maintained at –80 °C until processed for analysis.

Figure 23: Crab caught in crab trap. Non–target organisms were released alive when possible.

The goal of this research was to collect ten male and ten female crabs from each location. As mentioned previously, since those numbers were not achieved, the decision was made to speciate arsenic from locations where at least 2 or 3 males were collected. Three lake locations and three East White Lake oil and gas field locations were chosen. A further decision was made to speciate arsenic only in male crabs for consistency since insufficient numbers of female crabs were collected. Figure 28 shows the anatomical differences in the abdomen and claws between female and male blue crabs.

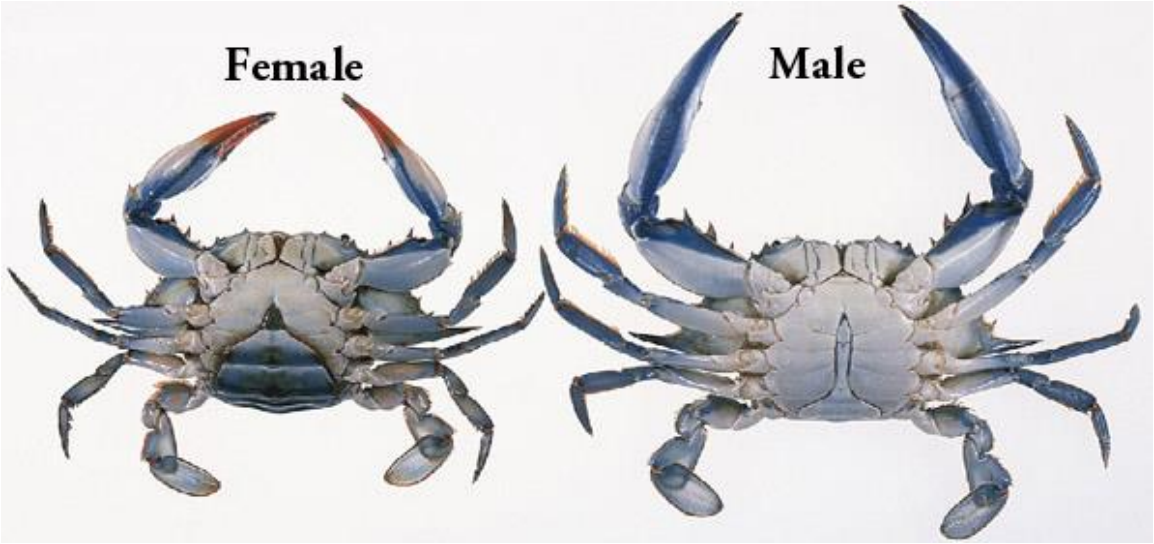


Figure 24: Abdominal apron and claw tip differences in female and male blue crabs. Note the wide apron and orange–tipped claws indicative of female crabs and the narrow, inverted "T" shaped apron and uniform blue claws indicative of male crabs. Modified from FDA (2015).

Crab processing

Frozen crabs were weighed for total body mass. Carapace width was measured from lateral spine to lateral spine as indicated in Figure 25.

Figure 25: Measuring carapace width from lateral spine to lateral spine.

The frozen crabs were then cut down the midline using a Craftsman 10–inch band saw (Figure 26). Figure 27 shows the medial aspect of two crab halves. The right half of the crab (looking from the dorsal anterior aspect) was immediately placed back in the freezer and the left half was weighed for total mass and dissected to obtain muscle meat, shell, and hepatopancreas for analysis.

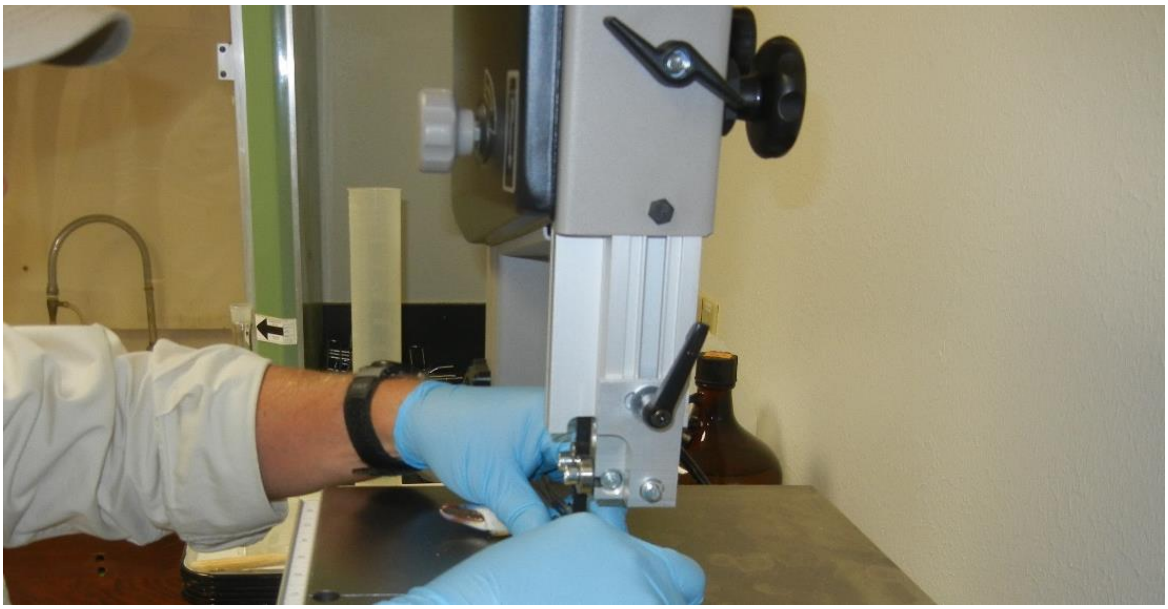


Figure 26: Crabs were cut along the medial line using a band saw.

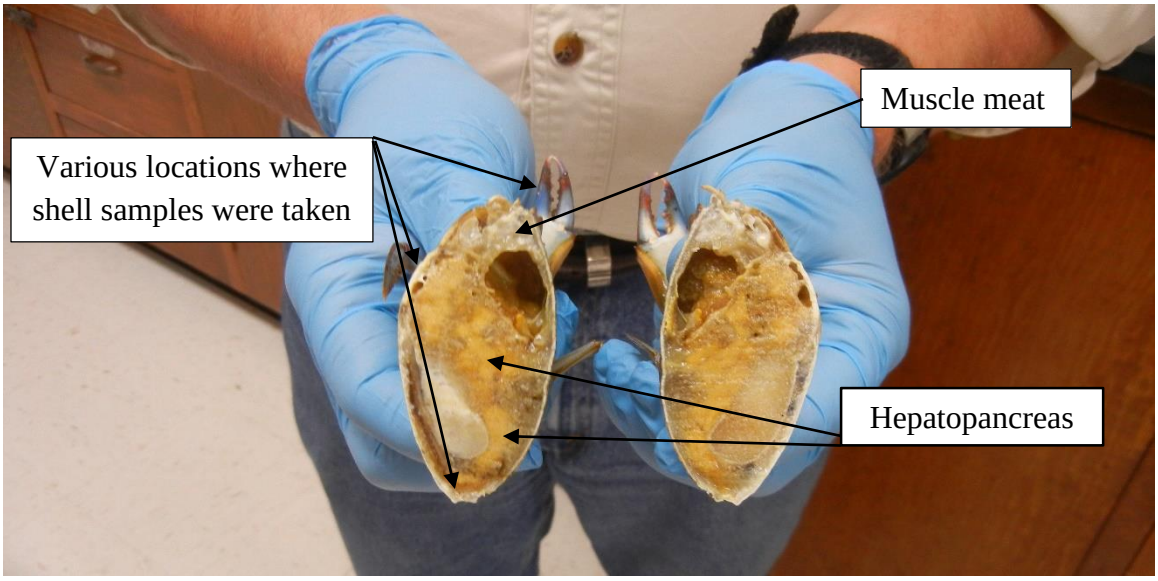


Figure 27: Blue crab cut down the medial aspect showing where samples were obtained.

There is no detailed procedure for determining a size–age relationship of blue crabs, which are considered mature anywhere from age one to eighteen months (Guillory et al., 2001). Accurately determining a blue crab's age and growth can be difficult since growth is a stepwise process occurring during molting (Guillory et al., 2001). Perry (1975) and Tagatz (1968) published the estimations listed in Table 6 based on mean percent growth per molt, mean molt interval, and pre– and post–molt carapace width measured from lateral spine to lateral spine. Tagatz (1968) and Fischler (1959) noted a negative correlation between size and salinity of water where molting occurs.

Table 6: Carapace widths (CW) of age one blue crabs.

Reference	Perry (1975)	Perry (1975)	Tagatz (1968)
Sex	Female	Male	
Pre–molt CW	119 mm	120 mm	142 mm mean
Post–molt CW	163 mm	151 mm	

Based on the guidelines published by Perry (1975) and Tagatz (1968), age estimations were determined for the crabs processed for arsenic speciation analysis (Table 7).

Table 7: Crab masses, carapace widths, anomalies, and age estimations using guidelines published by Perry (1975) and Tagatz (1968).

Crab	Whole crab wet weight (g)	Carapace width (cm)	Age estimation	Left half wet weight (g)	Anomalies
L–1–1	258.5	18.4	1+ year; post–molt	148.9	None noted; Figure 28
L–1–2	100.1	12.8	1 year; pre–molt	46.1	None noted; Figure 28
L–1–3	86.6	12.0	1 year; pre–molt	41.0	None noted; Figure 28
L–2–1	121.4	14.4	1 year; pre–molt	62.6	None noted; Figure 29
L–2–2	103.9	14.5	1 year; pre–molt	61.3	Black, glassy mass anterior HP; Figure 30
L–2–3	252.2	19.0	1+ year; post–molt	120.9	None noted; Figure 29
L–3–1	105.7	13.5	1 year; pre–molt	54.0	None noted; Figure 31
L–3–2	113.4	13.0	1 year; pre–molt	56.1	None noted; Figure 31
L–3–3	160.3	15.0	1+ year; post–molt	82.5	None noted; Figure 31
S–1–1	152.2	13.9	1 year; pre–molt	77.9	None noted; Figure 32
S–1–2	88.3	12.1	1 year; pre–molt	43.7	None noted; Figure 32
S–1–3	69.0	11.4	Juvenile	32.9	Very soft, fragile shell; Figure 32
S–2–1	77.5	11.3	Juvenile	37.6	None noted; Figure 33
S–2–2	116.2	14.0	1 year; pre–molt	60.8	None noted; Figure 33
S–2–3	132.4	13.6	1 year; pre–molt	62.7	None noted; Figure 33
S–6–1	215.1	16.5	1+ year; post–molt	105.2	Scratch resistant brown shell; Figures 34 and 35
S–6–2	138.0	14.0	1 year; pre–molt	66.8	None noted; Figures 34 and 35
S–6–3	155.1	13.9	1 year; pre–molt	75.1	Scratch resistant brown shell; Figures 34 and 35

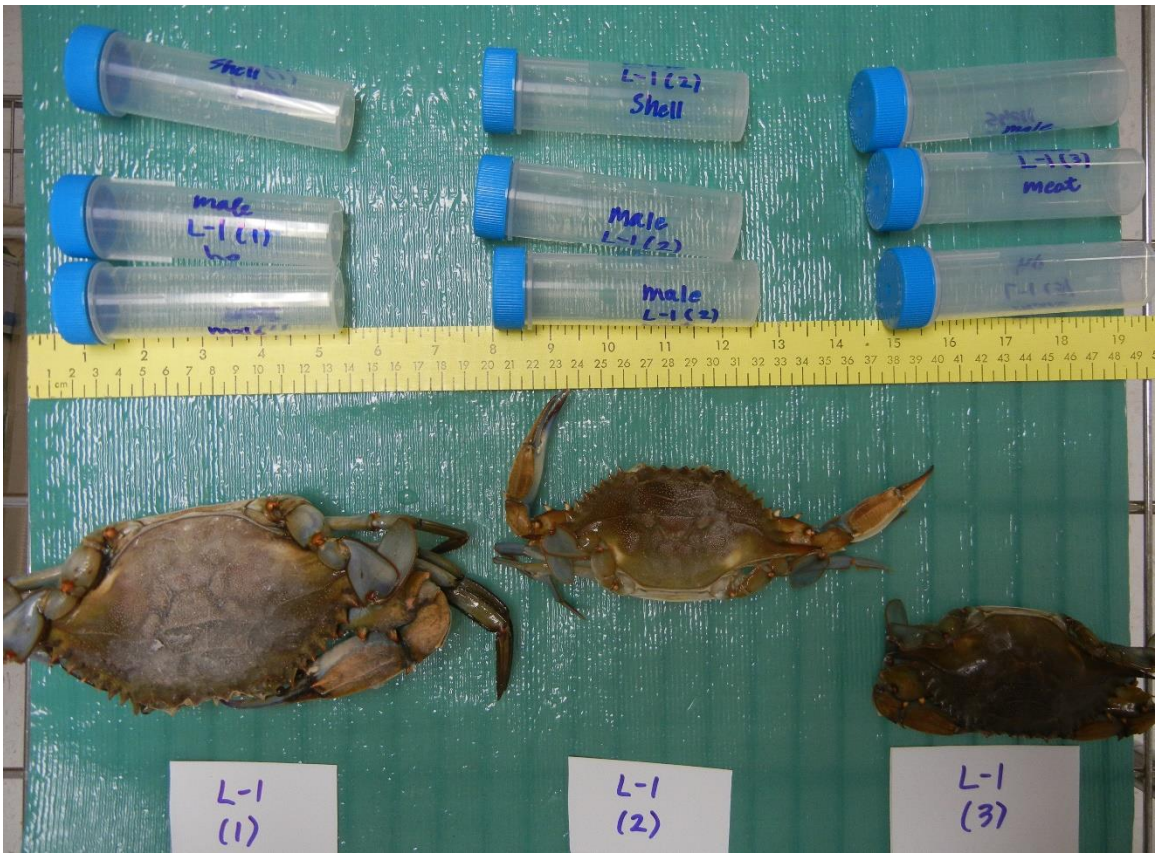


Figure 28: Dorsal view of crabs from the L–1 location.

Figure 29: Dorsal view of crabs from the L–2 location.

Figure 30: Black, glassy mass found in crab number 2 from the L–2 location.

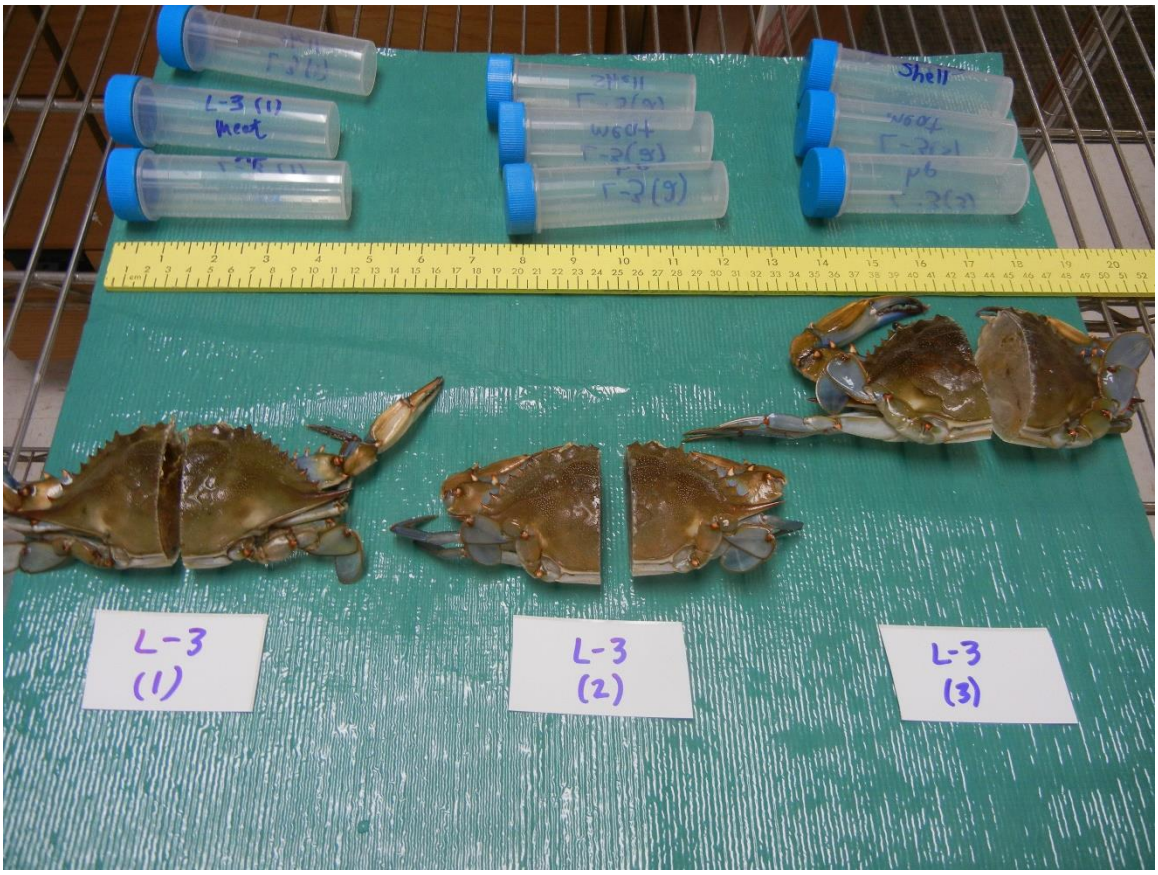


Figure 31: Crabs from the L–3 location cut along the medial line.

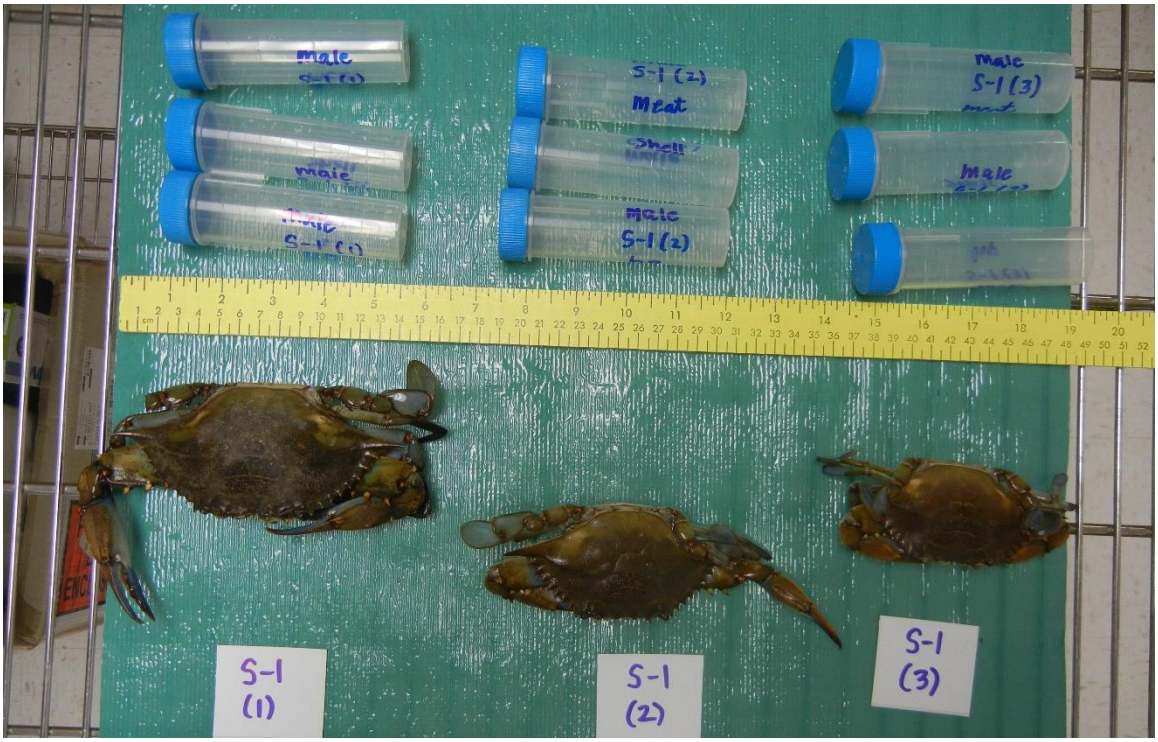


Figure 32: Crabs from the S–1 location.

Figure 33: Crabs from the S–2 location.

Figure 34: Dorsal view of crabs from the S–6 location showing discolorations, likely from hydrocarbons.

Figure 35: Ventral view of crabs from the S–6 location showing discolorations, likely from hydrocarbons.

Experimental

Instrumentation

Ion exchange chromatography/inductively coupled plasma–mass spectrometry (IEC/ICP–MS) was performed on a Thermo Scientific™ Dionex™ ICS 5000^+ IC system equipped with an AS–DV autosampler, a single pump all–PEEK™ (polyether ether ketone) pathway, an injection valve with a 20 µL injection loop, an IonPac™ AG7 guard column, and an IonPac™ AS7 anion exchange column (2 mm i.d. x 250 mm; 10 µm particle diameter) (all from Thermo Scientific™ Dionex™ , Waltham, MA). The IonPac™ AS7 anion exchange column contains a three–region patented packing including an inert and stable PEEK™ core, a sulfonated core surface region, and a permanent outer layer—2% divinylbenzene (DVB) cross–linked sub–micron anion exchange MicroBeads™ with a high loading capacity and short diffusion path (Figure 36). The IC system was coupled to a Thermo Scientific™ iCAP™ Q ICP–MS equipped with a Teflon™ PFA–ST MicroFlow nebulizer (Elemental Scientific, Omaha, NE), quartz torch, and quartz cyclonic spray chamber (Thermo Scientific™ Dionex™ , Waltham, MA) via a 60–cm long PEEK™ transfer line (150 µm i.d.). For total As determination, a concentric glass nebulizer (Thermo Scientific™ Dionex™ , Waltham, MA)—connected to a CETAC Technologies (Omaha, NE) ASX–520 autosampler via an inline internal standard mixing kit—was used in place of the MicroFlow nebulizer, and the solutions were delivered using a peristaltic pump and orange/yellow (0.508 mm i.d.) flexible polyvinyl chloride (PVC) tubing for internal standard and black/black (0.508 mm i.d.) flexible polyvinyl chloride (PVC) tubing for samples.

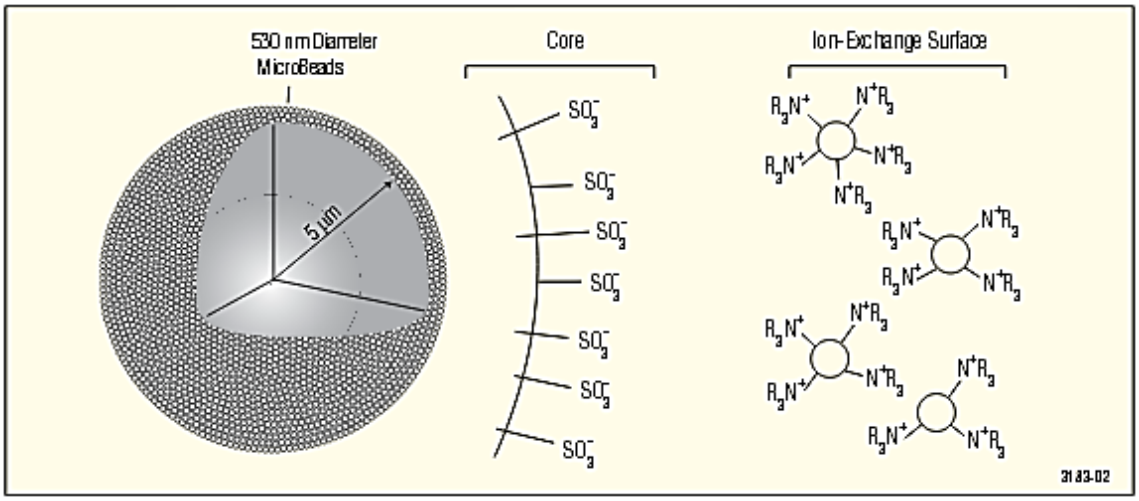


Figure 36: IonPac™ AS7 anion exchange column packing (Dionex Corporation, 2006).

Instrument performance parameters including analyte stability and sensitivity, torch position, ion optics, and detector voltages were optimized prior to analysis using Thermo Scientific™ 1ppb Tune B solution in 2% HNO_3 and 0.5% HCl. Analyte signal responses were monitored using the real time display option in the instrument software. Instrument operating conditions are given in Tables 8 and 9.

Samples for total arsenic determination were digested on a DigiPREP MS graphite digestion block with a keypad controller (SCP Science, Champlain, NY).

Table 8: IC/ICP–MS specifications and operating conditions, Run 1.

IC Specifications and Parameters	
Guard column	IonPac™ AG7 guard column
Analytical column	IonPac™ AS7 anion exchange column
Flow rate	4.0 mL/min
Mobile phase A	20 mM ammonium carbonate
Mobile phase B	200 mM ammonium carbonate
Gradient program	0–2.5 min: 100% A, 2.5–11 min: slope 5 to 100% B
Run time	11 minutes
ICP Specifications and Parameters	
Cooling gas flow	13.8 L/min Ar
Auxiliary gas flow	0.8 L/min He
Nebulizer gas flow	0.97 L/min Ar
Plasma power	1550 W
Pirani pressure	1.690 mbar
Penning pressure	4.220×10^{-7} mbar

Table 9: IC/ICP–MS specifications and operating conditions, Run 2.

IC Specifications and Parameters	
Guard column	IonPac™ AG7 guard column
Analytical column	IonPac™ AS7 anion exchange column
Flow rate	4.0 mL/min
Mobile phase A	20 mM ammonium carbonate
Mobile phase B	200 mM ammonium carbonate
Gradient program	0–3 min: 100% A, 3–13 min: slope 5 to 100% B
Run time	13 minutes
ICP Specifications and Parameters	
Cooling gas flow	13.8 L/min Ar
Auxiliary gas flow	0.8 L/min He
Nebulizer gas flow	0.93 L/min Ar
Plasma power	1548 W
Pirani pressure	1.654 mbar
Penning pressure	6.767×10^{-7} mbar

Chemicals and reagents

All solutions and dilutions were prepared using an in–house Elga PURELAB® Classic Ultra Pure Type 1 (18.2 MΩ cm) reverse osmosis (RO) water system (Woodridge, IL). 20 mM and 200 mM ammonium carbonate buffers (Alfa Aesar, 99.999%, Lancashire, UK) were prepared as the IC eluents for separation and 2% HNO_3 (BDH Aristar® Ultra, 70%, VWR International) was prepared as the ICP rinse for total As. Standard As solutions of concentrations 0.1 ppb, 1 ppb, 5 ppb, and 10 ppb were prepared from a 1000 ug/mL stock solution for the total As calibration curve (CPI International, Palo Alto, CA). A 5 ppb independent calibration verification (ICV) solution was prepared from a 1000 ug/mL As stock solution from a different vendor (BDH Aristar® Plus, VWR International) and different lot number to verify the total As calibration curve. Standard solutions of the individual As species of concentrations 0.05 ppb, 0.1 ppb, 1 ppb, 5 ppb, and 10 ppb from the following stock reagents: As (III) in 2% HCl, (SPEX CertiPrep, 1001 µg/mL ± 5 µg/mL, Metuchen, NJ), As (V) in TraceSELECT® Ultra (18.2 MΩ cm, 0.22 µm filtered water), (Fluka–Sigma Aldrich, 1001 mg/L ± 3 mg/L, St. Louis, MO), (methylarsonic acid (MMA (V)), (Chem Service, Inc., 99.5% ± 0.5%, West Chester, PA), and cacodylic acid (DMA (V)), (Alfa Aesar, 98%, Lancashire, UK). For total As determinations, a 20 ppm internal standard solution was prepared from TraceCERT® germanium, indium, and scandium (Fluka–Sigma Aldrich, 1000 mg/L in HNO_3, St. Louis, MO).

Quality control (QC)

Table 10 lists the quality control measures employed to verify instrument performance and ensure sample result validity.

Certified reference materials DORM–4 (fish protein homogenate) and TORT–3 (lobster hepatopancreas) from the National Research Council of Canada (Ottawa, Ontario) were analyzed as certified reference materials (CRM).

Table 10: Quality control measures employed in arsenic analysis.

QC Measure	Analysis Frequency	Value and/or Tolerance
Calibration blank and curve	Every batch	$R^2 > 0.997$
Digestion blank	Every batch	~ calibration blank
Independent Calibration Verification (ICV)	Every curve	±10%
Continuous Calibration Verification (CCV)	Every 10 samples	±10%
Matrix spike	Every 15–20 samples	±10%
Internal standard (total As)	Continuous	60–125%
DORM–4	Every batch	6.43–7.31 ±10 %
TORT–3	Every batch	55.7–63.3 ±10 %
Extraction efficiencies		95–105 %

Arsenic speciation

Blue crab, HP, and shell samples ranging from 50–500 mg were weighed in DigiTUBE digestion vessels (SCP Science, Champlain, NY), 10 mL of 80% methanol/RO water was added (BDH, 99.8% ACS grade methanol from VWR International), and samples were digested for 6 minutes at 80 °C. After cooling, samples were sonicated for one hour, and centrifuged at 3500 rpm for 5 minutes. The supernatants

were quantitatively transferred to 100–mL polymethylpentene (PMP) volumetric flasks and diluted to volume with RO water. Aliquots were filtered through 0.45 µm MicroLiter polytetrafluoroethylene (PTFE/Teflon®) filters (Wheaton, Millville, NJ) prior to analysis. Arsenic speciation to determine As (III), As (V), MMA, and DMA was performed by IC/ICP–MS with Thermo Scientific™ Qtegra™ ISDS software with a Dionex™ Chromeleon® Xpress chromatography plug–in. Through the plug–in, peak integration and species concentration was calculated using operator–entered mass and volume values and the Chromatography Data System (CDS) Cobra™ peak detection algorithm. All results were reported in µg/kg.

The complexities of soil and sediment matrices makes accurate and representative extraction and species quantification extremely difficult. In order to quantify every species present in sediment, a multi–step and multi–chemical extraction is typically performed. However, since the dominant species present was expected to be As (V), the sediment samples previously prepared for total arsenic were used for arsenic speciation analysis.

Extraction efficiencies

Extraction efficiencies in the CRMs and sediment samples were determined by calculating the ratio between total arsenic in the sample extracts (by addition of species concentration) and total arsenic in the samples (as measured after acid digestion). Extraction efficiencies were between 95.1% and 100.7%, with an average of 97.7%—numbers consistent with literature extraction efficiencies (Amayo et al., 2011; Petursdottir et al., 2014; Zmolinski et al., 2015).

CHAPTER III

RESULTS AND DISCUSSION

The objective of this research was to determine the species of arsenic present in sediment and blue crabs collected from the East White Lake oil and gas field. Both As (III) and As (V) were present in the sediment, but As (V) dominated (Figure 37). This was consistent with literature reports of arsenic distribution in sediment (Ferguson and Gavis, 1972; Mamindy-Pajany et al., 2013). The primary arsenic species (~ 90%) found in the sediment was the toxic inorganic As (V) species. The sediment averaged ~ 5.5 % of the organic metabolite DMA and ~ 3 % of the most toxic arsenic species, As (III). The presence of DMA in the sediment likely can be attributed to microbial transformations of the inorganic As (V) and As (III) species (Sharma and Sohn, 2009).

Crabs can be exposed to arsenic through 1) their omnivorous diet that includes benthic organisms and other crabs (Hong et al., 2014) and 2) biosorption of arsenate on the crab shell (Vijayaraghavan et al., 2009). Therefore, the blue crabs most likely took up the inorganic As (V) from the sediment, metabolizing almost 90% of it to the less toxic DMA (Figure 38). The crabs still contained, on average, about 6.5% As (V), and they also retained the As (III). The crabs contained more As (III) than was measured in the

sediment (~ 4.7%), an elevation which is likely due to As (V) metabolism since crabs are known to biotransform and retain arsenic (Hong et al., 2014). Even though most of the As (V) metabolized in the crab to DMA, it is important to note that even though DMA is less toxic than the inorganic As (III) and (V), it is still toxic and classified as a possible human carcinogen that forms toxic intermediates (ATSDR, 2007). DMA is also known to demethylate to MMA, the most toxic species of arsenic, as well as transforming back to the toxic inorganic arsenic species (Gao and Burau, 1997; Huang et al., 2007). It must therefore be considered when assessing human and ecological health risk and not written off as a less toxic arsenic fraction.

In this study, there were no samples taken to assess an anthropogenic arsenic background concentration, and no blue crab data was found in the literature reviewed. However, arsenic in *clams* was detected from 6–15 times higher than ambient levels in a cluster of more than ten samples collected from the Gulf of Mexico, southwest of Chenier au Tigre in Vermilion Parish (Tittlebaum, 1987). Clams are favored biological indicators for arsenic monitoring in marine and freshwater ecosystems with a lack of baseline data due to positive correlation between metal concentration in clam tissue and metal concentration in the water (Rayment and Barry, 2000). Although the water of the East White Lake oil and gas field was not analyzed for arsenic, it has been well documented how arsenic cycles from water into sediment (Ferguson and Gavis, 1972; Jain and Ali, 2000; Matschullat 2000).

Tables 11 and 12 summarize the arsenic species analyzed in three tissue types of blue crabs and sediment from the East White Lake oil and gas field.

Table 11: Blue crab arsenic species concentrations in µg/kg.

Sample		MMA	DMA	As (III)	As (V)	Species sum
L–1 (1)	meat	ND	ND	ND	ND	ND
	hepatopancreas	ND	146	67	ND	213
	shell	ND	ND	ND	ND	ND
L–1 (2)	meat	ND	ND	ND	ND	ND
	hepatopancreas	ND	108	ND	ND	108
	shell	ND	ND	ND	ND	ND
L–1 (3)	meat	ND	ND	ND	ND	ND
	hepatopancreas	10	43	6	ND	59
	shell	ND	ND	ND	ND	ND
L–2 (1)	meat	ND	306	ND	597	903
	hepatopancreas	ND	2936	ND	102	3038
	shell	ND	340	ND	35	375
L–2 (2)	meat	ND	402	ND	20	422
	hepatopancreas	ND	1073	42	21	1136
	shell	ND	390	ND	8	398
L–2 (3)	meat	ND	89	ND	15	104
	hepatopancreas	ND	700	ND	64	764
	shell	ND	220	ND	12	232
L–3 (1)	meat	ND	373	ND	ND	373
	hepatopancreas	ND	ND	ND	ND	ND
	shell	ND	131	ND	ND	131
L–3 (2)	meat	ND	ND	ND	ND	ND
	hepatopancreas	14	102	ND	ND	116
	shell	ND	ND	ND	ND	ND
L–3 (3)	meat	ND	158	ND	ND	158
	hepatopancreas	11	209	10	ND	230
	shell	ND	ND	ND	ND	ND
S–1 (1)	meat	ND	14	ND	ND	14
	hepatopancreas	ND	67	32	ND	99
	shell	ND	ND	ND	ND	ND
S–1 (2)	meat	ND	39	ND	ND	39
	hepatopancreas	ND	ND	ND	ND	ND
	shell	ND	ND	ND	ND	ND
S–2 (1)	meat	ND	262	ND	2	264
	hepatopancreas	ND	731	19	49	799
	shell	ND	338	ND	13	351
S–2 (2)	meat	ND	211	ND	2	213
	hepatopancreas	ND	671	83	16	770
	shell	ND	170	ND	3	173
S–2 (3)	meat	ND	288	ND	2	290
	hepatopancreas	3	576	237	2	818
	shell	ND	223	ND	7	230
S–6 (1)	meat	ND	87	ND	2	89
	hepatopancreas	ND	283	65	ND	348
	shell	ND	1	ND	44	45
S–6 (2)	meat	ND	125	ND	1	126
	hepatopancreas	ND	641	85	5	731
	shell	ND	66	ND	12	78
S–6 (3)	meat	ND	530	ND	ND	530
	hepatopancreas	ND	657	100	1	758
	shell	ND	422	ND	45	467

ND = Not Detected

Table 12: Sediment total arsenic and species concentrations in µg/kg.

Sediment Sample	Total Arsenic	DMA	% of Total	As (III)	% of Total	As (V)	% of Total
L–1	1061	NS	NS	NS	NS	NS	NS
L–2	1352	108	8.0	60	4.4	1118	82.7
L–3	1474	NS	NS	NS	NS	NS	NS
S–1	1627	NS	NS	NS	NS	NS	NS
S–2	2209	107	4.8	59	2.7	2058	93.2
S–6	2910	135	4.6	77	2.6	2590	89.0

NS = Not Speciated
Note: MMA Not Detected

These results agree with a 1985–1986 U.S. Fish and Wildlife study performed in the Aransas Bay Complex surrounding the Aransas National Wildlife Refuge on the Texas Gulf Coast—an area impacted by oil and gas production activities. The total arsenic concentrations analyzed in the sediment averaged ~ 1,800 µg/kg, while composited and homogenized blue crab concentrations averaged ~ 1,400 µg/kg (Gamble et al., 1989). The differences in crab arsenic concentrations can possibility be attributed to the blue crabs' diet, mobility, and age. A study by Barrett (2014) reported arsenic values in produced water from U.S. oilfields ranging from not detected to ~ 1,600 µg/kg.

There are multiple ways to deal with ND values in statistics including substitution of zero, half the detection limit, or the low point on a calibration curve (Helsel, 1990). Due to the small sample size and an inability to perform robust statistics on the reported values, I chose to substitute the value of the low point on the calibration curve (0.00005 ppm). The instrument detection limit was analyzed by the author as 0.00003 ppm.

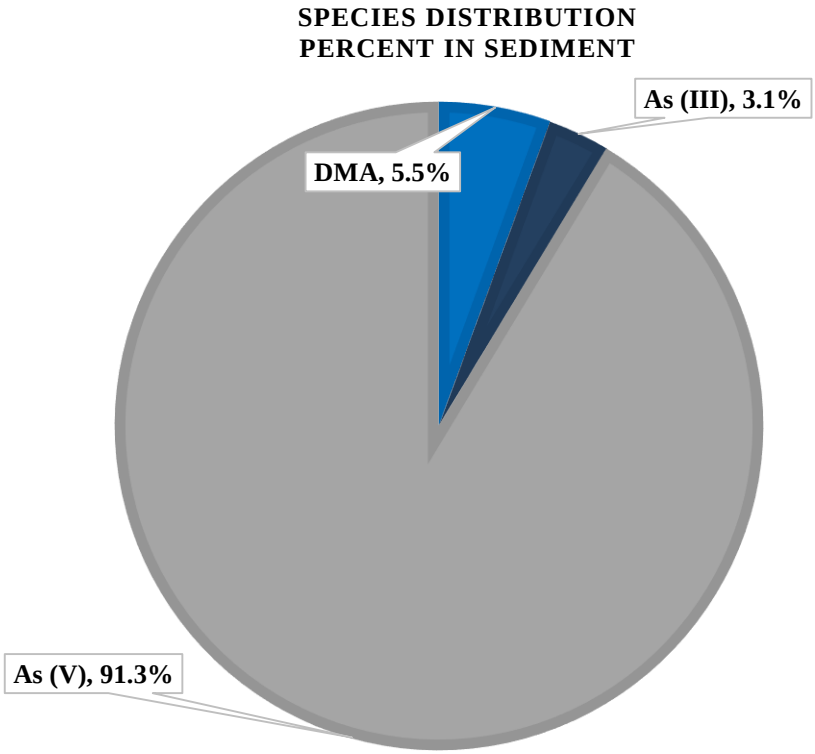


Figure 37: Overall distribution by percent of arsenic species in sediment.

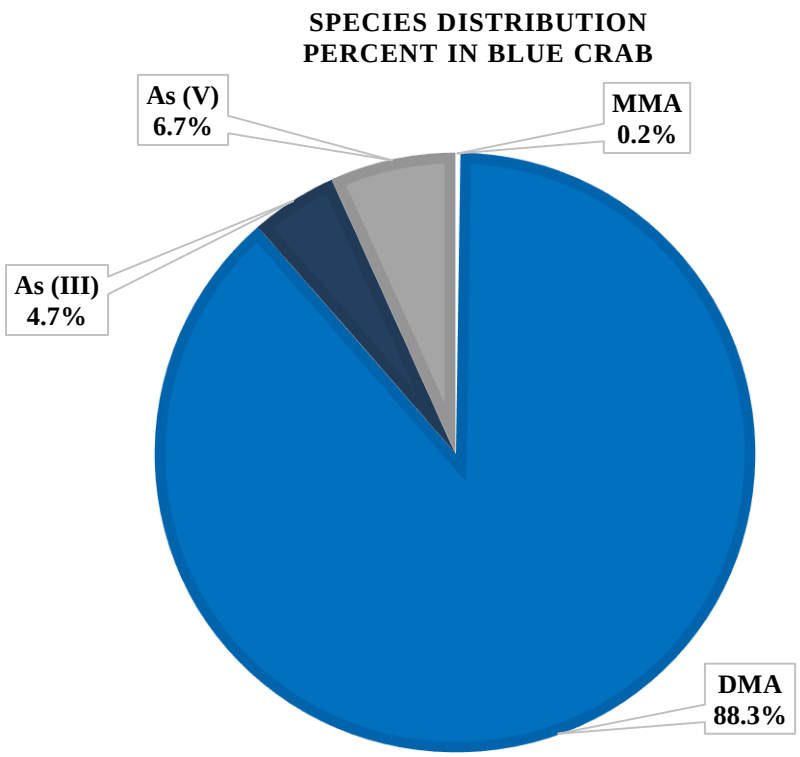


Figure 38: Overall distribution by percent of arsenic species in blue crab.

Statistical analysis and interpretation

The possible difference in mean total arsenic concentrations between the lake and oilfield sediment samples (Table 12) was checked by independent samples t–test and the Kruskal-Wallis test. While East White Lake oil and gas field sediment sample concentrations, on average, were about 1.75 times that of the lake sediment sample concentrations, neither the t–test nor Kruskal-Wallis test showed statistical significance between those sample means (Figure 53).

Independent t–test and the Kruskal-Wallis analysis for sediment means in Table 12:

H_0: means are equal

H_A: means are not equal

$\alpha = 0.05$

$p = 0.071$ (t–test)

$p = 0.368$ (Kruskal–Wallis)

Conclusion: $p \not< 0.05$ $\therefore$ fail to reject the null hypothesis and conclude that the means are equal.

Even though the arsenic concentration differences between the lake and oilfield sediments are not statistically significant, they are still important, suggesting that the ~ 1.75x difference is likely due to arsenic–laden produced water being discharged into the oilfield freshwater marsh. It is likely that had a larger number of samples been analyzed a statistically significant difference would be observed. Correlations were determined at the 0.05 and 0.01 levels between numerous variables (Table 13 and Figure 39). Analysis was performed using SPSS analytics software from IBM. Regression curves were created for all data showing correlations (Appendix C).

Table 13: Significance correlations between arsenic species.

Arsenic species	Arsenic species	Correlation level	Pearson r	p value
As (V) HP	As (V) meat	0.01	0.781	0.000
As (V) meat	DMA HP	0.01	0.896	0.000
As (V) HP	DMA HP	0.01	0.858	0.000
As (V) HP	DMA shell	0.05	0.573	0.016
As (V) shell	DMA HP	0.05	0.519	0.033
As (V) shell	DMA shell	0.05	0.502	0.040
DMA meat	DMA shell	0.01	0.860	0.000
DMA HP	DMA shell	0.01	0.661	0.004
As (V) sediment	As III sediment	0.05	0.744	0.022
As (V) sediment	DMA sediment	0.05	0.756	0.018

Correlations

		ASIIIHP	ASVM	ASVHP	ASVS	MMAHP	DMAM	DMAHP	DMAS	ASVSED	ASIIISED	DMASED	CARWIDTH
ASIIIHP	Pearson Correlation	1	-.187	-.255	.198	-.176	.324	.017	.238	.477	.107	.118	.072
	Sig. (2-tailed)		.471	.322	.447	.499	.205	.949	.357	.194	.784	.763	.784
	N	17	17	17	17	17	17	17	17	9	9	9	17
ASVM	Pearson Correlation	-.187	1	.781**	.407	-.134	.222	.896**	.354	-.496	-.244	-.252	.037
	Sig. (2-tailed)	.471		.000	.105	.609	.391	.000	.163	.175	.527	.512	.889
	N	17	17	17	17	17	17	17	17	9	9	9	17
ASVHP	Pearson Correlation	-.255	.781**	1	.353	-.266	.247	.858**	.573*	-.747*	-.542	-.551	.184
	Sig. (2-tailed)	.322	.000		.165	.302	.339	.000	.016	.021	.132	.124	.479
	N	17	17	17	17	17	17	17	17	9	9	9	17
ASVS	Pearson Correlation	.198	.407	.353	1	-.329	.480	.519*	.502*	.308	.635	.631	.202
	Sig. (2-tailed)	.447	.105	.165		.197	.051	.033	.040	.421	.066	.068	.437
	N	17	17	17	17	17	17	17	17	9	9	9	17
MMAHP	Pearson Correlation	-.176	-.134	-.266	-.329	1	-.314	-.272	-.385	.079	-.271	-.264	-.215
	Sig. (2-tailed)	.499	.609	.302	.197		.219	.291	.127	.840	.480	.493	.408
	N	17	17	17	17	17	17	17	17	9	9	9	17
DMAM	Pearson Correlation	.324	.222	.247	.480	-.314	1	.462	.860**	-.054	-.040	-.041	-.119
	Sig. (2-tailed)	.205	.391	.339	.051	.219		.062	.000	.889	.919	.918	.649
	N	17	17	17	17	17	17	17	17	9	9	9	17
DMAHP	Pearson Correlation	.017	.896**	.858**	.519*	-.272	.462	1	.661**	-.609	-.350	-.359	.091
	Sig. (2-tailed)	.949	.000	.000	.033	.291	.062		.004	.082	.356	.343	.728
	N	17	17	17	17	17	17	17	17	9	9	9	17
DMAS	Pearson Correlation	.238	.354	.573*	.502*	-.385	.860**	.661**	1	-.451	-.393	-.397	-.039
	Sig. (2-tailed)	.357	.163	.016	.040	.127	.000	.004		.223	.296	.290	.882
	N	17	17	17	17	17	17	17	17	9	9	9	17
ASVSED	Pearson Correlation	.477	-.496	-.747*	.308	.079	-.054	-.609	-.451	1	.744*	.756*	-.325
	Sig. (2-tailed)	.194	.175	.021	.421	.840	.889	.082	.223		.022	.018	.394
	N	9	9	9	9	9	9	9	9	9	9	9	9
ASIIISED	Pearson Correlation	.107	-.244	-.542	.635	-.271	-.040	-.350	-.393	.744*	1	1.000**	.109
	Sig. (2-tailed)	.784	.527	.132	.066	.480	.919	.356	.296	.022		.000	.781
	N	9	9	9	9	9	9	9	9	9	9	9	9
DMASED	Pearson Correlation	.118	-.252	-.551	.631	-.264	-.041	-.359	-.397	.756*	1.000**	1	.098
	Sig. (2-tailed)	.763	.512	.124	.068	.493	.918	.343	.290	.018	.000		.803
	N	9	9	9	9	9	9	9	9	9	9	9	9
CARWIDTH	Pearson Correlation	.072	.037	.184	.202	-.215	-.119	.091	-.039	-.325	.109	.098	1
	Sig. (2-tailed)	.784	.889	.479	.437	.408	.649	.728	.882	.394	.781	.803	
	N	17	17	17	17	17	17	17	17	9	9	9	17

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Figure 39: SPSS correlations table between crab and sediment species (HP = hepatopancreas, S = shell, and M = meat).

For all species correlations in Table 13:

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

$\alpha = 0.05$

p = all p values are 0.04 or less

Conclusion: $p < 0.05$ ∴ reject the null hypothesis and conclude that the data are correlated.

The Pearson r correlation coefficient, which varies between –1.0 and 1.0, measures the strength of the linear relationship between two variables; the closer to 1.0 the value is, the stronger the positive correlation is (as *x* increases, *y* increases), and the converse holds true for negative correlation. Values near zero indicate no linear relationship between the variables. The strongest positive correlation exists between As (V) in the sediment and As (III) and DMA in the sediment—as one increases in concentration, so does the other (Figures 62 and 63). There are also strong positive correlations between DMA in the meat and DMA in the shell (Figure 60), and between As (V) in the hepatopancreas and As (V) in the meat (Figure 54) and As (V) in the hepatopancreas and DMA in the hepatopancreas (Figure 56).

There are weak correlations between As (III) and DMA in the sediment to DMA in the shell and between As (V) in the shell and hepatopancreas to DMA in the hepatopancreas and shell, respectively. There were no correlations found between the

major arsenic species in sediment, (i.e., As (V)), and other arsenic species concentrations in the crabs.

There are likely multiple explanations why strong correlations were not found between the arsenic species in sediments and crabs: 1) the complex metabolic processes happening in the crabs cause significant arsenic biotransformation, thus the form of arsenic in the sediment and crab may differ significantly temporally, 2) the crabs collected in this research were free ranging and their location and duration of exposure to the sediments sampled may not correspond closely. A solution to this is to cage the crabs in the same location for a known duration. Additional research using biomonitoring methods might resolve these discrepancies (Barbee et al., 2008).

CHAPTER IV

SUMMARY AND CONCLUSIONS

Summary

Arsenic can mobilize and enter the environment from the natural breakdown of petroleum hydrocarbons and from the produced water that is a byproduct of oil and gas exploration (Cozzarelli et al., 2015; Sadiq and Zaidi, 1985). As an oil reservior ages, more water than oil typically is produced. In the U.S. where most oilfields are mature, oil and gas E&P waste can contain up to 98% produced water (Ahmadun et al., 2009; Veil et al., 2004). The East White Lake oil and gas field is considered a mature oilfield since it has been operating for around eight decades. So arsenic exposure and ingestion via tainted crabs is a huge concern due to the large volume of arsenic–laden produced water that has been discharged into and contaminated this freshwater marsh.

Arsenic in oilfield-contaminated sediment occurs predominantly in the inorganic and toxic As (V) form (Cavalca et al., 2013; Gamble et al., 1989; Mamindy–Pajany et al., 2013). It is taken up by the crab, biotransformed into other toxic arsenic species and distributed in the shell, meat, and hepatopancreas—all potential sources of arsenic exposure and ingestion (Gao and Burau, 1997; Rahman et al., 2012; Vijayaraghavan et al., 2009).

Coastal Louisiana residents eat approximately 10–15 times the average seafood consumption of 100 g per week as reported by the National Oceanic and Atmospheric Administration Fisheries Division (NOAA, 2015). The crabs that the locals are eating from the East White Lake marshes contain concerning levels of arsenic that accumulate over time in the hair, fingernails, and organs of humans.

Research has suggested that cooking does not promote transformation of inorganic arsenicals to the non toxic organic forms, nor does it eliminate toxins (FDA, 2008; Sartal et al., 2012). In Louisiana, residents often boil and use the entire crab, with the resulting stock being reused as seasoning, which is a potential long–term source of arsenic exposure in humans. In addition, the primary arsenic sink in crabs, the hepatopancreas—also called crab fat, mustard, or tomalley—is considered a delicacy (Liu, 2015). Previous studies concerning arsenic exposure from seafood have analyzed only what are considered the edible portions of the blue crab, that is, the claw and muscle meat (Sweat, 2007). These studies have not included the exposure from ingestion of the hepatopancreas or shell sources such as the seasoning stock or consumption of an entire "soft shell" crab. Thus, human dietary exposures to arsenic that consider only the edible portions are erroneously low estimations of total arsenic exposure and ingestion.

Some literature reports that seafood ingestion poses little to no health risk because seafood contains primarily the organic arsenic species (Kaise and Fukui, 1992; Li et al., 2003), while others assert the need for speciation analysis in assessing health risk from arsenic–contaminated shellfish (Francesconi 2010; Peshut et al., 2008; Sirot et al., 2009). This research has shown that the presence of the toxic inorganic species As (III) and As

(V) as well as the organic metabolites MMA and DMA in the blue crab (*Callinectes sapidus*) does indicate a need for further investigation into arsenic speciation in seafood before a true determination can be made regarding the health risk of seafood ingestion, especially from a site with known arsenic contamination.

Conclusions

In addition to humans, higher trophic level ecological populations are exposed to toxic arsenic species through food and water ingestion (Caumette et al, 2012; Rahman et al, 2012). To properly assess the extent of arsenic bioaccumulation and biomagnification in human and ecological populations, future research should include the following:

- A total flora and fauna investigation that includes all species living or growing in the marsh wetlands that are potentially affected by the arsenic contamination. This list includes, but is not limited to the following:
    - Shrimp, trout, flounder, mullet, catfish, menhaden, bass, bluegill, alligator gar, crappie, and other fishes
    - Ferns, petunia, holly, milkweed, swamp thistle, coneflower, pinweed, oak, and other plants (it would be particularly interesting to determine arsenic levels in ferns, since they are known arsenic hyperaccumulators)
    - Alligators, shorebirds, and migratory birds
- A human population study that analyzes arsenic levels in blood and urine of a representative cross–section of local subsistence residents.
- An in depth analysis of the cooking and eating habits of the local population to ensure their seafood ingestion rates are accurately reported and measured.

- Determination of true and/or anthropogenic background arsenic species concentrations in south Louisiana sediments and biota. For example, it might be necessary to go farther out in White Lake, farther inland, possibly at the White Lake Wetlands Conservation Area, and perhaps all along the Louisiana Gulf Coast.

REFERENCES

Abbott, S.W. 1889. "Some historical and statistical facts pertaining to the use of arsenic as a poison." *Boston Medical and Surgical Journal CXX(20)* 477–480.

Abdul, K.S.M., Jayasinghe, S.S., Chandana, E.P.S., Jayasumana, C., Mangala, P., and De Silva, C.S. 2015. "Arsenic and human health effects: a review." *Environmental Toxicology and Pharmacology 40* 828–846.

Agarwal, K., Patel, M., and Agarwal, V. 2015. "A complicated case of acute promyelocytic leukemia in the second trimester of pregnancy successfully treated with all–trans–retinoic acid." *Case Reports in Hematology 2015* 1–4.

Ahmadun, F.R., Pendashteh, A., Abdullah, L. C., Biak, D.R.A., Madaeni, S.S., and Abidin, Z.Z. 2009. "Review of technologies for oil and gas produced water treatment." *Journal of Hazardous Materials 170* 530–551.

Amayo, K.O., Petursdottir, A.H., Newcombe, C., Gunnlaugsdottir, H., Raab, A., Krupp, E.M., and Feldmann, J. 2011. "Identification and quantification of arsenolipids using reversed–phase HPLC coupled simultaneously to high–resolution ICP–MS and high–resolution electrospray MS without species–specific standards ." *Analytical Chemistry 83(9)* 3589–3595.

Amayo, K.O., Raab, A., Krupp, E.M., and Feldmann, J. 2014a. "Identification of arsenolipids and their degradation products in cod–liver oil." *Talanta 118* 217–223.

Amayo, K.O., Raab, A., Krupp, E.M., Marschall, T., Horsfall Jr, M., and Feldmann, J. 2014b. "Arsenolipids show different profiles in muscle tissues of four commercial fish species." *Journal of Trace Elements in Medicine and Biology 28* 131–137.

Anawar, H.M., Akai, J., Mostofa, K.M.G., and Tareq, S.M. 2002. "Arsenic poisoning in groundwater: health risk and geochemical sources in Bangladesh." *Environment International 27* 597–604.

Argos, M., Kalra, T., Rathouz, P.J., Chen, Y., Pierce, B., Parvez, F., Islam, T., Ahmed, A., Rakibuz–Zaman, M., Hasan, R., Sarwar, G., Slavkovich, V., van Geen, A., Graziano, J., and Ahsan, H. 2010. "Arsenic exposure from drinking water, and all–cause and chronic–disease mortalities in Bangladesh (HEALS): a prospective cohort study." *Lancet 376(9737)* 252–258.

ATSDR. 2009. *Case Studies in Environmental Medicine.* Atlanta, GA: U.S. Department of Health and Human Services.

ATSDR. 2013. *Priority list of hazardous substances.* Atlanta, GA: ATSDR.

ATSDR. 2007. *Toxicological profile for arsenic.* Atlanta, GA: U.S. Department of Health and Human Services.

Baptist, D.L. and Leslie, N.S. 2008. "Children playing with poison: arsenic exposure from CCA–treated wood." *The Journal for Nurse Practitioners 4(1)* 48–53.

Barats, A., Feraud, G., Potot, C., Philippini, V., Travi, Y., Durrieu, G., Dubar, M., and Simler, R. 2014. "Naturally dissolved arsenic concentrations in the Alpine/Mediterranean Var River watershed (France)." *Science of the Total Environment 473–474* 422–436.

Barbee, G.C., Barich, J., Duncan, B., Bickham, J.W., Matson, C.W., Hintze, C.J., Autenrieth, R.L., Zhou, G.D., McDonald, T.J., Cizmas, L., Norton, D., and Donnelly, K.C. 2008. "*In situ* biomonitoring of PAH-contaminated sediments using juvenile coho salmon (Oncorhynchus kisutch). *Ecotoxicology and Environmental Safety 71(2)* 454-464.

Barbee, G.C. and Castille, G.J. 2010. "Investigation of historical land use and environmental impacts on the Vermilion Parish School Board property, Section 16, T. 15 S.–R. 1 E., Vermilion Parish, Louisiana." Canyon, TX and Baton Rouge, LA.

Barrett, M.L. 2014. *"Historic oilfield arsenic sources: implications for pit groundwater models."* 21st International Petroleum Environmental Conference. October 14–16, 2014. Houston, TX.

Bartrip, P. 1992. "A pennurth of arsenic for rat poison: the Arsenic Act, 1851 and prevention of secret poisoning ." *Medical History 36* 53–69.

Barwick, M. and Maher, W. 2003. "Biotransference and biomagnification of selenium copper, cadmium, zinc, arsenic and lead in a temperate seagrass ecosystem from Lake Macquarie Estuary, NSW, Australia." *Marine Environmental Research 56(4)* 471–502.

Beene, L.C., Halluer, J., Yoshinaga, M., Hamdi, M., and Liu, Z. 2011. "Pentavalent arsenate transport by zebrafish phosphate transporter NaPi–llb1." *Zebrafish 8(3)* 125–131.

Bhattacharya, P., Welch, A.H., Stollenwerk, K.G., McLaughlin, M.J., Bundschuh, J., and Panaullah, G. 2007. "Arsenic in the environment: biology and chemistry." *Science of the Total Environment 379* 109–120.

Bin Sayeed, M.S., Ratan, M., Hossen, F., Hassan, F., Faisal, M., and Kadir, M.F. 2012. "Arsenosugar induced blood and brain oxidative stress, DNA damage, and neurobehavioral impairments." *Neurochemical Research 38(2)* 405–412.

Bissen, M. and Frimmel, F.H. 2003. "Arsenic–a review. Part 1: occurrence, toxicity, speciation, mobility." *Acta Hydrochimica et Hydrobiologica 31* 9–18.

BLM. 2008. *Reasonably foreseeable development scenario for fluid minerals.* Jackson, MS: Bureau of Land Management. U.S. Department of the Interior.

Boehringer, B. and MacKenzie, T. 2011. *Whooping cranes return celebrated at white lake wetlands conservation area.* Baton Rouge, LA: USFWS.

Bolan, N.S., Choppala, G., Kunhikrishnan, A., Park, J., and Naidu, R. 2013. "Microbial transformation of trace elements in soils in relation to bioavailability and remediation." *Reviews of Environmental Contamination and Toxicology 225* 1–56.

Bowen, H.J. 1972. "The biochemistry of the trace elements." *Nuclear Activation Techniques in the Life Sciences 1972.* Vienna, Austria: International Atomic Energy Agency. 393–405.

Britsch, L.D., Dillon, D.L., and Markewich, H.W. 1998. *Carbon storage and Late Holocene chronostratigraphy of a Mississippi River deltaic marsh, St. Bernard Parish, Louisiana.* Reston, VA: U.S. Geological Survey.

Byrne, J.V., LeRoy, D.O., and Riley, C.M. 1959. "The Chenier Plain and its stratigraphy, Southwestern Louisiana." *Gulf Coast Association of Geological Societies 9* 237–260.

Caumette, G., Koch, I., and Reimer, K.J. 2012. "Arsenobetaine formation in plankton: a review of studies at the base of the aquatic food chain." *Journal of Environmental Monitoring 14* 2841–2853.

Cavalca, L., Corsini, A., Zaccheo, P., Andreoni, V., and Muyzer, G. 2013. "Microbial transformations of arsenic: perspectives for biological removal of arsenic from water." *Future Microbiology 8(6)*753–768.

Challenger, F. 1979. "Biosynthesis of organometallic and organomettaloidal compounds, Chapter 1." *ACS Symposium Series 82.* Washington, D.C.: American Chemical Society. 1–22.

Chen, C.J., Chuang, Y.C., Lin, T.M., and Wu, H.Y. 1985. "Malignant neoplasms among residents of a Blackfoot Disease–endemic area in Taiwan: high–arsenic artesian well water and cancers." *Cancer Research 45* 5895–5899.

Chen, C.J. and Wang, C.J. 1990. "Ecological correlation between arsenic levels in well water and age–adjusted mortality from malignant neoplasms." *Cancer Research 50* 5470–5474.

Chen, S.L., Dzeng, S.R., Yang, M.H., Chiu, K.H., Shieh, G.M., and Wai, C.M. 1994. "Arsenic species in groundwaters of the blackfoot disease area, Taiwan." *Environmental Science and Technology 28* 877–881.

Cheng, P.S., Weng, S.F., Chiang, C.H., and Lai, F.J. 2015. "Relationship between arsenic–containing drinking water and skin cancers in the arseniasis endemic areas in Taiwan." *Journal of Dermatology 42(11)* 1–6.

Choong, T.S.Y., Chuah, T.G., Robiah, Y., Gregory, F.L., and Azni, K.I. 2007. "Arsenic toxicity, health hazards and removal techniques from water: an overview." *Desalination 217* 139–166.

Chung, C.J., Huang, Y.L., Huang, Y.K., Wu, M.M., Chen, S.Y., Hsueh, Y.M., and Chen, C.J. 2013. "Urinary arsenic profiles and the risks of cancer mortality: a population–based 20–year follow–up study in arseniasis–endemic areas of Taiwan." *Environmental Research 122* 25–30.

Circle Star Energy. 2015. *Gulf Coast.* http://www.circlestarenergy.com/projects/texas/gulf–coast.

Cozzarelli, I.M., Schreiber, M.E., Erickson, M.L., and Ziegler, B.A. 2015. "Arsenic cycling in hydrocarbon plumes: secondary effects of natural attenuation." *Groundwater 53(6)* 1–11.

Cullen, W.R. 2008. *Is arsenic an aphrodisiac?: the sociochemistry of an element.* London, England: The Royal Society of Chemistry.

Cullen, W.R., McBride, B.C., Manji, H., Pickett, A.W., and Reglinski, J. 1989. "The metabolism of methylarsine oxide and sulfide." *Applied Organometallic Chemistry 3(1)* 71–78.

De Felippo, M. 2014. *Produced water facts.* Houston, TX: The Produced Water Society.

DeLaune, R.D., Jugsujunda, A., Gambrell, R.P., and Miao, S. 2008. "Metal concentrations and trace metal Al and Fe ratios in soil of the Chenier Plain, Southwest Louisiana coastal zone." *Journal of Environmental Science and Health Part A 43* 300–312.

Dembitsky, V.M. and Levitsky, D.O. 2004. "Arsenolipids." *Progress in Lipid Research 43(5)* 403–448.

Dionex Corporation. 2006. "Product Specification Sheet LPN 0843 IonPac AS7 Anion–Exchange Column." Sunnyvale, CA.

Dixit, S. and Hering, J. 2003. "Comparison of arsenic (V) and arsenic (III) sorption onto iron oxide minerals: implications for arsenic mobility." *Environmental Science and Technology 37(18)* 4182–4189.

Doyle, D. 2009. "Notoriety to respectability: a short history of arsenic prior to its present day use in haematology." *British Journal of Haematology 145* 309–317.

Drobna, Z., Styblo, M., and Thomas, D.J. 2009. "An Overview of Arsenic Metabolism and Toxicity." *Current Protocols in Toxicology 42(431)* 1–6.

Dunn, M.G., Ed. 1993. *Exploring your world: the adventure of geography.* Washington, D.C.: National Geographic Society.

Ebert, F., Leffers, L., Weber, T., Berndt, S., Mangerich, A., Beneke, S., Burkle, A., and Schwerdtle, T. 2014. "Toxicological properties of the thiolated inorganic arsenic and arsenosugar metabolite thio–dimethylarsinic acid in human bladder cells." *Journal of Trace Elements in Medicine and Biology 28(2)* 138–146.

EIA. 2015. August U.S. Energy Information Administration. https://www.eia.gov/state/rankings/#/series/46.

Eiduss, J., Kalendarev, R., Rodionov, A., Sazonov, A., and Chikvaidze, G. 1996. "Some aspects of the solid state physics of yellow arsenic." *Physica Status Solidi (b) 193(3)* 3–23.

Elias, M., Wellner, A., Goldin–Azulay, K., Chabriere, E., Vorholt, J.A., Erb, T.J., and Tawfik, D.S. 2012. "The molecular basis of phosphate discrimination in arsenate–rich environments ." *Nature 1;491(7422)* 134–137.

EPA. 2001. *Drinking water standard for arsenic, EPA 815–F–00–015.* Washington, D.C: Office of Water.

EPA. 1985. *Methods manual for bottom sediment sample collection.* Chicago, IL: Great Lakes National Program Office.

EPA. 2009. *Organic arsenicals; amendment to reregistration eligibility decision, Docket ID EPA–HQ–OPP–2009–0191.* Washington, D.C: EPA.

EPA. 2008. *Reregistration Eligibility Decision for Chromated Arsenicals, List A, Case No. 0132.* Washington, D.C.: Office of Prevention, Pesticides, and Toxic Substances.

FDA. 2008. *FDA advises against consumption of tomalley from American lobster (also known as "Maine Lobster") regulators find dangerous levels of toxins that cause paralytic shellfish poisoning in tomalley of American lobsters.* Silver Spring, MD: FDA.

FDA. 2015. "RFE Page 1 for Callinectes sapidus, Blue Crab, FDA Market Name: Crab, Blue." *Regulatory Fish Encyclopedia (RFE).* 9 1. http://www.fda.gov/Food/FoodScienceResearch/RFE/ucm078750.htm.

Feldmann, J. and Krupp, E.M. 2011. "Critical review or scientific opinion paper: arsenosugars–a class of benign arsenic species or justification for developing partly speciated arsenic fractionation in foodstuffs?" *Analytical and bioanalytical chemistry 399(5)* 1735–1741.

Feng, Q., Zhang, Z., Chen, Y., Liu, L., Zhang, Z., and Chen, C. 2013. "Adsorption and desorption characteristics of arsenic on soils: kinetics, equilibrium, and effect of $Fe(OH)_3$ colloid, H_2SiO_3 colloid and phosphate." *Procedia Environmental Sciences 18* 26–36.

Fenneman, N.M. 1906. *Oil fields of the Texas–Louisiana Gulf Coastal Plain.* Washington, D.C.: Government Printing Office.

Ferguson, J.F. and Gavis, J. 1972. "A review of the arsenic cycle in natural waters." *Water Research 6* 1259–1274.

Fischler, K.J. 1959. "Occurrence of extremely small ovigerous crabs (Callinectes sp.) in coastal North Carolina." *Ecology 40* 720.

Fisher, R.S. 1995. "Naturally occurring radioactive materials (NORM) in produced water and scale from Texas oil, gas, and geothermal wells: geographic, geologic, and geochemical controls." *Geological Circular 95–3, University of Texas at Austin, Bureau of Economic Geology* 1–48.

Francesconi, K.A. 2010. "Arsenic species in seafood: origin and human health implications." *Pure and Applied Chemistry 82(2)* 373–381.

Frazier, D.E. and Osanik, A. 1969. "Recent peat deposits–Louisiana coastal plain." *GSA Special Papers 114* 63–86.

Freeman, A.M., Jose, F., Roberts, H.H., and Stone, G.W. 2015. "Storm induced hydrodynamics and sediment transport in a coastal Louisiana lake." *Estuarine, Coastal and Shelf Science 161* 65–75.

Frith, J. 2013. "Arsenic–the "poison of kings" and the "saviour of syphylis"." *Journal of Military and Veterans' Health 21(4)* 11–17.

Gahalaut, P., Rastogi, M.K., Mishra, N., and Chauhan, S. 2012. "Multiple pigmented Bowen's disease: a diagnostic and therapeutic dilemma." *Case Reports in Oncological Medicine 2012* 1–6.

Gallegos–Garcia, M., Ramirez–Muniz, K, and Song, S. 2012. "Arsenic removal from water by adsorption using iron oxide minerals as adsorbents: a review." *Mineral Processing and Extractive Metallurgy Review 33* 301–315.

Gamble, L.R., Jackson, G., and Maurer, T.C. 1989. *"Contaminant investigation of the Aransas Bay Complex, Texas, 1985–1986."* U. S. Fish and Wildlife Service. Corpus Christi, TX.

Gao, S. and Burau, R. 1997. "Environmental factors affecting rates of arsine evolution from and mineralization of arsenicals in soil." *Journal of Environmental Quality 26(3)* 753–763.

Geng, W., Komine, R., Ohta, T., Nakajima, T., Takanashi, H., and Ohki, A. 2009. "Arsenic speciation in marine product samples: comparison of extraction–HPLC method and digestion–cryogenic trap method." *Talanta 79(2)* 369–375.

Ghaeni, M., Pour, N.A., and Hosseini, M. 2015. "Bioaccumulation of polychlorinated biphenyl (PCB), polycyclic aromatic hydrocarbon (PAH), mercury, methylmercury, and arsenic in blue crab Portunus segnis from Persian Gulf." *Environmental Monitoring and Assessment 187(253)* 1–9.

Goessler, W., Maher, W., Irgolic, K.J., Kuehnelt, D., Schlagenhaufen, C., and Kaise, T. 1997. "Arsenic compounds in a marine food chain." *Fresenius Journal of Analytical Chemistry 359* 434–437.

Goh, K.H. and Lim, T.T. 2005. "Arsenic fractionation in a fine soil fraction and influence of various anions on its mobility in the subsurface environment." *Applied Geochemistry 20(2)* 229–239.

Goldberg, S., Lesch, S.M., Suarez, D.L., and Basta, N.T. 2005. "Predicting arsenate adsorption by soils using soil chemical parameters in the constant capacitance model." *Soil Science Society of America Journal 69* 1389-1398.

Gomez, G.M. 1992. "Whooping cranes in Southwest Louisiana: history and human attitudes." *Proceedings of the Sixth North American Crane Workshop*. Grand Island, NE: North American Crane Working Group. 19-23.

Gomez, G.M. 2008. *The Louisiana Coast: Guide to an American Wetland.* College Station, TX: Texas A&M University Press.

Gomez–Caminero, A., Howe, P., Hughes, M., Kenyon, E., Lewis, D.R., Moore, M., Ng, J., Aitio, A., and Becking, G. 2001. *Arsenic and arsenic compounds.* Geneva, Switzerland: World Health Organization.

Google Earth. 2015. "Google Imagery, Digital Globe, USDA Farm Service Agency." *Google Earth.*

Gorny, J., Billon, G., Lesven, L., Dumoulin, D., Made, B., and Noiriel, C. 2015. "Arsenic behavior in river sediments under redox gradient: a review." *Science of the Total Environment 505* 423–434.

Guerra, K., Dahm, K., and Dundorf, S. 2011. *Oil and gas produced water management and beneficial use in the Western United States.* Denver, CO: Bureau of Reclamation.

Guillory, V., Perry, H., Steele, P., Wagner, T., Keithly, W., Pellegrin, B., Petterson, J., Floyd, T., Buckson, B., Hartman, L., Holder, E., and Moss, C. 2001. *The blue crab fishery of the Gulf of Mexico, United States: a regional management plan.* Ocean Springs, MS: Gulf States Marine Fisheries Commission.

Halliwell, B. 1992. "Reactive oxygen species and the central nervous system." In *Free Radicals in the Brain*, by L., Prilipko, L., and Christen, Y., Eds. Packer, 21–40. Heidelberg, Germany: Springer Berlin Heidelberg.

Harold, F.M. and Baarda, J.R. 1966. "Interaction of arsenate with phosphate–transport systems in wild–type and mutant Streptococcus faecalis." *Journal of Bacteriology 91(6)* 2257–2262.

Harris, G.D. 1910. *Oil and gas in Louisiana with a brief summary of their occurrence in adjacent states.* Washington, D.C.: Government Printing Office.

Helsel, D.R. 1990. "Less than obvious: statistical treatment of data below the detection limit." *Environmental Science & Technology 24(12)* 1766-1774.

Henderson, W.G., Andersen, L.C., and McGimsey, C.R. 2002. "Distinguishing natural and archaeological deposits: stratigraphy taxonomy, and taphonomy of Holocene shell–rich accumulations from the Louisiana Chenier Plain." *Palaios 17(2)* 192–205.

Henke, K.R. and Hutchison, A. 2009. "Arsenic Chemistry." In *Arsenic: Environmental Chemistry, Health Threats and Waste Treatment*, by K.R., Ed. Henke, 9–68. Hoboken, NJ: John Wiley & Sons, Ltd.

Hong, S., Khim, J.S., Park, J., Son, H.S., Choi, S.D., Choi, K., Ryu, J., Kim, C.Y., Chang, G.S., and Giesy, J.P. 2014. "Species– and tissue–specific bioccumulation of arsenicals in various aquatic organisms from a highly industrialized area in the Pohang City, Korea." *Environmental Pollution 192* 27–35.

Huang, J., Scherr, F., and Matzner, E. 2007. ”Demethylation of dimethylarsinic acid and arsenobetaine in different organic soils." *Water, Air, and Soil Pollution 182(1–4)* 31–41.

Hughes, M.F. 2002. "Arsenic toxicity and potential mechanisms of action." *Toxicology Letters 133* 1–16.

Hughes, M.F., Beck, B.D., Chen, Y., Lewis, A.S., and Thomas, D.J. 2011. "Arsenic exposure and toxicology: a historical perspective." *Toxicological Sciences 123(2)* 305–332.

Huq, S.M.I., Ara, Q.A.J., Islam, K., Zaher, A., and Naidu, R. 2001. "The possible contamination from arsenic through food chain." *Groundwater Arsenic Contamination in the Bengal Delta Plain of Bangladesh.* Bangladesh: Kungl Tekniska Hogskolan. 91–96.

IARC. 2012. *IARC Monographs on the Evaluation of Carcinogenic Risks on Humans 100C.* Lyon, France: International Agency for Research on Cancer. World Health Organization.

Jain, C.K. and Ali, I. 2000. "Arsenic: occurrence, toxicity and speciation techniques." *Water Research 34(17)* 4304–4312.

Jiang, Y., Zeng, X., Fan, X., Chao, S., Zhu, M., and Cao, H. 2015. "Levels of arsenic pollution in daily foodstuffs and soils and its associated human health risk in a town in Jiangsu Province, China." *Ecotoxicology and Envirnmental Safety 122* 198–204.

Joseph, T., Dubey, B., and McBean, E.A. 2015. "Human health risk assessment from arsenic exposures in Bangladesh." *Science of the Total Environment 527–528* 552–560.

Kaise, T. and Fukui, S. 1992. "The chemical form and acute toxicity of arsenic compounds in marine organisms." *Applied Organometallic Chemistry 6* 155–160.

Kalendarev, R.I., Sazonov, A.I., Rodionov, A.N., Chikvaidze, G.V., and Eiduss, J.A. 1984. "Properties of vapour–deposited yellow arsenic films at various condensation conditions." *Materials Research Bulletin 19(1)* 11–15.

Karttunen, A.J., Linnolahti, M., and Pakkanen, T.A. 2007. "Icosahedral and ring–shaped allotropes of arsenic." *ChemPhysChem 8* 2373–2378.

Khorasanipour, M. and Esmaeilzadeh, E. 2015. "Geo–genic arsenic contamination in the Kerman Cenozoic Magmatic Arc, Kerman, Iran: implications for the source identification and regional analysis." *Applied Geochemistry xxx Article In Press* 1–13.

Klaunig, J.E., Kamendulis, L.M., and Hocevar, B.A. 2010. "Oxidative stress and oxidative damage in carcinogenesis." *Toxicologic pathology 38(1)* 96–109.

Koch, L., McPherson, K., Smith, P., Easton, L., Doe, K.G., and Reimer, K.J. 2007. "Arsenic bioaccessibility and speciation in clams and seaweed from a contaminated marine environment." *Marine Pollution Bulletin 54* 586–594.

Langmuir, D., Chrostowski, P., Vigneault, B., and Chaney, R. 2005. "Issue paper on the environmental chemistry of metals." Washington, D.C.: USEPA.

Laparra, J.L., Velez, D., Barbera, D., Montoro, R., and Farre, R. 2007. "Bioaccessibility and transport by Caco–2 cells of organoarsenical species present in seafood." *Journal of Agricultural and Food Chemistry 55* 5892–5897.

LDEQ. 2012. *Tissue screening level guidelines for issuance of public health advisories for selected contaminants and supporting documentation.* Baton Rouge: Louisiana Department of Environmental Quality.

LDWF. 2004. *White Lake Wetlands Conservation Area.* Baton Rouge: Louisiana Department of Wildlife and Fisheries.

Leffers, L., Ebert, F., Taleshi, M.S., Francesconi, K.A., and Schwerdtle, T. 2013a. "In vitro toxicological characterization of two arsenosugars and their metabolites." *Molecular Nutrition & Food Research 57(7)* 1270–1282.

Leffers, L., Wehe, C.A., Huwel, S., Bartel, M., Ebert, F., Taleshi, M.S., Galla, H.J., Karst, U., Francesconi, K.A., and Schwerdtle, T. 2013b. "In vitro intestinal bioavailability of arsenosugar metabolites and presystemic metabolism of thio–dimethylarsinic acid in Caco–2 cells." *The Royal Society of Chemistry 5* 1031–1042.

Levy, J.L., Stauber, J.L., Adams, MS., Maher, W.A., Kirby, J.K., and Jolley, D.F. 2005. "Toxicity, biotransformation, and mode of action of arsenic in two freshwater microalgae (Chlorella sp. and Monoraphidium arcuatum)." *Environmental Toxicology and Chemistry 24* 2630–2639.

Li., W., Wei, C., Zhang, C., van Hulle, M., Cornelis, R., and Zhang, X. 2003. "A survey of arsenic species in Chinese seafood." *Food and Chemical Toxicology 41* 1103–1110.

Liu, J. 2015. *Maryland crabs: a guide to the East Coast's essential summer feast.* Washington, D.C.: Eater, D.C.

Lloyd, J.R. and Oremland, R.S. 2006. "Microbial transformations of arsenic in the environment: from soda lakes to aquifers." *Elements 2* 85–90.

Louisiana Geological Survey. 2010. *Generalized geology of Louisiana.* Baton Rouge: Louisiana State University.

Lushchak, V.I. 2011. "Environmentally induced oxidative stress in aquatic animals." *Aquatic Toxicology 101* 13–30.

Maher, W.A., Foster, S.D., Taylor, A.M., Krikowa, F., Duncan, E.G., and Chariton, A.A. 2011. "Arsenic distribution and species in two Zostera capricorni seagrass ecosystems, New South Wales, Australia." *Environmental Chemistry 8* 9–18.

Mamindy–Pajany, Y., Bataillard, P., Seby, F., Crouzet, C., Moulin, A., Guezennec, A.G., Hurel, C., Marmier, N., and Battaglia–Brunet, F. 2013. "Arsenic in marina sediments from the Mediterranean coast: speciation in the solid phase and occurrence of thioarsenates." *Soil and Sediment Contamination 22* 984–1002.

Mandal, B.K. and Suzuki, K.T. 2002. "Arsenic round the world: a review." *Talanta 58* 201–235.

Marlborough, S.J. and Wilson, V.L. 2015. "Arsenic speciation driving risk based corrective action." *Science of the Total Environment 520* 253–259.

Mass, M.J., Tennant, A., Roop, B.C., Cullen, W.R., Styblo, M., Thomas, D.J., and Kligerman, A.D. 2001. "Methylated trivalent arsenic species are genotoxic." *Chemical Research in Toxicology 14* 355–361.

Masscheleyn, P.H., Delaune, R.D., and Patrick, Jr., W.H. 1991. "Effect of redox potential and pH on arsenic speciation and solubility in a contaminated soil." *Environmental Science & Technology 25(8)* 1414–1419.

Mast, R.F., Root, D.H., Williams, L.P., Beeman, W.R., and Barnett, D.L. 1998. "Areas of historical oil and gas exploration and production in the conterminous United States: U.S. Geological Survey Oil and Gas Investigations Map I–2582".

Matschullat, J. 2000. "Arsenic in the geosphere a review." *Science of the Total Environment 249* 297–312.

McBride, R.A., Taylor, M.J., and Byrnes, M.R. 2007. "Coastal morphodynamics and Chenier–Plain evolution in southwestern Louisiana, USA: a geomorphic model." *Geomorphology 88(3–4)* 367–422.

McGeer, J., Henningsen, G., Lanno, R., Fisher, N., Sappington, K., and Drexler, J. 2004. *Issue paper on the bioavailability and bioaccumulation of metals.* Lexington, MA: Eastern Research Group, Inc.

McNaught, A.D. and Wilkinson, A. 1997. *IUPAC Compendium of Chemical Terminology, 2nd ed. (the "Gold Book").* Oxford, England: Blackwell Scientific Publications.

Mertz, W. 1981. "The essential elements." *Science 213(4514)* 1332–1338.

Mo, Y., Momen, B., and Kearney, M.S. 2015. "Quantifying moderate resolution remote sensing phenology of Louisiana coastal marshes." *Ecological Modelling 312* 191–199.

Mohan, D. and Pittman, Jr., C.U. 2007. "Arsenic removal from water/wastewater using adsorbents—a critical review." *Journal of Hazardous Materials 142* 1–53.

Molinari, A., Guadagnini, L., Marcaccio, M., Straface, S., Sanchez–Vila, X., and Guadagnini, A. 2013. "Arsenic release from deep natural solid matrices under experimentally controlled redox conditions." *Science of the Total Environment 444* 231–240.

Moreda–Pineiro, J., Alonso–Rodriguez, E., Romaris–Hortas, V., Moreda–Pineiro, A., Lopez–Mahia, P., Muniategui–Lorenzo, S., Prada–Rodriguez, D., and Bernejo–Barrera, P. 2012. "Assessment of the bioavailability of toxic and non–toxic arsenic species in seafood samples." *Food Chemistry 130* 552–560.

Mukherjee, A., Verma, S., Gupta, S., Henke, K.R., and Bhattacharya, P. 2014. "Influence of tectonics, sedimentation and aqueous flow cycles on the origin of global groundwater arsenic: paradigms from three continents." *Journal of Hydrology 518* 284–299.

Murgo, A.J. 2001. "Clinical trials of arsenic trioxide in hematologic and solid tumors: overview of the National Cancer Institute Cooperative Research and Development Studies." *Oncologist 6(Suppl 2)* 22–28.

Murray, L.A., Raab, A., Marr, I.L., and Feldmann, J. 2003. "Biotransformation of arsenate to arsenosugars by Chlorella vulgaris." *Applied Organometallic Chemistry 17(9)* 669–674.

National Research Council. 2003. *Bioavailability of Contaminants in Soils and Sediments: Processes, Tools, and Applications.* Washington, D.C.: National Academies Press, 119–215.

Navratilova, J., Raber, G., Fisher, S.J., and Francesconi, K.A. 2011. "Arsenic cycling in marine systems: degradation of arsenosugars to arsenate in decomposing algae, and preliminary evidence for the formation of recalcitrant arsenic." *Environmental Chemistry 8(1)* 44–51.

Neff, J.M. 1997. "Ecotoxicology of arsenic in the marine environment." *Environmental Toxicology and Chemistry 16(5)* 917–927.

Nelson, W.C., Lykins, M. H., Mackey, J., Newill, V.A., Finklea, J.F., and Hammer, D.I. 1973. "Mortality among orchard workers exposed to lead arsenate spray: a cohort study." *Journal of Chronic Disease 26* 105–118.

Nemeti, B. and Gregus, Z. 2013. "Reduction of dimethylarsinic acid to the highly toxic dimethylarsinous acid by rats and rat liver cytosol." *Chemical Research in Toxicology 26(3)* 432–443.

Ng, J.C. 2005. "Environmental contamination of arsenic and its toxicological impact on humans." *Environmental Chemistry 2* 146–160.

Nichol, S.L., Boyd, R., and Penland, S. 1992. "Hydrology of a wave–dominated estuary: Lake Calcasieu, Southwest Louisiana." *Gulf Coast Association of Geological Societies, Volume 42* 835–844.

NOAA. 2015. *Seafood and Human Health.* December 5. http://www.nmfs.noaa.gov/aquaculture/faqs/faq_seafood_health.html#8what.

Nowell, L.H., Capel, P.D., and Dileanis, P.D. 1999. *Pesticides in stream sediment and aquatic biota: distribution, trends, and governing factors.* Boca Raton, FL: Lewis Publishers.

Nriagu, J.O. 2002. "Arsenic poisoning through the ages." In *Environmental Chemistry of Arsenic*, by Jr., W.T. Frankenberger, 1–26. New York City, NY: Marcel Dekker, Inc.

OGP. 2005. *Fate and effects of naturally occurring substances in produced water on the marine environment.* Brussels, Belgium: International Association of Oil and Gas Producers.

Oremland, R.S. and Stolz, J.F. 2003. "The ecology of arsenic." *Science 300* 939–944.

Osters, O., Nilges, T., Bachhuber, F., Pielnhofer, F., Weihrich, R., Schoneich, M., and Schmidt, P. 2012. "Synthesis and identification of metastable compounds: black arsenic –science or fiction?" *Angenwandte Chemie International Edition 51(12)* 2994–2997.

Otton, J.K. and Mercier, T. Undated pdf produced by USGS. *Produced water brine and stream salinity.* Accessed 2015: United States Geological Survey.

Pandey, S., Rai, R., and Rai, L.C. 2015. "Biochemical and molecular basis of arsenic toxicity and tolerance in microbes and plants." In *Handbook of Arsenic Toxicology*, by S.J.S. Flora, 627–674. Amsterdam, Netherlands: Elsevier.

Paul, D., Kazy, ,S.K., Banerjee, T.D., Gupta, A.K., Pal, T., and Sar, P. 2015. "Arsenic biotransformation and release by bacteria indigenous to arsenic contaminated groundwater." *Bioresource Technology 188* 14–23.

Pavao, C.G., Franca, G.S., Bianchi, M., de Almeida, D., and von Huelsen, M.G. 2013. "Upper–lower crust thickness of the Borborema Province, NE Brazil, using receiver function." *Journal of South American Earth Sciences 42* 242–249.

Perry, H.M. 1975. The blue crab fishery in Mississippi. *Gulf Research Reports 5(1).*

Peryea, F. J. 1998. "Historical use of lead arsenate insecticides, resulting soil contamination and implications for soil remediation." *16th World Congress on Soil Science.* Montpelier, France.

Peshut, P.J., Morrison, R.J., and Brooks, B.A. 2008. "Arsenic speciation in marine fish and shellfish from American Samoa." *Chemosphere 71(3)* 484–492.

Petrick, J.S., Ayala–Fierro, F., Cullen, W.R., Carter, D.E., and Aposhian, H.V. 2000. "Monomethylarsonous acid (MMA III) is more toxic than arsenite in Chang human hepatocytes." *Toxicology and Applied Pharmacology 163(2)* 203–207.

Petrick, J.S., Jagadish, B., Mash, E.A., and Aposhian, H.V. 2001. "Monomethylarsonous acid (MMA III) and arsenite: LD50 in hamsters and in vitro inhibition of pyruvate dehydrogenase." *Chemical Research in Toxicology 14(6)* 651–656.

Petursdottir, A.H., Gunnlaugsdottir, H., Krupp, E.M., and Feldmann, J. 2014. "Inorganic arsenic in seafood: does the extraction method matter?" *Food chemistry 150* 353–359.

Phillips, J.D. 2003. "Toledo Bend reservoir and geomorphic response in the lower Sabine River ." *River Research and Applications 19(2)* 137–159.

Pichon, A. 2013. "Mellow yellow." *Nature Chemistry 5* 78.

Pugesek, B.H., Baldwin, M.J., and Stehn, T. 2013. "The relationship of blue crab abundance to winter mortality of whooping cranes ." *The Wilson Journal of Ornithology 125(3)* 658–661.

Rahman, M.A., Hasegawa, H., and Lim, R.P. 2012. "Bioaccumulation, biotransformation and trophic transfer of arsenic in the aquatic food chain." *Environmental Research 116* 118–135.

Ratnaike, R.N. 2003. "Acute and chronic arsenic toxicity." *Postgraduate Medical Journal 79* 391–396.

Rayment, G.E. and Barry, G.A. 2000. "Indicator tissues for heavy metal monitoring–additional attributes." *Marine Pollution Bulletin 41(7–12)* 353–358.

Reddy, K.R., D'Angelo, E.M., and Harris, W.G. 2000. "Biogeochemistry of Wetlands." In *Handbook of Soil Science,* by M.E., Editor–in–Chief Sumner, G89–119. Boca Raton, FL: CRC Press.

Ritsema, R., Dukan, L., Roig i Navarro, T., van Leeuwen, W., Oliveira, N., Wolfs, P., and Lebret, E. 1998. "Speciation of arsenic compounds in urine by LC–ICP MS." *Applied Organometallic Chemistry 12(8–9)* 591–599.

Rodionov, A., Kalendarev, R., and Eiduss, J. 1995. "Photostimulated structural changes in yellow arsenic." *Journal of Physics: Condensed Matter 7* 5805–5814.

Rodionov, A., Kalendarev, R., Eiduss, J., and Zhukovskii, Y. 1996. "Polymerization of yellow (molecular) arsenic." *Journal of Molecular Structure 380(3)* 257–266.

Rosen, B.P., Ajees, A.A., and McDermott, T.R. 2011a. "Life and death with arsenic, arsenic life: an analysis of the recent report "A bacterium that can grow by using arsenic instead of phosphorus"." *Bioessays 33* 350–357.

Rosen, T. and Xu, Y.J. 2011b. "Riverine sediment inflow to Louisiana Chenier Plain in the Northern Gulf of Mexico." *Estuarine, Coastal and Shelf Science 95(2–3)* 279–288.

Rust, D.M. and Soignet, S.L. 2000. "Risk/benefit profile of arsenic trioxide." *The Oncologist 6(Suppl 2)* 29–32.

Ruttens, A., Blanpain, A.C., De Temmerman, L., and Waegeneers, N. 2012. "Arsenic speciation in food in Belgium Part 1: Fish, molluscs and crustaceans." *Journal of Geochemical Exploration 121* 55–61.

Sadiq, M. and Zaidi, T.H. 1985. "Metal concentrations in the sediments from the Arabian gulf coast of Saudi Arabia." Bulletin of Environmental Contamination and Toxicology 34 565–571.

Safe Drinking Water Foundation. 2009. "Mining and Water Pollution." Saskatoon, SK.

Sartal, C.G., del Carmen Barciela–Alonso, M., and Bermejo–Barrera, P. 2012. "Effect of the cooking procedure on the arsenic speciation in the bioavailable (dialyzable) fraction from seaweed." *Microchemical Journal 105* 65–71.

Scheindlin, S. 2005. "The duplicitous nature of inorganic arsenic." *Molecular Interventions 5(2)* 60–64.

Schintu, M., Marrucci, A., Marras, B., Meloni, P., and Contu, A. 2012. "Health risk assessment for inorganic arsenic in seafood from a polluted lagoon in Italy." *Understanding the Geological and Medical Interface of Arsenic.* London, England: Taylor & Francis Group. 222–223.

Schmeisser, E., Goessler, W., and Francesconi, K.A. 2006. "Human metabolism of arsenolipids present in cod liver oil." *Analytical and Bioanalytical Chemistry 385(2)* 367–376.

Schroeder, H.A. and Balassa, J.J. 1966. "Abnormal trace metals in man: arsenic." *Journal of Chronic Diseases 19(5)* 85–106.

Schroeder, H.A., Nason, A.P., and Tipton, I.H. 1969. "Essential metals in man magnesium." *Journal of Chronic Diseases 21* 815–841.

Scott, L.C. 2014. *The energy sector: still a giant economic engine for the Louisiana economy.* Baton Rouge, LA: Loren C. Scott and Associates, Inc.

Sekeres, M.S. 2007. "New data with arsenic trioxide in leukemias and myelodysplastic syndromes." *Clin. Lymphoma Myeloma 8(Suppl. 1)* S7–S12.

Sele, V., Sloth, J.J., Lundebye, A.K., Larsen, E.H., Berntssen, M.H.G., and Amlulnd, H. 2012. "Arsenolipids in marine oils and fats: a review of occurrence, chemistry, and future research needs." *Food Chemistry 133* 618–630.

Shankar, S., Shanker, U., and Shikha. 2014. "Arsenic contamination of groundwater: a review of sources, prevalence, health risks, and strategies for mitigation." *The Scientific World Journal 2014* 1–18.

Sharma, V.K. and Sohn, M. 2009. "Aquatic arsenic: toxicity, speciation, transformations, and remediation." *Environment International 35* 743–759.

Shen, Z.X., Chen, G.Q., Ni, J.H., Li, X.S., Xiong, S.M., Qiu, Q.Y., Zhu, J., Tang, W., Sun, G.L., Yang, K.Q., Chen, Y., Zhou, L., Fang, Z.W., Wang, Y.T., Ma, J., Zhang, P., Zhang, T.D., Chen, S.J., Chen, Z., and Wang, Z.Y. 1997. "Use of arsenic trioxide (As2O3) in the treatment of acute promyelocytic leukemia (APL): II. Clinical efficacy and pharmacokinetics in relapsed patients." *Blood 89(9)* 3354–3360.

Simeonova, P.P. and Luster, M.I. 2000. "Mechanisms of arsenic carcinogenicity: genetic or epigenetic mechanisms?" *Journal of Environmental Pathology, Toxicology, and Oncology 19* 281–286.

Singh, A.P., Goel, R.K., and Kaur, T. 2011. "Mechanisms pertaining to arsenic toxicity." *Toxicology International 18(2*)87–93.

Singh, R., Singh, S., Parihar, P., Singh, V.P., and Prasad, S.M. 2015. "Arsenic contamination, consequences and remediation techniques: a review." *Ecotoxicology and Environmental Safety* 247–270.

Sirot, V., Guerin, T., Volatier, J.L., and Leblanc, J.C. 2009. "Dietary exposure and biomarkers of arsenic in consumers of fish and shellfish from France." *Science of the Total Environment 407* 1875–1885.

Smedley, P.L. and Kinniburgh, D.G. 2002. "A review of the source, behaviour and distribution of arsenic in natural waters." *Applied Geochemistry 17* 517–568.

Smith, A.H., Lingas, E.O., and Rahman, M. 2000. "Contamination of drinking–water by arsenic in Bangladesh: a public health emergency." *Bulletin of the World Health Organization 78(9)* 1093–1103.

Spinney, H.A., Piro, N.A., and Cummins, C.C. 2009. "Triple–bond activity of an AsP complex intermediate: synthesis stemming from molecular arsenic, As4." *Journal of the American Chemical Society 131(44)* 16233–16243.

Styblo, M., Del Razo, L.M., Vega, L., Germolec, D.R., LeCluyse, E.L., Hamilton, G.A., Reed, W., Wang, C., Cullen, W.R., and Thomas, D.J. 2000. "Comparative toxicity of trivalent and pentavalent inorganic and methylated arsenicals in rat and human cells." *Archives of Toxicology 74(6)* 289–299.

Styblo, M., Drobna, Z., Jaspers, I., Lin, S., and Thomas, D.J. 2002. "The role of biomethylation in toxicity and carcinogenicity of arsenic: a research update." *Environmental Health Perspectives 110(Suppl 5)* 767–771.

Sun, Q., Song, Y., Liu, S., Wang, F., Zhang, L., Xi, S., and Sun, G. 2015. "Arsenic exposure levels in relation to different working departments in a copper mining and smelting plant." *Atmospheric Environment 118* 1–6.

Sweat, D.E. 2007. *Fish facts for Florida consumers blue crab, SGEF–120.* Gainesville: Florida Sea Grant College Program.

Tagatz, M.E. 1968. "Growth of juvenile blue crabs, Callinectes sapidus Rathbun, in the St. Johns River, Florida." *Fishery Bulletin 67(2)* 281–288.

Thompson, D.J. 1993. "A chemical hypothesis for arsenic methylation in mammals." *Chemico–Biological Interactions 88(2–3)* 89–114.

Tittlebaum, M.E. 1987. *Inventory of heavy metals data: Louisiana coastal zone.* Washington, D.C.: U.S. Government Printing Office.

Tseng, C.H., Chong, C.K., Chen, C.J., and Ty, T.Y. 1996. "Dose–response relationship between peripheral vascular disease and ingested inorganic arsenic among residents in blackfoot disease endemic villages in Taiwan." *Atherosclerosis 120(1–2)* 125–133.

Tseng, W.P. 1977. "Effects and dose–response relationships of skin cancer and blackfoot disease with arsenic." *Environmental Health Perspectives 19* 109–119.

Umunnakwe, J.E. and Ogamba, A.S. 2013. "Bioaccumulation of heavy metals in crabs at Bundu–Ama community, Port Harcourt." *Journal of Environmental and Earth Science 3(14)* 11–21.

USACE. 2009. "Mermentau Basin Water Salinity Monitoring Sites." *U.S. Army Corps of Engineers.* July 1. http://www2.mvn.usace.army.mil/ops/sms/verm.asp.

USDA. 2015. *Web Soil Survey.* http://websoilsurvey.sc.egov.usda.gov/App/WebSoilSurvey.aspx.

USGS. 2011. *Subsidence and wetland loss related to fluid energy production, Gulf Coast Basin.* St. Petersburg, FL: St. Petersburg Coastal and Marine Science Center.

USGS. 2013. *USGS research on saline waters co–produced with energy resources, fact sheet FS–003–97.* Denver, CO: Central Region Energy Resources Team.

Vahidnia, A., van der Voet, G.B., and de Wolff, D.A. 2007. "Arsenic neurotoxicity – a review." *Human and Experimental Toxicology 26* 823–832.

Vahter, M. 1999. "Methylation of inorganic arsenic in different mammalian species and population groups." *Science Progress 82(Pt 1)* 69–88.

Vahter, M. 1994. "What are the chemical forms of arsenic in urine, and what can they tell us about exposure?" *Clinical Chemistry 40(5)* 679–680.

Veil, J. 2015. *U.S. produced water volumes and management practices in 2012.* Annapolis, MD: Veil Environmental, LLC.

Veil, J.A., Puder, M.G., Elcock, D., and Redweik Jr., R.J. 2004. *A white paper describing produced water from production of crude oil, natural gas, and coal bed methane.* Lemont, IL: Argonne National Laboratory.

Vijayaraghavan, K., Arun, M., Joshi, U.M., and Balasubramanian, R. 2009. "Biosorption of As (V) onto the shells of the crab (*Portunus sanguinolentus*): equilibrium and kinetic studies." *Industrial & Engineering Chemistry Research 48* 3589–3594.

Visser, J.M., Sasser, C.E., Chabreck, R.H., and Linscombe, R.G. 2000. "Marsh vegetation types of the Chenier Plain, Louisiana, USA." *Estuaries 23(3)* 318–327.

Wang, Z.G., Rivi, R., Delva, L., Konig, A., Scheinberg, D.A., Gambacorti–Passerini, C., Gabrilove, J.L., Warrell, Jr., R.P., and Pandolfi, P.P. 1998. "Arsenic trioxide and melarsoprol induce programmed cell death in myeloid leukemia cell lines and function in a PML and PML–RAR independent manner." *Blood, 92(5)* 1497–1504.

Wedepohl, K.H. 1995. "The composition of the continental crust." *Geochimica et Cosmochimica Acta 59(7)* 1217–1232.

Welch, A.H., Westjohn, D.B., Helsel, D.R., and Wanty, R.B. 2000. "Arsenic in groundwater of the United States: occurrence and geochemistry." *Groundwater 38(4)* 589–604.

Wenzel, W.W. 2013. "Arsenic." In *Heavy Metals in Soils*, by B.J. Alloway, 241–282. Glasgow, Scotland: Chapman & Hall.

Williams, J.D. and Dodd, C.K. 1978. *Importance of wetlands to endangered and threatened species.* Washington, D.C.: U.S. Fish and Wildlife Service.

Williams, S.J. 1995. *Louisiana coastal wetlands: a resource at risk.* Woods Hole, MA: U.S. Geological Survey.

Woolson, E.A., Moore, L., Fleischer, M., Kearney, P.C., Buck, W.B., Peoples, S.A., and Calvert, C.C. 1977. *Arsenic: Medical and Biologic Effects of Environmental Pollutants.* Washington, D.C.: National Academy of Sciences.

Zmolinski, A.V., Llorente–Mirandes, T., Lopez–Sanchez, J.F., and da Silva, M.M. 2015. "Establishment of a method for determination of arsenic species in seafood by LC–ICP–MS." *Food Chemistry 173* 1073–1082.

APPENDIX A

ICP–MS TUNING AND PERFORMANCE REPORTS

Report 1: ICP–MS Autotune Report, 10–26–2015, 12 pp.

Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified

Summary

The autotuning was successful.

Intensity Changes

Analyte	Original Intensity [cps]	Tuned Intensity [cps]
115In	61240	71544
140Ce	184159	223572
140Ce.16O	2998	1447
7Li	82	110
59Co	42213	51164
238U	534353	564160
140Ce.16O/140Ce	0.0163	0.0065

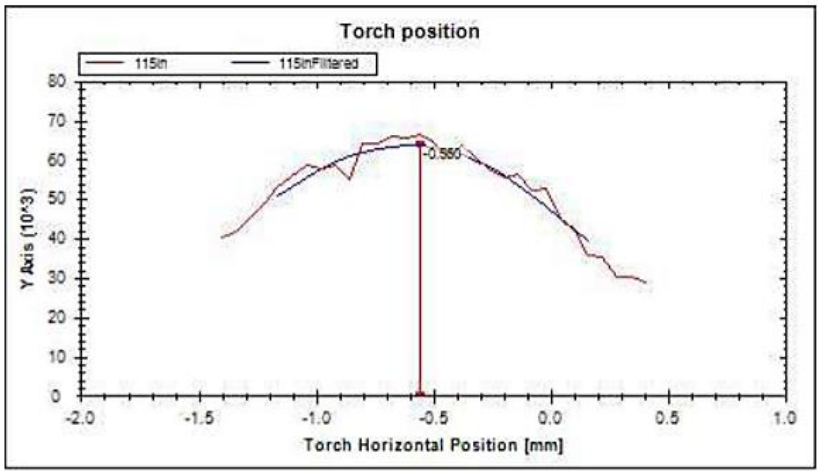
Control Changes

Control		Original Value	Tuned Value
Nebulizer Flow	[l/min]	0.965	0.94
Torch Horizontal Position	[mm]	-0.47	-0.68
Torch Vertical Position	[mm]	-0.56	-0.48
Extraction Lens 2	[V]	-116.3	-132
CCT Focus Lens	[V]	-2.76	-3.36

Autotune Report: **2015-10-26 16:32**

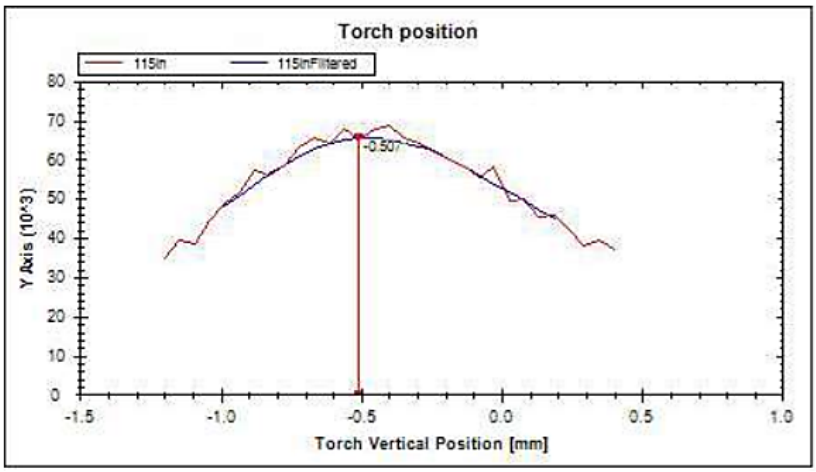
Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified

Graphical Results



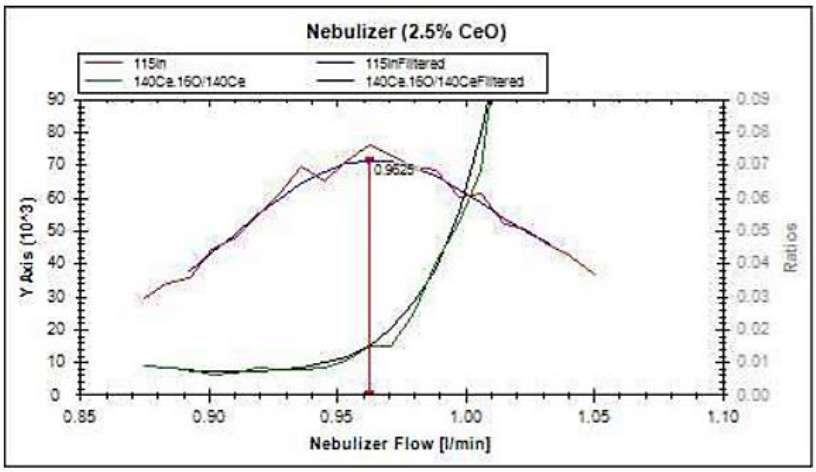
Autotune Report: **2015-10-26 16:32**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



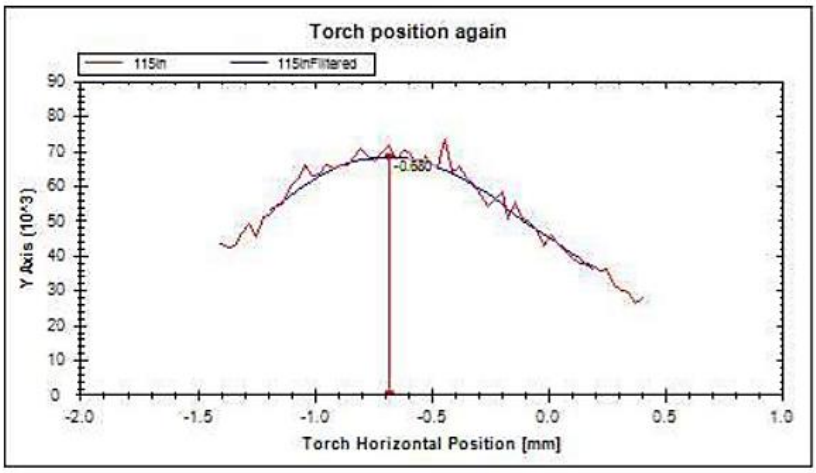
Autotune Report: **2015-10-26 16:32**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



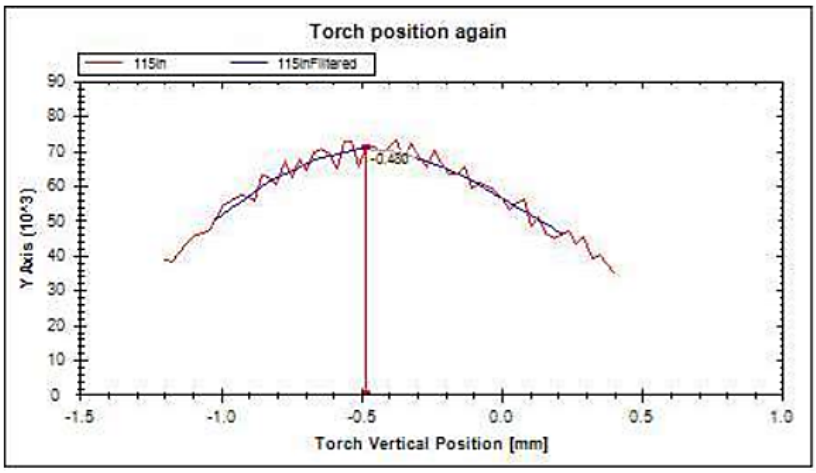
Autotune Report: **2015-10-26 16:32**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



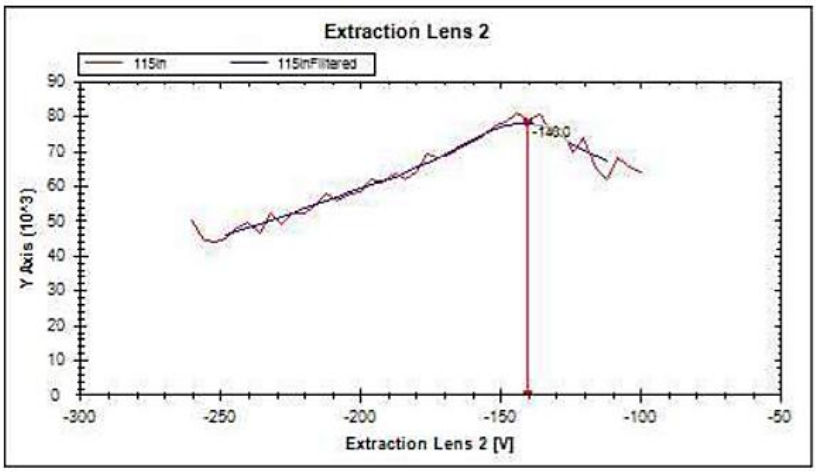
Autotune Report: **2015-10-26 16:32**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



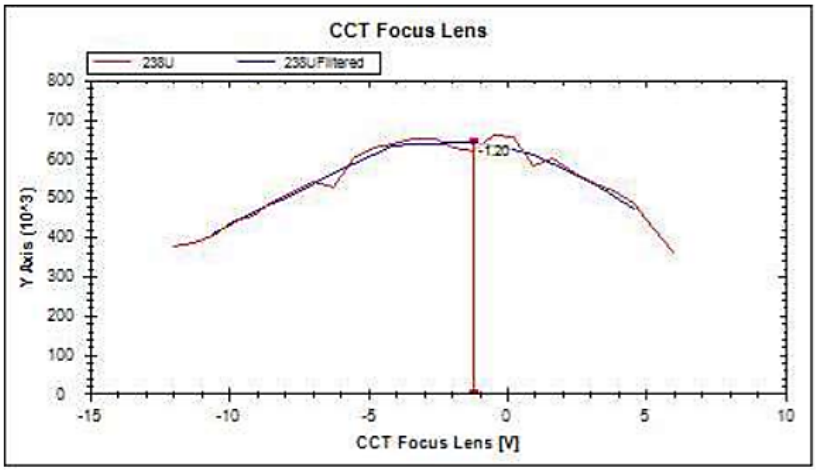
Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified



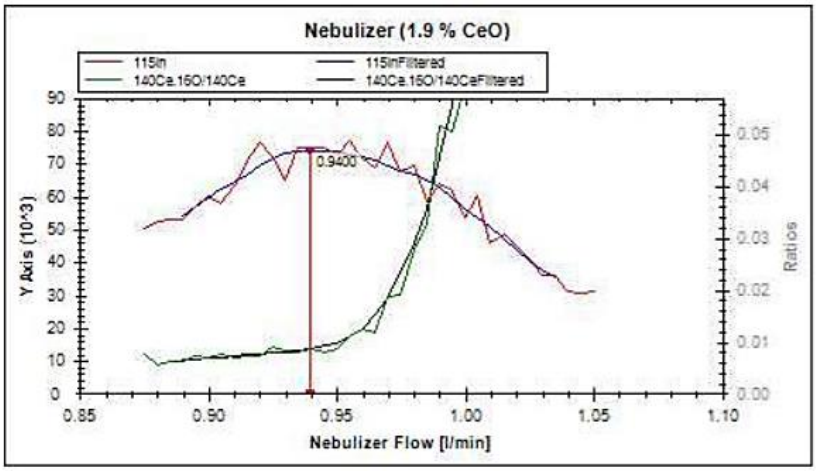
Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified



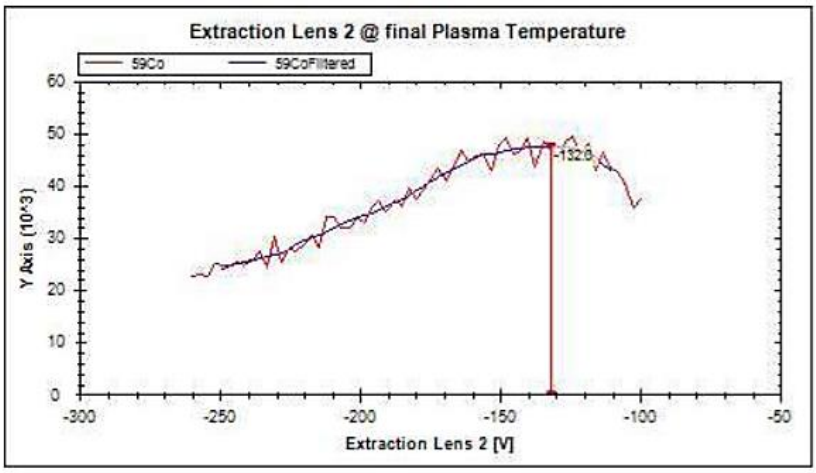
Autotune Report: **2015-10-26 16:32**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	PDRC Source Tune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



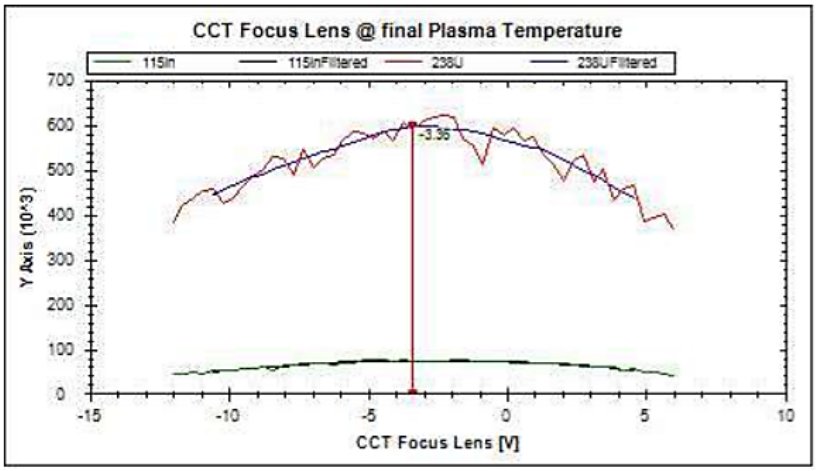
Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified



Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified



Resulting Tune Settings

Parameter	Value
Additional Gas Flow 1	0.00
Additional Gas Flow 2	0.00
Additional Gas Flow 3	0.00
CCT Entry Lens	-86.00
Angular Deflection	-377.00
Deflection Entry Lens	-35.00
Extraction Lens 1 Polarity	0.00
Extraction Lens 1 Negative	0.00

Autotune Report: **2015-10-26 16:32**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: PDRC Source Tune High Matrix
Serial Number: Undefined
Solution: No solution specified

Extraction Lens 1 Positive	0.00
Spray Chamber Temperature	2.70
Peristaltic Pump Speed	40.00
Cool Flow	14.00
Sampling Depth	5.00
Plasma Power	1550.00
Auxilliary Flow	0.80
Nebulizer Flow	0.94
Torch Horizontal Position	-0.68
Torch Vertical Position	-0.48
Extraction Lens 2	-132.00
CCT Focus Lens	-3.36
CCT Bias	-21.00
CCT Exit Lens	-40.00
Focus Lens	-8.00
D1 Lens	-350.00
D2 Lens	-160.00
Quad Entry Lens	-30.00
Pole Bias	-18.00
CCT1 Flow	4.80
CCT2 Flow	0.00
CCT1 Shut-Off Valve	1.00
CCT2 Shut-Off Valve	0.00
Virtual CCT Mass to Dac Factor	60.00
Virtual CCT Mass to Dac Offset	0.00
Virtual CCT Mass parameter b	1.00
Virtual CCT Mass Maximum Dac Limit Set	4095.00

Report 2: ICP–MS Autotune Report, 11–11–2015, 12 pp.

Autotune Report: **2015-11-11 13:59**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: SourceTune High Matrix
Serial Number: Undefined
Solution: No solution specified

Summary

The autotuning was successful.

Intensity Changes

Analyte	Original Intensity [cps]	Tuned Intensity [cps]
115In	71401	68356
140Ce	221372	212643
140Ce.16O	1611	1750
7Li	86	120
59Co	47480	45402
238U	561858	590506
140Ce.16O/140Ce	0.0073	0.0082

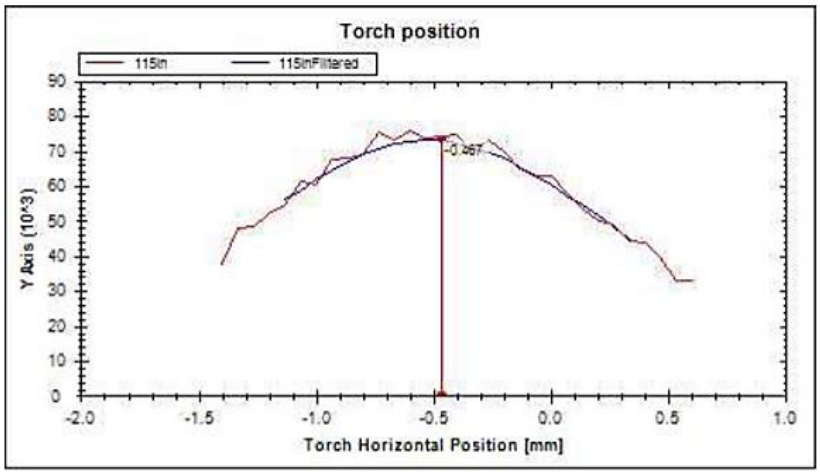
Control Changes

Control		Original Value	Tuned Value
Nebulizer Flow	[l/min]	0.94	0.95
Torch Horizontal Position	[mm]	-0.68	-0.47
Torch Vertical Position	[mm]	-0.48	-0.39
Extraction Lens 2	[V]	-132	-116.3
CCT Focus Lens	[V]	-3.36	-3.48

Autotune Report: **2015-11-11 13:59**

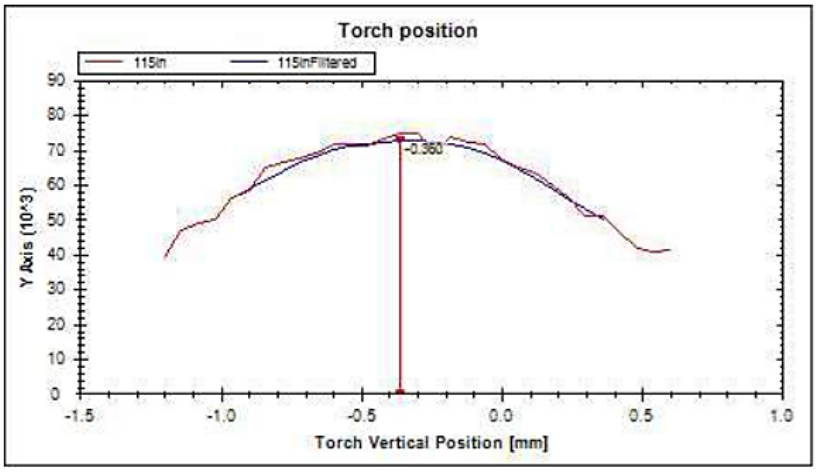
Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified

Graphical Results



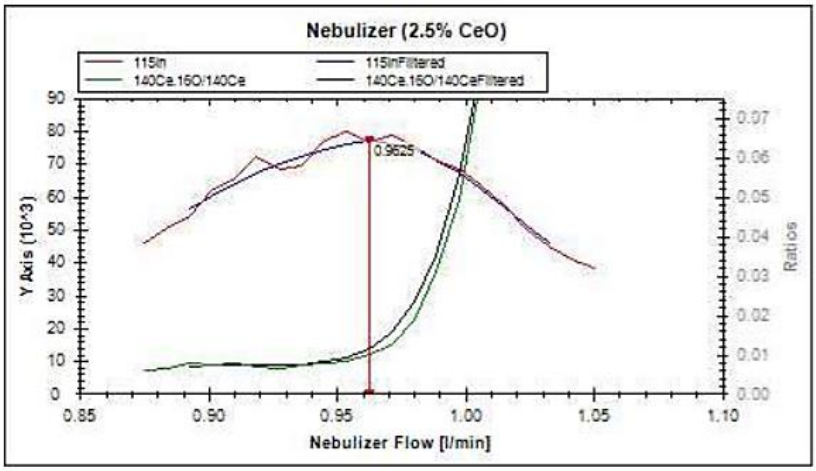
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



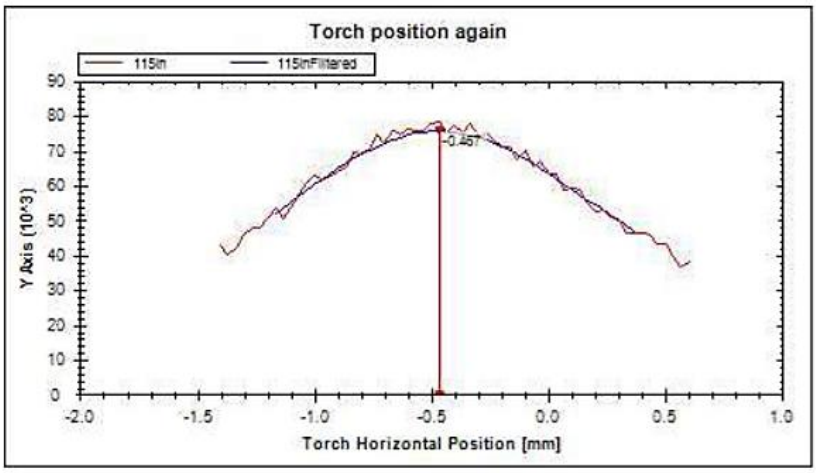
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



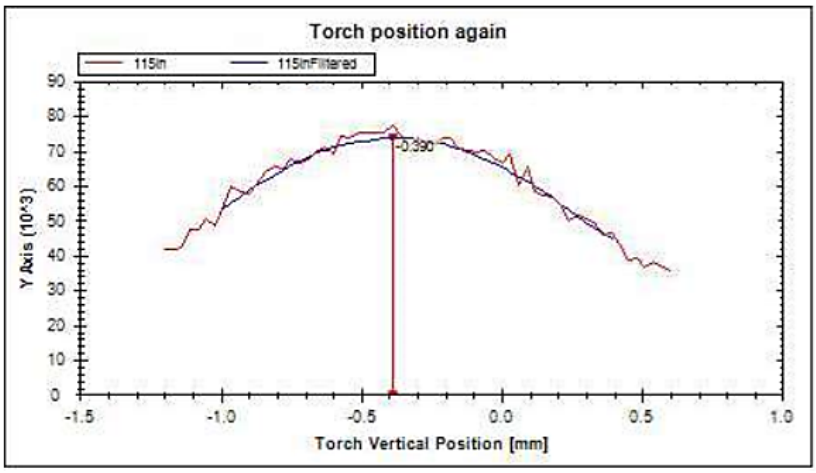
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



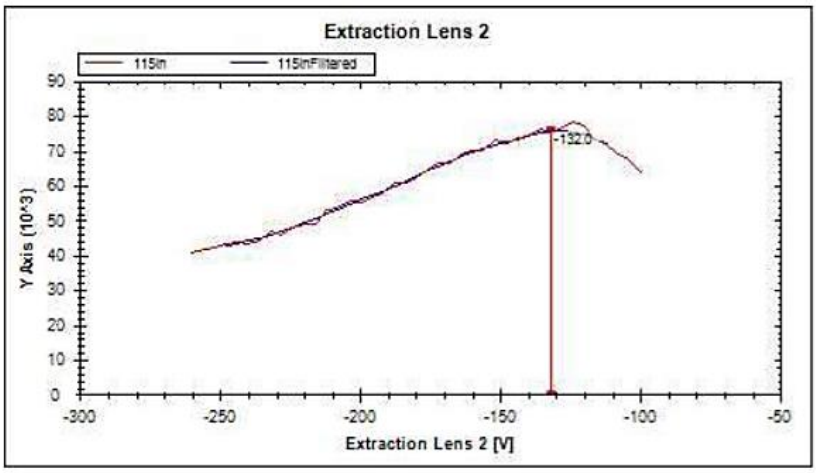
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



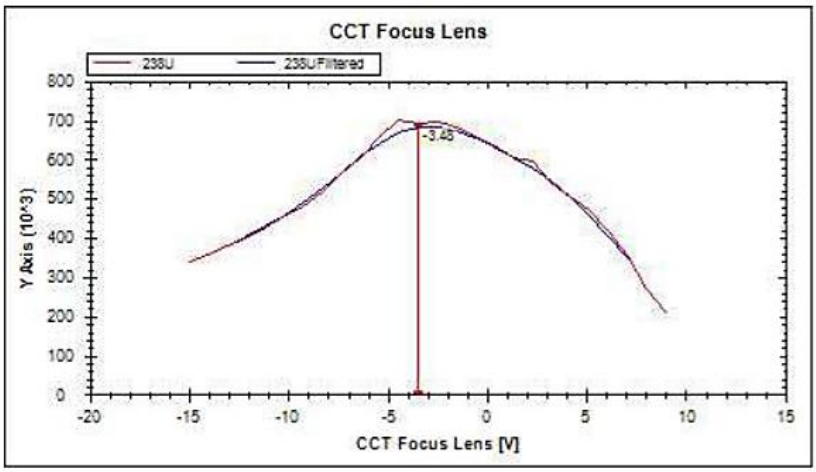
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



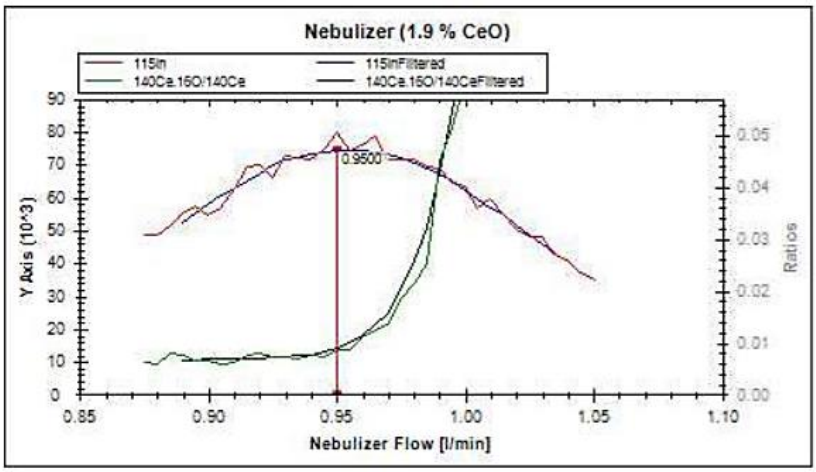
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



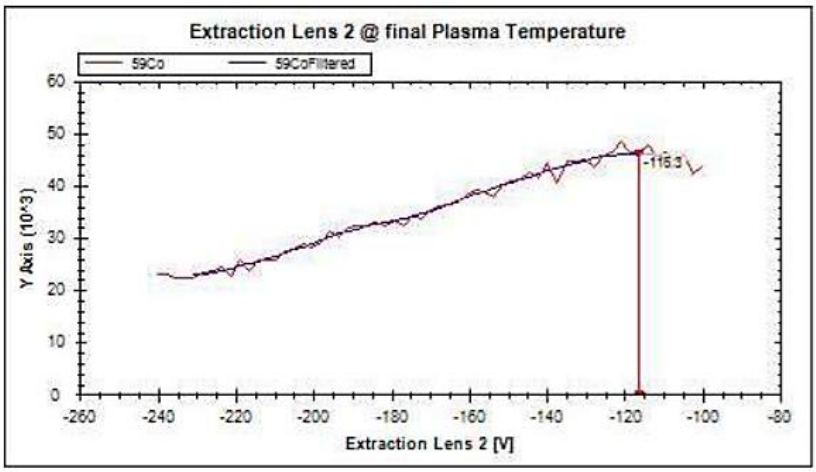
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



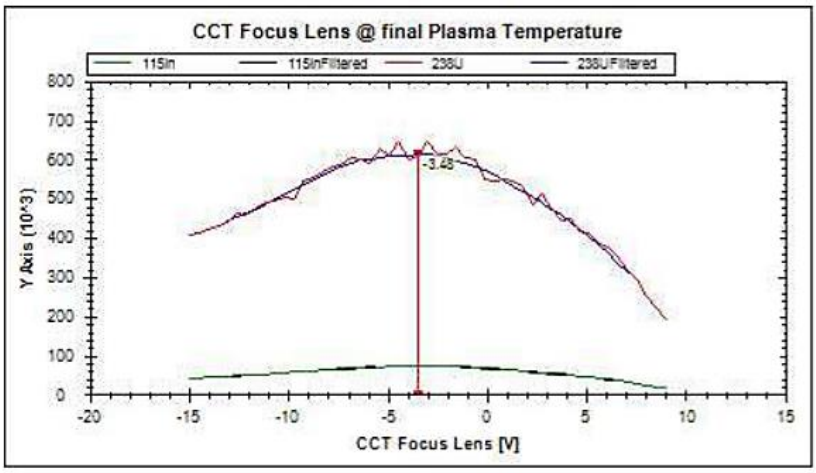
Autotune Report: **2015-11-11 13:59**

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Sequence:	SourceTune High Matrix
Serial Number:	Undefined
Solution:	No solution specified



Autotune Report: **2015-11-11 13:59**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: SourceTune High Matrix
Serial Number: Undefined
Solution: No solution specified



Resulting Tune Settings

Parameter	Value
Additional Gas Flow 1	0.00
Additional Gas Flow 2	0.00
Additional Gas Flow 3	0.00
CCT Entry Lens	-86.00
Angular Deflection	-377.00
Deflection Entry Lens	-35.00
Extraction Lens 1 Polarity	0.00
Extraction Lens 1 Negative	0.00

Autotune Report: **2015-11-11 13:59**

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Sequence: SourceTune High Matrix
Serial Number: Undefined
Solution: No solution specified

Extraction Lens 1 Positive	0.00
Spray Chamber Temperature	2.70
Peristaltic Pump Speed	40.00
Cool Flow	14.00
Sampling Depth	5.00
Plasma Power	1550.00
Auxilliary Flow	0.80
Nebulizer Flow	0.95
Torch Horizontal Position	-0.47
Torch Vertical Position	-0.39
Extraction Lens 2	-116.33
CCT Focus Lens	-3.48
CCT Bias	-21.00
CCT Exit Lens	-40.00
Focus Lens	-8.00
D1 Lens	-350.00
D2 Lens	-160.00
Quad Entry Lens	-30.00
Pole Bias	-18.00
CCT1 Flow	4.80
CCT2 Flow	0.00
CCT1 Shut-Off Valve	1.00
CCT2 Shut-Off Valve	0.00
Virtual CCT Mass to Dac Factor	60.00
Virtual CCT Mass to Dac Offset	0.00
Virtual CCT Mass parameter b	1.00
Virtual CCT Mass Maximum Dac Limit Set	4095.00

Report 3: ICP–MS Performance Report, 10–22–2015, 6 pp.

Performance Report: 2015-10-22 12:08

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	STD
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Signal **Result: Failed**

Runs: 10

Sweeps: 60

Analyte			Dwell time [s]		Result
137Ba			0.1		**Passed**
	Stability Test	Limit:	0	Result: 0.8 %	
	Sensitivity Test		50,335.0		0.0
137Ba++			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.1 %	
	Sensitivity Test		1,279.0		0.0
137Ba++/137Ba			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.4 %	
	Sensitivity Test		0.0254	Less than	0.03
140Ce			0.1		**Passed**
	Stability Test	Limit:	0	Result: 0.4 %	
	Sensitivity Test		370,159.0		0.0
140Ce.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.2 %	
	Sensitivity Test		12,063.0		0.0

Performance Report: 2015-10-22 12:08

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Template: STD
Serial Number: Undefined
Last Autotune: Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution: 1 ppb Tune B in 2% HNO3 and 0.5% HCl.

140Ce.16O/140Ce			0.1		**Failed**
	Stability Test	Limit:	0	Result: 1.1 %	
	Sensitivity Test		0.0326	Less than	0.02
Bkg4.5			0.1		**Passed**
	Stability Test	Limit:	0	Result: NaN	
	Sensitivity Test		0.0 CPS	Less than	1.0 CPS
7Li			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.6 %	
	Sensitivity Test		78,340.0 CPS	Greater than	50,000.0 CPS
59Co			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.5 %	
	Sensitivity Test		189,139.0 CPS	Greater than	100,000.0 CPS
115In			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.6 %	
	Sensitivity Test		385,582.0 CPS	Greater than	220,000.0 CPS
Bkg220.7			0.1		**Passed**
	Stability Test	Limit:	0	Result: 84.4 %	
	Sensitivity Test		0.27 CPS	Less than	2.0 CPS
238U			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.6 %	
	Sensitivity Test		409,267.0 CPS	Greater than	300,000.0 CPS

Performance Report: 2015-10-22 12:08

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	STD
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

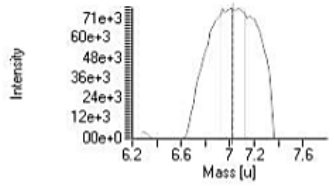
Masscalibration Result: Passed The mass calibration verification has passed

Sweeps:	5	Channels:	75
Measurement width [u]:	1.5	Point spacing [u]:	0.02
		Dwell time [s]:	0.04

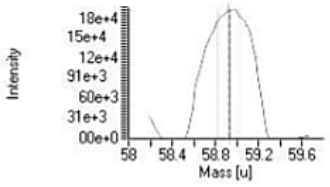
Analyte	Centroid Mass [u]	Peak width [u]:	Error [u]:	Peak width [u]		Result:
				Min.	Max.	
7Li	7.0224	0.6927	0.00645	0.65	0.85	Passed
59Co	58.9214	0.7044	0.01177	0.65	0.85	Passed
115In	114.8898	0.7138	0.01410	0.65	0.85	Passed
238U	238.0752	0.7372	0.02444	0.65	0.85	Passed

Peaks

7Li



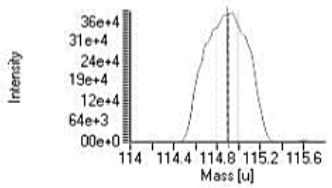
59Co



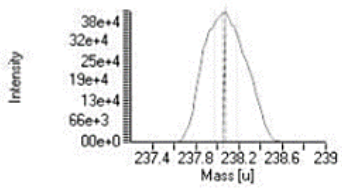
Performance Report: 2015-10-22 12:08

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	STD
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

115In



238U



Tune Settings

Parameter	Value
Additional Gas Flow 1	0.00
Additional Gas Flow 2	0.00
Additional Gas Flow 3	0.00
CCT Entry Lens	-86.00
Angular Deflection	-377.00
Deflection Entry Lens	-35.00
Extraction Lens 1 Polarity	0.00
Extraction Lens 1 Negative	0.00
Extraction Lens 1 Positive	0.00
Spray Chamber Temperature	2.70
Peristaltic Pump Speed	40.00
Cool Flow	14.00

Performance Report: 2015-10-22 12:08

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	STD
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Sampling Depth	5.00
Plasma Power	1550.00
Auxilliary Flow	0.80
Nebulizer Flow	0.96
Torch Horizontal Position	-0.71
Torch Vertical Position	-0.51
Extraction Lens 2	-108.00
CCT Focus Lens	-5.28
CCT Bias	-2.00
CCT Exit Lens	-160.00
Focus Lens	18.00
D1 Lens	-195.20
D2 Lens	-80.00
Quad Entry Lens	-18.20
Pole Bias	-1.00
CCT1 Flow	0.00
CCT2 Flow	0.00
CCT1 Shut-Off Valve	0.00
CCT2 Shut-Off Valve	0.00
Virtual CCT Mass to Dac Factor	130.00
Virtual CCT Mass to Dac Offset	-240.00
Virtual CCT Mass parameter b	0.65
Virtual CCT Mass Maximum Dac Limit Set	4095.00

Performance Report: 2015-10-22 12:08

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Template: STD
Serial Number: Undefined
Last Autotune: Autotune-SourceTune High Matrix-20151019-163458759.imatdat
Solution: 1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Vacuum Check

Parameter	Value	Rating
Analyzer Pressure	4.31E-07	Vacuum ok
Interface Pressure	1.69E+00	

Detector Voltages

Counting	1000.0
Analog	-1775.0

Report 4: ICP–MS Performance Report, 10–26–2015, 6 pp.

Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Signal **Result: Passed**

Runs: 10

Sweeps: 60

Analyte			Dwell time [s]		Result
35Cl.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.0 %	
	Sensitivity Test		1,307.0		0.0
59Co			0.1		**Passed**
	Stability Test	Limit:	2	Result: 1.2 %	
	Sensitivity Test		47,578.0 CPS	Greater than	30,000.0 CPS
59Co/35Cl.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.7 %	
	Sensitivity Test		36.4	Greater than	18.0
140Ce			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.0 %	
	Sensitivity Test		222,761.0		0.0
140Ce.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.2 %	
	Sensitivity Test		1,876.0		0.0

10/26/2015 4:46:24 PM 1 / 6

Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

140Ce.16O/140Ce			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.9 %	
	Sensitivity Test		0.0084	Less than	0.01
Bkg4.5			0.3		**Passed**
	Stability Test	Limit:	0	Result: 140.5 %	
	Sensitivity Test		0.033 CPS	Less than	0.5 CPS
115In			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.9 %	
	Sensitivity Test		72,286.0 CPS	Greater than	30,000.0 CPS
Bkg220.7			0.1		**Passed**
	Stability Test	Limit:	0	Result: 76.0 %	
	Sensitivity Test		0.35 CPS	Less than	2.0 CPS
238U			0.1		**Passed**
	Stability Test	Limit:	2	Result: 1.0 %	
	Sensitivity Test		572,157.0 CPS	Greater than	80,000.0 CPS

Masscalibration	**Result:**	**Passed**	The mass calibration verification has passed		
Sweeps:	5		Channels:	75	
Measurement width [u]:	1.5		Point spacing [u]:	0.02	
			Dwell time [s]:	0.04	

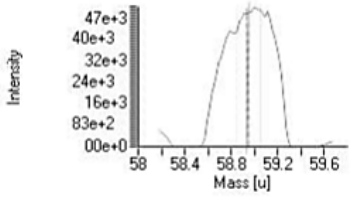
Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

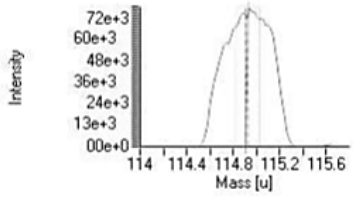
Analyte	Centroid Mass [u]	Peak width [u]:	Error [u]:	Peak width [u]		Result:
				Min.	Max.	
59Co	58.9492	0.7003	0.01596	0.65	0.85	Passed
115In	114.9256	0.6996	0.02175	0.65	0.85	Passed
238U	238.0922	0.7015	0.04140	0.65	0.85	Passed

Peaks

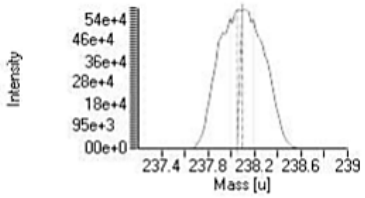
59Co



115In



238U



Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

TuneSettings

Parameter	Value
Additional Gas Flow 1	0.00
Additional Gas Flow 2	0.00
Additional Gas Flow 3	0.00
CCT Entry Lens	-86.00
Angular Deflection	-377.00
Deflection Entry Lens	-35.00
Extraction Lens 1 Polarity	0.00
Extraction Lens 1 Negative	0.00
Extraction Lens 1 Positive	0.00
Spray Chamber Temperature	2.70
Peristaltic Pump Speed	40.00
Cool Flow	14.00
Sampling Depth	5.00
Plasma Power	1550.00
Auxilliary Flow	0.80
Nebulizer Flow	0.94
Torch Horizontal Position	-0.68
Torch Vertical Position	-0.48
Extraction Lens 2	-132.00
CCT Focus Lens	-3.36

Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

CCT Bias	-21.00
CCT Exit Lens	-40.00
Focus Lens	-8.00
D1 Lens	-350.00
D2 Lens	-160.00
Quad Entry Lens	-30.00
Pole Bias	-18.00
CCT1 Flow	4.80
CCT2 Flow	0.00
CCT1 Shut-Off Valve	1.00
CCT2 Shut-Off Valve	0.00
Virtual CCT Mass to Dac Factor	60.00
Virtual CCT Mass to Dac Offset	0.00
Virtual CCT Mass parameter b	1.00
Virtual CCT Mass Maximum Dac Limit Set	4095.00

Vacuum Check

Parameter	Value	Rating
Analyzer Pressure	7.40E-07	
Interface Pressure	1.62E+00	

Detector Voltages

Performance Report: 2015-10-26 16:46

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-PDRC Source Tune High Matrix-20151026-163223068.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Counting	1000.0
Analog	-1775.0

Report 5: ICP–MS Performance Report, 11–11–2015, 6 pp.

Performance Report: 2015-11-11 14:13

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Signal **Result: Passed**

Runs: 10

Sweeps: 60

Analyte			Dwell time [s]		Result
35Cl.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.5 %	
	Sensitivity Test		1,383.0		0.0
59Co			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.6 %	
	Sensitivity Test		44,511.0 CPS	Greater than	30,000.0 CPS
59Co/35Cl.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 1.8 %	
	Sensitivity Test		32.2	Greater than	18.0
140Ce			0.1		**Passed**
	Stability Test	Limit:	0	Result: 0.5 %	
	Sensitivity Test		216,641.0		0.0
140Ce.16O			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.4 %	
	Sensitivity Test		1,900.0		0.0

Performance Report: 2015-11-11 14:13

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

140Ce.16O/140Ce			0.1		**Passed**
	Stability Test	Limit:	0	Result: 2.3 %	
	Sensitivity Test		0.0088	Less than	0.01
Bkg4.5			0.3		**Passed**
	Stability Test	Limit:	0	Result: 225.0 %	
	Sensitivity Test		0.017 CPS	Less than	0.5 CPS
115In			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.7 %	
	Sensitivity Test		70,383.0 CPS	Greater than	30,000.0 CPS
Bkg220.7			0.1		**Passed**
	Stability Test	Limit:	0	Result: 111.0 %	
	Sensitivity Test		0.27 CPS	Less than	2.0 CPS
238U			0.1		**Passed**
	Stability Test	Limit:	2	Result: 0.8 %	
	Sensitivity Test		593,542.0 CPS	Greater than	80,000.0 CPS

Masscalibration	**Result: Passed**	The mass calibration verification has passed	
Sweeps:	5	Channels:	75
Measurement width [u]:	1.5	Point spacing [u]:	0.02
		Dwell time [s]:	0.04

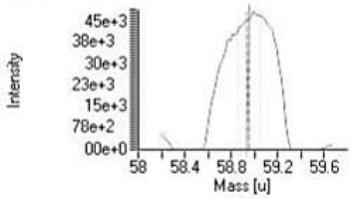
Performance Report: 2015-11-11 14:13

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

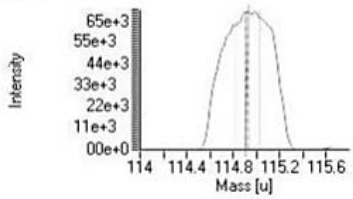
Analyte	Centroid Mass [u]	Peak width [u]:	Error [u]:	Peak width [u]		Result:
				Min.	Max.	
59Co	58.9528	0.6993	0.01964	0.65	0.85	Passed
115In	114.9259	0.7035	0.02201	0.65	0.85	Passed
238U	238.0885	0.7136	0.03771	0.65	0.85	Passed

Peaks

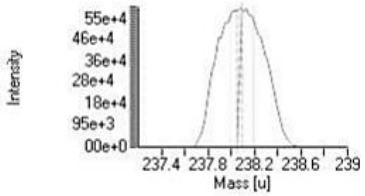
59Co



115In



238U



Performance Report: 2015-11-11 14:13

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

TuneSettings

Parameter	Value
Additional Gas Flow 1	0.00
Additional Gas Flow 2	0.00
Additional Gas Flow 3	0.00
CCT Entry Lens	-86.00
Angular Deflection	-377.00
Deflection Entry Lens	-35.00
Extraction Lens 1 Polarity	0.00
Extraction Lens 1 Negative	0.00
Extraction Lens 1 Positive	0.00
Spray Chamber Temperature	2.70
Peristaltic Pump Speed	40.00
Cool Flow	14.00
Sampling Depth	5.00
Plasma Power	1550.00
Auxilliary Flow	0.80
Nebulizer Flow	0.95
Torch Horizontal Position	-0.47
Torch Vertical Position	-0.39
Extraction Lens 2	-116.33
CCT Focus Lens	-3.48
CCT Bias	-21.00

Performance Report: 2015-11-11 14:13

Instrument: iCAP Q
Operator: WTAMU-PC\lab2
Template: KED
Serial Number: Undefined
Last Autotune: Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution: 1 ppb Tune B in 2% HNO3 and 0.5% HCl.

CCT Exit Lens	-40.00
Focus Lens	-8.00
D1 Lens	-350.00
D2 Lens	-160.00
Quad Entry Lens	-30.00
Pole Bias	-18.00
CCT1 Flow	4.80
CCT2 Flow	0.00
CCT1 Shut-Off Valve	1.00
CCT2 Shut-Off Valve	0.00
Virtual CCT Mass to Dac Factor	60.00
Virtual CCT Mass to Dac Offset	0.00
Virtual CCT Mass parameter b	1.00
Virtual CCT Mass Maximum Dac Limit Set	4095.00

Vacuum Check

Parameter	Value	Rating
Analyzer Pressure	7.57E-07	
Interface Pressure	1.69E+00	

Detector Voltages

Counting 1000.0

Performance Report: 2015-11-11 14:13

Instrument:	iCAP Q
Operator:	WTAMU-PC\lab2
Template:	KED
Serial Number:	Undefined
Last Autotune:	Autotune-SourceTune High Matrix-20151111-135919054.imatdat
Solution:	1 ppb Tune B in 2% HNO3 and 0.5% HCl.

Analog	-1775.0

APPENDIX B

IC/ICP–MS RAW DATA

Report 6: ICP–MS sediment total arsenic raw data, 10 pp.

Comprehensive

10/22/2015 1:47:49 PM

QTEGRA

Instrument Summary:

Instrument ID: iCAP Q
Serial Number: Undefined

Labbook Summary:

Path and filename:	_Application Data\Workspace\LabBooks\Heavy Metals (As, Cd, Pb) Sediments and PW 10 22 2015.imexp
Labbook created by:	lab2
Based on template:	
Hardware configuration:	iCAP Q with ASX 520
Labbook type:	eQuant
Qtegra version (last saved):	2.4.1800.192
Labbook started:	10/22/2015 12:44:14 PM
Labbook finished:	10/22/2015 1:45:06 PM

Method Summary:

Isotope	Mode	Dwell time (s)	# of Channels	Spacing	Resolution	IS
45Sc (STD)	STD	0.01	1	0.1	Normal	
73Ge (KED)	KED	0.01	1	0.1	Normal	
75As (KED)	KED	0.1	1	0.1	Normal	
111Cd (STD)	STD	0.05	1	0.1	Normal	
115In (STD)	STD	0.01	1	0.1	Normal	
208Pb (STD)	STD	0.05	1	0.1	Normal	

Isotope	Mode	Dwell time (s)	# of Channels	Spacing	Resolution
45Sc (STD)	STD	0.01	1	0.1	Normal
73Ge (KED)	KED	0.01	1	0.1	Normal
75As (KED)	KED	0.1	1	0.1	Normal
111Cd (STD)	STD	0.05	1	0.1	Normal
115In (STD)	STD	0.01	1	0.1	Normal
208Pb (STD)	STD	0.05	1	0.1	Normal

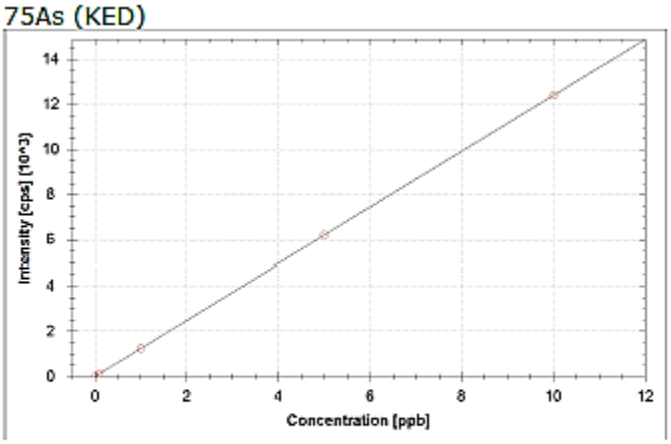
No. of sweeps	Time per sweep[s]	Time per main run[s]
10	0.23	2.3

Comprehensive
10/22/2015 1:47:49 PM

QTEGRA

Calibration Curve

75As (KED)



f(x) = 1239.3440*x + 10.0000
R^2 = 1.0000
BEC = 0.008 ppb
LoD = N/A

Comprehensive

10/22/2015 1:47:49 PM

QTEGRA

Standards:

Analysis Index: 2
Analysis Name: 0.1 ppb
Analysis Type: STD
Analysis Started at: 10/22/2015 12:45:56 PM
Standard (Stock): 0.1 ppb
Standard DF: 1
Rack: 0
Vial: 2

Category	Concentration average	Concentration RSD	Standard Concentration
45Sc (STD)	98.615 %		
73Ge (KED)	90.822 %		
75As (KED)	0.092 ppb		0.100 ppb
111Cd (STD)	0.097 ppb		0.100 ppb
115In (STD)	97.169 %		
208Pb (STD)	0.122 ppb		0.100 ppb

Standards:

Analysis Index: 3
Analysis Name: 1 ppb
Analysis Type: STD
Analysis Started at: 10/22/2015 12:47:30 PM
Standard (Stock): 1 ppb
Standard DF: 1
Rack: 0
Vial: 3

Category	Concentration average	Concentration RSD	Standard Concentration
45Sc (STD)	98.089 %		
73Ge (KED)	100.493 %		
75As (KED)	1.008 ppb		1.000 ppb
111Cd (STD)	0.988 ppb		1.000 ppb
115In (STD)	99.161 %		
208Pb (STD)	1.011 ppb		1.000 ppb

Comprehensive

10/22/2015 1:47:49 PM

QTEGRA

Standards:

Analysis Index: 4
Analysis Name: 5 ppb
Analysis Type: STD
Analysis Started at: 10/22/2015 12:49:05 PM
Standard (Stock): 5 ppb
Standard DF: 1
Rack: 0
Vial: 4

Category	Concentration average	Concentration RSD	Standard Concentration
45Sc (STD)	98.547 %		
73Ge (KED)	100.517 %		
75As (KED)	5.002 ppb		5.000 ppb
111Cd (STD)	5.033 ppb		5.000 ppb
115In (STD)	97.725 %		
208Pb (STD)	5.047 ppb		5.000 ppb

Standards:

Analysis Index: 5
Analysis Name: 10 ppb
Analysis Type: STD
Analysis Started at: 10/22/2015 12:50:40 PM
Standard (Stock): 10 ppb
Standard DF: 1
Rack: 0
Vial: 5

Category	Concentration average	Concentration RSD	Standard Concentration
45Sc (STD)	98.261 %		
73Ge (KED)	97.764 %		
75As (KED)	9.998 ppb		10.000 ppb
111Cd (STD)	9.985 ppb		10.000 ppb
115In (STD)	97.076 %		
208Pb (STD)	9.975 ppb		10.000 ppb

Comprehensive
10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 1
Analysis name: Blank
Analysis started at: 10/22/2015 12:44:22 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 1

Category	Concentration average	Concentration RSD
75As (KED)	0.000 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	100 %	13088039.2 cps	

Analysis index: 2
Analysis name: 0.1 ppb
Analysis started at: 10/22/2015 12:45:56 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 2

Category	Concentration average	Concentration RSD
75As (KED)	0.092 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.6 %	12906802.9 cps	

Analysis index: 3
Analysis name: 1 ppb
Analysis started at: 10/22/2015 12:47:30 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 3

Category	Concentration average	Concentration RSD
75As (KED)	1.008 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.1 %	12837938.7 cps	

Comprehensive

10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 4
Analysis name: 5 ppb
Analysis started at: 10/22/2015 12:49:05 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 4

Category	Concentration average	Concentration RSD
75As (KED)	5.002 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.5 %	12897935.1 cps	

Analysis index: 5
Analysis name: 10 ppb
Analysis started at: 10/22/2015 12:50:40 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 5

Category	Concentration average	Concentration RSD
75As (KED)	9.998 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.3 %	12860454.7 cps	

Analysis index: 6
Analysis name: ICV
Analysis started at: 10/22/2015 12:52:16 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 6

Category	Concentration average	Recovery Percentage 1	Concentration RSD
75As (KED)	5.088 ppb	101.757 %	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98 %	12831634.2 cps	

Comprehensive
10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 7
Analysis name: LFB
Analysis started at: 10/22/2015 12:53:53 PM
User name: WTAMU-PC\lab2
Rack: 1
Vial: 1

Category	Concentration average	Concentration RSD
75As (KED)	0.006 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	101.6 %	13292137.8 cps	

Analysis index: 8
Analysis name: DORM
Analysis started at: 10/22/2015 12:55:26 PM
User name: WTAMU-PC\lab2
Rack: 1
Vial: 2

Category	Concentration average	Concentration RSD
75As (KED)	7,060.312 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.2 %	12851171.2 cps	

Analysis index: 9
Analysis name: TORT
Analysis started at: 10/22/2015 12:57:01 PM
User name: WTAMU-PC\lab2
Rack: 1
Vial: 3

Category	Concentration average	Concentration RSD
75As (KED)	74,589.031 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	97.5 %	12763670.6 cps	

Comprehensive
10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 13
Analysis name: L-1-SS
Analysis started at: 10/22/2015 1:03:24 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 4

Category	Concentration average	Concentration RSD
75As (KED)	1,060.746 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	104.5 %	13677541.9 cps	

Analysis index: 14
Analysis name: L-2-SS
Analysis started at: 10/22/2015 1:05:00 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 5

Category	Concentration average	Concentration RSD
75As (KED)	1,352.045 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	99 %	12962642.5 cps	

Analysis index: 15
Analysis name: L-3-SS
Analysis started at: 10/22/2015 1:06:36 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 6

Category	Concentration average	Concentration RSD
75As (KED)	1,474.331 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	97.4 %	12745588.6 cps	

Comprehensive
10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 16
Analysis name: S-1-SS
Analysis started at: 10/22/2015 1:08:12 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 7

Category	Concentration average	Concentration RSD
75As (KED)	1,626.587 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	101.3 %	13252578.9 cps	

Analysis index: 17
Analysis name: S-2-SS
Analysis started at: 10/22/2015 1:09:48 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 8

Category	Concentration average	Concentration RSD
75As (KED)	2,209.629 ppb	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	102.9 %	13469009.4 cps	

Analysis index: 21
Analysis name: QC Check 5 ppb As Cd Pb
Analysis started at: 10/22/2015 1:16:13 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 4

Category	Concentration average	Recovery Percentage 1	Concentration RSD
75As (KED)	5.024 ppb	100.482 %	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	98.4 %	12876042.6 cps	

Comprehensive

10/22/2015 1:47:49 PM

QTEGRA

Sample Summary

Instrument ID: iCAP Q

Analysis index: 24
Analysis name: S-6-SS
Analysis started at: 10/22/2015 1:21:01 PM
User name: WTAMU-PC\lab2
Rack: 3
Vial: 13

Category	Concentration average	Concentration RSD
75As (KED)	2,910.258 ppb	

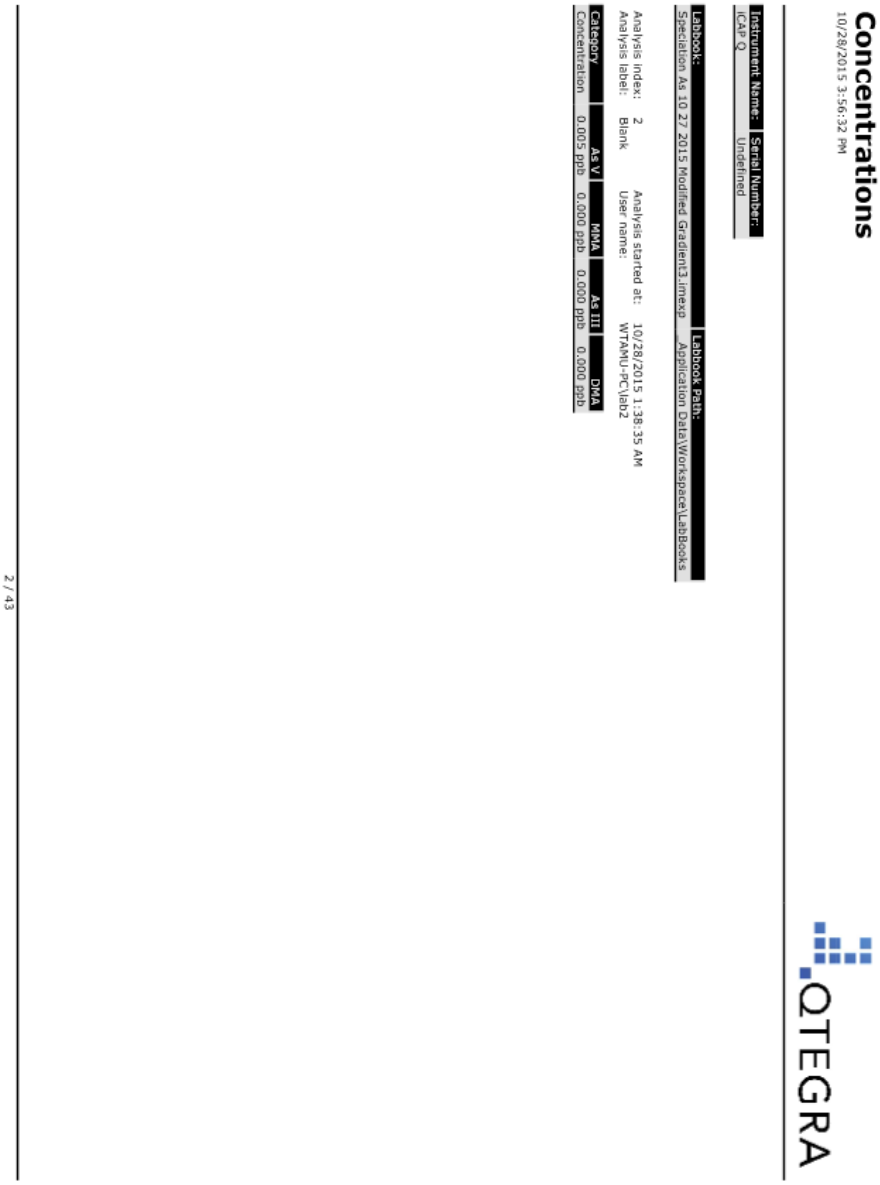
Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	97.3 %	12732841.1 cps	

Analysis index: 28
Analysis name: QC Check 5 ppb As Cd Pb
Analysis started at: 10/22/2015 1:27:27 PM
User name: WTAMU-PC\lab2
Rack: 0
Vial: 4

Category	Concentration average	Recovery Percentage 1	Concentration RSD
75As (KED)	5.030 ppb	100.595 %	

Category	Concentration average	Intensity average	Intensity RSD
45Sc (STD)	97.9 %	12819718.1 cps	

Report 7: IC/ICP–MS speciation raw data, 10–28–2015, 42 pp.



Concentrations

10/28/2015 3:56:32 PM

QTEGRA

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.inexp	Application Data\Workspace\LabBooks

Analysis index: 2
Analysis label: Blank

Analysis started at: 10/28/2015 1:38:35 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.005 ppb	0.000 ppb	0.000 ppb	0.000 ppb

2 / 43

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 3
Analysis label: 0.05 ppb
Analysis started at: 10/28/2015 1:51:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.119 ppb	0.003 ppb	0.091 ppb	0.240 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 4
Analysis label: 0.1 ppb
Analysis started at: 10/28/2015 2:04:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.125 ppb	0.068 ppb	0.022 ppb	0.004 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 5
Analysis label: 1 ppb
Analysis started at: 10/28/2015 2:17:14 AM
User name: WTAMU-PC\Jab2

Category	As V	MMA	As III	DMA
Concentration	0.905 ppb	1.043 ppb	0.898 ppb	0.938 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 6
Analysis label: 5 ppb
Analysis started at: 10/28/2015 2:30:12 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	4.977 ppb	5.079 ppb	5.257 ppb	4.923 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 7
Analysis label: 10 ppb
Analysis started at: 10/28/2015 2:43:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	10.021 ppb	9.957 ppb	9.882 ppb	10.045 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 8
Analysis label: L-2 (1) Meat
Analysis started at: 10/28/2015 2:56:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	597.412 ppb	-9.203 ppb	-44.419 ppb	306.218 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 9
Analysis label: L-2 (1) HP
Analysis started at: 10/28/2015 3:09:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	102.363 ppb	0.000 ppb	-15.365 ppb	2,936.592 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 10
Analysis label: L-2 (1) Shell
Analysis started at: 10/28/2015 3:22:12 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	35.274 ppb	0.000 ppb	0.000 ppb	340.300 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 11
Analysis label: L-2 (2) Meat
Analysis started at: 10/28/2015 3:35:10 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	19.900 ppb	0.000 ppb	0.000 ppb	402.050 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 12
Analysis label: L-2 (2) HP
Analysis started at: 10/28/2015 3:48:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	20.582 ppb	0.000 ppb	41.968 ppb	1,072.814 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 13
Analysis label: L-2 (2) Shell
Analysis started at: 10/28/2015 4:01:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	7.676 ppb	0.000 ppb	0.000 ppb	389.501 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 14
Analysis label: L-2 (3) Meat
Analysis started at: 10/28/2015 4:14:09 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	15.376 ppb	0.000 ppb	0.000 ppb	89.007 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 15
Analysis label: L-2 (3) HP
Analysis started at: 10/28/2015 4:27:10 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	64.287 ppb	0.000 ppb	0.183 ppb	700.448 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 16
Analysis label: L-2 (3) Shell
Analysis started at: 10/28/2015 4:40:10 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	12.137 ppb	0.000 ppb	-38.946 ppb	220.376 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 17
Analysis label: S-2 (1) Meat
Analysis started at: 10/28/2015 4:53:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	2.428 ppb	0.000 ppb	0.000 ppb	261.542 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 18
Analysis label: QC Check 5 ppb
Analysis started at: 10/28/2015 5:06:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	5.168 ppb	5.313 ppb	5.303 ppb	5.330 ppb

Concentrations

10/28/2015 3:56:32 PM

QTEGRA

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 19
Analysis label: S-2 (1) HP
Analysis started at: 10/28/2015 5:19:09 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	48.742 ppb	-10.436 ppb	19.192 ppb	731.220 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 20
Analysis label: S-2 (1) Shell
Analysis started at: 10/28/2015 5:32:10 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	12.737 ppb	0.000 ppb	-33.445 ppb	337.927 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 21
Analysis label: S-2 (2) Meat
Analysis started at: 10/28/2015 5:45:10 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1.703 ppb	0.000 ppb	0.000 ppb	211.445 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 22
Analysis label: S-2 (2) HP
Analysis started at: 10/28/2015 5:58:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	15.969 ppb	0.000 ppb	82.590 ppb	670.679 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 23
Analysis label: S-2 (2) Shell
Analysis started at: 10/28/2015 6:11:11 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	2.806 ppb	0.000 ppb	0.000 ppb	170.180 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 24
Analysis label: S-2 (3) Meat
Analysis started at: 10/28/2015 6:24:12 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1.674 ppb	0.000 ppb	0.000 ppb	288.222 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 25
Analysis label: S-2 (3) HP
Analysis started at: 10/28/2015 6:37:12 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1.516 ppb	3.479 ppb	237.381 ppb	575.680 ppb

Concentrations

10/28/2015 3:56:32 PM

QTEGRA

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 26
Analysis label: S-2 (3) Shell
Analysis started at: 10/28/2015 6:50:13 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	6.561 ppb	0.000 ppb	-100.137 ppb	222.607 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 27
Analysis label: S-6 (1) Meat
Analysis started at: 10/28/2015 7:03:13 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	2.205 ppb	0.000 ppb	0.000 ppb	87.366 ppb

Concentrations

10/28/2015 3:56:32 PM

QTEGRA

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 28
Analysis label: S-6 (1) HP
Analysis started at: 10/28/2015 7:16:13 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.000 ppb	0.000 ppb	64.851 ppb	282.810 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 29
Analysis label: QC Check 5 ppb
Analysis started at: 10/28/2015 7:29:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	4.299 ppb	5.003 ppb	5.760 ppb	5.677 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 30
Analysis label: S-6 (1) Shell
Analysis started at: 10/28/2015 7:42:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	43.629 ppb	0.000 ppb	-20.833 ppb	0.577 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 31
Analysis label: S-6 (2) Meat
Analysis started at: 10/28/2015 7:55:15 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1.191 ppb	0.000 ppb	0.000 ppb	125.410 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 32
Analysis label: S-6 (2) HP
Analysis started at: 10/28/2015 8:08:15 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	5.032 ppb	0.000 ppb	84.662 ppb	640.962 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 33
Analysis label: S-6 (2) Shell
Analysis started at: 10/28/2015 8:21:16 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	12.371 ppb	0.000 ppb	0.000 ppb	66.157 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 34
Analysis label: S-6 (3) Meat
Analysis started at: 10/28/2015 8:34:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.000 ppb	0.000 ppb	0.000 ppb	529.646 ppb

Concentrations

10/28/2015 3:56:32 PM

QTEGRA

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 35
Analysis label: S-6 (3) HP
Analysis started at: 10/28/2015 8:47:15 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	0.770 ppb	-2.528 ppb	99.936 ppb	657.435 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 36
Analysis label: S-6 (3) Shell
Analysis started at: 10/28/2015 9:00:15 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	44.863 ppb	0.000 ppb	-45.495 ppb	421.552 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 37
Analysis label: L-2 (SS)
Analysis started at: 10/28/2015 9:13:16 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1,117.984 ppb	0.000 ppb	59.570 ppb	108.183 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 38
Analysis label: S-2 (SS)
Analysis started at: 10/28/2015 9:26:16 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	2,058.224 ppb	0.000 ppb	58.776 ppb	106.955 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 39
Analysis label: S-6 (SS)
Analysis started at: 10/28/2015 9:39:15 AM
User name: WTAMU-PC\Jab2

Category	As V	MMA	As III	DMA
Concentration	2,590.369 ppb	0.000 ppb	76.530 ppb	134.871 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 40
Analysis label: DORM
Analysis started at: 10/28/2015 9:52:15 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	521.914 ppb	0.000 ppb	0.000 ppb	23,241.896 ppb

Concentrations

10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	_Application Data\Workspace\LabBooks

Analysis index: 41
Analysis label: TORT
Analysis started at: 10/28/2015 10:05:16 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	1,238.139 ppb	0.000 ppb	0.000 ppb	252,320.403 ppb

Concentrations

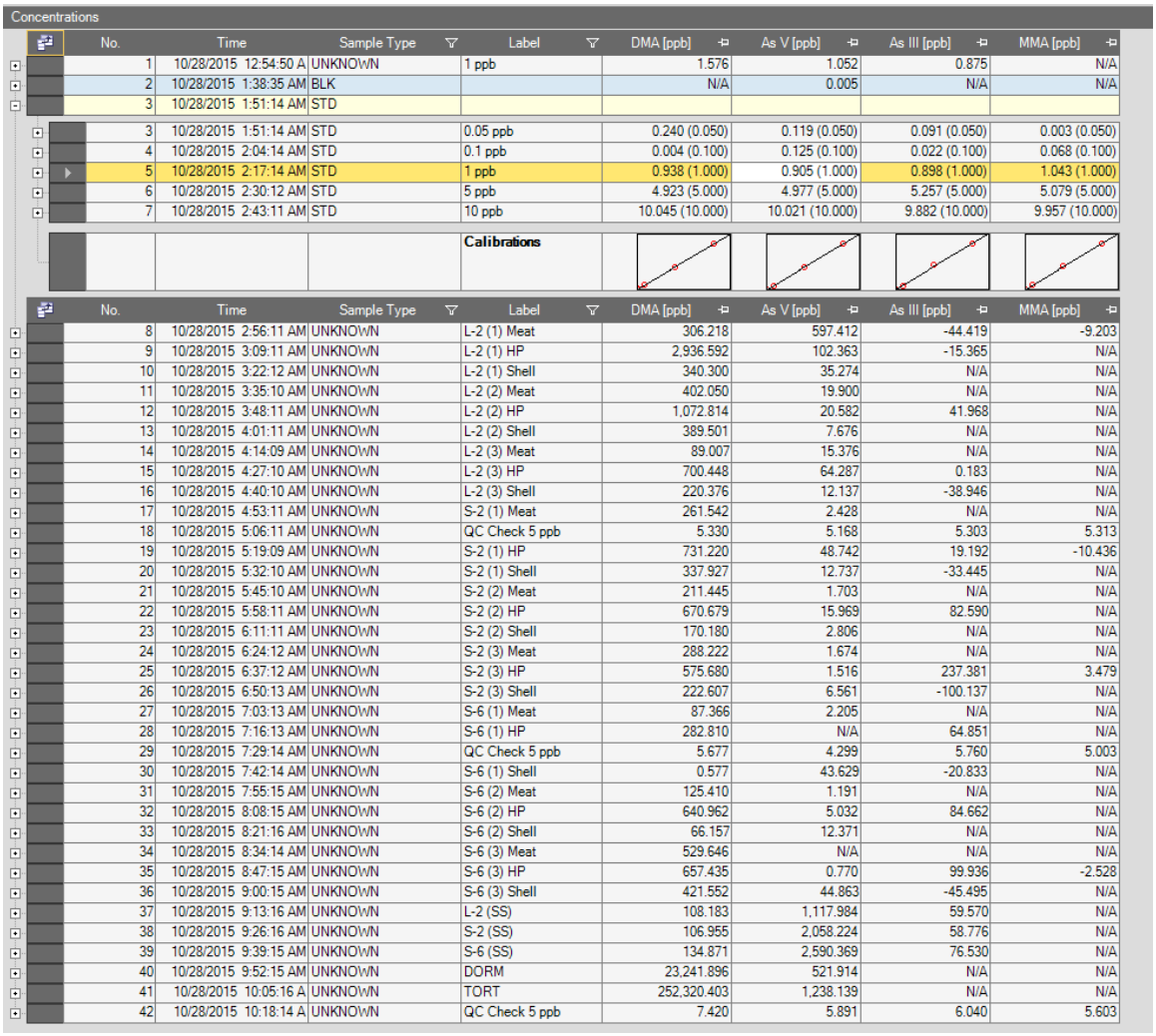
10/28/2015 3:56:32 PM

Instrument Name:	Serial Number:
iCAP Q	Undefined

Labbook:	Labbook Path:
Speciation As 10 27 2015 Modified Gradient3.imexp	Application Data\Workspace\LabBooks

Analysis index: 42
Analysis label: QC Check 5 ppb
Analysis started at: 10/28/2015 10:18:14 AM
User name: WTAMU-PC\lab2

Category	As V	MMA	As III	DMA
Concentration	5.891 ppb	5.603 ppb	6.040 ppb	7.420 ppb

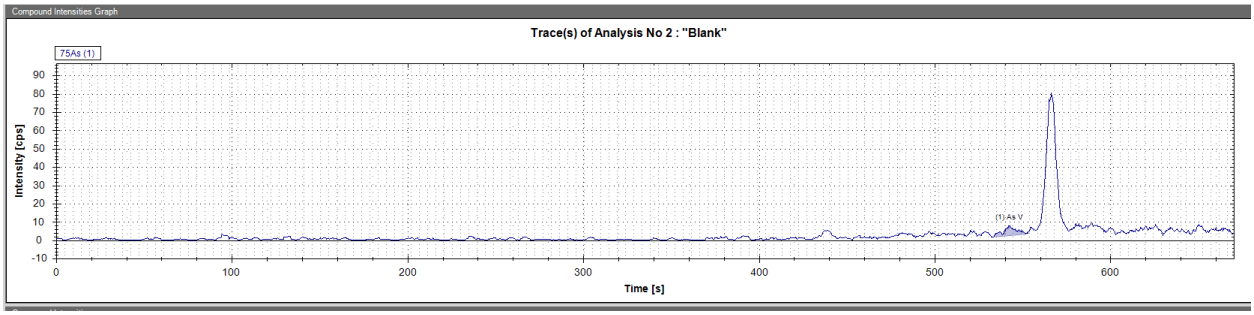


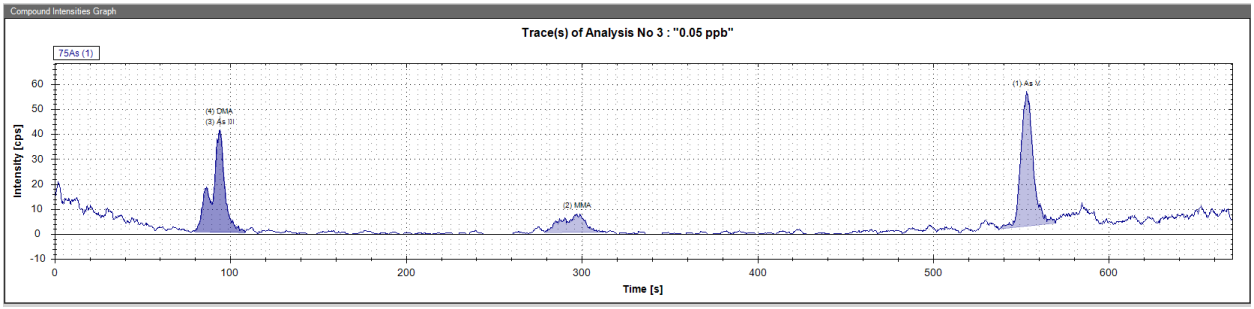
Concentrations

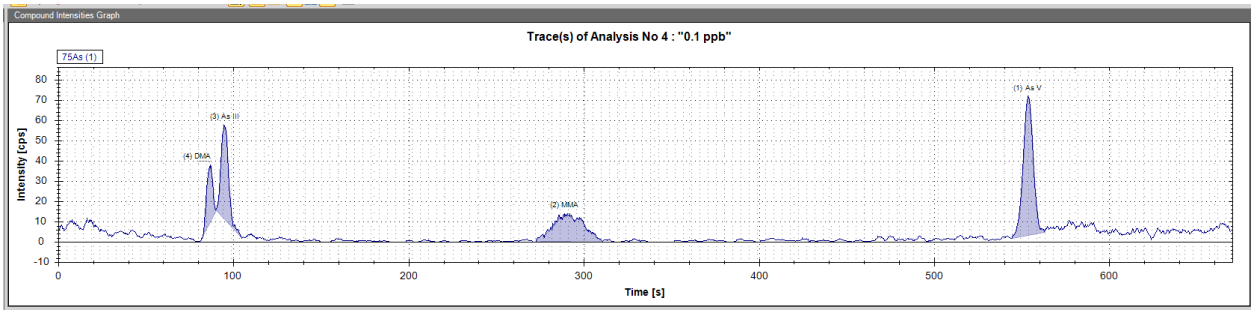
No.	Time	Sample Type	Label	DMA [ppb]	As V [ppb]	As III [ppb]	MMA [ppb]
1	10/28/2015 12:54:50 A	UNKNOWN	1 ppb	1.576	1.052	0.875	N/A
2	10/28/2015 1:38:35 AM	BLK		N/A	0.005	N/A	N/A
3	10/28/2015 1:51:14 AM	STD					
3	10/28/2015 1:51:14 AM	STD	0.05 ppb	0.240 (0.050)	0.119 (0.050)	0.091 (0.050)	0.003 (0.050)
4	10/28/2015 2:04:14 AM	STD	0.1 ppb	0.004 (0.100)	0.125 (0.100)	0.022 (0.100)	0.068 (0.100)
5	10/28/2015 2:17:14 AM	STD	1 ppb	0.938 (1.000)	0.905 (1.000)	0.898 (1.000)	1.043 (1.000)
6	10/28/2015 2:30:12 AM	STD	5 ppb	4.923 (5.000)	4.977 (5.000)	5.257 (5.000)	5.079 (5.000)
7	10/28/2015 2:43:11 AM	STD	10 ppb	10.045 (10.000)	10.021 (10.000)	9.882 (10.000)	9.957 (10.000)
			Calibrations				
8	10/28/2015 2:56:11 AM	UNKNOWN	L-2 (1) Meat	306.218	597.412	-44.419	-9.203
9	10/28/2015 3:09:11 AM	UNKNOWN	L-2 (1) HP	2,936.592	102.363	-15.365	N/A
10	10/28/2015 3:22:12 AM	UNKNOWN	L-2 (1) Shell	340.300	35.274	N/A	N/A
11	10/28/2015 3:35:10 AM	UNKNOWN	L-2 (2) Meat	402.050	19.900	N/A	N/A
12	10/28/2015 3:48:11 AM	UNKNOWN	L-2 (2) HP	1,072.814	20.582	41.968	N/A
13	10/28/2015 4:01:11 AM	UNKNOWN	L-2 (2) Shell	389.501	7.676	N/A	N/A
14	10/28/2015 4:14:09 AM	UNKNOWN	L-2 (3) Meat	89.007	15.376	N/A	N/A
15	10/28/2015 4:27:10 AM	UNKNOWN	L-2 (3) HP	700.448	64.287	0.183	N/A
16	10/28/2015 4:40:10 AM	UNKNOWN	L-2 (3) Shell	220.376	12.137	-38.946	N/A
17	10/28/2015 4:53:11 AM	UNKNOWN	S-2 (1) Meat	261.542	2.428	N/A	N/A
18	10/28/2015 5:06:11 AM	UNKNOWN	QC Check 5 ppb	5.330	5.168	5.303	5.313
19	10/28/2015 5:19:09 AM	UNKNOWN	S-2 (1) HP	731.220	48.742	19.192	-10.436
20	10/28/2015 5:32:10 AM	UNKNOWN	S-2 (1) Shell	337.927	12.737	-33.445	N/A
21	10/28/2015 5:45:10 AM	UNKNOWN	S-2 (2) Meat	211.445	1.703	N/A	N/A
22	10/28/2015 5:58:11 AM	UNKNOWN	S-2 (2) HP	670.679	15.969	82.590	N/A
23	10/28/2015 6:11:11 AM	UNKNOWN	S-2 (2) Shell	170.180	2.806	N/A	N/A
24	10/28/2015 6:24:12 AM	UNKNOWN	S-2 (3) Meat	288.222	1.674	N/A	N/A
25	10/28/2015 6:37:12 AM	UNKNOWN	S-2 (3) HP	575.680	1.516	237.381	3.479
26	10/28/2015 6:50:13 AM	UNKNOWN	S-2 (3) Shell	222.607	6.561	-100.137	N/A
27	10/28/2015 7:03:13 AM	UNKNOWN	S-6 (1) Meat	87.366	2.205	N/A	N/A
28	10/28/2015 7:16:13 AM	UNKNOWN	S-6 (1) HP	282.810	N/A	64.851	N/A
29	10/28/2015 7:29:14 AM	UNKNOWN	QC Check 5 ppb	5.677	4.299	5.760	5.003
30	10/28/2015 7:42:14 AM	UNKNOWN	S-6 (1) Shell	0.577	43.629	-20.833	N/A
31	10/28/2015 7:55:15 AM	UNKNOWN	S-6 (2) Meat	125.410	1.191	N/A	N/A
32	10/28/2015 8:08:15 AM	UNKNOWN	S-6 (2) HP	640.962	5.032	84.662	N/A
33	10/28/2015 8:21:16 AM	UNKNOWN	S-6 (2) Shell	66.157	12.371	N/A	N/A
34	10/28/2015 8:34:14 AM	UNKNOWN	S-6 (3) Meat	529.646	N/A	N/A	N/A
35	10/28/2015 8:47:15 AM	UNKNOWN	S-6 (3) HP	657.435	0.770	99.936	-2.528
36	10/28/2015 9:00:15 AM	UNKNOWN	S-6 (3) Shell	421.552	44.863	-45.495	N/A
37	10/28/2015 9:13:16 AM	UNKNOWN	L-2 (SS)	108.183	1,117.984	59.570	N/A
38	10/28/2015 9:26:16 AM	UNKNOWN	S-2 (SS)	106.955	2,058.224	58.776	N/A
39	10/28/2015 9:39:15 AM	UNKNOWN	S-6 (SS)	134.871	2,590.369	76.530	N/A
40	10/28/2015 9:52:15 AM	UNKNOWN	DORM	23,241.896	521.914	N/A	N/A
41	10/28/2015 10:05:16 A	UNKNOWN	TORT	252,320.403	1,238.139	N/A	N/A
42	10/28/2015 10:18:14 A	UNKNOWN	QC Check 5 ppb	7.420	5.891	6.040	5.603

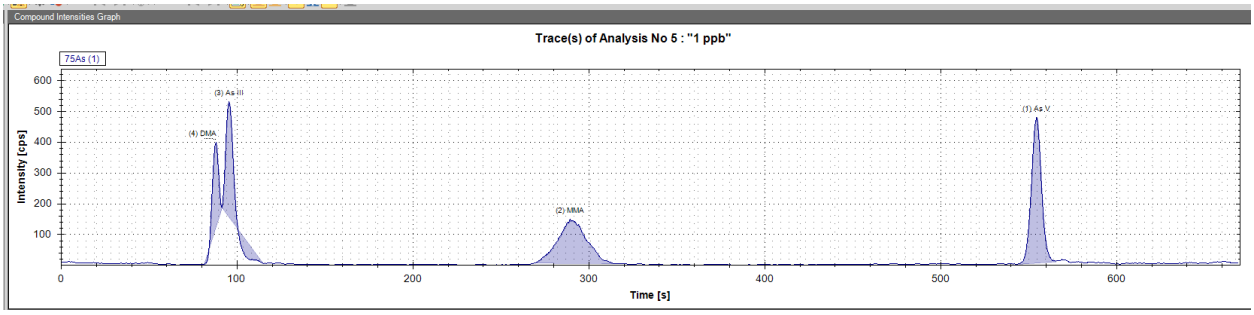
Figure 40: Screen capture of Thermo Scientific™ Qtegra™ ISDS calculated sample concentrations, 10–28–2015.

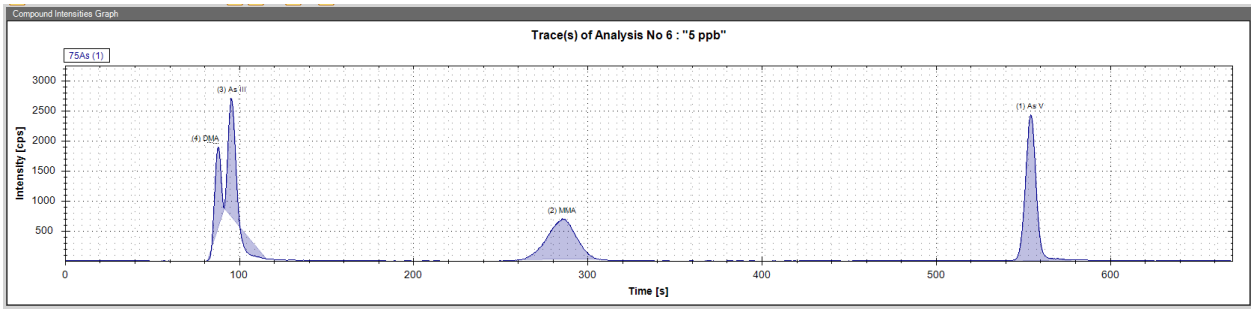
Report 8: Calibration, L–2, S–2, and S–6 chromatograms, 10–28–2015, 11 pp. Chromatography and peak detection was performed using the Dionex™ Chromeleon® Xpress chromatography plug–in through Qtegra™ ISDS and the Chromatography Data System (CDS) Cobra™ peak detection algorithm.

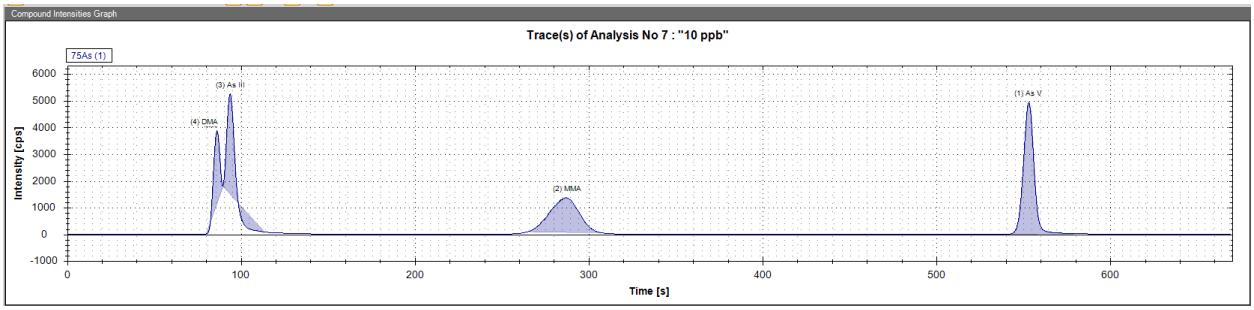


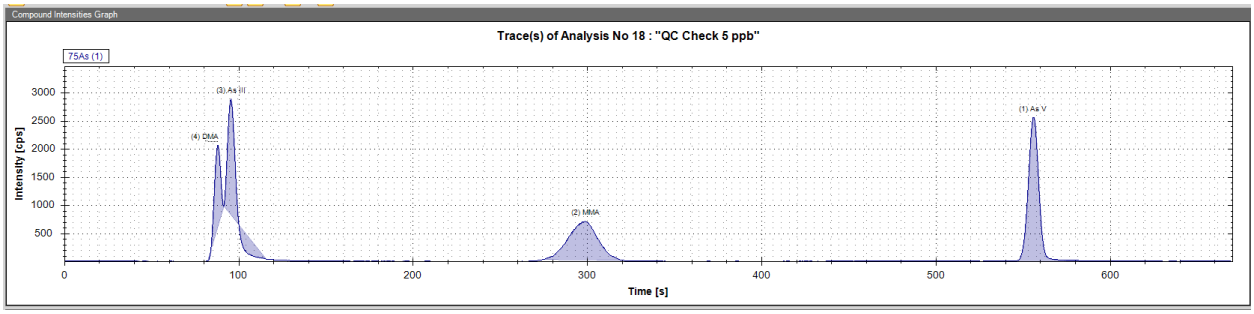


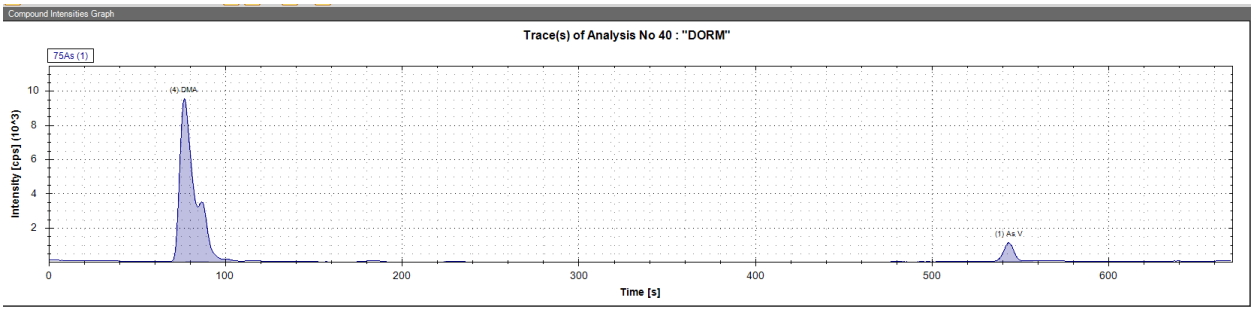


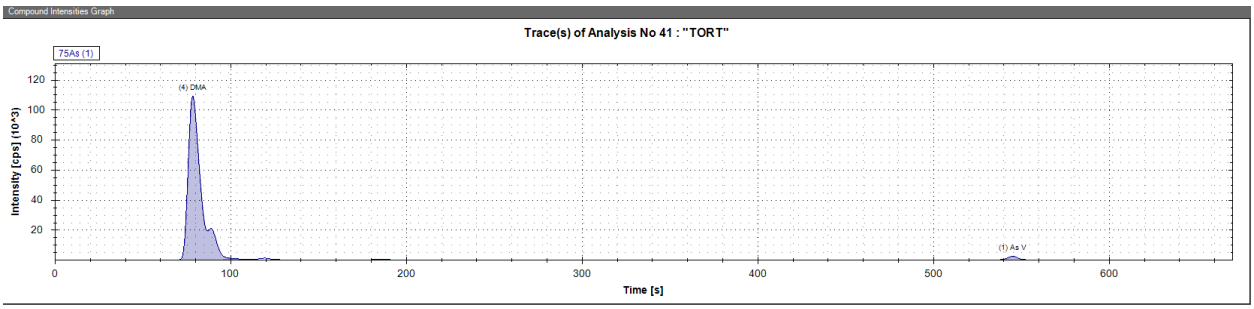


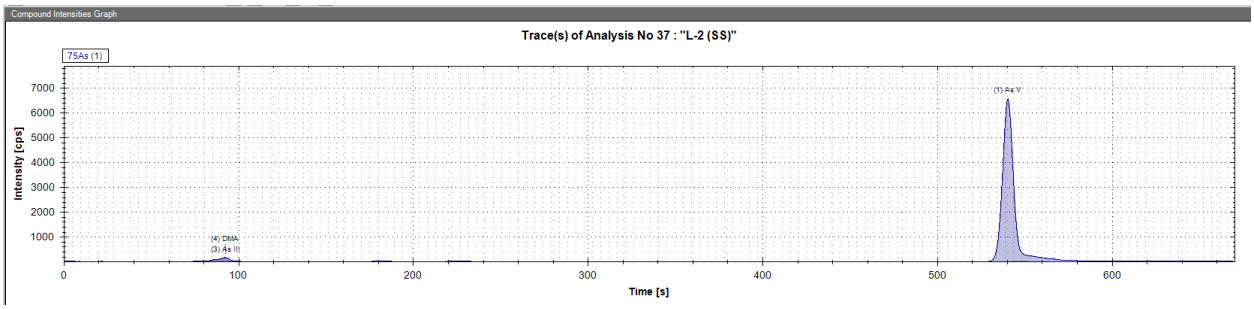

Compound Intensities Graph
Trace(s) of Analysis No 6 : "5 ppb"
75As (1)
(3) As III
(4) DMA
(2) MMA
(1) As V
Intensity [cps]
Time [s]

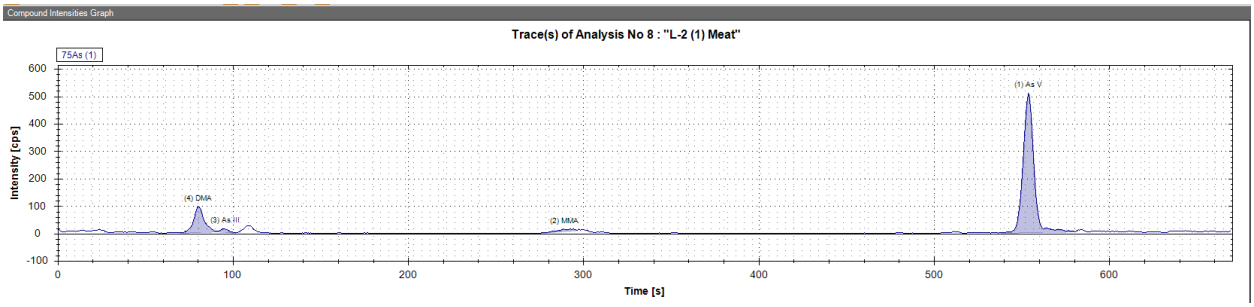

Compound Intensities Graph
Trace(s) of Analysis No 7 : "10 ppb"
75As (1)
(3) As III
(4) DMA
(2) MMA
(1) As V
Intensity [cps]
Time [s]

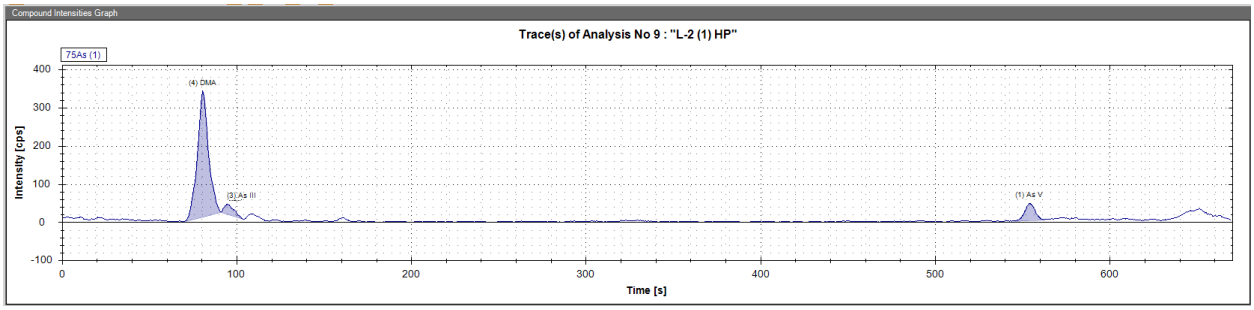

Compound Intensities Graph
Trace(s) of Analysis No 18 : "QC Check 5 ppb"
75As (1)
(3) As III
(4) DMA
(2) MMA
(1) As V
Intensity [cps]
Time [s]

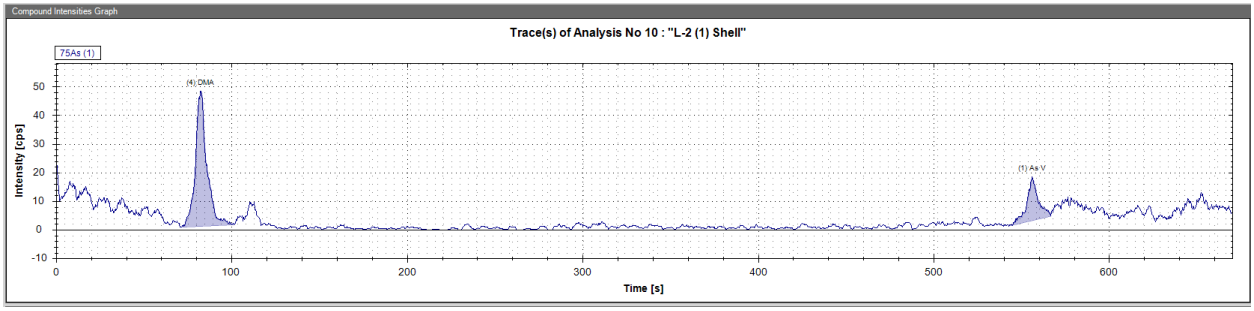

Compound Intensities Graph
Trace(s) of Analysis No 40 : "DORM"
75As (1)
(4) DMA
(1) As V
Intensity [cps] (10^3)
Time [s]

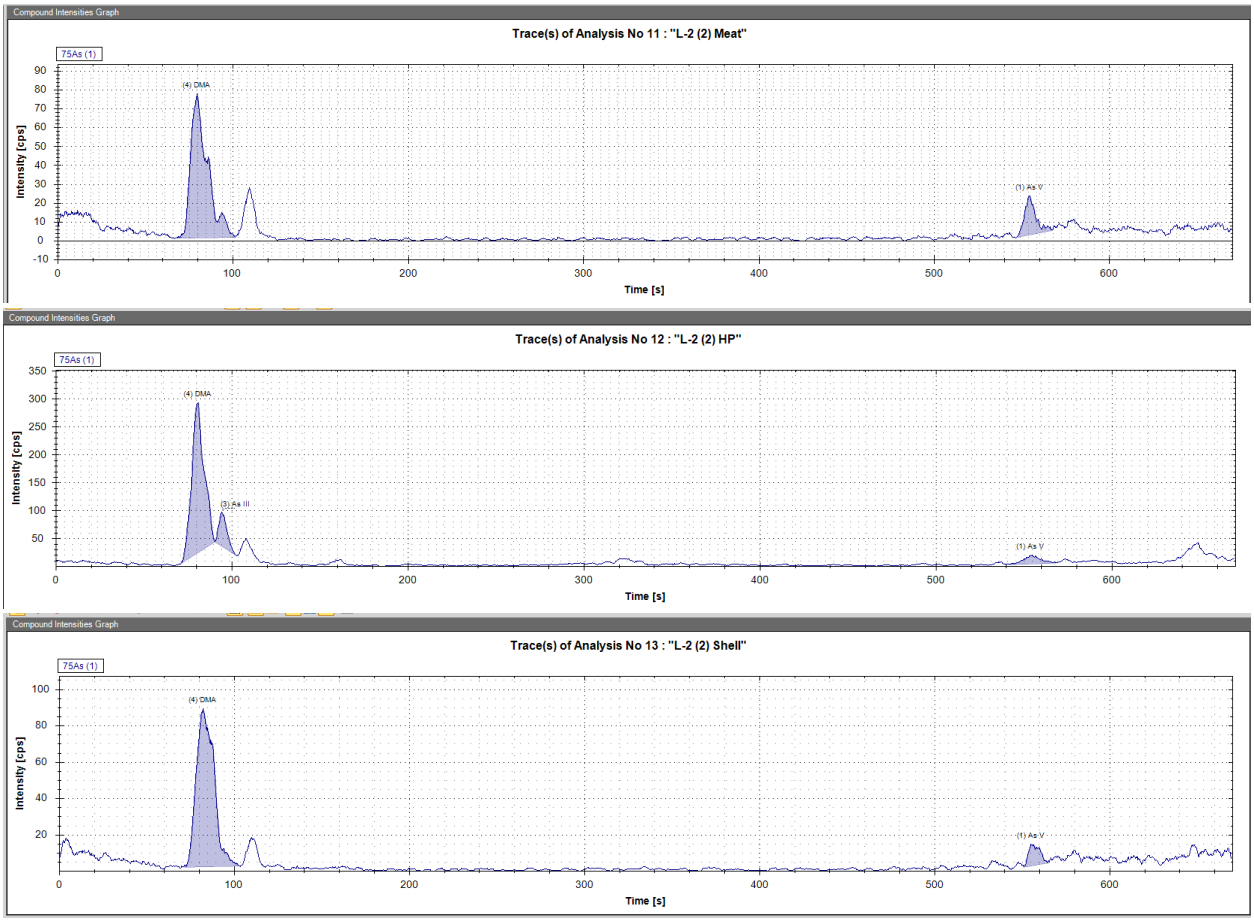

Compound Intensities Graph
Trace(s) of Analysis No 41 : "TORT"
75As (1)
(4) DMA
(1) As V
Intensity [cps] (10^3)
Time [s]

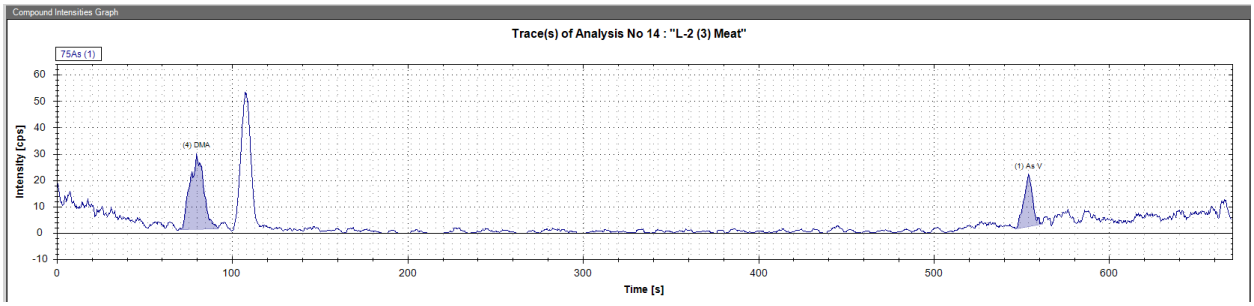

Compound Intensities Graph
Trace(s) of Analysis No 37 : "L-2 (SS)"
75As (1)
Intensity [cps]
Time [s]
(4) DMA
(3) As III
(1) As V

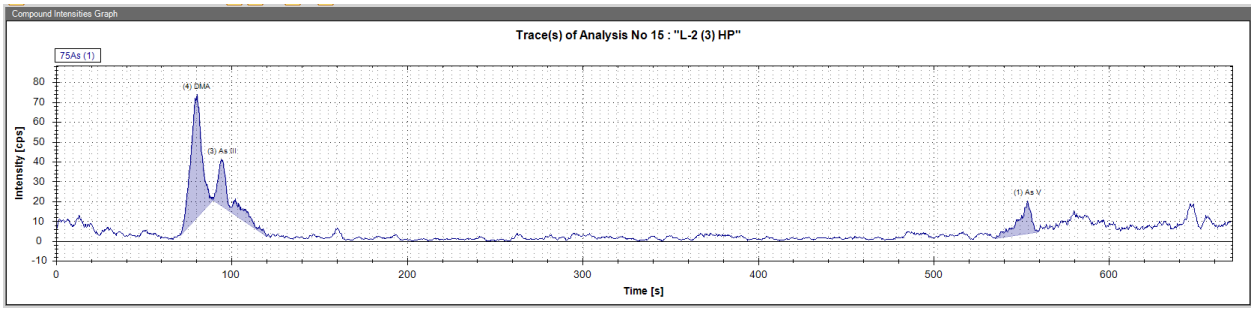

Compound Intensities Graph
Trace(s) of Analysis No 8 : "L-2 (1) Meat"
75As (1)
Intensity [cps]
Time [s]
(4) DMA
(3) As III
(2) MMA
(1) As V

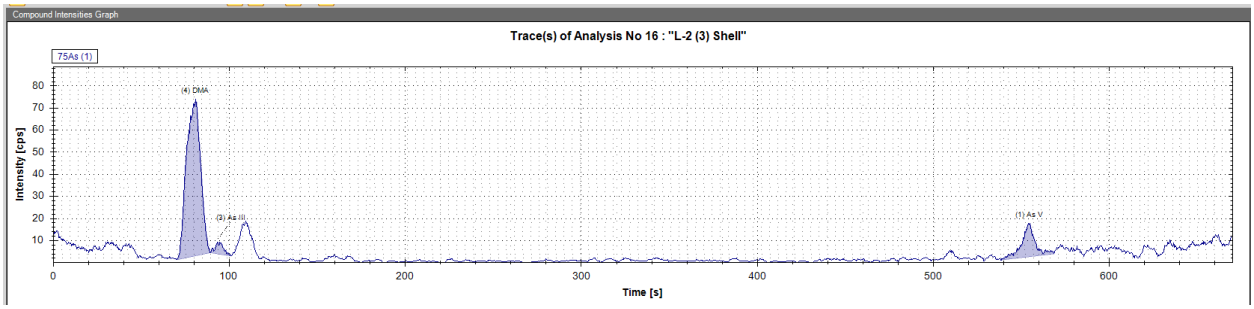

Compound Intensities Graph
Trace(s) of Analysis No 9 : "L-2 (1) HP"
75As (1)
Intensity [cps]
Time [s]
(4) DMA
(3) As III
(1) As V

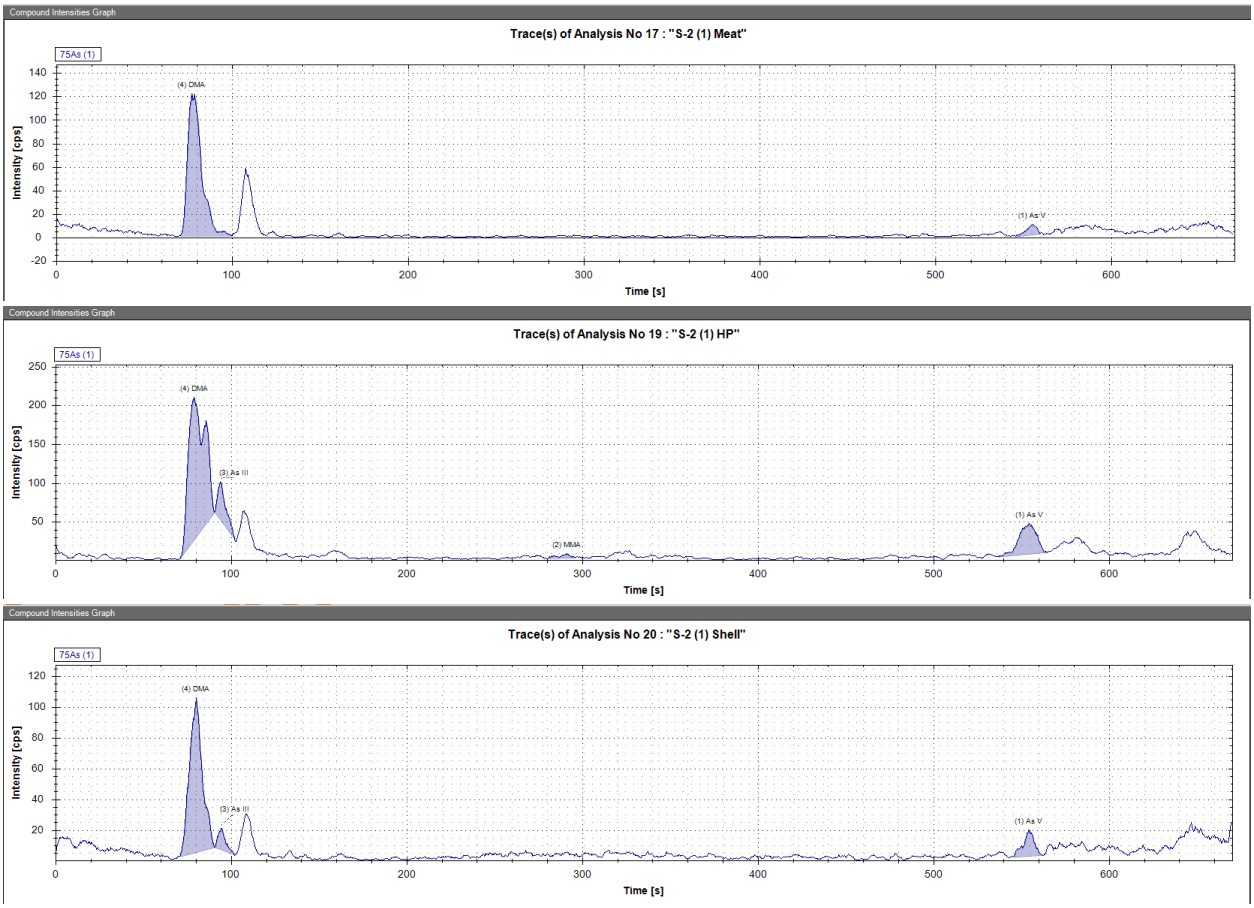

Compound Intensities Graph
Trace(s) of Analysis No 10 : "L-2 (1) Shell"
75As (1)
Intensity [cps]
Time [s]
(4) DMA
(1) As V

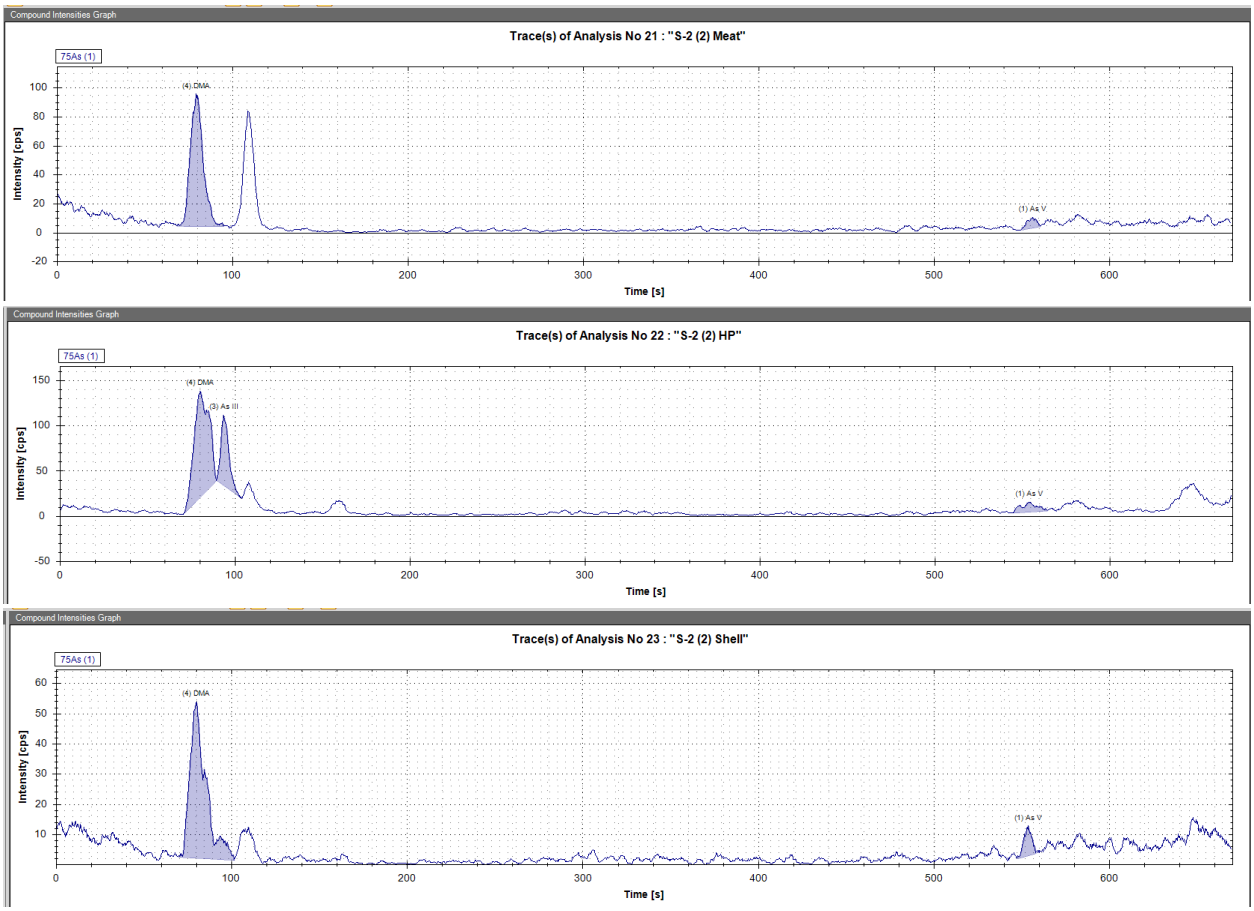

Compound Intensities Graph
Trace(s) of Analysis No 11 : "L-2 (2) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 12 : "L-2 (2) HP"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 13 : "L-2 (2) Shell"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]

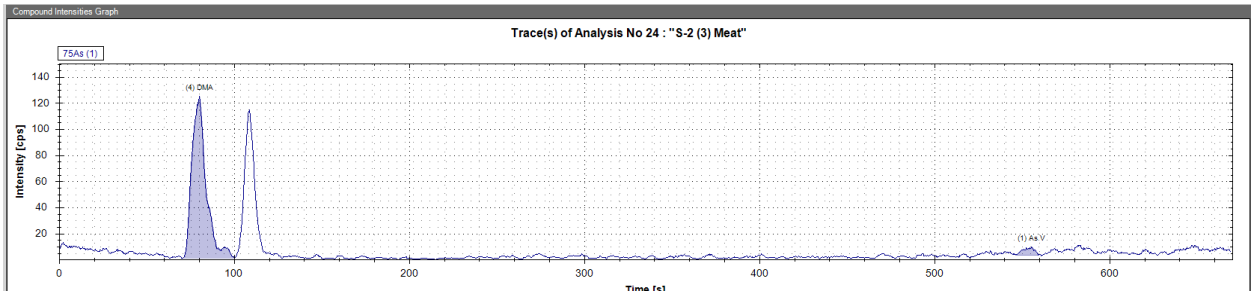

Compound Intensities Graph
Trace(s) of Analysis No 14 : "L-2 (3) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]

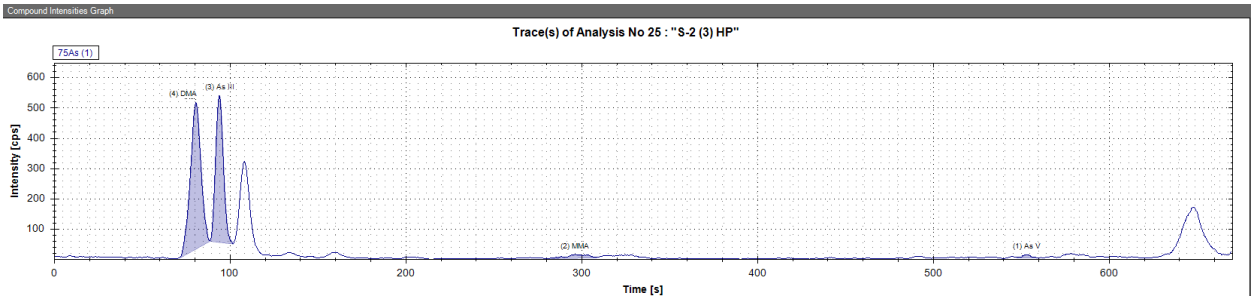

Compound Intensities Graph
Trace(s) of Analysis No 15 : "L-2 (3) HP"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

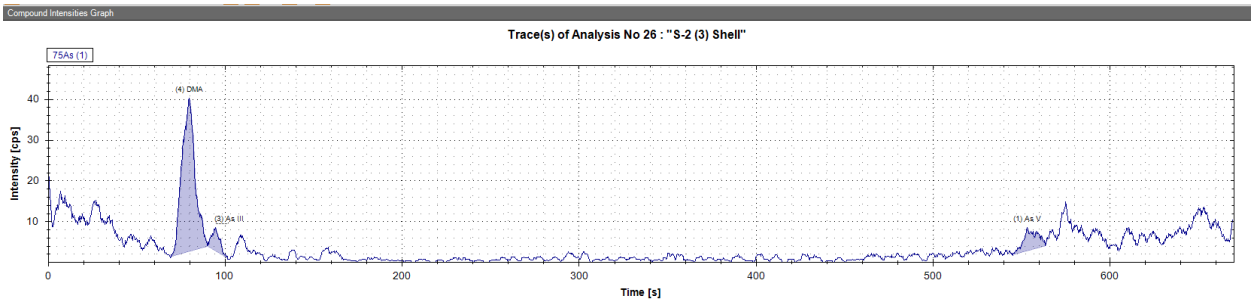

Compound Intensities Graph
Trace(s) of Analysis No 16 : "L-2 (3) Shell"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

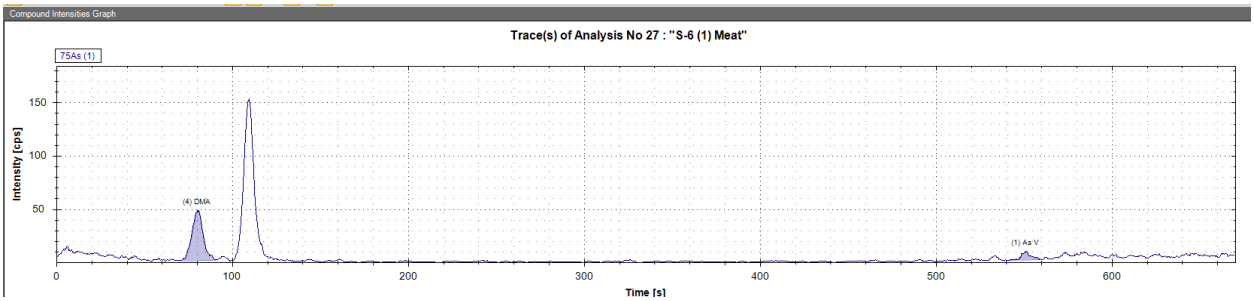

Compound Intensities Graph
Trace(s) of Analysis No 17 : "S-2 (1) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 19 : "S-2 (1) HP"
75As (1)
(4) DMA
(3) As III
(2) MMA
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 20 : "S-2 (1) Shell"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

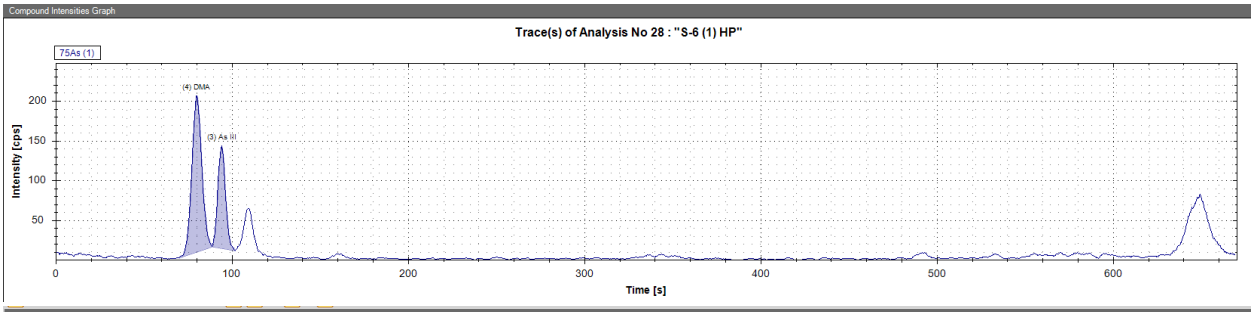

Compound Intensities Graph
Trace(s) of Analysis No 21 : "S-2 (2) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 22 : "S-2 (2) HP"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 23 : "S-2 (2) Shell"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]

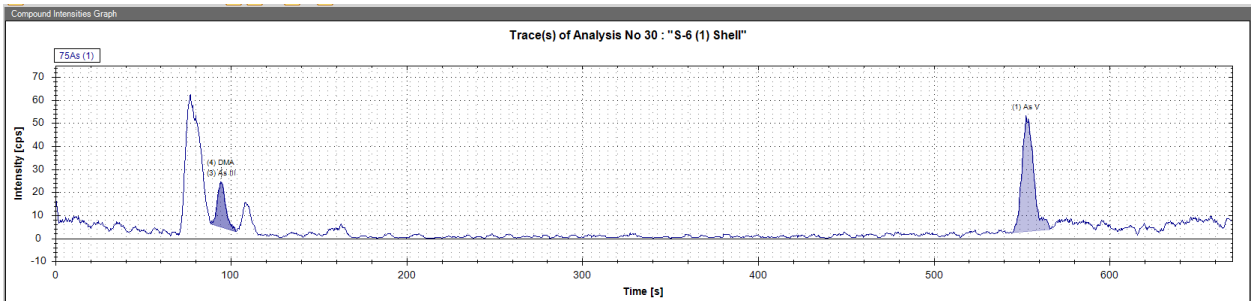

Compound Intensities Graph
Trace(s) of Analysis No 24 : "S-2 (3) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]

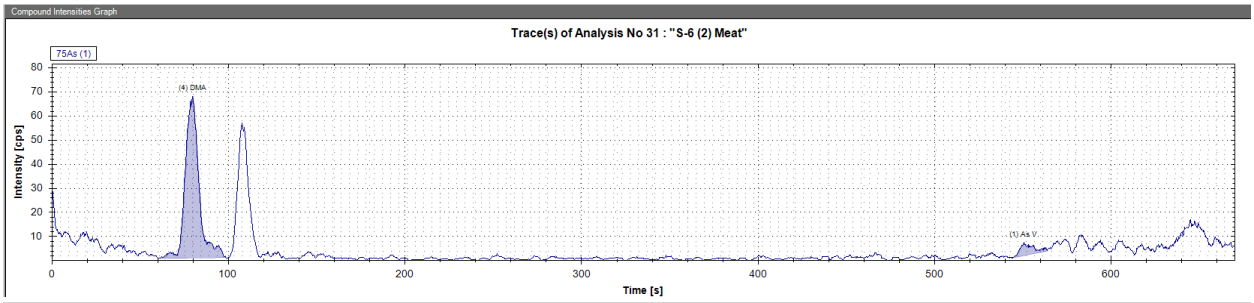

Compound Intensities Graph
Trace(s) of Analysis No 25 : "S-2 (3) HP"
75As (1)
(4) DMA
(3) As III
(2) MMA
(1) As V
Intensity [cps]
Time [s]

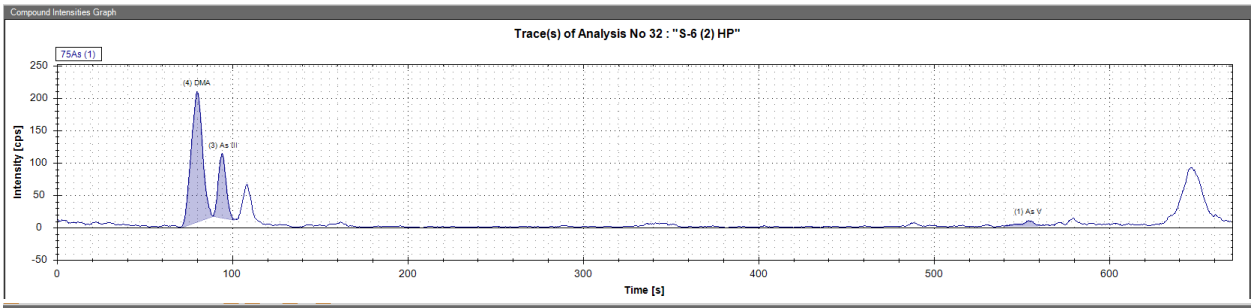

Compound Intensities Graph
Trace(s) of Analysis No 26 : "S-2 (3) Shell"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

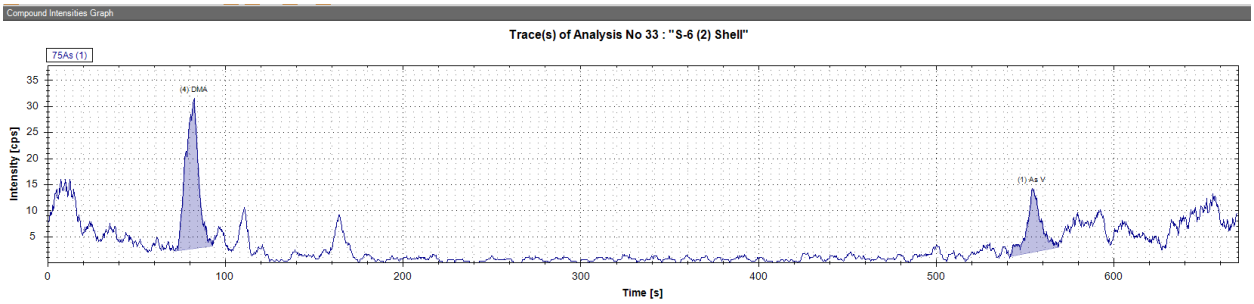

Compound Intensities Graph
Trace(s) of Analysis No 27 : "S-6 (1) Meat"
75As (1)
Intensity [cps]
(4) DMA
(1) As V
Time [s]

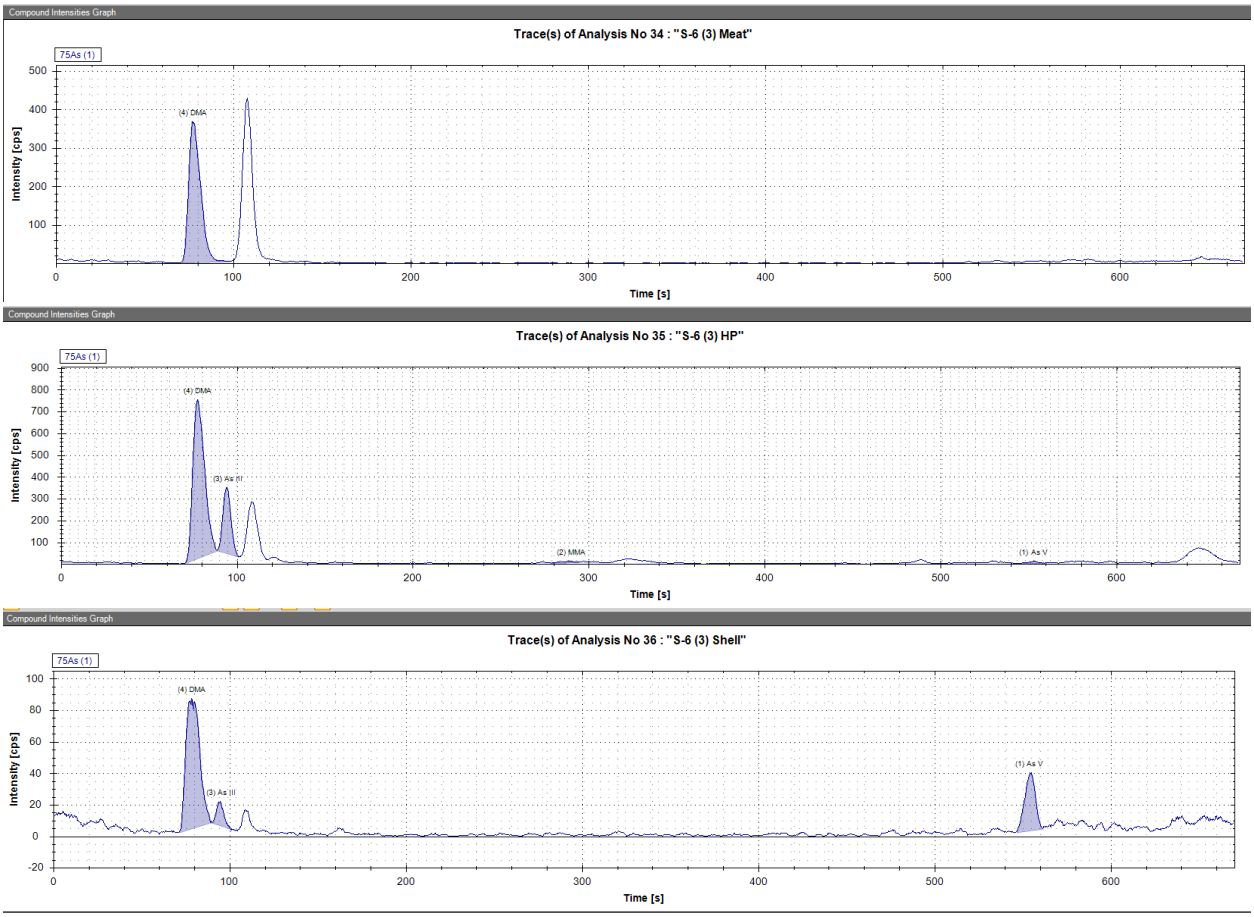

Compound Intensities Graph
Trace(s) of Analysis No 28 : "S-6 (1) HP"
75As (1)
Intensity [cps]
(4) DMA
(3) As III
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 30 : "S-6 (1) Shell"
75As (1)
Intensity [cps]
(4) DMA
(3) As III
(1) As V
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 31 : "S-6 (2) Meat"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 32 : "S-6 (2) HP"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

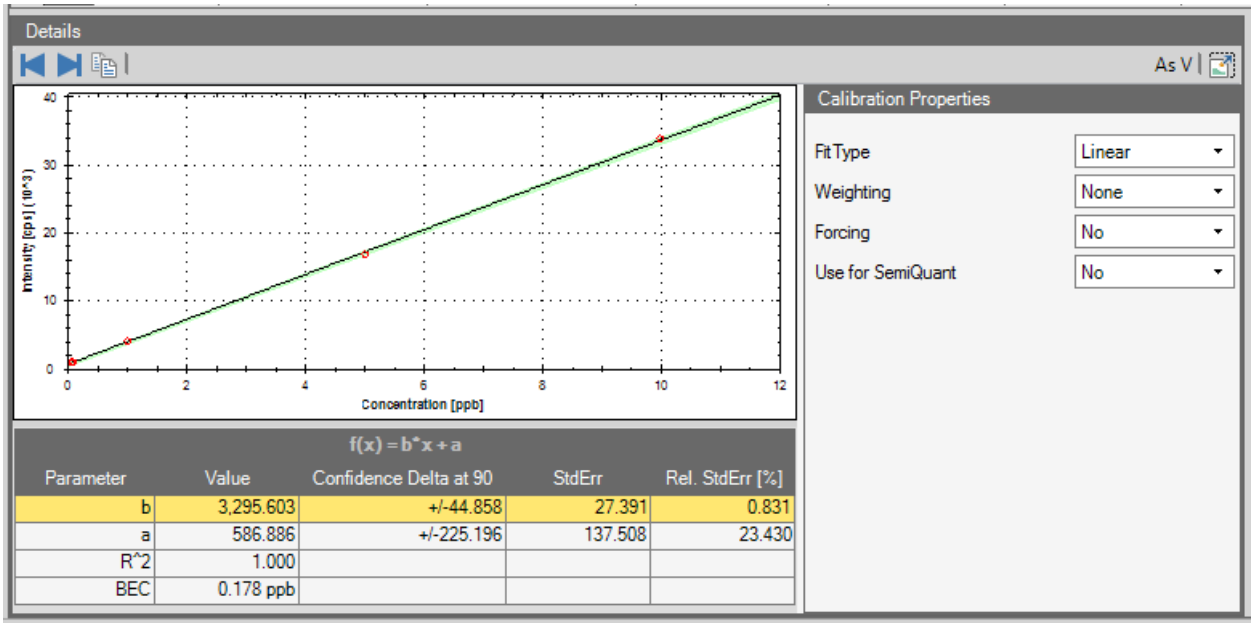

Compound Intensities Graph
Trace(s) of Analysis No 33 : "S-6 (2) Shell"
75As (1)
(4) DMA
(1) As V
Intensity [cps]
Time [s]

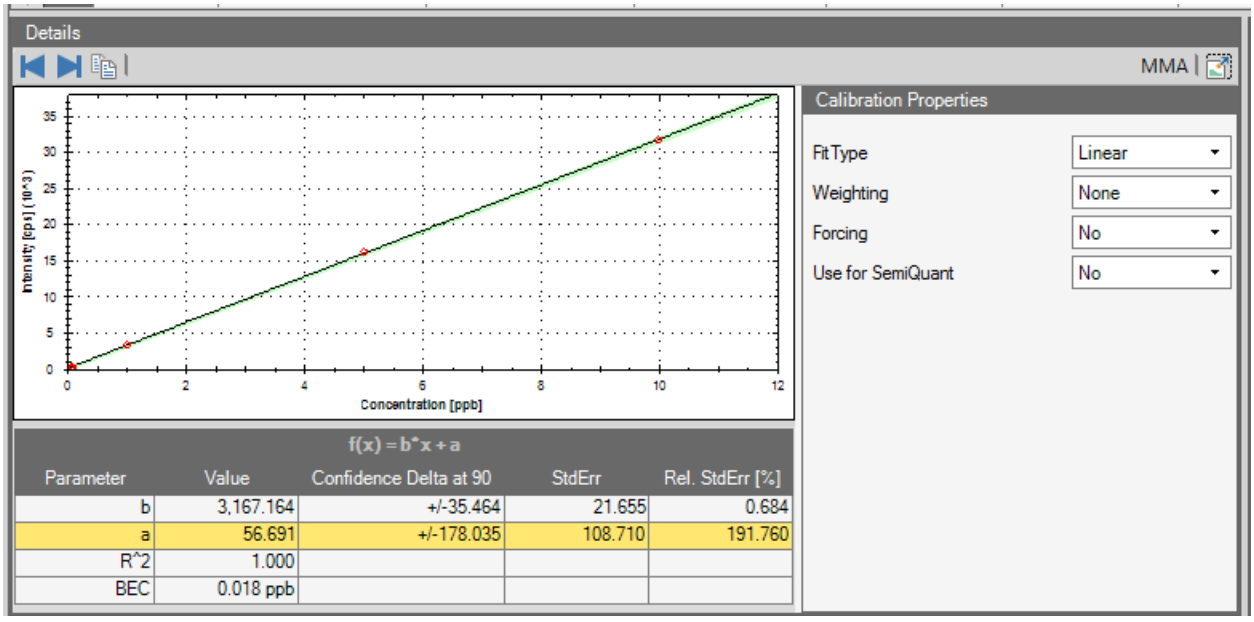

Compound Intensities Graph
Trace(s) of Analysis No 34 : "S-6 (3) Meat"
75As (1)
(4) DMA
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 35 : "S-6 (3) HP"
75As (1)
(4) DMA
(3) As III
(2) MMA
(1) As V
Intensity [cps]
Time [s]
Compound Intensities Graph
Trace(s) of Analysis No 36 : "S-6 (3) Shell"
75As (1)
(4) DMA
(3) As III
(1) As V
Intensity [cps]
Time [s]

Report 9: Calibration, L–1, L–3, and S–1 chromatograms, 11–11–2015, 7 pp. Chromatography and peak detection was performed using the Dionex™ Chromeleon® Xpress chromatography plug–in through Qtegra™ ISDS and the Chromatography Data System (CDS) Cobra™ peak detection algorithm.

Concentrations

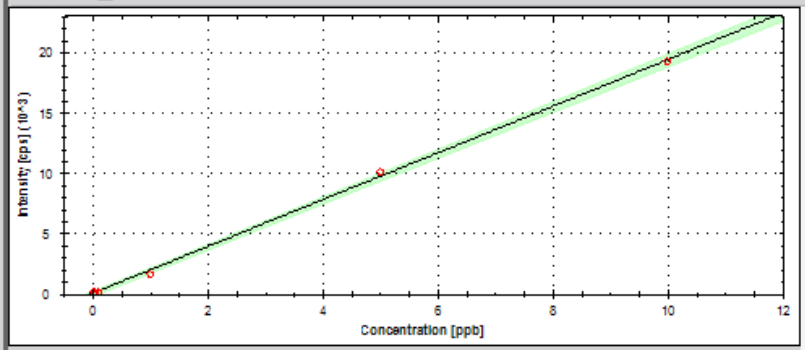
No.	Time	Sample Type	Label	As V [ppb]	MMA [ppb]	As III [ppb]	DMA [ppb]
1	11/12/2015 6:09:04 PM	UNKNOWN	Blank	N/A	N/A	N/A	N/A
2	11/12/2015 6:24:06 PM	UNKNOWN	1 ppb	N/A	N/A	N/A	N/A
3	11/12/2015 6:34:01 PM	UNKNOWN	1 ppb	N/A	N/A	N/A	N/A
4	11/12/2015 6:48:57 PM	BLK		N/A	N/A	0.044	0.043
5	11/12/2015 7:04:16 PM	STD					
10	11/12/2015 8:20:47 PM	BLK		0.000	0.000	0.000	0.000
11	11/12/2015 8:36:05 PM	UNKNOWN	L-1 (1) Meat	N/A	N/A	N/A	N/A
12	11/12/2015 8:51:23 PM	UNKNOWN	L-1 (1) HP	N/A	N/A	67.237	146.214
13	11/12/2015 9:06:43 PM	UNKNOWN	L-1 (1) Shell	N/A	N/A	N/A	N/A
14	11/12/2015 9:22:03 PM	UNKNOWN	L-1 (2) Meat	N/A	N/A	N/A	N/A
15	11/12/2015 9:37:21 PM	UNKNOWN	L-1 (2) HP	N/A	N/A	N/A	107.521
16	11/12/2015 9:52:41 PM	UNKNOWN	L-1 (2) Shell	N/A	N/A	N/A	N/A
17	11/12/2015 10:08:00 P	UNKNOWN	L-1 (3) Meat	N/A	N/A	N/A	N/A
18	11/12/2015 10:23:19 P	UNKNOWN	L-1 (3) HP	N/A	10.259	5.856	42.887
19	11/12/2015 10:38:38 P	UNKNOWN	L-1 (3) Shell	N/A	N/A	N/A	N/A
20	11/12/2015 10:53:57 P	STD					
21	11/12/2015 11:09:15 P	BLK		0.000	N/A	0.000	0.000
22	11/12/2015 11:24:34 P	UNKNOWN	L-3 (1) Meat	N/A	N/A	N/A	373.491
23	11/12/2015 11:39:52 P	UNKNOWN	L-3 (1) HP	N/A	N/A	-41.799	N/A
24	11/12/2015 11:55:09 P	UNKNOWN	L-3 (1) Shell	N/A	N/A	N/A	131.467
25	11/13/2015 12:10:28 A	UNKNOWN	L-3 (2) Meat	N/A	N/A	N/A	N/A
26	11/13/2015 12:25:47 A	UNKNOWN	L-3 (2) HP	N/A	14.091	-15.052	102.164
27	11/13/2015 12:41:05 A	UNKNOWN	L-3 (2) Shell	N/A	N/A	N/A	N/A
28	11/13/2015 12:56:23 A	UNKNOWN	L-3 (3) Meat	N/A	N/A	N/A	157.746
29	11/13/2015 1:11:43 AM	UNKNOWN	L-3 (3) HP	N/A	11.102	9.736	208.991
30	11/13/2015 1:27:02 AM	UNKNOWN	L-3 (3) Shell	N/A	N/A	N/A	-43.840
31	11/13/2015 1:42:22 AM	STD					
32	11/13/2015 1:57:40 AM	BLK		N/A	N/A	N/A	0.000
33	11/13/2015 2:12:59 AM	UNKNOWN	S-1 (1) Meat	N/A	N/A	N/A	14.169
34	11/13/2015 2:28:17 AM	UNKNOWN	S-1 (1) HP	N/A	N/A	32.308	66.544
35	11/13/2015 2:43:35 AM	UNKNOWN	S-1 (1) Shell	N/A	N/A	N/A	N/A
36	11/13/2015 2:58:54 AM	UNKNOWN	S-1 (2) Meat	N/A	N/A	N/A	38.659
37	11/13/2015 9:23:24 AM	UNKNOWN	S-1 (2) HP	N/A	N/A	N/A	N/A
38	11/13/2015 9:35:09 AM	UNKNOWN	S-1 (2) Shell	N/A	N/A	N/A	N/A
39	11/13/2015 2:04:35 PM	BLK		N/A	N/A	N/A	N/A
40	11/13/2015 2:19:33 PM	STD					


Details
As V
Calibration Properties
Fit Type
Linear
Weighting
None
Forcing
No
Use for SemiQuant
No
Intensity [cps] (10^3)
Concentration [ppb]
f(x) = b*x + a
Parameter | Value | Confidence Delta at 90 | StdErr | Rel. StdErr [%]
b | 3,295.603 | +/-44.858 | 27.391 | 0.831
a | 586.886 | +/-225.196 | 137.508 | 23.430
R^2 | 1.000
BEC | 0.178 ppb


Details
MMA
Calibration Properties
Fit Type
Linear
Weighting
None
Forcing
No
Use for SemiQuant
No
Intensity [cps] (10^3)
Concentration [ppb]
f(x) = b*x + a
Parameter | Value | Confidence Delta at 90 | StdErr | Rel. StdErr [%]
b | 3,167.164 | +/-35.464 | 21.655 | 0.684
a | 56.691 | +/-178.035 | 108.710 | 191.760
R^2 | 1.000
BEC | 0.018 ppb

Details

As III



Calibration Properties

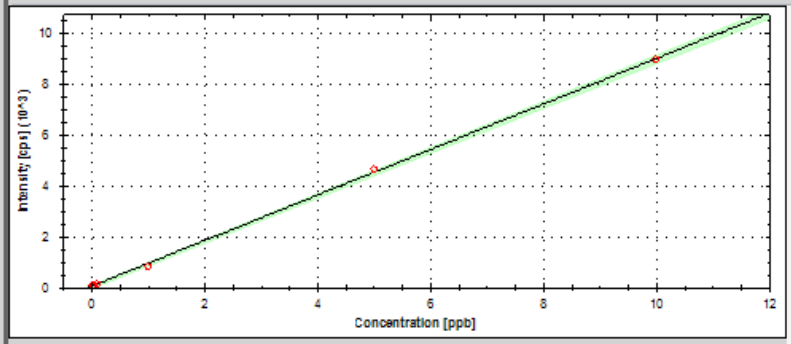
Fit Type	Linear
Weighting	None
Forcing	No
Use for SemiQuant	No

$f(x) = b^*x + a$

Parameter	Value	Confidence Delta at 90	StdErr	Rel. StdErr [%]
b	1,940.207	+/-49.453	32.254	1.662
a	28.955	+/-226.631	147.816	510.511
R^2	0.999			
BEC	0.015 ppb			

Details

DMA

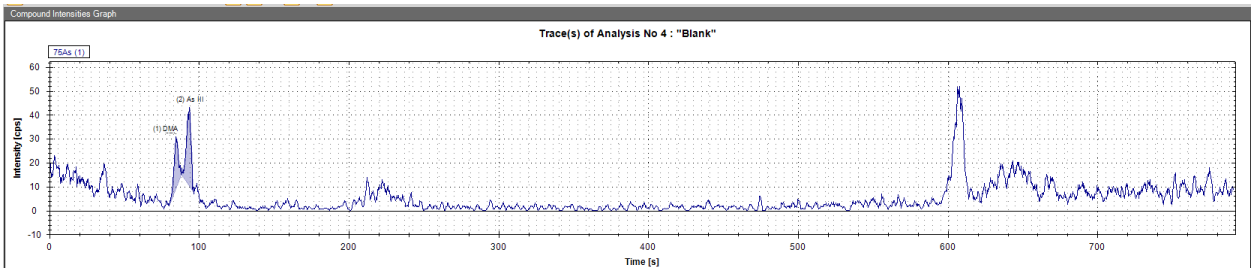


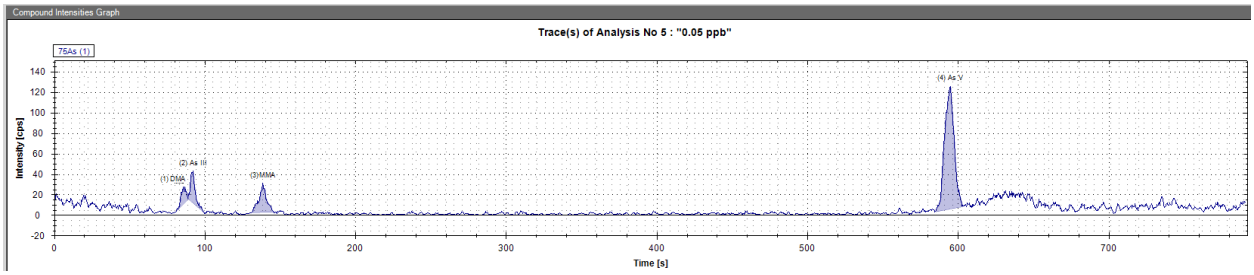
Calibration Properties

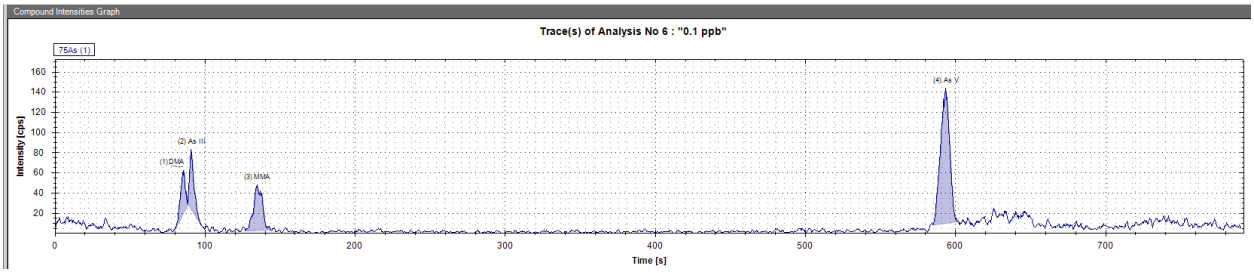
Fit Type	Linear
Weighting	None
Forcing	No
Use for SemiQuant	No

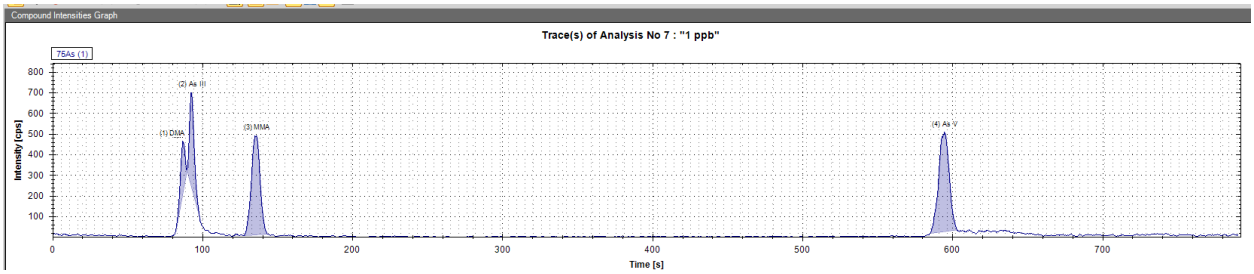
$f(x) = b^*x + a$

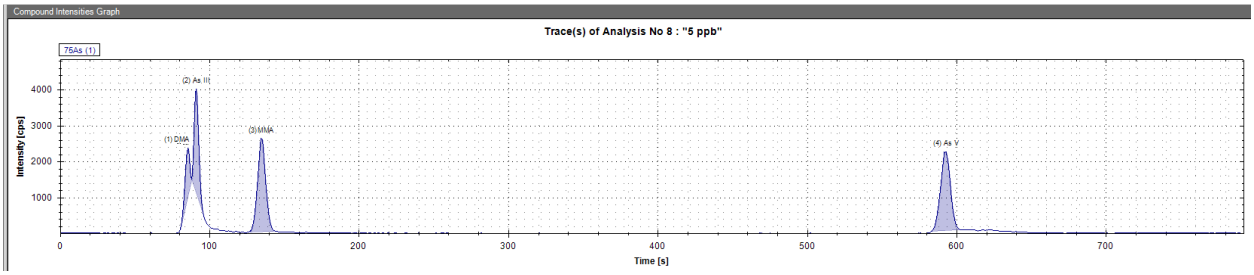
Parameter	Value	Confidence Delta at 90	StdErr	Rel. StdErr [%]
b	895.826	+/-15.412	10.052	1.122
a	27.667	+/-70.632	46.068	166.506
R^2	0.999			
BEC	0.031 ppb			

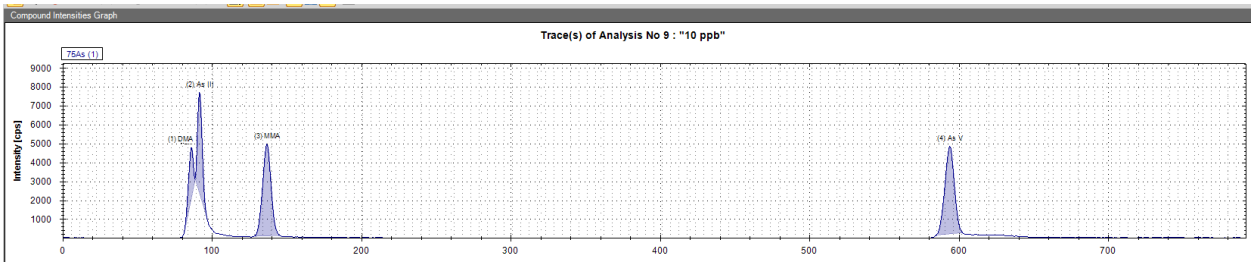


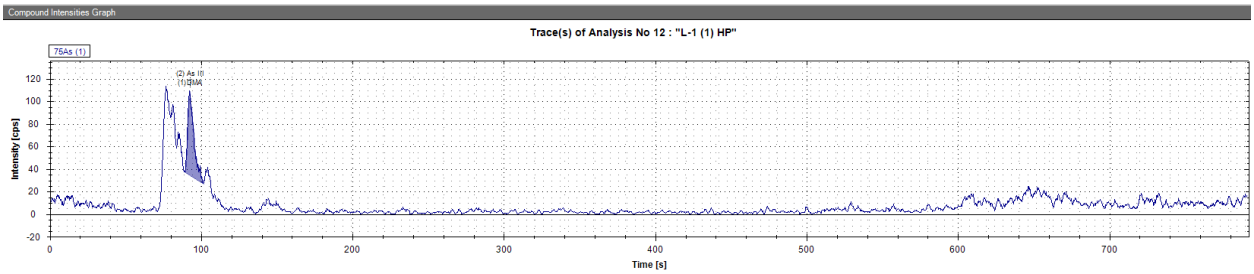

Compound Intensities Graph
Trace(s) of Analysis No 5 : "0.05 ppb"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA
(4) As V

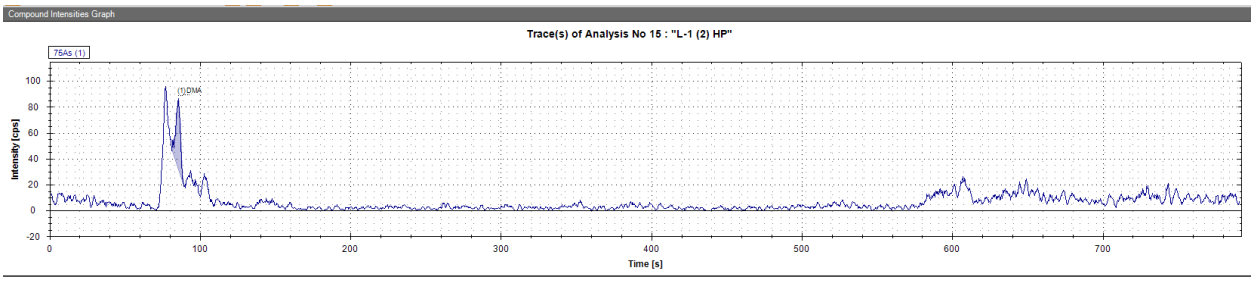

Compound Intensities Graph
Trace(s) of Analysis No 6 : "0.1 ppb"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA
(4) As V

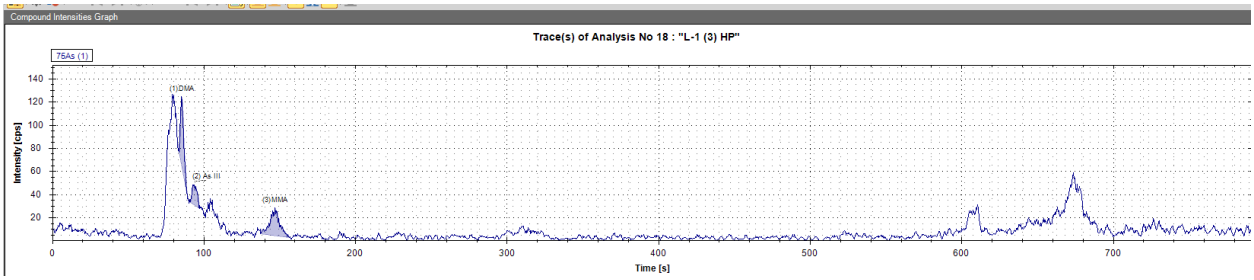

Compound Intensities Graph
Trace(s) of Analysis No 7 : "1 ppb"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA
(4) As V

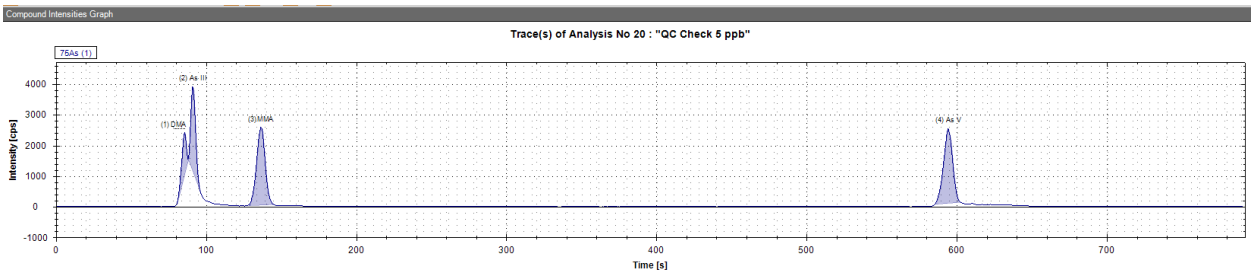

Compound Intensities Graph
Trace(s) of Analysis No 8 : "5 ppb"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA
(4) As V

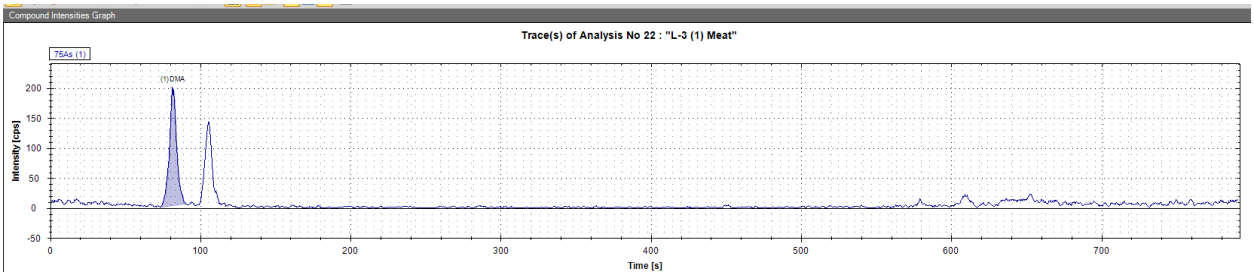

Compound Intensities Graph
Trace(s) of Analysis No 9 : "10 ppb"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA
(4) As V

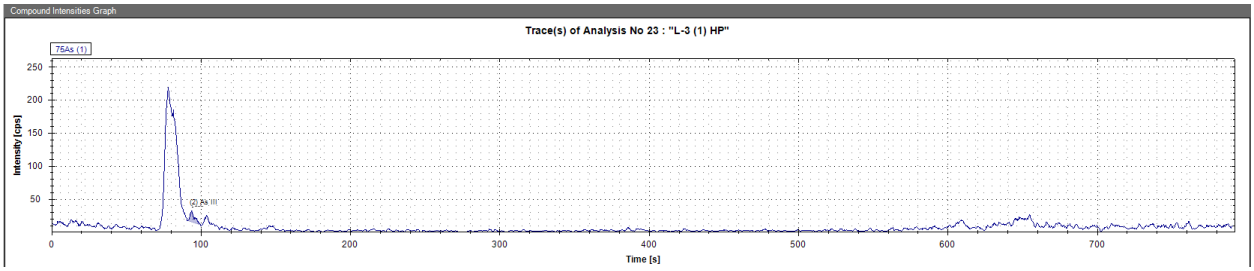

Compound Intensities Graph
Trace(s) of Analysis No 12 : "L-1 (1) HP"
75As (1)
(2) As III
(1) DMA
Intensity [cps]
Time [s]

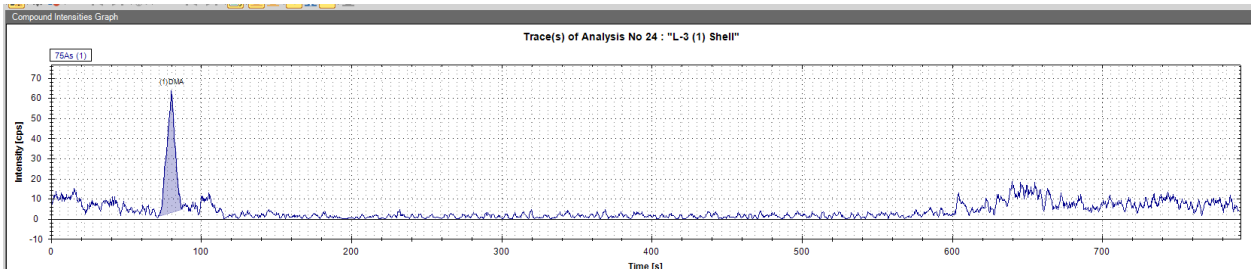

Compound Intensities Graph
Trace(s) of Analysis No 15 : "L-1 (2) HP"
75As (1)
(1) DMA
Intensity [cps]
Time [s]

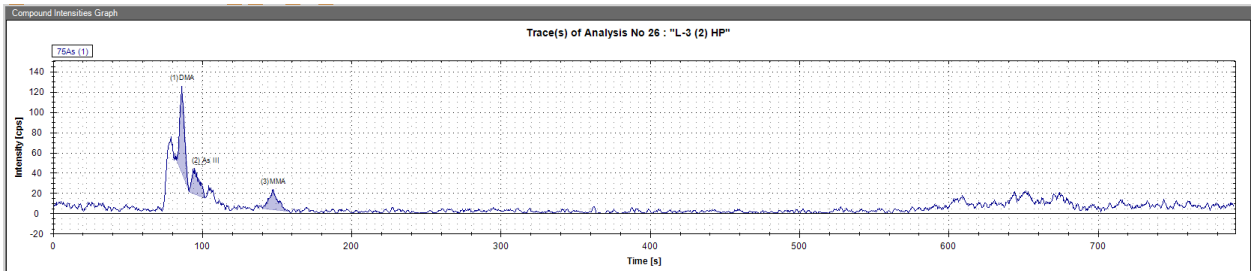

Compound Intensities Graph
Trace(s) of Analysis No 18 : "L-1 (3) HP"
75As (1)
(1) DMA
(2) As III
(3) MMA
Intensity [cps]
Time [s]

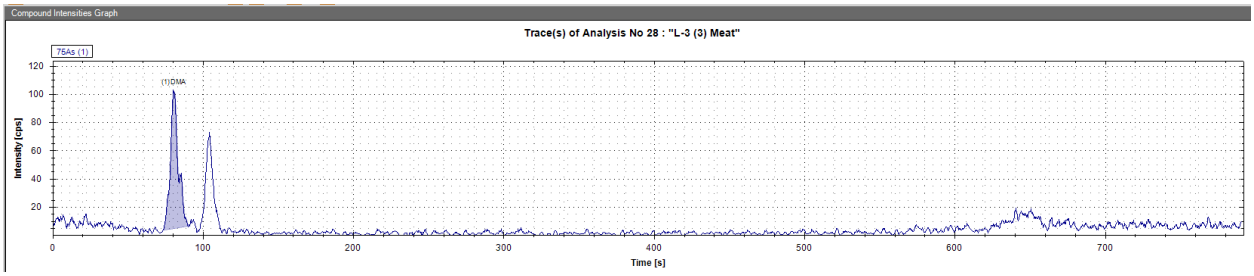

Compound Intensities Graph
Trace(s) of Analysis No 20 : "QC Check 5 ppb"
75As (1)
(2) As III
(1) DMA
(3) MMA
(4) As V
Intensity [cps]
Time [s]

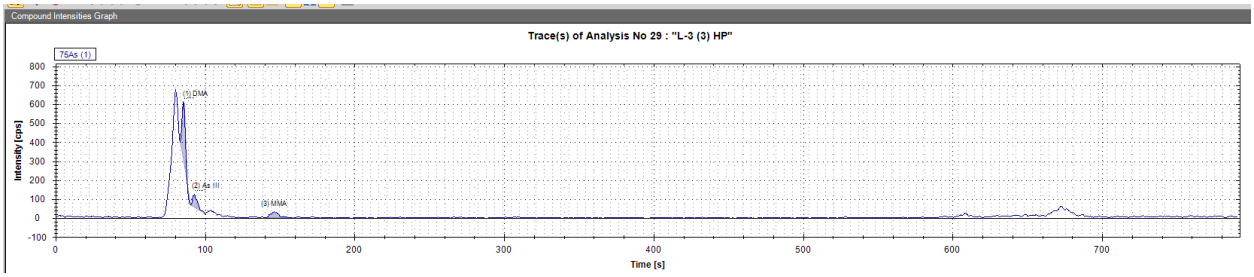

Compound Intensities Graph
Trace(s) of Analysis No 22 : "L-3 (1) Meat"
75As (1)
(1) DMA
Intensity [cps]
Time [s]

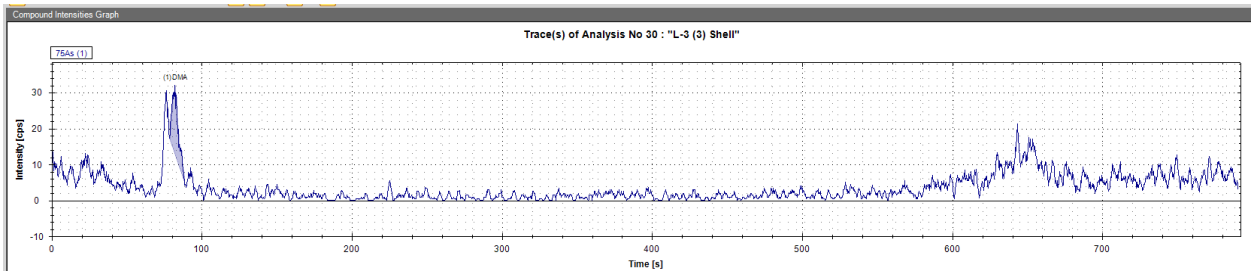

Compound Intensities Graph
Trace(s) of Analysis No 23 : "L-3 (1) HP"
75As (1)
Intensity [cps]
Time [s]
(2) As III

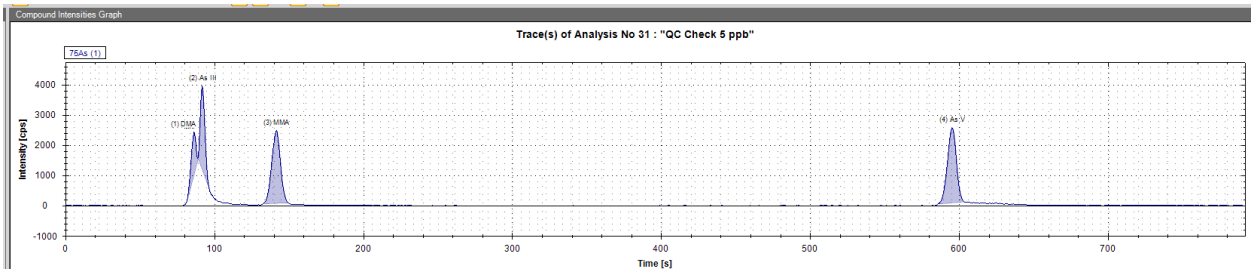

Compound Intensities Graph
Trace(s) of Analysis No 24 : "L-3 (1) Shell"
75As (1)
Intensity [cps]
Time [s]
(1) DMA

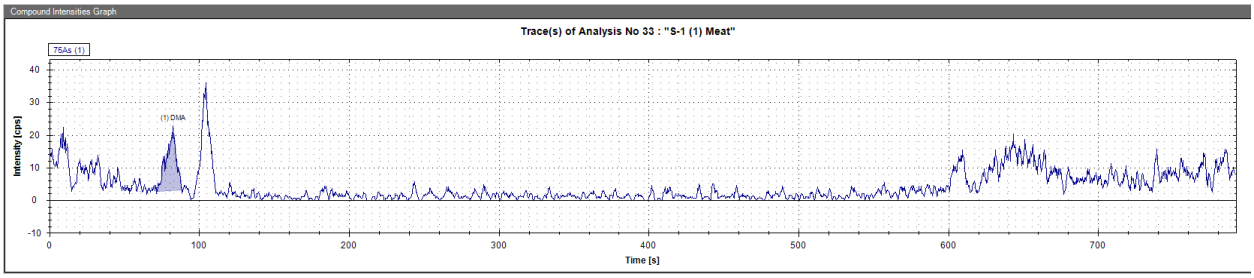

Compound Intensities Graph
Trace(s) of Analysis No 26 : "L-3 (2) HP"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA

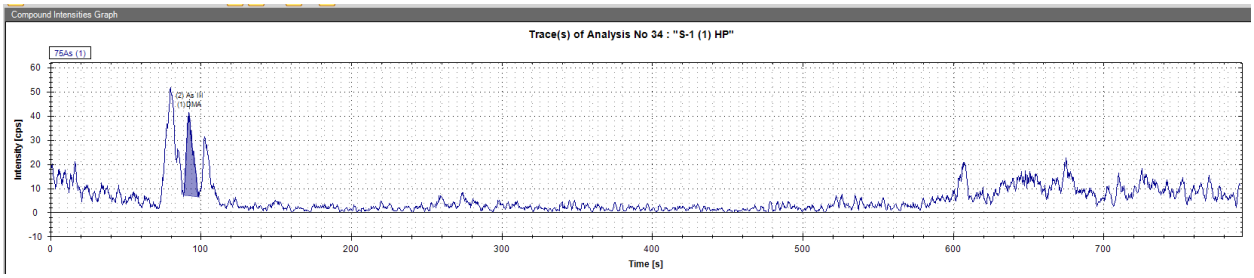

Compound Intensities Graph
Trace(s) of Analysis No 28 : "L-3 (3) Meat"
75As (1)
Intensity [cps]
Time [s]
(1) DMA

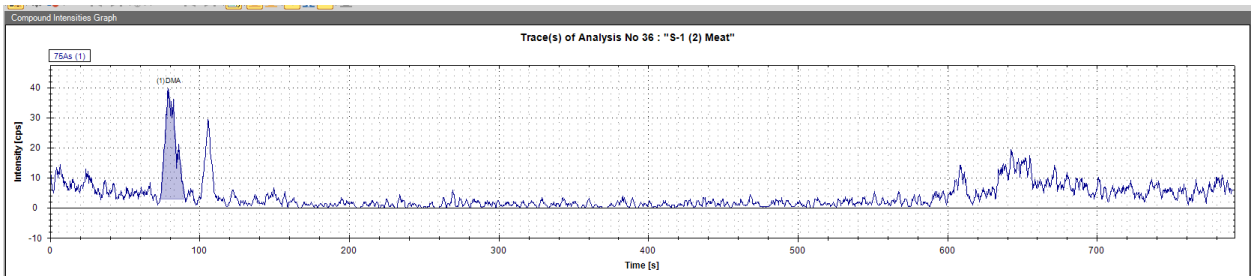

Compound Intensities Graph
Trace(s) of Analysis No 29 : "L-3 (3) HP"
75As (1)
Intensity [cps]
Time [s]
(1) DMA
(2) As III
(3) MMA


Compound Intensities Graph
Trace(s) of Analysis No 30 : "L-3 (3) Shell"
75As (1)
(1) DMA
Intensity [cps]
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 31 : "QC Check 5 ppb"
75As (1)
(2) As III
(1) DMA
(3) MMA
(4) As V
Intensity [cps]
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 33 : "S-1 (1) Meat"
75As (1)
(1) DMA
Intensity [cps]
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 34 : "S-1 (1) HP"
75As (1)
(2) As III
(1) DMA
Intensity [cps]
Time [s]


Compound Intensities Graph
Trace(s) of Analysis No 36 : "S-1 (2) Meat"
75As (1)
(1) DMA
Intensity [cps]
Time [s]

APPENDIX C

SPSS AND EXCEL STATISTICAL REPORTS

Table 14: Summary table of samples speciated from East White Lake

2015 East White Lake Crabs																
Sample ID	Whole Crab Wet Weight (g)	Lateral Width (spine to spine) (cm)	Left Half Crab Wet Weight (g)	Mass of Dissected Meat (g)	Meat Split Mass for Speciation	Total Mass of Meat (g)	Total Arsenic Load Meat (mg)	Mass of Dissected Shell (g)	Shell Split Mass for Speciation	Total Mass of Shell (g)	Total Arsenic Load Shell (mg)	Mass of Dissected HP (g)	HP Split Mass for Speciation	Total Mass of HP (g)	Total Arsenic Load HP (mg)	Total Arsenic Load (Crab) (mg)
L-1 (1)	258.5	18.4	148.9	0.6512	0.2885	12.6512	0	0.4408	0.2224	57.4408	0	0.5531	0.3093	3.3531	0.0007	0.0007
L-1 (2)	100.1	12.8	46.1	0.3835	0.2258	2.4835	0	0.3426	0.1773	22.7426	0	0.4193	0.2458	1.7193	0.0002	0.0002
L-1 (3)	86.6	12.0	41.0	0.9454	0.3341	2.6454	0	0.3125	0.1753	16.5125	0	0.5862	0.2450	2.3862	0.0001	0.0001
L-2 (1)	121.4	14.4	62.6	0.6408	0.1722	3.8408	0.0035	0.3164	0.0711	24.6164	0.0092	0.4685	0.0781	2.7685	0.0084	0.0211
L-2 (2)	103.9	14.5	61.3	0.4165	0.1792	2.4165	0.0010	0.3976	0.2321	21.5976	0.0086	0.5165	0.1919	1.9165	0.0022	0.0118
L-2 (3)	252.2	19.0	120.9	0.4295	0.1648	4.3295	0.0005	0.5276	0.2447	49.7276	0.0115	0.2921	0.0488	3.6921	0.0028	0.0148
L-3 (1)	105.7	13.5	54.0	0.4969	0.2186	4.6969	0.0018	0.3370	0.1681	20.3370	0.0027	0.2046	0.0519	1.7046	0	0.0044
L-3 (2)	113.4	13.0	56.1	0.5829	0.2051	3.8829	0	0.3263	0.2163	20.1263	0	0.2233	0.0861	2.5233	0.0003	0.0003
L-3 (3)	160.3	15.0	82.5	0.6598	0.3648	9.8598	0.0016	0.5008	0.2529	29.7008	0	0.6593	0.3091	1.4593	0.0003	0.0019
S-1 (1)	152.2	13.9	77.9	0.5387	0.1293	4.5387	6.35418E-05	0.2926	0.1984	33.3926	0	0.3289	0.2013	1.7289	0.0002	0.0002
S-1 (2)	88.3	12.1	43.7	0.8174	0.2474	4.0174	0.0002	0.3473	0.1533	16.2473	0	0.6723	0.3550	2.2723	0	0.0002
S-1 (3)	69.0	11.4	32.9	0.9445	0.3493	2.1445	Not analyzed	0.3207	0.1514	13.2207	Not analyzed	1.0980	0.4171	2.1980	Not analyzed	X
S-2 (1)	77.5	11.3	37.6	0.7093	0.38222	3.4093	0.0009	0.4535	0.2153	13.2535	0.0047	0.3917	0.2526	1.5917	0.0013	0.0068
S-2 (2)	116.2	14.0	60.8	0.4652	0.3121	3.9652	0.0008	0.4903	0.2530	21.7903	0.0038	0.4660	0.1540	1.5660	0.0012	0.0058
S-2 (3)	132.4	13.6	62.7	0.469	0.3576	4.569	0.0013	0.2320	0.0951	21.2320	0.0049	0.9662	0.5547	2.1662	0.0018	0.0080
S-6 (1)	215.1	16.5	105.2	0.6041	0.2753	7.3041	0.0007	0.6596	0.2389	43.5596	0.0020	0.5925	0.4110	2.1925	0.0008	2.1951
S-6 (2)	138.0	14.0	66.8	0.7888	0.3855	5.9888	0.0008	0.4464	0.1952	25.1464	0.0020	0.4897	0.2158	2.4897	0.0018	2.4924
S-6 (3)	155.1	13.9	75.1	0.9207	0.5084	4.7207	0.0025	0.4669	0.1516	25.5669	0.0119	1.2749	0.8263	2.4749	0.0019	2.4893

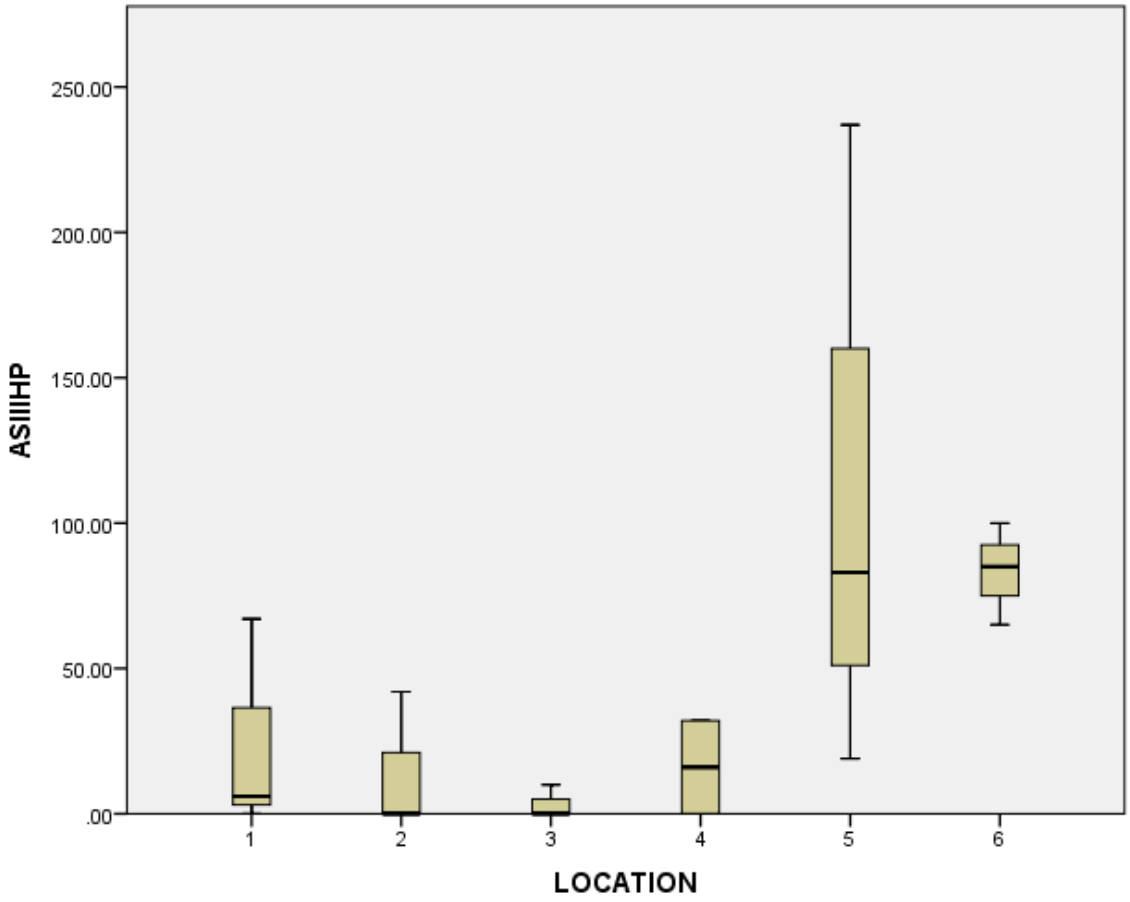


Figure 41: Boxplot of As (III) hepatopancreas at 6 sampling locations

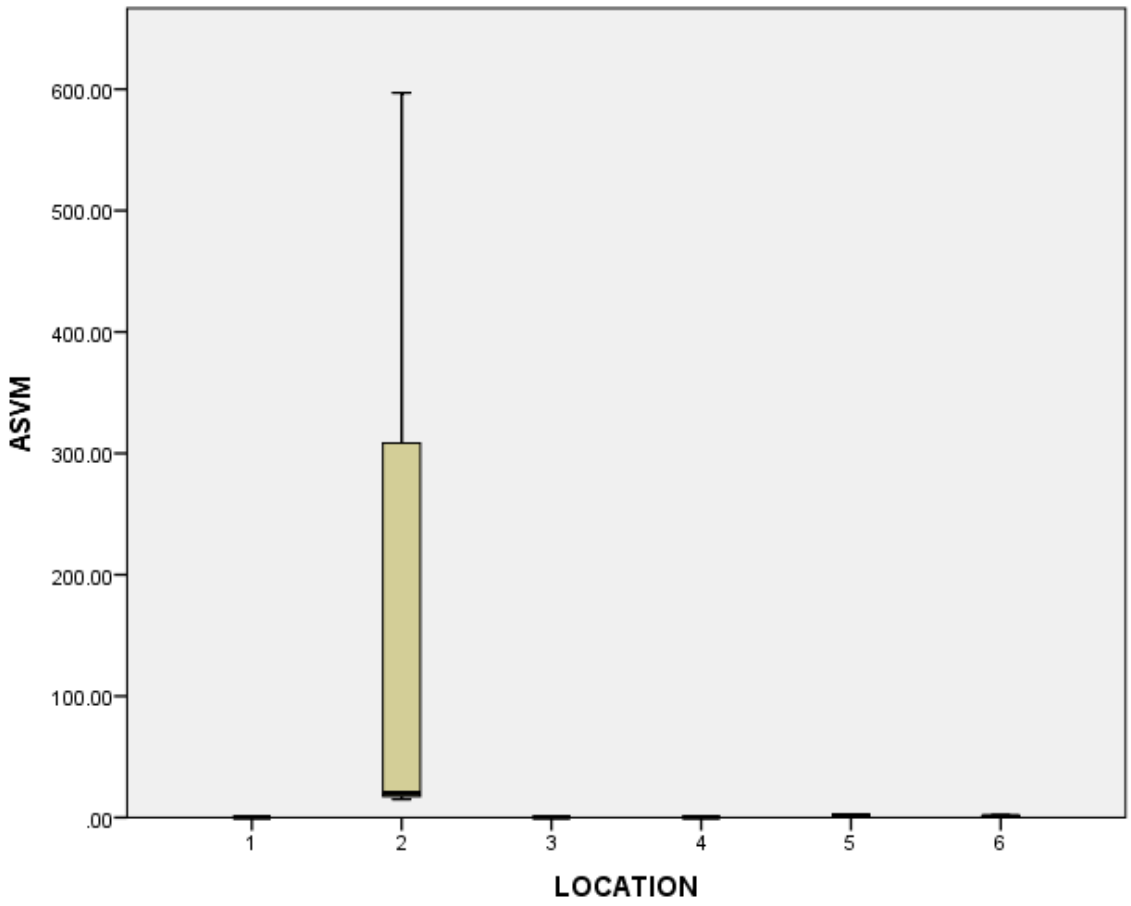


Figure 42: Boxplot of As (V) meat at 6 sampling locations

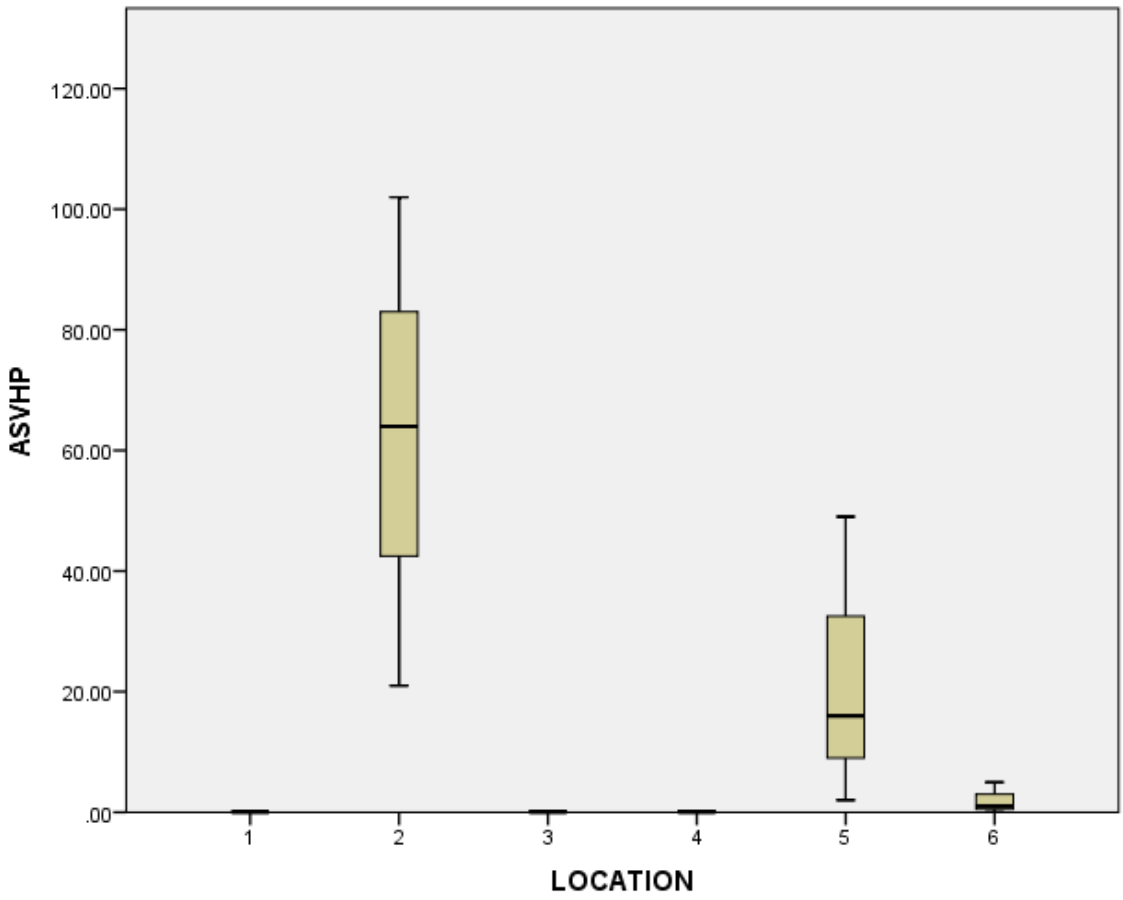


Figure 43: Boxplot of As (V) hepatopancreas at 6 sampling locations

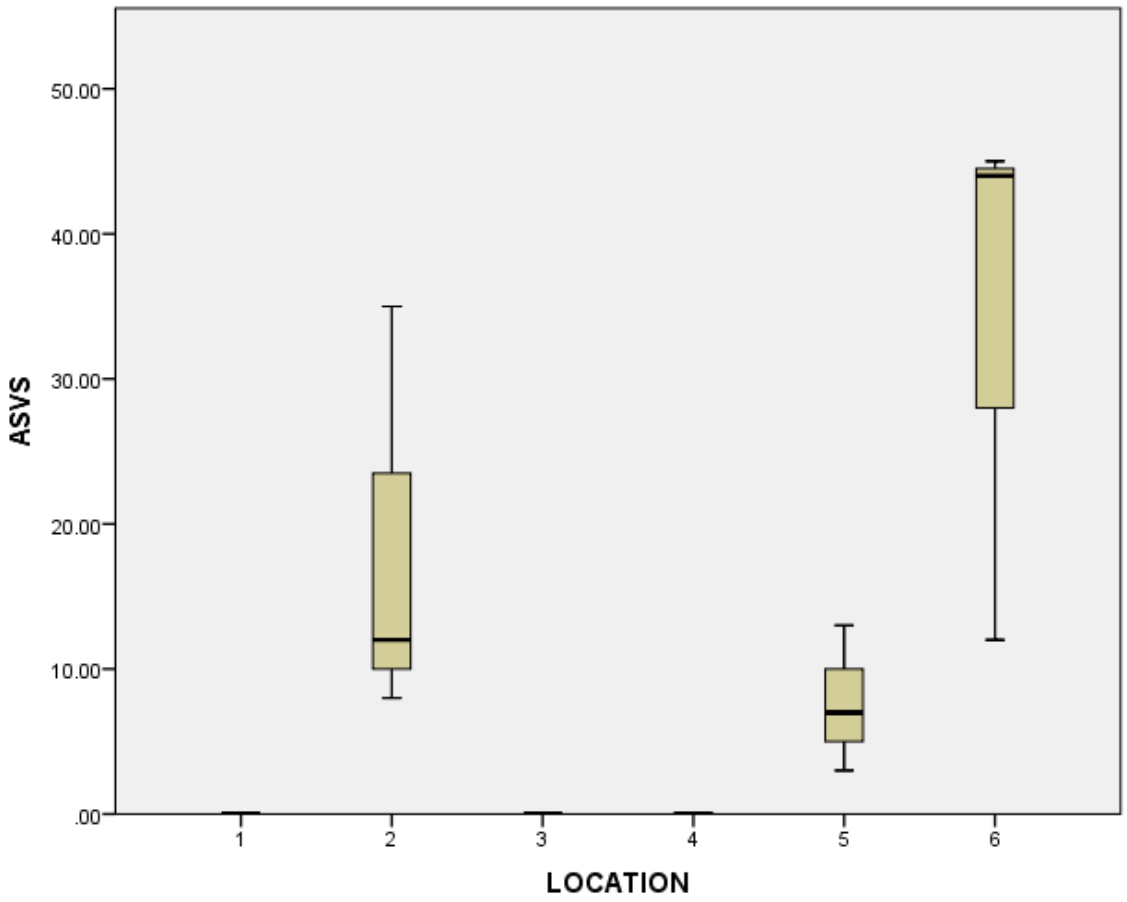


Figure 44: Boxplot of As (V) shell at 6 sampling locations

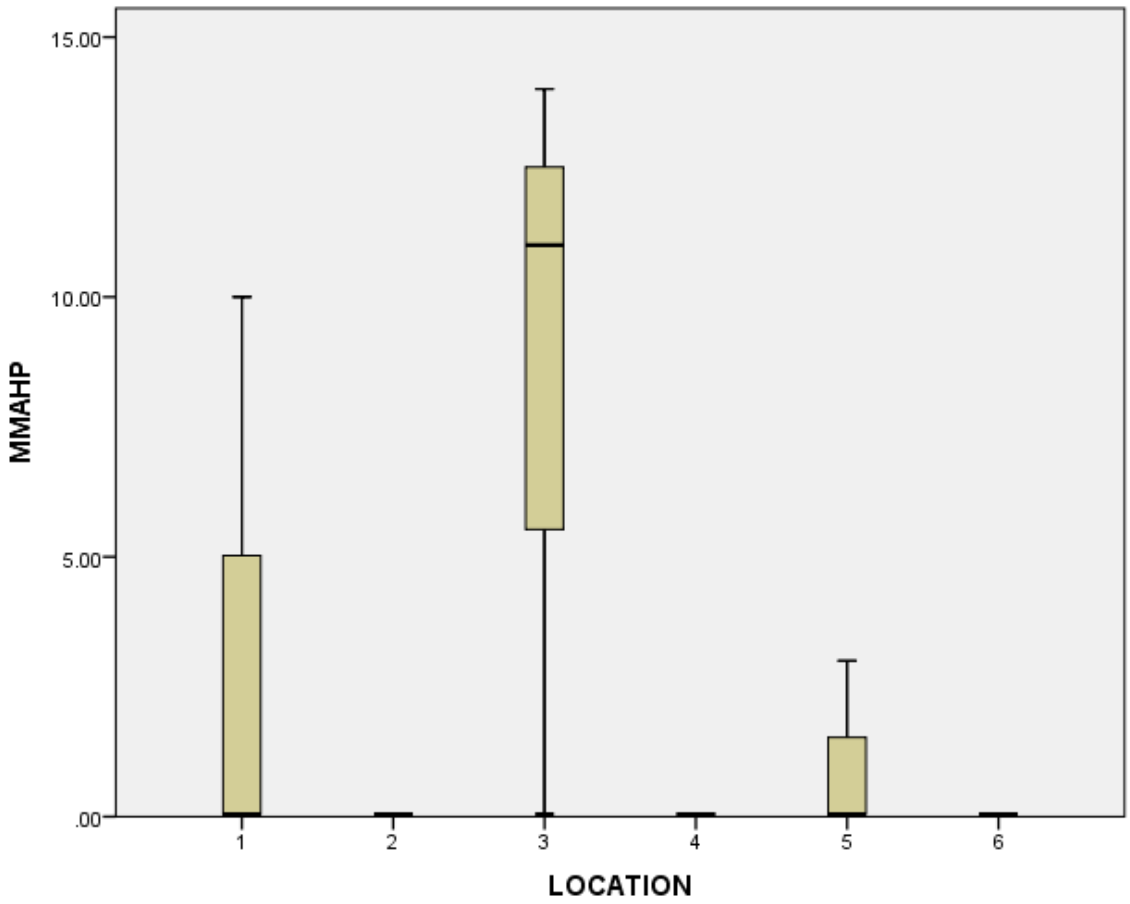


Figure 45: Boxplot of MMA hepatopancreas at 6 sampling locations

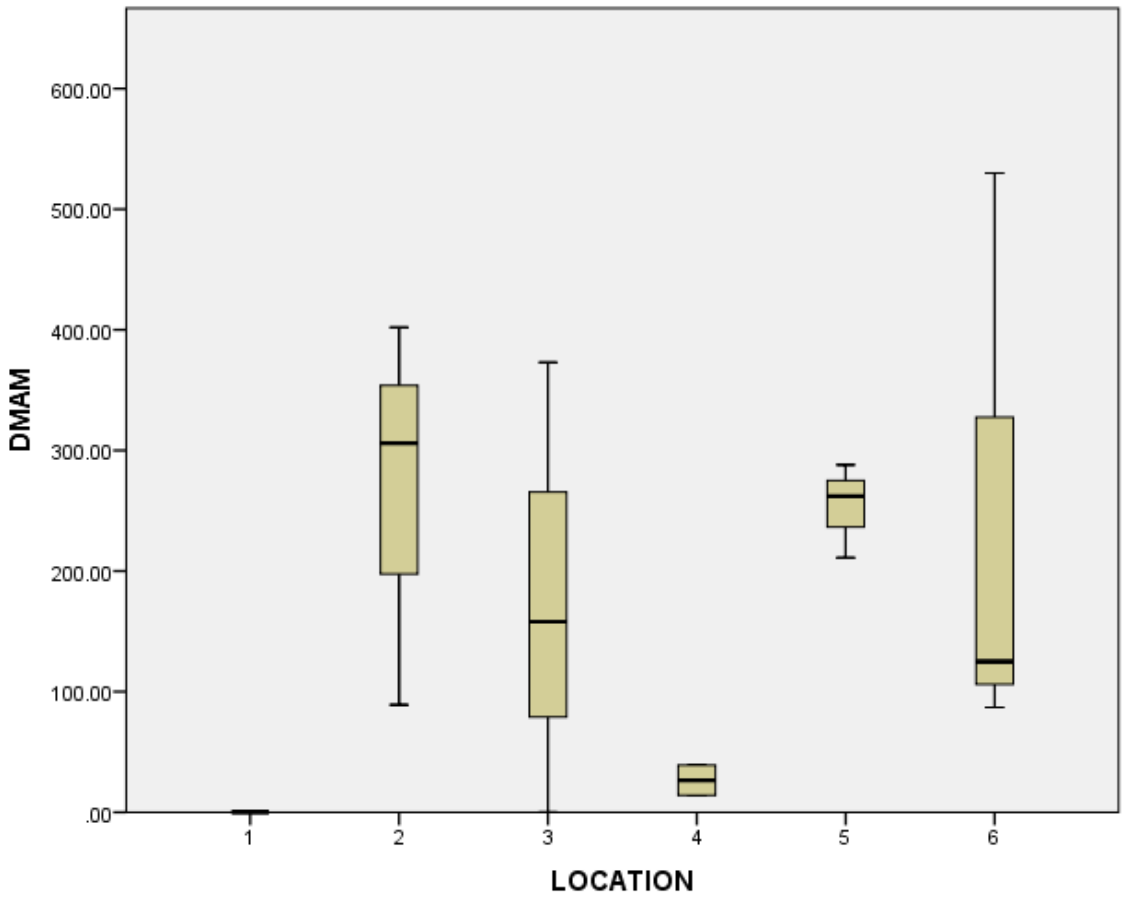


Figure 46: Boxplot of DMA meat at 6 sampling locations

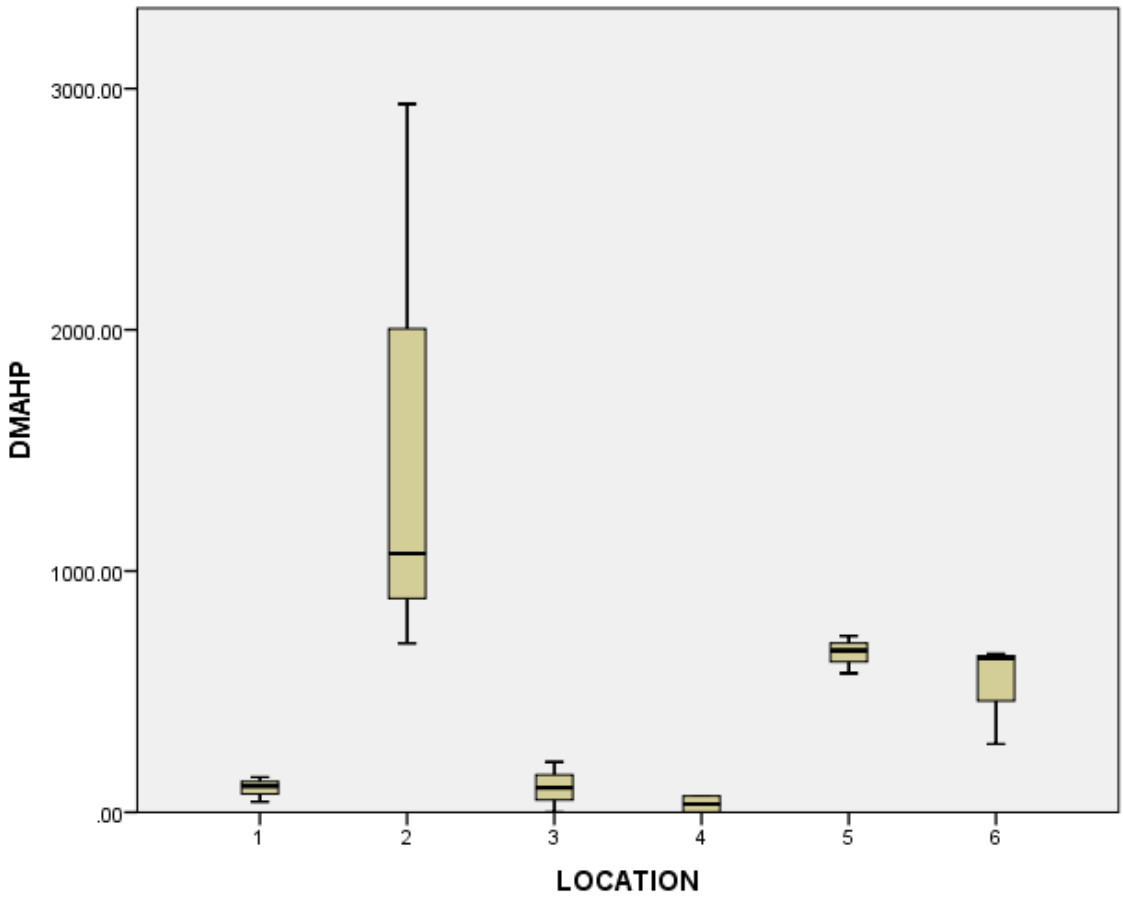


Figure 47: Boxplot of DMA hepatopancreas at 6 sampling locations

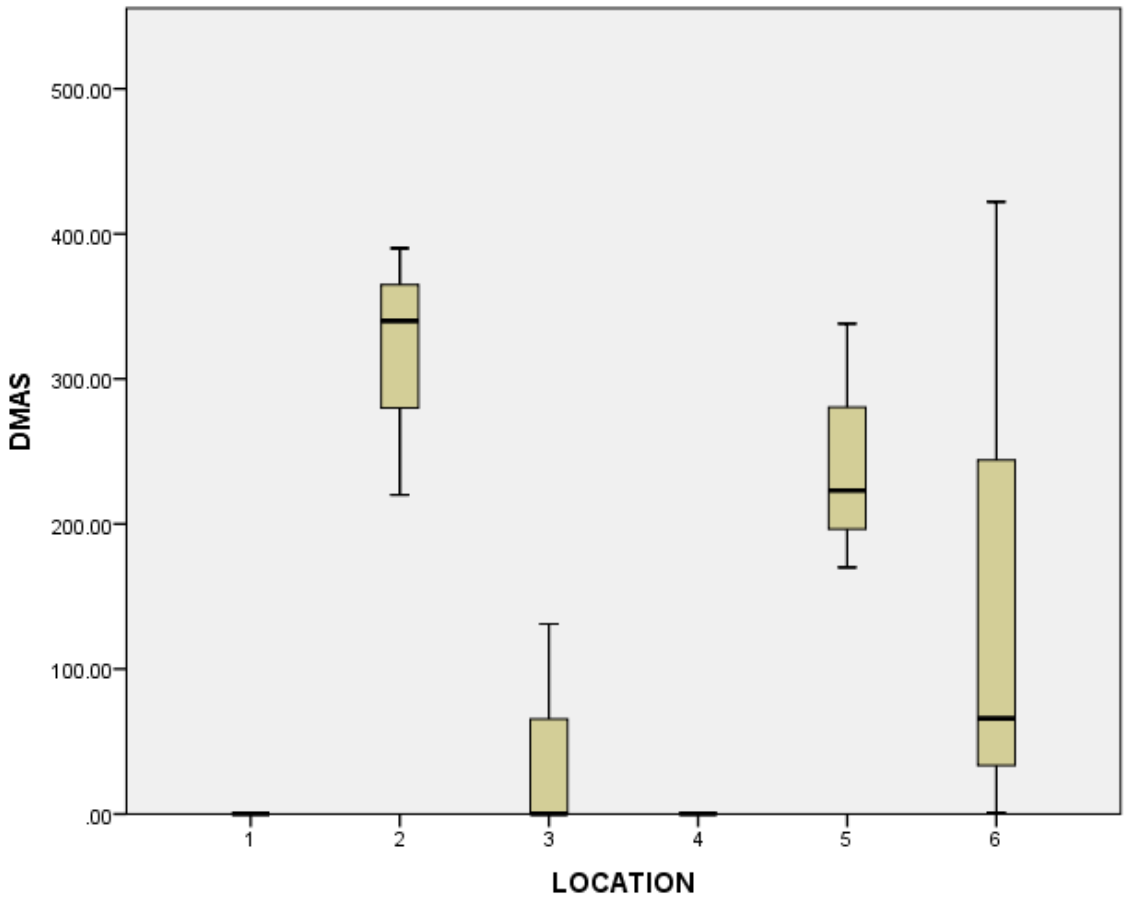


Figure 48: Boxplot of DMA shell at 6 sampling locations

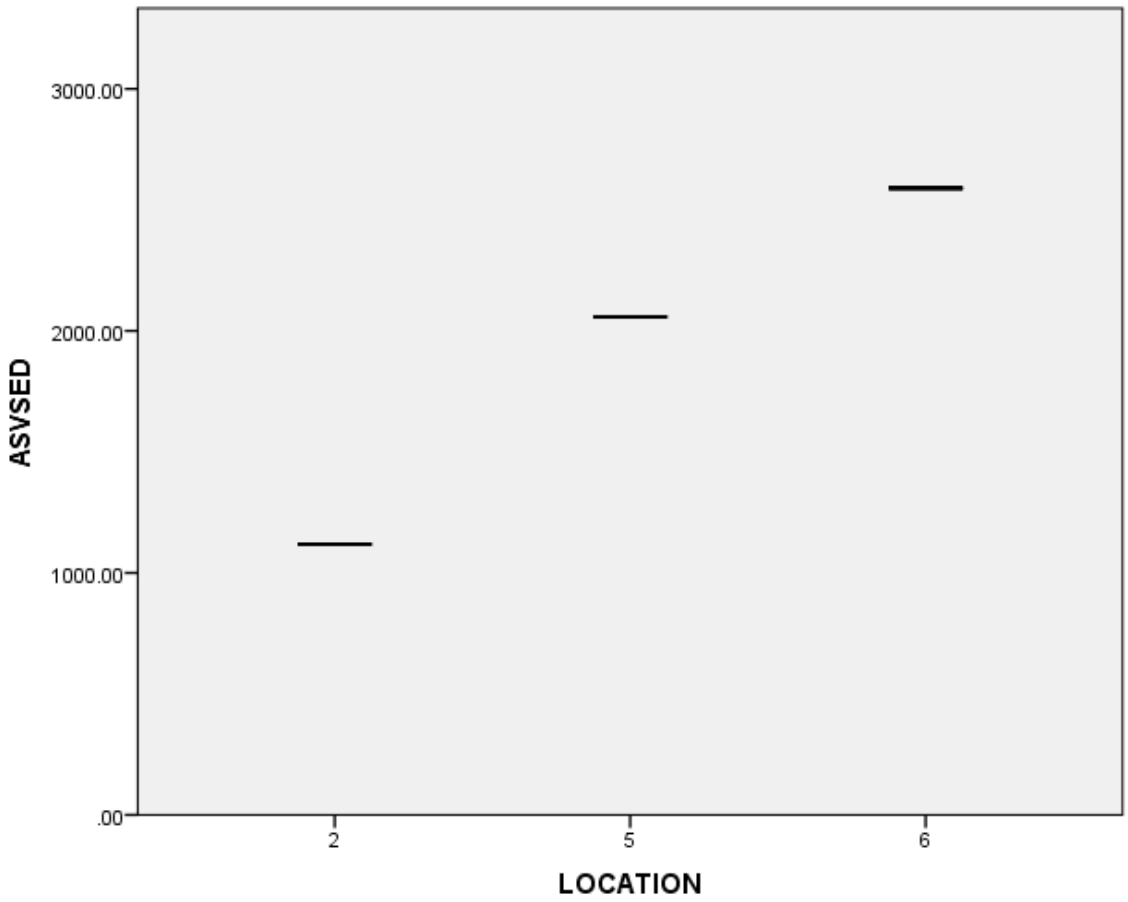


Figure 49: Boxplot of As (V) sediment at 3 sampling locations (sediment samples from Locations L-1, L-3, and S-1 were not speciated).

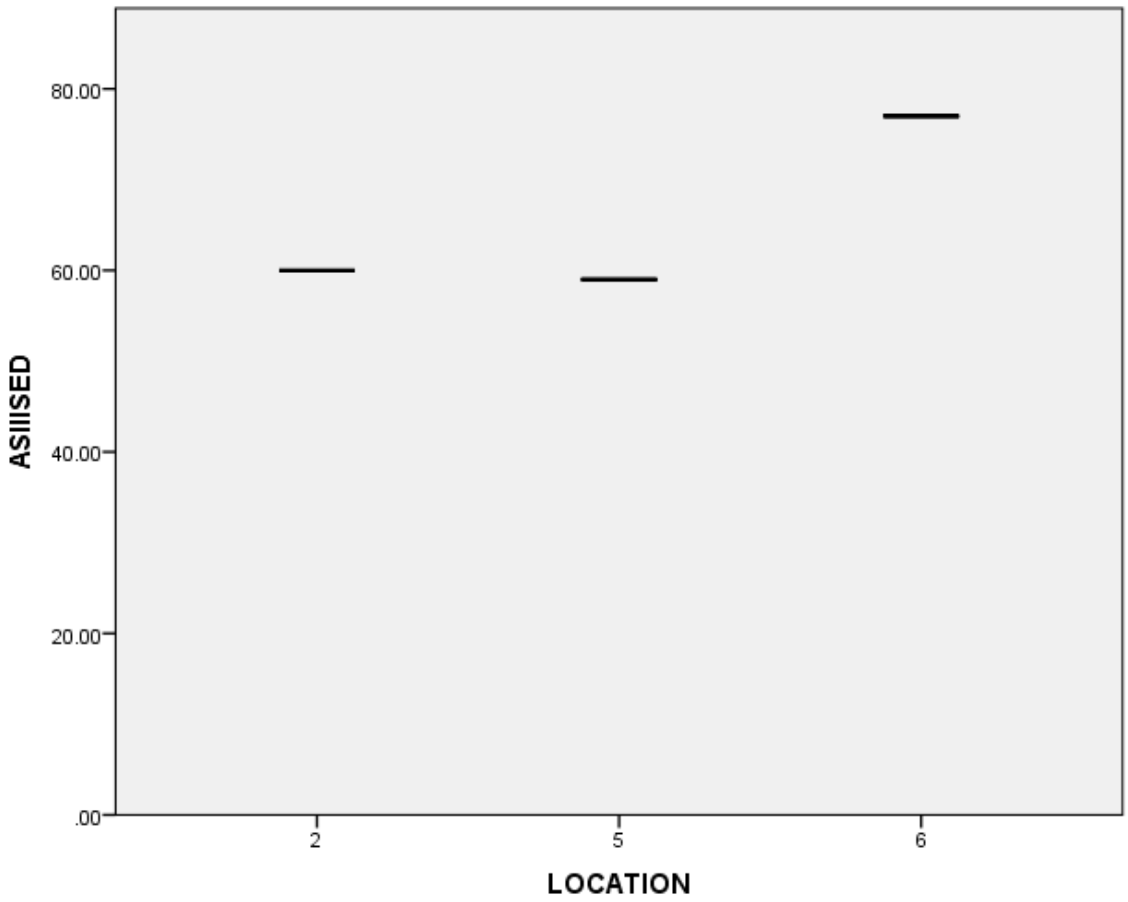


Figure 50: Boxplot of As (III) sediment at 3 sampling locations (sediment samples from Locations L-1, L-3, and S-1 were not speciated).

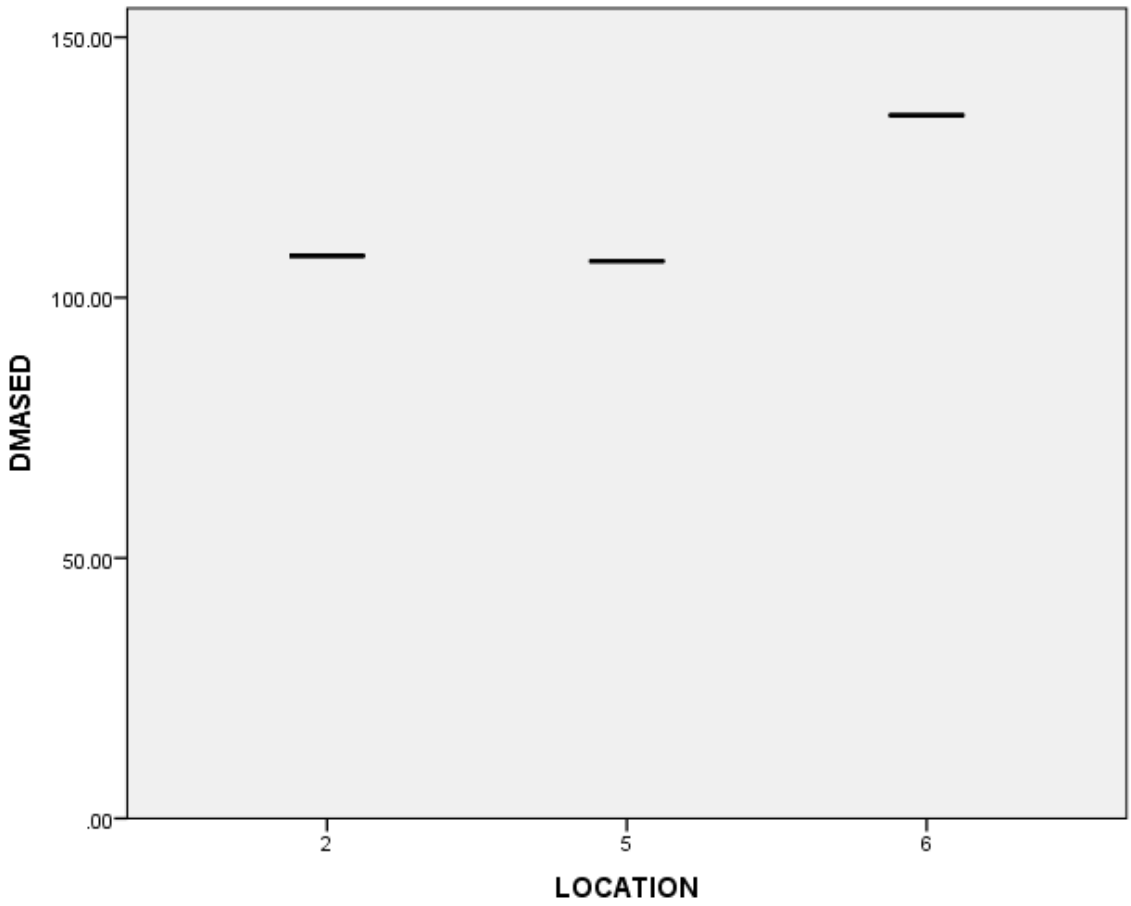


Figure 51: Boxplot of DMA sediment at 3 sampling locations (sediment samples from Locations L-1, L-3, and S-1 were not speciated).

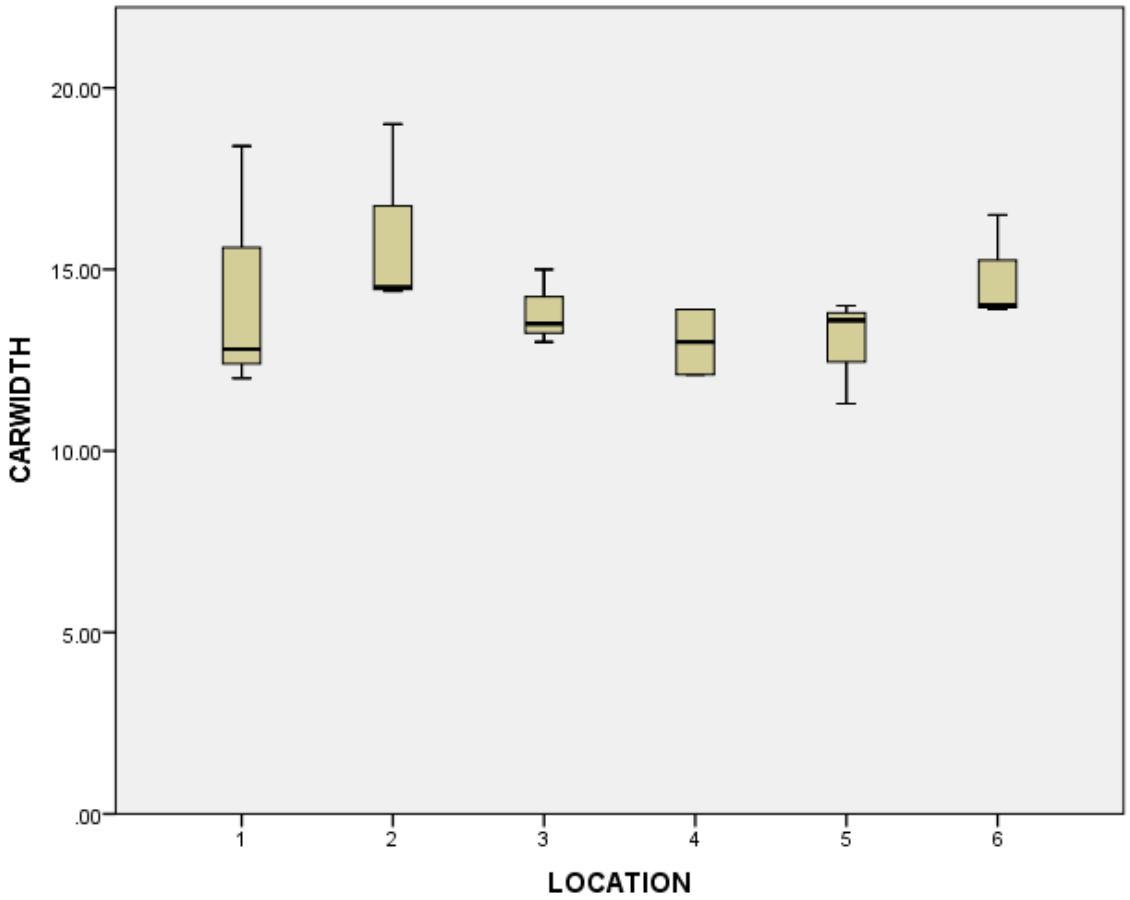


Figure 52: Boxplot of carapace width at 6 sampling locations

Group Statistics

	LOCATION	N	Mean	Std. Deviation	Std. Error Mean
TOTALARSENIC	1	3	1295.67	212.185	122.505
	2	3	2248.67	642.419	370.901

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
TOTALARSENIC	Equal variances assumed	1.875	.243	-2.440	4	.071	-953.000	390.608	-2037.503	131.503
	Equal variances not assumed			-2.440	2.431	.112	-953.000	390.608	-2377.955	471.955

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The distribution of ASVSED is the same across categories of LOCATION.	Independent-Samples Kruskal-Wallis Test	.368	Retain the null hypothesis.
2	The distribution of DMASED is the same across categories of LOCATION.	Independent-Samples Kruskal-Wallis Test	.368	Retain the null hypothesis.
3	The distribution of ASIIISED is the same across categories of LOCATION.	Independent-Samples Kruskal-Wallis Test	.368	Retain the null hypothesis.

Asymptotic significances are displayed. The significance level is .05.

Figure 53: Independent samples t–test and Kruskal-Wallis test of total arsenic in lake versus East White Lake oil and gas field sediment samples.

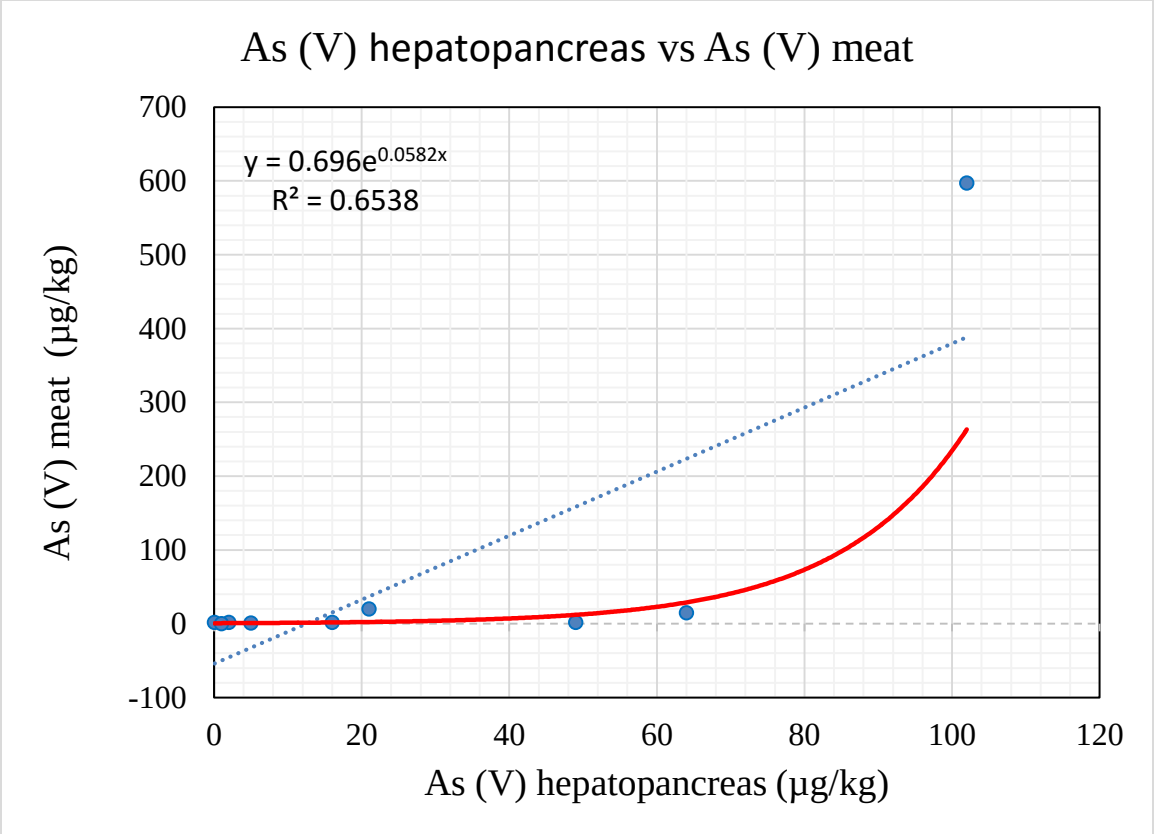


Figure 54: Regression curve of As (V) hepatopancreas vs As (V) meat

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

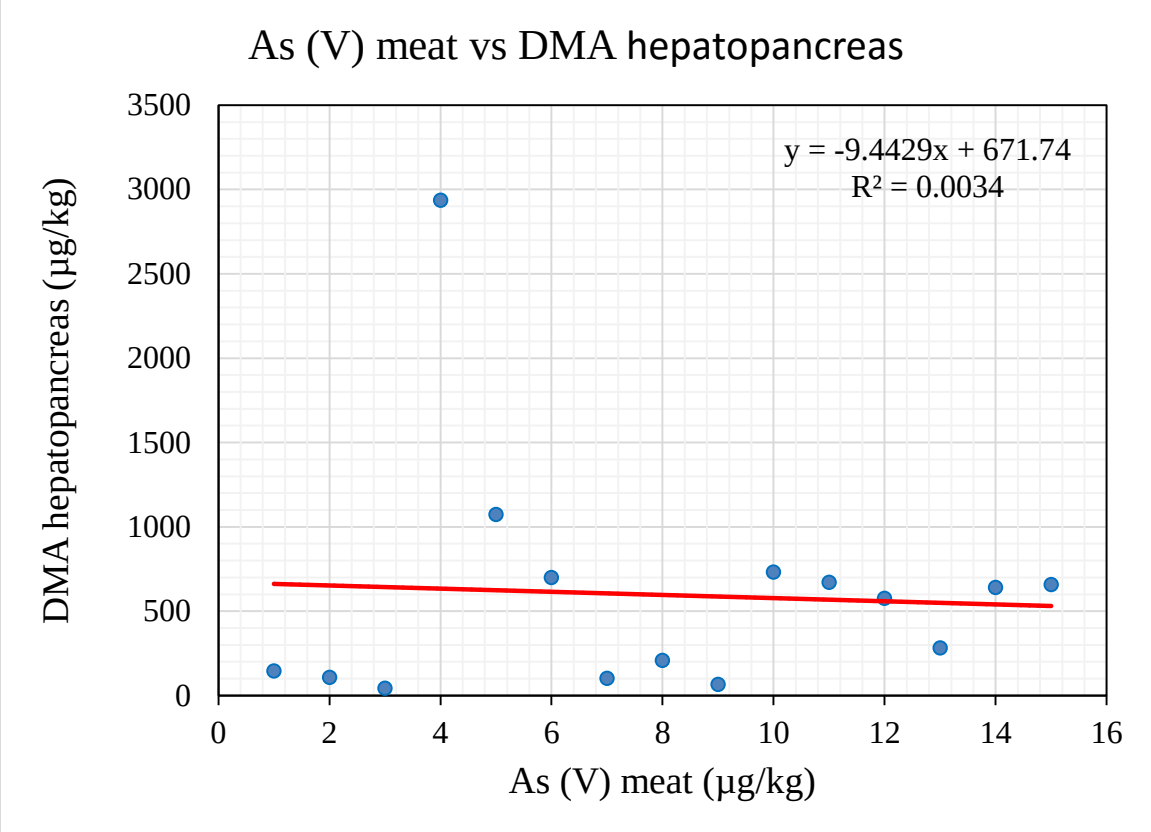


Figure 55: Regression curve of As (V) meat vs DMA hepatopancreas

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

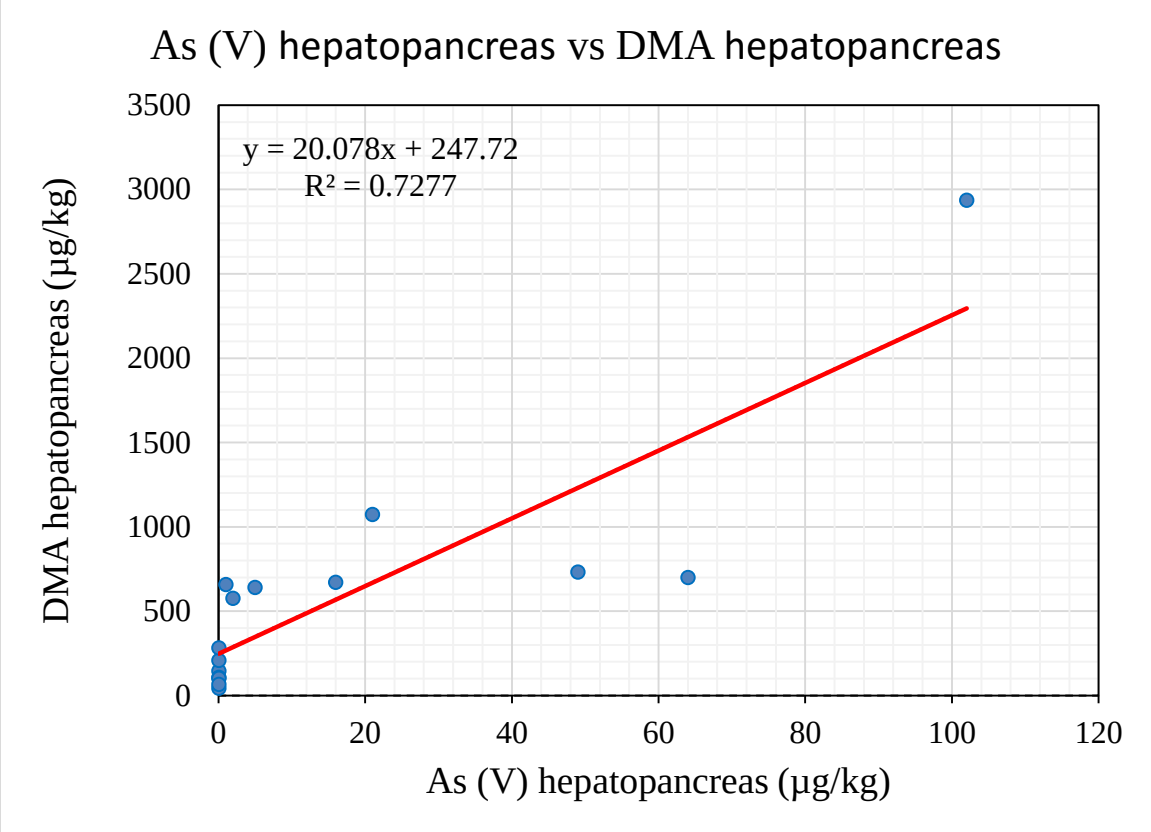


Figure 56: Regression curve of As (V) hepatopancreas vs DMA hepatopancreas

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

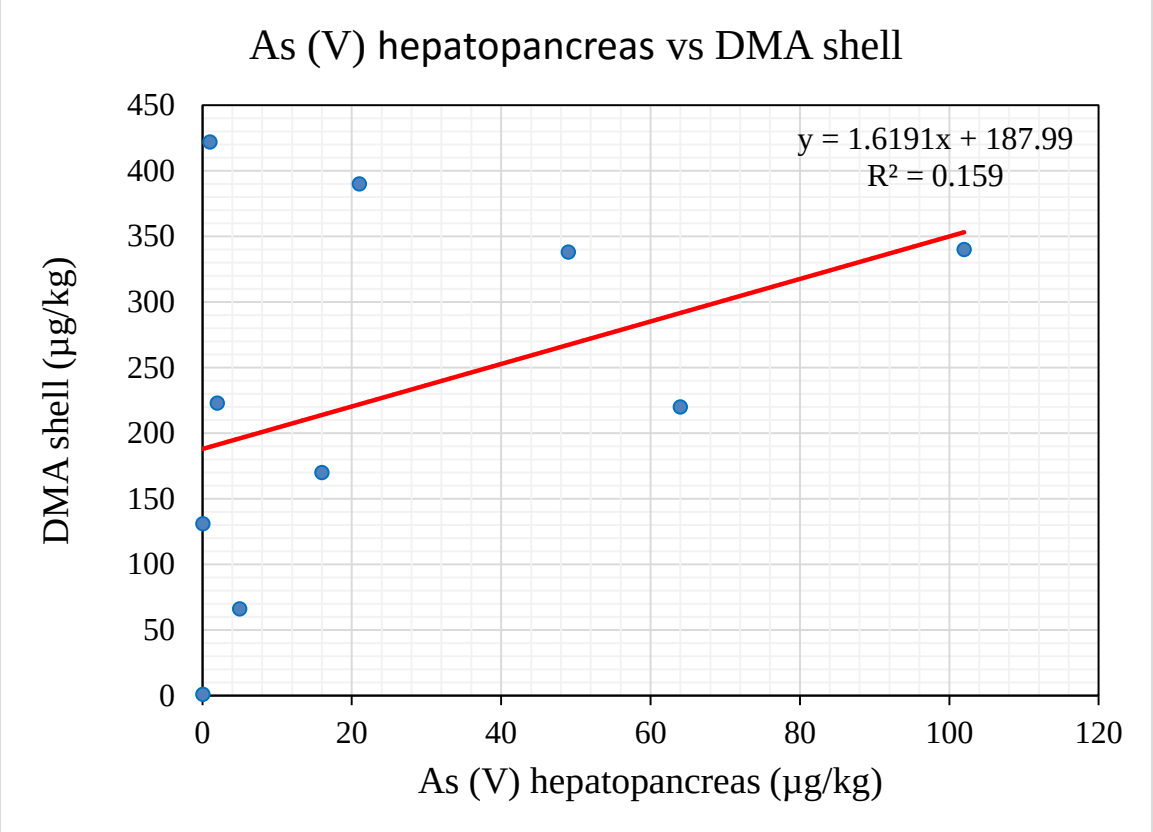


Figure 57: Regression curve of As (V) hepatopancreas vs DMA shell

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

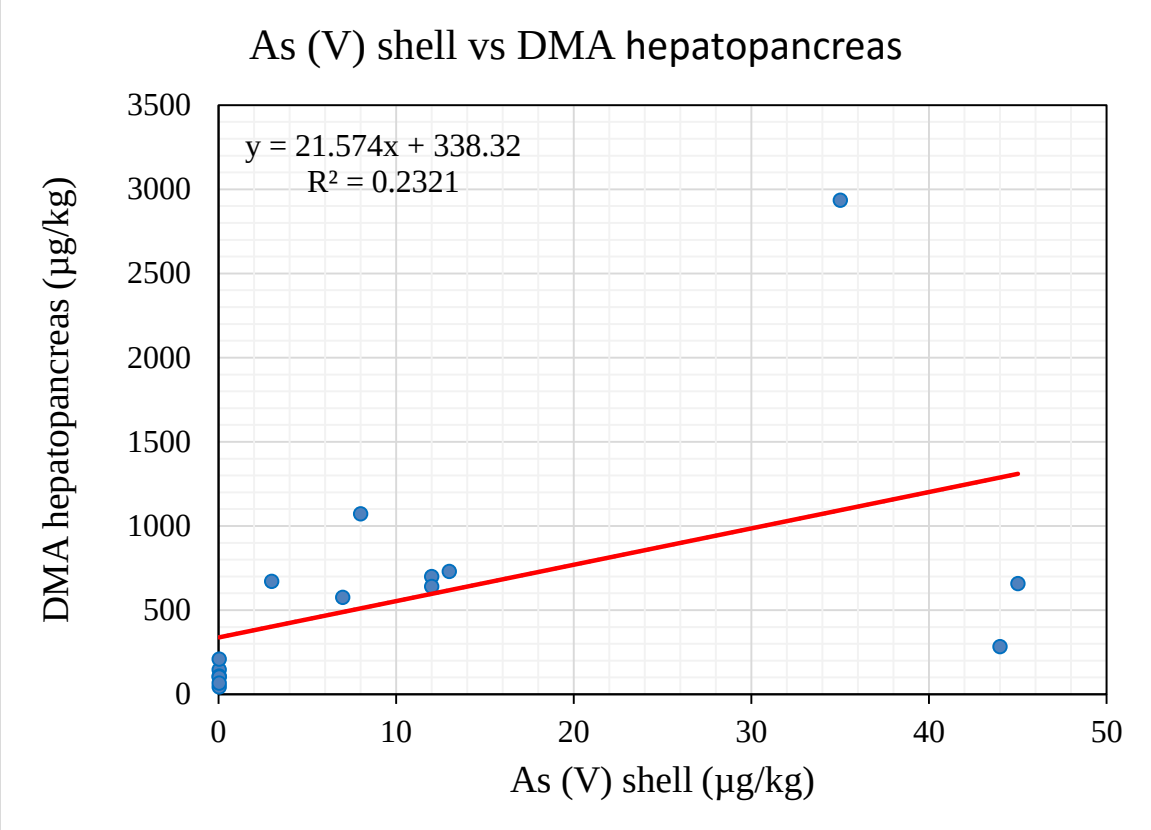


Figure 58: Regression curve of As (V) shell vs DMA hepatopancreas

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

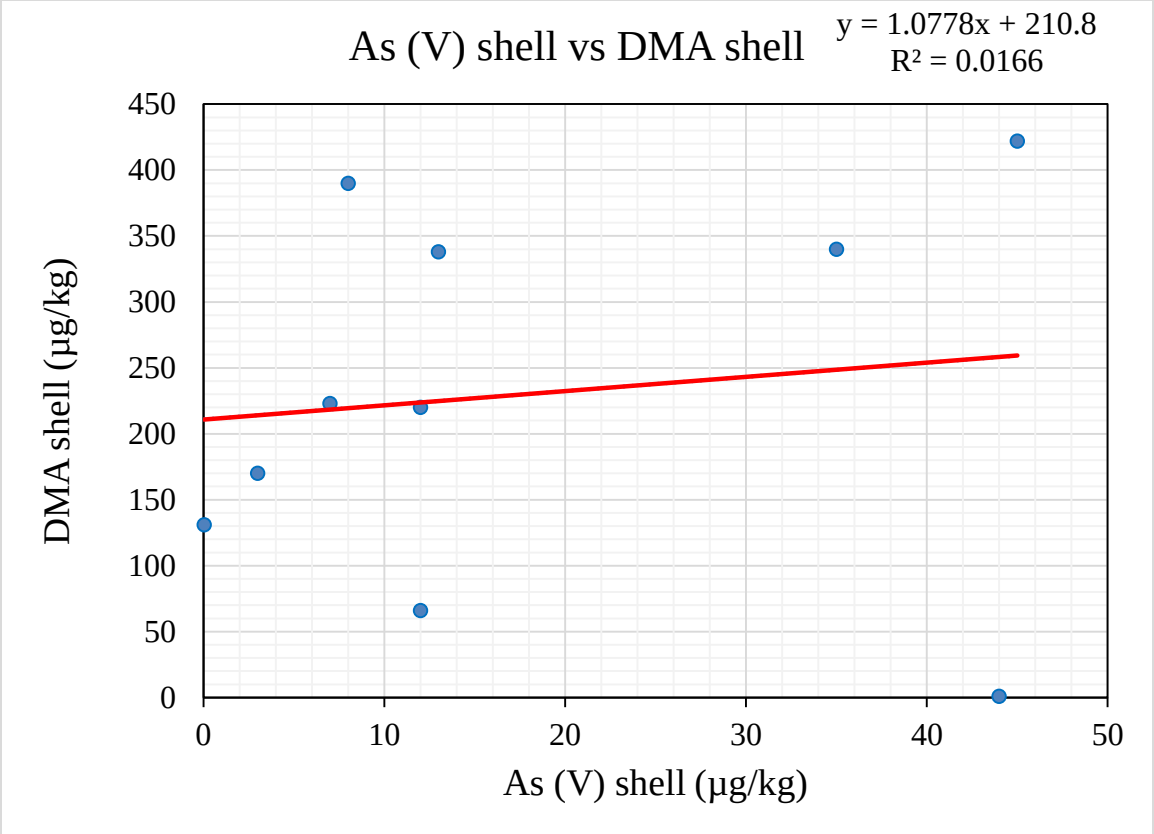


Figure 59: Regression curve of As (V) shell vs DMA shell

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

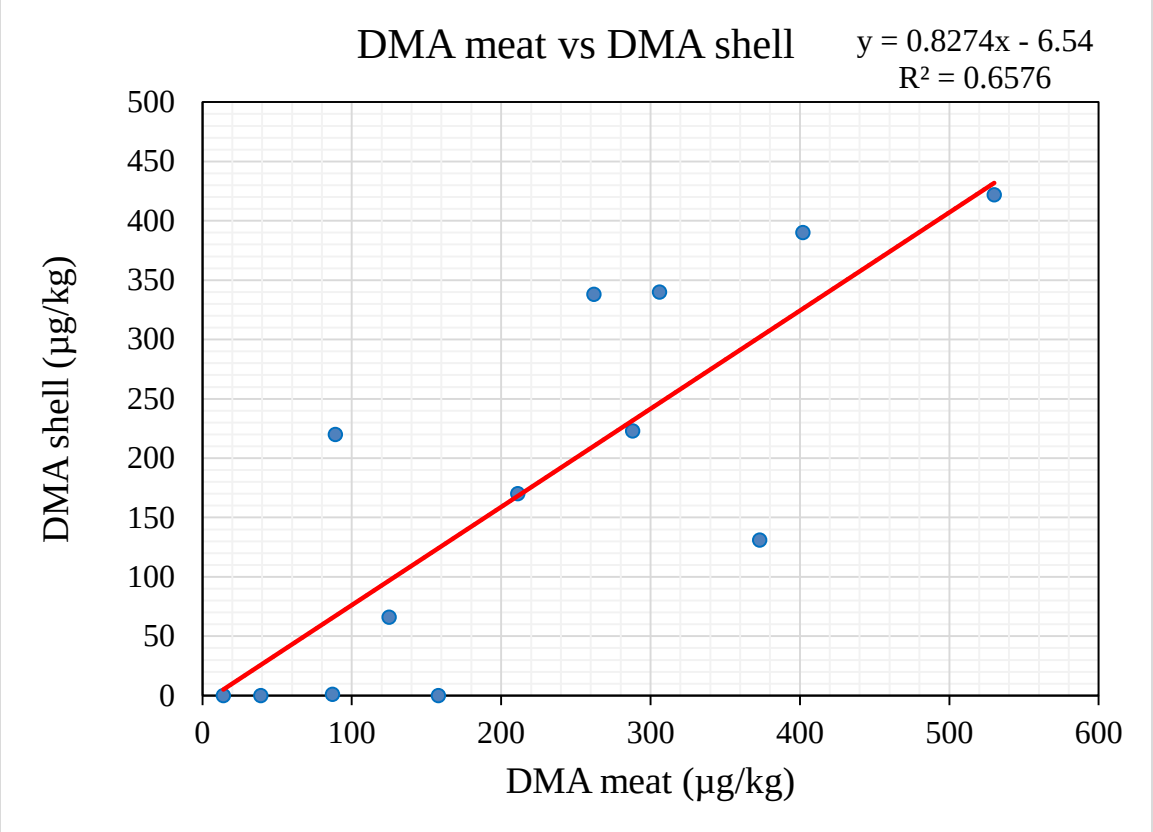


Figure 60: DMA meat vs DMA shell

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

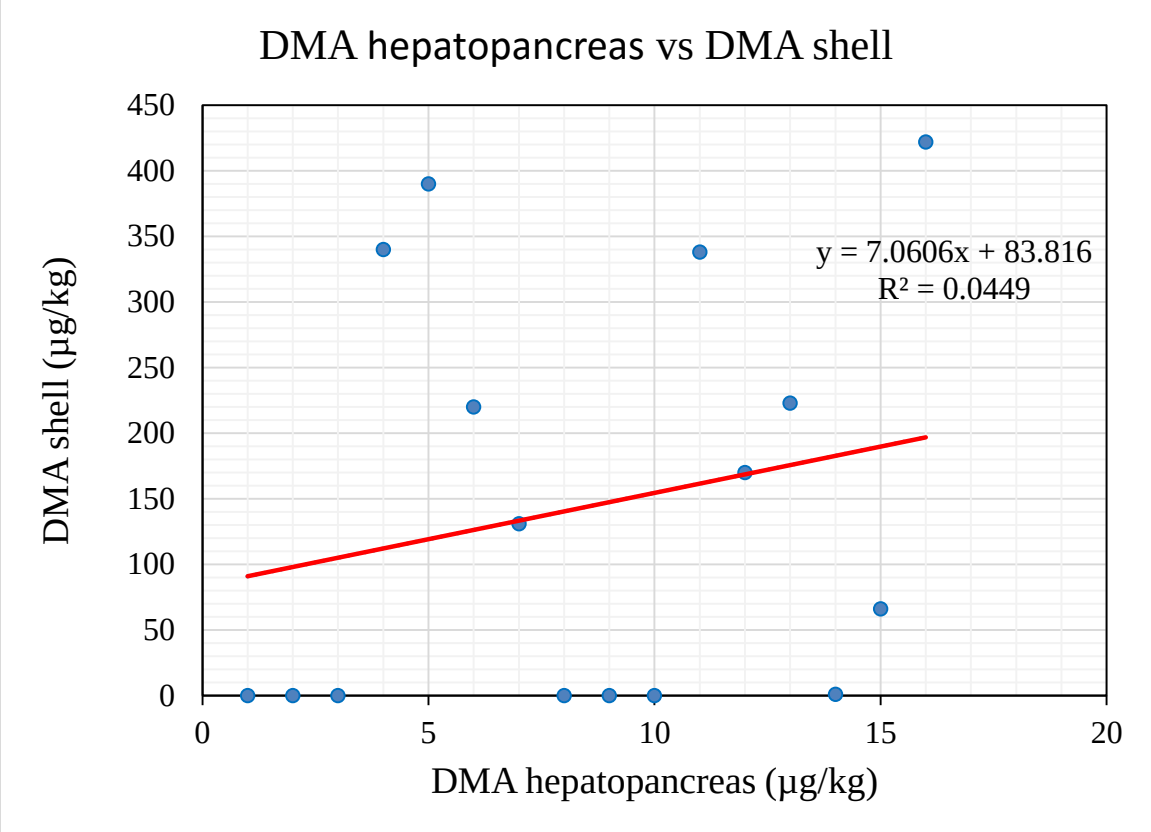


Figure 61: Regression curve of DMA hepatopancreas vs DMA shell

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

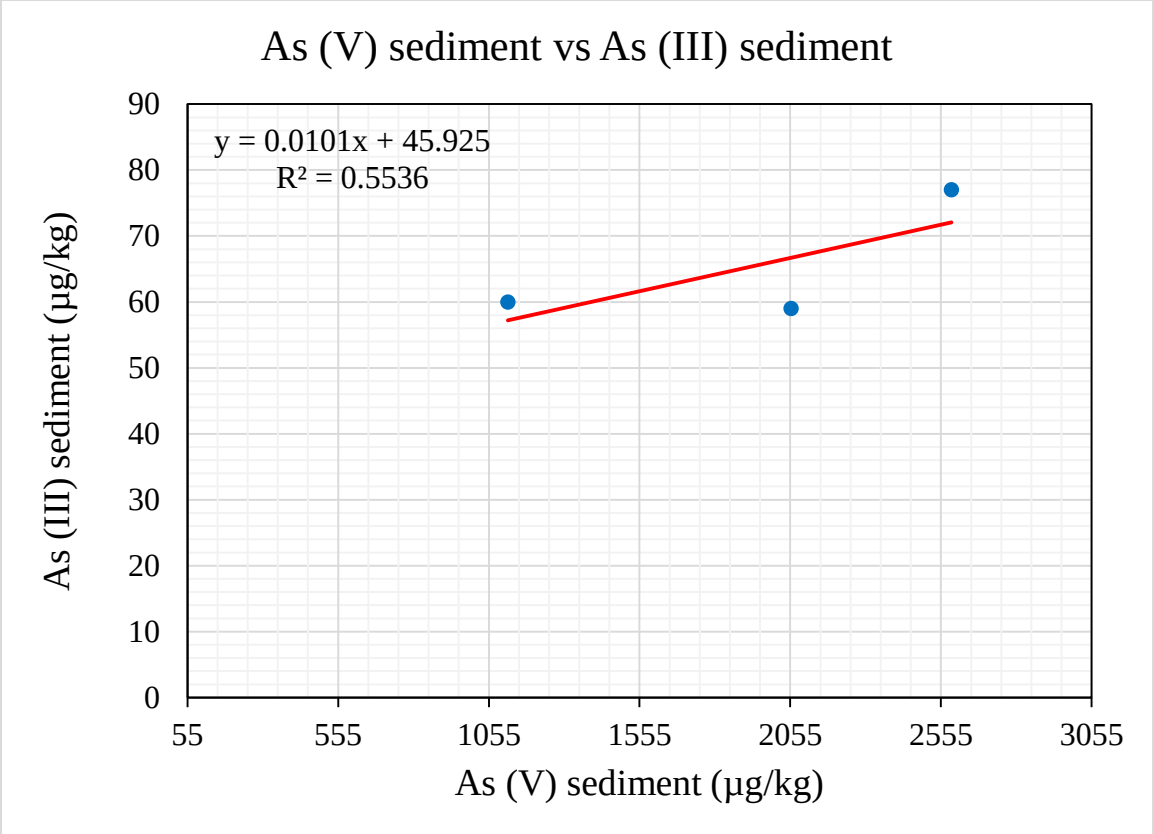


Figure 62: Regression curve of As (V) sediment vs As (III) sediment

H_0: slope of the line = 0; the data are not correlated

H_A: slope of the line ≠ 0; the data are correlated

Conclusion: The slope of the line ≠ 0 ∴ reject the null hypothesis and conclude that the data are correlated

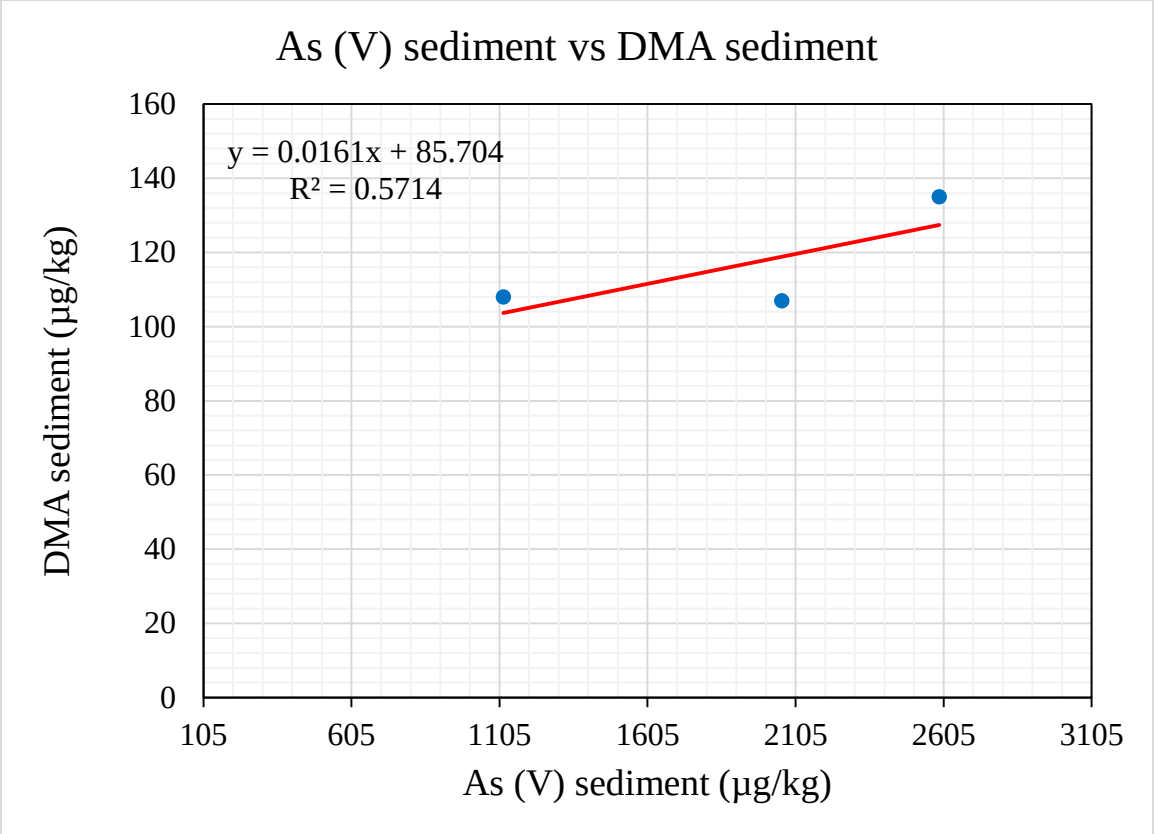


Figure 63: Regression curve of As (V) sediment vs DMA sediment