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PURITY AND ADULTERANT ANALYSIS OF COCAINE SEIZED IN GHANA

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THE REQUIREMENT OF

M. Phil IN FORENSIC SCIENCE

BY

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DECLARATION

I hereby declare that this submission is my own work towards the MPhil and that, to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

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ABSTRACT

There is no report on the purity of cocaine and its adulterants in white powder seized as cocaine in Ghana. This leaves a big gap on data for hazard analysis such as causes of acute cocaine toxicity, overdoses, and fatal reactions. The main goal of the study was to determine the quality of cocaine seized in Ghana by detecting and quantifying the presence of adulterants in seized cocaine samples and also quantify the actual amounts of cocaine in these samples. This work analyzed 45 cocaine samples seized in Ghana in the year 2010, 2013 and 2014. The suspected cocaine samples were analyzed at the forensic laboratory of the Ghana Standards Authority using gas-chromatography / mass spectrometry. The average cocaine content determined were 9.60 mg/g, 9.44mg/g and 7.43mg/g for 2010, 2013 and 2014 respectively. The purity levels were 81.72 ± 4.96 , 80.38 ± 12.23 and 63.25 ± 20.23 . Analysis of variance for the cocaine purity for 2010, 2013 and 2014 at the time of arrest showed a statistically significant difference (P-value 0.0129). With differences arising from samples arrested in 2010 and those in 2014. On the contrary, analysis of variance of cocaine purity for the various years after storage time showed no statistically significant difference (P-value 0.9337). This is testament to the effects of storage conditions on the integrity of the cocaine samples. Adulterants detected were Lidocaine and Caffeine with mean concentrations of 4.30667 x 10⁻⁰⁵ and 0.147064 respectively. None of the samples tested positive for the presence of the adulterant Procaine. There was no significant relationship between the presence of adulterants and the cocaine content. Based on these adulterant findings, measures should be put in place to screen illicit drugs for the presence of these adulterants on arrest.

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CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND

Illicit drugs are known to contain some other substances apart from their purported active ingredient (Fukishima, 2014). Several studies have shown that these additional substances can have unfavorable health effect or can even lead to premature death. The high demand of illicit drugs makes it susceptible to adulteration and dilution and this also enables dealers to increase profit (Fukushima, 2014).

According to Preble and Casey (1969), in New York, through analyzing the various distribution points heroin, it was discovered that a bag of heroin bought on the street in minute quantity was adulterated about twenty-four times. Additionally, a mixture of other substances was added to white powder heroin and cocaine more frequently (Perry, 1975).

Adulterant is a term used to describe compounds that have properties that are pharmacologically, physically and chemically similar to the drug to which they are intentionally added in order to stimulate the drug's effect, to dilute the active ingredient and to increase the weight and volume of the drug to be trafficked according to Bermejo-Barrera *et al.* (1999). Cocaine hydrochloride is mostly marketed in the adulterated form. In the illicit market, apart from the drug being adulterated or diluted, it is found to be contaminated naturally by impurities and compounds that result from the different the production processes also referred to as refining and hydrolysis (Casale and Waggoner, 1991).

In a forensic literature review relating to drug impurities, 48 additives were identified in cocaine analysis comprising of 35 pharmacologically active ingredients, 9 inert additives and 4 volatile compounds (Shesser *et al.*, 1991). Drug dealers, drug users and the general public have made some suggestions for cutting substances which include brick dust, gravy powder, mannitol, chalk, codeine, ground glass, rat poison, household cleaning products, sugar, among others (Best *et al.*, 2004).

Addiction or dependence on illicit drugs leads to a number of public health problems. About 21.8 million Americans above 11 years old had used an illicit drug in 2009 representing 8.7% of the population in this age group. According to the United Nations Office on Drugs and Crimes (UNODC) (2015) world drug report, about 246 million people, representing over 5% of those between 14 and 65 years worldwide used an illicit drug in 2013.

The report likewise shows that coca cultivation kept on declining in 2013 achieving its most reduced levels since 1990. Cocaine utilization however stays high in Eastern and Central Europe, North America and Oceania although recent information demonstrates a declining pattern (UNODC, 2015).

Africa is now the second worldwide trafficker and consumer of illicit drugs with an estimate of 28 million drug users according to the United Nations (UN). The figure for the United States and Canada stands at 32 million. According to the UN the rate of consumption of illicit drugs in Africa is on the rise and this they have attributed to the political instability that exists in the corridors of Africa as well as the porously guarded borders. West Africa has been identified as completely weak in terms of border control

and as such has become a safe haven for big drug cartels from Colombia and Latin America to use as a way to reach Europe (UNODC, 2015).

1.2 PROBLEM STATEMENT

There is no report on the percentage purity of cocaine and its adulterants in white powder seized as cocaine in Ghana. This leaves a big gap on data for hazard analysis such as causes of cocaine overdoses, acute cocaine toxicity and disastrous reactions. With the menace associated with the trafficking of drugs, cocaine is also doctored from the production chain till it reaches the final consumer on the street. The purity of cocaine from street samples from chemical analysis has been found to range between 14 to 75%, with a present average of 40%. Thus from a mathematical point of view, when one buys 1g of cocaine, he/she would have bought 400mg cocaine and more than 600mg of other substances. In individuals whose day by day intake of cocaine is several grams, the imaginable outcome is that, these adulterants may prompt critical toxicological impacts that intensify those of cocaine. Due to the fact that almost all seized cocaine is adulterated, there is indeed no certainty that the myriad of "new" toxicological syndromes associated with cocaine abuse are not as a result of cocaine adulterants.

1.3 OBJECTIVES

1.3.1 General Objective

The objective of this project is to examine the quality of cocaine seized in Ghana.

1.3.2 Specific objectives

- To determine cocaine concentration in seized suspected cocaine powder in Ghana
- To quantify the purity of cocaine in seized suspected cocaine powder in Ghana
- To identify and quantify adulterants and their proportions
- To compare the current purity levels determined to the time of arrest
- To determine the stability levels of seized cocaine on storage

1.4 JUSTIFICATION

There is difficulty in comparing adulteration practices by country or over time because we lack standardized analysis which provides a detailed report on these practices. Most analyses are interested in identifying the kind of adulterants that are present in illicit drug samples. These studies lack the detailed report on the overall constituents of the drug and how much of the adulterants are present in the drug. Generally, giving a report on the percentage of drugs without any adulteration is not a standard practice. However, having both information, further important information concerning adulteration practices would be provided, as well as the threats that they pose to the public health. It has been the practice of several countries in gathering information on the adulteration of illicit drugs that are seized in their countries, but most of this data is not reported on a routine basis. The understanding and public health responses to the adulteration of illicit drugs would be enhanced by providing an early warning system for the identification and rapid adverse effects of these practices.

CHAPTER TWO

LITERATURE REVIEW

2.1 COCA PLANT

The coca plant, known as *Erythroxylum coca* Lamarck is a tropical shrub belonging to the plant family Erythroxylaceae and the order Geraniales (Krol, 1998). This plant family consists of about 250 species with two tropical genera being dicotyledons. Plants from this family has undivided leaves which alternate and toothless. They have small flowers which are in clusters from the axils of the leaves. They also have persistent calyces which have five sepals, five petals which often come with appendages, three styles and ten stamens which are persistent and merged at their bases. They have fruits with single



seeds (Krol, 1998).

Fig 2.1: Leaves of a coca plant (UNODC, 2011)

It has been known to grow to a height of about 2-4 m which is approximately 8 feet and thrives best in warm and moist situations, like the clearing of forests. The leaves are most preferred in the dry state and hence are mostly obtained in drier locations like on the hillside (Krol, 1998).

2.1.1 The Coca Plant Cultivation

Coca productions mostly occurs in marginalized and isolated areas which are qualified by limited control from the government, poverty and violence, lack of or inadequate infrastructure, unclear land rights, that are fields where a small number of international development agencies tend to operate. As a result of quick returns from products that are non-perishable, farmers tend to grow this crop more (UNODC, 2015). Erythroxylum coca is mostly grown in Southeast Asia, Africa, Taiwan and northern South America; however three countries [Colombia, Peru and the Plurinational State of Bolivia] produce most of the world's coca plant. In Peru, coca bush cultivation has declined over the years which was mainly driven by an 18% reduction in the growing of coca shrub cultivation (from 60,400 hectares to 49,800 hectares), reaching its minimum level in the year 2013 from estimates made since the middle 1980s. Also, there was a 9 percent reduction in the Plurinational State of Bolivia from 25,300 hectares in 2012 to 23,000 hectares. Enhanced coca leaf yields and changes in the methods used to separate the extracts are accepted to have balanced decreases in the zone under coca cultivation. There is no report of it being grown in Ghana.

COUNTRY	YEAR								
	2006	2007	2008	2009	2010	2011	2012	2013	
Bolivia	27,500	28,900	30,500	30,900	31,000	27,200	25,300	23,000	
Colombia	78,000	99,000	81,000	73,000	62,000	64,000	48,000	48,000	
Peru						62,500	60,400	49,800	
Peru	51,400	53,700	56,100	59,900	61,200	64,400			
Ghana	ND	ND	ND	ND	ND	ND	ND	ND	
Total	156,900	181,600	167,600	163,800	154,200	155,600 ^d	133,700	120,800	

 Table 2.1: Illicit coca bush cultivation at the global level from 2006 - 2013 in

 hectares

ND – No Data

(UNODC, 2015)

2.1.2 Uses of the Coca Plant

The coca plant was popularly used for producing Coca-Cola soft drink (Plowman, 1982) but has been discontinued since 1903 (Krol, 1998). Chewing of coca leaves decreases hunger and pain. Some workers claim that the leaves of coca plant energize them to work for a prolonged number of hours at high altitudes and often in very cold environments (Krol and America, 2000). In India, the Yukunas are known to consume coca leaves in large quantities on a daily basis and these people have been known to be the healthiest and hard-working Indians. This has not been an issue because these people are also able to make time to cultivate their crops, go on hunting and fishing, and supply their food (Krol, 1998). It has been suggested that, in South America, majority of the old coca plant

usage in South America was for various religious rituals and shaman practices. The use of the coca helps the shaman to easily enter into a state of trance where he is able to communicate with his spiritual deities and call upon them to help him/her with some issues. Late in the 1800's where the Europeans and the North Americans got to know about coca plant, they began to import it for elixirs and use in medicines which they patented (Krol, 1998). It was used as a stimulant, a local anesthetic, and as a "cure" for morphine dependence. This plant was formerly used in the treatment of various diseases especially shingles. It has also been found to possess effective bactericide properties against bacteria that are gram-negative and coccus (Krol, 1998). Currently, this plant is popularly known for the production of cocaine drug.

2.2 COCAINE PRODUCTION

Cocaine, an active sympathomimetic drug is isolated from the leaves of coca plant. The alkaloid in it varies in the leaves with respect to its distribution. Overall alkaloids isolated are $\sim 0.7 - 1.0$ %. In general, lower altitudes provide lower alkaloid contents. The alkaloid content is not uniform in the leaves and decreases after bud break. According to report by Johnson (1996), the stem contains the highest amount of alkaloids. The acidity of the solvent directly affects the solubility of the cocaine and this forms the basis for the extraction process (Krol, 1998). In order to extract, the leaves are dried for a period of time, carbonated water is used to wet them and further dried. This is then followed by soaking in kerosene so as to obtain or extract the alkaloid. Afterwards, sulphuric acid is used to precipitate the extract, which leads to the formation of cocaine sulfate. This cocaine sulfate is then dissolved in water. This solution is precipitated with soda, lime or ammonia, after which a whitish mass, which is an impure basic alkaloid, is obtained. This

whitish mass is mostly used as basic cocaine paste. In the case where potassium permanganate is used to oxidize and separate impurities, the washed basic paste is obtained which is then dissolved in ether or acetone, treated with hydrochloric acid, and made to crystallize as cocaine (Krol, 1998).

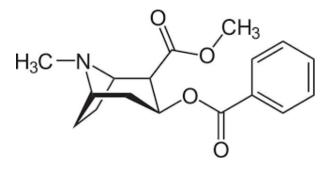


Fig. 2.2: Chemical structure of cocaine

Table 2.2: The Potential for manufacturing 100% pure cocaine in	n selected countries
in tons	

COUNTRY	YEAR								
	2005	2006	2007	2008	2009	2010	2011	2012	2013
Bolivia	80	94	104	113	NA	NA	NA	NA	NA
Colombia	680	660	630	450	488	424	384	333	290
Range								240- 377	249- 331
Peru	260	280	290	302	n.a	n.a	n.a	n.a	n.a
Total	1,020	1,034	1,024	865	842- 1,111	788- 1,060	776- 1,051	714- 973	662- 902

n.a = Not available

(UNODC, 2015).

2.2.1 Methods of Using Cocaine

Snorting is the most common method by which cocaine powder is utilized. This involves sniffing the powder into the nostril. Other ways of using cocaine is by intravenous injection, oral ingestion and sometimes by rubbing on the gums of the user. In the powdered form, cocaine can be smoked. These uses mostly sprinkle the cocaine powder on cigarettes. The powder can be processed into rock form. In this form, users mostly smoke it as crack cocaine or 'freebase' which causes intense feelings of energy and alertness called a high (within 10 to 15 seconds) because of the fact that smoking allows it to reach the brain more rapidly than other methods. As a result, it makes the drug more addictive.

2.2.2 Mechanism of Cocaine Action

As a strong stimulant of the central nervous system, cocaine increases the dopamine level in the brain circuit, thereby regulating the pleasure and movement. Under normal circumstances, the release of dopamine by the neurons in these circuits occurs as a result of response to potential rewards or stimuli like the aroma of delicious food. After its release, it is recycled back to its original form and this shuts off the signal between the neurons. On the contrary, in the presence of cocaine, the dopamine is prevented from being recycled. This causes excessive quantities to accumulate in the synapse, or the junction between the neurons. When this happens, the dopamine signal is amplified and this leads to the ultimate disruption of the normal brain communication and eventually, causes the cocaine's characteristic high as a result of the flood of dopamine. Addiction to cocaine is caused by the continuous use of the drug which further leads to chronic variations in the brain's reward system and other systems in the brain. The continuous usage of cocaine can also lead to the development of cocaine tolerance. According to most cocaine abusers, they seek for more because of the eventual failure to attain as much pleasure as they had when they were first exposed to cocaine. In this case, they try to increase their dosage in order to heighten and prolong their high. This can increase their risk of adverse psychological or physiological effects.

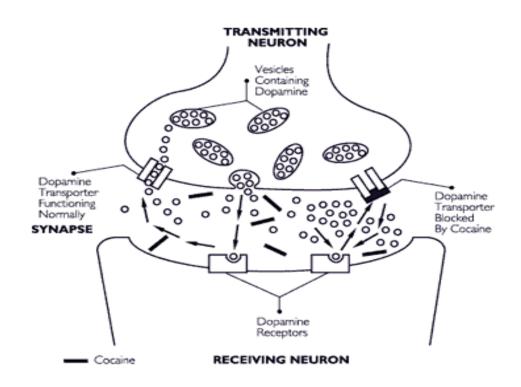


Fig 2.3: Action of cocaine when it enters the brain

2.2.3 Types of cocaine

1. Cocaine hydrochloride

This type of cocaine is mostly common in Australia. It is in the form of white powder which is mostly snorted (sniffed through the nose) or injected into the body. It is destroyed by burning and hence it cannot be smoked.

2. Freebase cocaine

This type of cocaine, also called the alkaloidal cocaine, is changed chemically and can be smoked. Its effect is felt quickly, i.e. the consumer feels high rapidly.

3. Crack cocaine

This is a type of freebase cocaine, mostly smoked, which comes in the form of small crystals or rocks. It is mostly mixed with other stuffs that look almost the same as the crack in order to make the drug go further. These adulterants can have some effects which are destructive or unpleasant. It is quite laborious to determine what the drug actually contains.

2.2.4 Mixing cocaine with other drugs

Cocaine users mostly mix the cocaine with other drugs before using it. They do this in order to manage the effects of the cocaine on their body. Examples of some of these drugs are alcohol, tranquilizers, cannabis or heroin which enables them to sleep. This makes them addict to the cocaine and these drugs because they would need to take cocaine every day to get them going and at the same time, they would have to take these other drugs, like tranquilizers in order to have a good night sleep. Dependence on these drugs can lead to severe physical and psychological problems and make users addicted to their use.

2.2.5 Absorption and Metabolism

There are a number of sufficiently sensitive biochemical assays that are available to evaluate the amount of cocaine and cocaine metabolite in the human body. This has made it possible for researchers to assess the significance of clinical data better and to design proper research studies according to Ambre *et al.* (1982).

Over the years, a number of cocaine studies in the laboratory have provided data from measured plasma cocaine levels in humans. This has led to the development of a fundamental understanding with respect to the relationship that exists between cocaine dose, route of administration and effects, even though some uncertainties still remain.

A group of enzymes known as esterases are known to play a vital role in cocaine metabolism. Esterase activity is higher in the plasma and liver of humans and other mammals whiles moderate esterase activity is observed in other organs such as the brain (Foldes, 1978).

Another group of enzymes known as cholinesterases, also play vital role in cocaine metabolism. These enzymes are also known as plasma cholinesterase or serum cholinesterase, pseudocholinesterase or nonspecific cholinesterase (Stewart *et al.* 1979; Inaba *et al.* 1978).

The level of cholinesterase in the body determines its activity, and the activity determines the rate of cocaine metabolism – lower the cholinesterase activity, the slower the cocaine is metabolized (Jatlow *et al.* 1979). In vitro activity of these enzymes becomes an important consideration in cocaine assay procedures (Jatlow and Bailey 1975).

The activity of plasma cholinesterase is different between individuals and also between species.

The dibucaine number, which is the percentage inhibition of esterase activity by dibucaine, is the usual clinical criterion that is used to test for unusual sensitivity to the muscle relaxant succinylcholine. Judging from in vitro tests, individuals who have low dibucaine numbers are seen to be slow metabolizers of succinylcholine and also slow metabolizers of cocaine (Jatlow *et al.* 1979; Stewart *et al.* 1979). However, other people may have higher activity of cholinesterase that is determined genetically and hence would be expected to be fast metabolizers of cocaine. Cholinesterase activity can be lowered by some disorders such as the presence of carcinoma, liver disease and anticholinesterase drug exposure (Foldes, 1978).

The activity of cholinesterase is higher in some species but not in other species. For instance, it is higher in humans, horses and chimpanzee (and other monkey species) and low in macaques. It is much lower in some mammals such as cats, rats, sheep and dogs and very low in cows. In mice, the cholinesterase active is four times different in various strains. In fetus, infants and aged males, the activity of cholinesterase is much lower and decreases more during pregnancy.

In vivo studies have not often measured levels of cocaine or its metabolite, let alone determined kinetics, but in vitro studies have provided enough evidence to show that the variation in the activity of cholinesterase in the range that is mostly encountered clinically can have some substantial effect on the in vitro cocaine metabolism and metabolites of cocaine.

The route of administration of cocaine may determine the functional impact of very low or very high cholinesterase activity, since metabolism of cocaine can be hepatic or nonhepatic. The impact of intravenously administered cocaine could be higher than smoking, intraperitoneal or subcutaneous administration route, and of course, the rate and the dosage could also interact (Cone *et al.*, 1998).

2.2.6 Prevalence of Cocaine

The cocaine supply worldwide is believed to have started in Plurinational State of Bolivia, Colombia and Peru. However, it has been reported that Western and Central Europe, as well as North and South America has the most prominent cocaine markets and highest rates of cocaine prevalence (UNODC, 2015). The second most prevalent drug in the Europe is cocaine, which is used by about 4.3 to 4.75 million people. According to report by UNODC, about one-third of the global users of cocaine are based in Europe. Approximately ninety per cent of these people are found in the central part of Europe where the prevalence rate of cocaine doubled between the year 1998 and 2006. For Africa, data on the extent of usage of cocaine is limited. However, in South, West and Central Africa, the cocaine usage prevalence has been estimated to be as high as almost 0.7% annual prevalence as at 2013 (UNODC, 2015). Africa has been used as a transshipment centre for the trafficking of cocaine to Europe. African countries such as Ghana, Tanzania and Nigeria have been described as transit countries. The official authorities of the United Republic of Tanzania suggested a change in the trafficking modus operandi because of the fact that traffickers smuggle minute quantities of cocaine in order not to be detected. Cocaine traffickers usually move the cocaine from South America Andean countries to North America and also to Europe across the Atlantic Ocean through the Caribbean or Africa. They do so through several means including air and sea (UNODC, 2015).

2.2.7 Cocaine related deaths

It is quite difficult to determine and name deaths associated with cocaine relative to deaths related to opioid overdose. Deaths due to cocaine results from a number of factors and often, the actual cause is hard to determine. Fatalities as a result of solely pharmacological overdose appear to be rare except for the heavy exposure as in the example of drug couriers who carry the cocaine internally. According to Ghuran and Nolan (2000), majority of cocaine deaths results from chronic toxicity which leads to cardiovascular complications like myocardial ischaemia and infarction and arrhythmias as well as neurological complications like haemorrhagic or ischaemic stroke).

Cocaine related deaths are not dose specific. Even though some users have died from single dose of around 100 mg, others have survived from doses of several grams. Death from cocaine can occur irrespective of the route of administration although it becomes much more risky when the level rises quickly in the brain.

Several cocaine deaths as a result of cardiovascular complications happen in individuals who have certain conditions or risk factors that existed already, caused by chronic cocaine usage. Darke *et al.* (2005) conducted a study in Australia which revealed the fact that, high levels of cardiovascular and cerebrovascular diseases were as a result of cocaine usage.

Deaths caused by cocaine (hard drug) usage may not be identified depending on how these deaths are investigated and recorded. Therefore, it is very important to improve on the method used in investigating deaths and health problems that are associated with the use of cocaine. Additionally, assessing the actual overall and cause-specific death among

the cocaine users would be of great value. This can be achieved through mortality followup studies, among others. Hard drug-drug victims usually recover impromptu, because the effects of hard drug are transient as a result of the short half-life of the drug. Excited delirium may be a common presentation of hard drug overdoses resulting in death (about ten percent) (Sztajnkrycer and Baez, 2005), though this condition will occur conjointly with alternative stimulants, in schizophrenic patients and in patients taking ataractic drug medication. The danger of hard drug toxicity looks to be influenced by the concomitant use of alternative substances, specifically alcohol and diacetylmorphine. This is often a general development in drug deaths, wherever polydrug use appears to extend the danger. In Europe generally, drug deaths involving each opioid and hard drug (and usually alternative substances) are thought of as opioid deaths, as deaths caused by the employment of illicit substances within the absence of opioids are comparatively rare. It's been instructed that hard drug could increase the danger of drug death among opioid users, as hard drug conjointly induces metabolism depression (Jaffe, 1990; Platt, 1997; Tseng et al., 1991). In Europe, hard drug is usually found alongside diacetylmorphine in drug deaths (EMCDDA, 2006), which can mirror the high level of polydrug use among problematic drug users. Over four hundred deaths were reported annually in 2005 and 2006 by the 2006 Reitox report that occurred as a result of hard drug usage. It absolutely was but tough to work out the reason for the death – whether or not it absolutely was due to poisoning of the hard drug, or the result of addition of some substances to the hard drug or a result of the existence of an existing pathological state within the user that was most likely precipitated by the employment of the hard drug. Following a field trial to boost data on substances implicated in drug deaths, many countries have improved their

reportage, however, any work continues to be necessary. The identification and classification of deaths related to hard drug use within Europe still remain unclear. Specifically, the potential that deaths occurring shortly, once are induced by hard drug use, however those aren't poisonings within the strict sense (e.g. deaths resulting from acute cardiac muscle infarctions, arrhythmias or stroke), these aren't known to be induced by hard drug, and thus aren't reported.

2.3 THE GHANAIAN MARKET

Ghana has been recognized as a prominent transit point for cocaine and heroin from South America and Asia respectively. Several Ghanaians are in prison in the USA, Europe and Asia for drug trafficking offences. Ghana does not have ample authority for narcotics and as such traffickers are taking advantage of the weak authority systems to divert and traffic these drugs. The rise in illicit trafficking of hard drugs is one of the contributing factors to the critical economic retrogression that Ghana has been experiencing. Cracked cocaine mixed with baking powder is sold in Ghana and smoked in pipes. The local names for crack cocaine include white powder, energy generator, crazy, Maggie powder, snow, coke, deck, white lady, fire on the mountain, Soroabofo, Aweabonsonsa, and Buu. In October, 2010, 125 kg of cocaine in a container were seized in Ghana and it came from the United States and passed through Panama. No data is currently available on the content, purity and adulterants of perceived cocaine seized samples. This has a lot of consequences because Ghana is used as a major trafficking point.

2.4 COCAINE PRICES

A kilogram of cocaine hydrochloride is sold in Colombia at an average price of US \$ 2,269 per kg, whereas the exact amount can be sold in Central American countries at prices ranging from USD \$2,800 to \$10,000. In Mexico, 1 kg of cocaine hydrochloride can be sold at a price ranging from USD \$15,000 and \$17,000, (UNODC, 2014). In Europe, prices may range between USD \$ 54,000 and USD 57,000, (UNODC, 2014). For an agricultural product, which requires chemical processing which is not expensive, with minimal cost of shipping absent interdiction, the price is quite high. The source of the cocaine and interdiction programs decrease the flow of drugs into a country and this leads to the shortage of supplies in local markets, with the result that sellers dilute the quality of the product and charge higher prices for the limited supply, (UNODC, 2014). As a result of this, source area and transit area programs almost certainly explain high wholesale prices. Although the wholesale price is only a small part of the retail price (closer to \$225,000 per pure kilogram), \$25,000 is not insignificant. Illicit drugs are usually sold at fixed prices which remains constant over time. For example, crack cocaine is usually sold as a \$10 rock and heroin is usually sold in a \$20 bag, regardless of purity or volume, (UNODC, 2014). The size of the rock and the purity of the bag change over time, however, this means that the price per pure gram, changes significantly over time, (UNODC, 2014).

Country	Cocaine retail prices (street prices), US\$ per gram							
	2005	2006	2007	2008	2009	2010		
Austria	101	78	99	110	97	97		
Belgium	51	60	67	72	71	67		
France	94	97	96	103	83	80		
Germany	79	74	86	91	87	87		
United Kingdom	79	87	91	74	62	62		
USA	138	136	129	169	189	169		

Sources: UNODC ARQ data (2008), EUROPOL and UNODC estimates

2.5 COCAINE SEIZURES

Cases on individual seizure of drugs that were reported to the UNODC revealed that trafficking through maritime has risen due to the transporting of cocaine in high amounts recently. This accounts for about sixty percent of the entire amounts that was seized (UNODC, 2015). One important factor that has had an effect on cocaine availability is a successful law enforcement efforts and conflicts that arose between transnational criminal groups. In spite of a reduced seizure in the two major markets of cocaine, that is the United States and Western and Central Europe, cocaine seizures have been stable between 2011 and 2012 as compared with 2012 and 2013 seizures. In the year 2013, about 687 tons of cocaine was seized and 684 tons in 2012 were seized. In the United States, there was 65% decrease in cocaine seizure from 104 to 37 tons. In the Western and central Europe, there was 18% decrease from 71 to 58 tons. (UNODC, 2015). A 2013 report shows that 290 kg of cocaine was seized in Nigeria, 901 kg seized in Ghana and 20

kg in Cote d'Ivoire, (UNODC, 2015). In between the year 2010 and 2012, there was a significant increase in cocaine seizure in the United Republic of Tanzania, in the Eastern part of Africa (UNODC, 2015). In July 2010, Nigerian authorities seized 450 kg of cocaine in the port of Lagos in a container originating from Chile. Two additional seizures of cocaine, making 275 kg, were affected in January 2011. In October 2010, a container from the United States which passed through Panama was seized in Ghana. It was reported to contain about 125 kg of cocaine. Exactly one year after this incident, Cape Verde also seized about 1.5 tons of cocaine. After these events, more cocaine has been seized in Africa and the America in the year 2011. Records show that over 1.4 tons of cocaine which had been disguised in consignment and was headed to Benin. Additionally, about 480 kg of cocaine headed for Nigeria were seized in Brazil in October 2011.

2.6 SCHEDULED DRUGS

Government agencies are provided with a means for managing drug abuse and illegal trade in drugs by the Controlled Substances Act (CSA) through systems that classify drugs according to a set criteria and this creates penalties, including monetary fines and jail terms for people who are involved in the use, sale, distribution and manufacture of controlled substances. Furthermore, CSA's may consist of provisions requiring harsher and minimum, mandatory penalties for specific or repeat offenses; authorizing forfeiture of assets associated with drug offenses; and particularly targeting illegal drug activity occurring near schools or involving minors, drug paraphernalia, safe houses and counterfeit or imitation drugs.

2.6.1 Classification of scheduled drugs

Under the Controlled Substances Act (CSA), drugs and other substances considered to be controlled substances can be sub-sectioned into five schedules. They are named under a particular schedule with respect to whether they have been accepted currently as a medical substance for disease treatment in the United States, their relative potential of being abused and the likelihood of causing dependence when they are abused.

2.6.2 Schedule I Controlled Substances

In the United States, substances belonging to this schedule have no currently accepted use in the medical field which is probably supervised. Hence, there is a high potential for abuse. Marijuana also known as Cannabis, heroin, peyote, lysergic acid diethylamide, among others are examples of Schedule I Substances.

2.6.3 Schedule II Controlled Substances

Substances in this schedule have a high potential for abuse which could lead to severe psychological or physical dependence. Examples of Schedule II narcotics include: hydromorphone, methadone, meperidine, oxycodone and fentanyl. There are other substances under this schedule which include codeine, hydrocodone, morphine and opium. According to the U.S. department of Justice, other controlled substances in this schedule include amobarbital, pentobarbital and glutethimide.

2.6.4 Schedule III Controlled Substances

As compared to substances in Schedule I and II, substances in schedule III have a higher abusing potential. They may lead to moderate or low dependence physically or high dependence psychologically. Products containing at most 90 mg of codeine per dose ((Tylenol with Codeine) belong to this schedule as well as buprenorphine.

2.6.5 Schedule IV Controlled Substances

Substances in this schedule have a low possibility for abuse relative to substances in Schedule III. They include the following substances: diazepam, alprazolam, temazepam carisoprodol, clonazepam, clorazepate, , lorazepam, midazolam and triazolam.

2.6.6 Schedule V Controlled Substances

In this schedule, there is a lower possibility for the abuse of substances as compared to Schedule IV substances. They are basically made up of preparations which contain a limited amount of specific narcotics. Cough syrups which contain at most 200 mg codein in 100 ml or 100 g (Phenergan with Codeine) and ezogabine (U.S Department of Justice controlled substances) are examples of substances in this schedule.

2.7 COCAINE ADULTERANTS

An adulterant is a drug which is added to illicit drugs to enhance it effects. Their function, on addition to the illicit drugs, is to increase the efficacy of the drug while at the same time, decreasing its purity (Cole, 2010). This activity increases the profit of dealers over very small amounts of the pure product extracted. Cocaine adulterants are mostly stimulants and aesthetics which can give the same or similar sense of euphoria and numbing sensation to avoid any form of suspicion by the buyer during testing (Wolford *et al.*, 2012). Some depressants, such as benzodiazepines, which have the ability to ease most of the unwanted side effects of cocaine are sometimes used (Maietti, *et al.*, 2009). Different types and forms of adulterants are used in various available drugs across the globe. A study in Spain identified procaine, lidocaine and caffeine as common adulterants present in seized cocaine samples (Barrio *et al.*, 1997)). A report from Rome, Italy showed the presence of hydroxyzine and levamisole, in seized cocaine samples (Fucci

and De Giovanni, 1998). The type and amount of adulterant added to cocaine vary widely from country to country and even within a country. These adulterants, although pretty harmless individually, have been found to cause various harmful effects on the user depending on how it is used. An article in Clinical Pharmacology and Therapeutics reported how a clot travelled through the blood stream to the lungs (pulmonary embolism) (Chang *et al.*, 2010). This occurs in drugs adulterated with a thick powder such as baking soda or baby powder. Methemoglobinemia, a condition in which there is abnormally high levels of methemoglobin in the blood, has also been associated with users of adulterated cocaine with benzocaine as an adulterant (McKinney *et al.*, 1992).

2.7.1 Phenacetin

At room temperature Phenacetin is a white, odorless monoclinic prism. It dissolves readily in water and its dissolution is temperature dependent. In hot water, it dissolves faster than in cold water. It also dissolves in alcohol, glycerol, and acetone and is slightly soluble in benzene. When exposed to oxidizing agents, iodine and nitrating agents, it appears to be unstable (IARC 1977). It is mainly used as an analgesic. For many years, it was utilized as a pain killer and fever-reducing agent in human beings as well as animals. In 1887, Phenacetin was introduced into the medical field for the disease treatment and it was mainly used for pain relieve until it was discovered to have some side effects such as its implication in kidney disease as a result of its abuse by users. This led to its withdrawal from the U.S. market in 1983 (Flower *et al.* 1985; FDA 1998, 1999 Ronco and Flahault, 1994). Previously, in hair bleaching preparations, phenacetin was used to stabilize hydrogen peroxide according to the IARC (1980) and HSDB (2009). As a result of its close resemblance to pure cocaine, it is one of the key chemicals added to cocaine.

In 1968, it was banned from general and analgesic use after it was discovered to have a link with bladder and kidney cancer. Its ban was later on called off but its legal usage is highly restricted because of its negative consequences. Constant use of this drug is linked with nephrotoxicity leading to incontinence and back and flank pain. A study in Netherland from 1999 to 2007 identified this drug in different percentages in cocaine powder analyzed. In 2006, 48% of phenacetin was present, out of a 593 samples analyzed and 40.6 % present out of 683 sample size, (Brunt *et al.*, 2009).

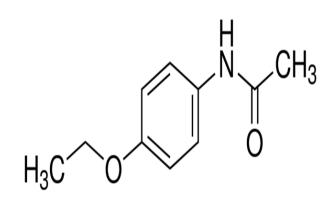


Fig 2.4 chemical structure of Phenacetin

2.7.2 Lidocaine

Lidocaine was first synthesized in 1943 and was used for many years as a local anesthetic agent. Its first reported use as an antiarrhythmic drug was in 1950. Anesthesiologists subsequently adopted lidocaine for treating arrhythmias occurring during surgery, and in 1963 its successful use in treating arrhythmias occurring during and after cardiac operations was described. Lidocaine has since been used extensively in treating ventricular arrhythmias, and administered intravenously is probably the most widely used agent for the treatment and prevention of cardiac arrhythmias after acute myocardial infarction. Lidocaine is a powdery white substance added to cocaine because it can

produce the same numbing effect as cocaine thereby giving the impression of a higher cocaine quality. Adverse cardiovascular and CNS reactions can occur at low doses whiles overdose increases the toxicity of cocaine. A Netherland study on cocaine powder identified 8.2 % of it in 593 samples and 6.4% in 683 samples in 2006 and 2007 respectively, (Brunt *et al.*, 2009).

Oral administration of lidocaine has low bioavailability and a relatively strong liver first pass effect. After intramuscular injection, it is completely absorbed and could be quickly absorbed in the heart, brain, kidney and other tissues with a rich blood supply. The apparent volume of distribution was approximately 1 L/kg; the protein binding rate was about 51%. It is immediately effective after intravenous injection (about45 to 90 s), for 10 to 20 min, $T1/2\alpha$ (distribution half-life) of 10 min, $T1/2\beta$ (elimination half-life) about 1 to 2 h.

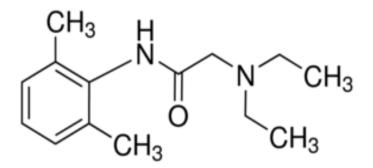


Fig 2.5 chemical structure of Lidocaine

2.7.3 Hydroxyzine

It is an antihistamine drug used primarily for the relief of itching caused by various allergic conditions. It is also used for treating anxiety and tension, and inducing sedation prior to or after anesthesia. The reason for adding it to cocaine is unknown, but potentially used in the final processing stages of cocaine manufacturing. When used in combination with sedative drugs, it can cause unconsciousness. A study on cocaine powder in Netherlands identified 2% of it in 593 samples and 4.4% in 683 samples in 2006 and 2007 respectively, (Brunt *et al.*, 2009).

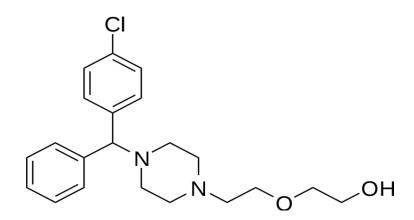


Fig 2.6 chemical structure of Hydroxyzine

2.7.4 Levamisole

Levamisole is a drug of choice for the treatment of ascariasis and also for cancer (antineoplastic) medication. Levamisole is primarily used in veterinary medicine to control parasites in livestock. It is available as an oral tablet or paste, boluses or gels. It also comes in the form of a soluble powdered substance, tropical solutions, feed premixes and injectable solutions. It has also been reported to be used as an adjuvant which aides in the treatment of human malignant diseases. Commercially, it is unavailable in the United States for human use. Levamisole interferes with the growth of cancer cells and slows their growth and spread in the body. It is unknown why it is added to cocaine, however, it is suggested to give a more intense high. It is highly toxic and no longer used in humans. It is known to cause fever and agranulocytosis. A study on cocaine powder in

Netherlands identified 4.5% of it in 593 samples and 11.6% in 683 samples in 2006 and 2007 respectively, (Brunt *et al.*, 2009). Levamisole is readily absorbed independent of the route of administration and is rapidly excreted, predominantly via the urine. An increasing fraction of the portion of the dose remaining in the animal is bound to larger molecules forming non-extractable residues.

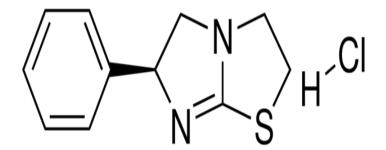


Fig 2.7 Chemical structure of Levamisole

2.7.5 Caffeine

Caffeine has become the most used substances in the world which is considered as a behavioural active substance. Majority of all caffeine sources are from diet especially in beverages from coffee and tea. Acute and especially, chronic caffeine intake appear to have only minor negative consequences on health. Government regulatory agencies have not imposed any restrictions on the usage of caffeine products because only a few users have reported loss of control over their intake of caffeine.

Caffeine is a psychoactive, stimulant drug and it is added to cocaine because it can create similar, although usually milder effect than cocaine. Large doses can cause considerable harms such as sleep disturbances, induce anxiety and increase the risk of variety of health problems, (Cole *et al.*, 2010). A study of street cocaine samples seized in five different Brazilian States between 2011 and 2014 identified 19% of caffeine in the cocaine hydrochloride samples, (Maldaner *et al.*, 2016).

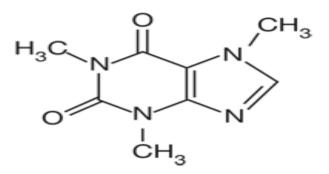


Fig 2.8 Chemical structure of Caffeine

2.8 EFFECTS OF COCAINE AND ADULTERATION

2.8.1 Social Effect

Addiction to illicit drugs such as crack cocaine has effects not only on the individual but his family, friends and the society as a whole. It affects marriages, education and increases crime rate in the society. A US national survey identified 11% of men and women who claimed that substance use (alcohol, marijuana and other hard drugs) played a role in their divorce, (Amato and Previti, 2003). People who are addicted to cocaine often turn to crime as a means of paying for their addiction. People tend to become irrational, agitated, excited or impulsive, which is the most harmful psychopharmacological effects of using crack cocaine. It makes them lose the ability to control their emotions or anger and they eventually vent it in the form of several physical assault. A report by Amato and Previti (2003) on how violent behavior is linked to the use of crack cocaine in one of the first studies clearly showed that almost half of the callers to a nationwide cocaine hotline in the United State had been involved in violent crimes or certain aggressive activities which include robbery, murder, child abuse and other physical assault while using crack cocaine.

2.8.2 Health effect

Cocaine is associated with various toxic effects on the human body, especially on the cardiovascular system (Gold 1997). Cocaine use decreases the blood flow to the brain, increases the heart rate, and elevates the blood components that promote clotting effects that can lead to stroke or heart attack even in those not otherwise at risk for these serious cardiovascular events (Gold 1997). Cocaine may also cause cerebrovascular effects resulting in cerebral ischemia and stroke (Devous *et al.*, 2000). Between 25% and 60% of cocaine-induced strokes can be attributed to cerebral ischemia, (Devous *et al.*, 2000). Although several studies have described the effects of cocaine on the body and nature of cocaine dependence (Foltin *et al.*, 1995) street cocaine usually differs considerably from the pharmaceutical grade cocaine which is used under laboratory conditions (EMCDDA, 2007). However, adverse reactions and other serious health hazards may occur, when a drug turns out to contain another pharmacologically active component (adulterant). Brunt *et al.*, 2009 identified caffeine, lidocaine, benzocaine, procaine and phenacetin adulterants in cocaine samples analyzed in Netherlands.

2.9 OTHER HEALTH EFFECTS OF COCAINE USE

The effects of cocaine use on the body comes in several ways. It constricts the blood vessels, enlarges the pupils and also increase the body temperature, as well as the rate at which the heart beats and the blood pressure. It also has other consequences such as headaches and complications in the gastrointestinal tract like abdominal pain and nausea.

Chronic cocaine users also tend to become malnourished because the use of this drug causes one to have decreased appetite.

Most seriously, people who use cocaine can suffer heart attacks or strokes, which could lead to sudden death. Cocaine-related deaths are usually a result of the heart stopping (cardiac arrest) followed by an arrest of breathing.

Even though cocaine users may not share needles or other drug paraphernalia, these people are also at risk of contracting HIV. The reason is that, when they become intoxicated with cocaine, their judgment is impaired and this can lead to risky sexual behavior.

The method of administration of cocaine determines some of its effect. For instance, regular cocaine snorting can lead to loss of the sense of smell, swallowing problems, bleeding from the nose, hoarseness and a chronic runny nose. Cocaine intake through the mouth can also lead to severe bowel gangrene because of reduced blood flow. When it is injected into the body, it can lead to severe allergic reactions and also an increased risk for contracting diseases like Hepatitis C, HIV and other blood-borne diseases.

Furthermore, irritability, anxiety and restlessness are as result of Bing-patterned cocaine. Abusers could be subjected to paranoia. This is a temporary state of full-blown paranoid psychosis, where victims lose the touch with what is happening in reality and mostly become victims of auditory hallucinations. Cocaine is more toxic when combined with other drugs or alcohol (poly-drug use). Speedball for instance, carries a particularly high risk of fatal overdose. Speedball is the combination of heroin and cocaine.

CHAPTER THREE

MATERIALS AND METHODS

3.1 SAMPLE COLLECTION AND PREPARATION

This study on the purity and adulterant analysis of cocaine seizures in Ghana over a four year interval was conducted between January 2016 and August 2016 in collaboration with the Ghana Standards Authority. Ethical approval for access to the cocaine samples to be used for the study was first sought from the Narcotics Control Board, Ghana. A further clearance was obtained from the Executive Director of the Ghana Standards Authority for the cocaine samples to be released.

As inclusion criteria, the cocaine samples were subjected to the cobalt thiocyanate test also known as the Scott test, which is a screening test for the presence of cocaine. Addition of the cobalt thiocyanate reagent to cocaine hydrochloride results in the surface of the particles turning a bright blue or faint blue for cocaine base. The solution changes back to pink upon adding one or two drops of hydrochloric acid and mixing. Addition of 10 drops of chloroform, vortexing and allowing the solution to settle results in a blue organic layer for both cocaine hydrochloride and cocaine base.

A total of 45 cocaine samples out of 123 samples seized within the four year period by law enforcement agencies and submitted to the forensic department of Ghana Standards Authority were used for this study. These samples had previously been tested for cocaine presence and purity only for law enforcement purposes. No further analyses were carried on these samples. The 45 samples were constituted by weighing and dissolving 0.01g of the white powder which had previously been analyzed for law enforcement purposes in 10mls methanol in a 10ml volumetric flask.

3.2 STANDARD PREPARATION

Standards used for the analysis included a reference cocaine standard of 85.16% purity. This reference cocaine standard was the same one that was used when the samples were previously analyzed. Adulterants used included caffeine, lidocaine and procaine as these are the commonly detected adulterants cited in literature.

The standards were constituted by weighing and dissolving 0.05g of each in methanol to form a single mixture containing all 4 standards in a 10ml volumetric flask. This was further diluted by taking 2mls of the mixture and topping it up to 10mls with methanol to give a solution with concentration of 1000ppm.

3.3 GAS CHROMATOGRAPHY

Analysis was performed at the Forensic laboratory and Pesticide laboratory of the Ghana Standards Authority. The samples were subjected to determination using gas – chromatography / mass spectrometry (GC/MS), an analytical method that combines the features of gas-chromatography and mass spectrometry to identify different substances within a test sample. Gas chromatography / mass spectrometry was performed in a Varian CP-3800 model gas chromatograph equipped with 30m VF-5ms (Agilent J&W) capillary column (0.25mm internal diameter, 0.25µm thickness +10m EZ-Guard) and a Mass Spectrometer Varian Satum-2200 model ion-trap system.

Oven conditions was started at 70°C and held for 1 minute after which it was moved to 180°C at a rate of 25°C and then to 300°C at a rate of 7°C. Flow rate was 1ml/min using Helium gas with an injector temperature of 250°C and injection volume of 1µL.

3.4 STATISTICAL ANALYSIS

Results were processed in a STAR web station. Data obtained was collated and entered into Microsoft Excel and imported into GraphPad Prism software version 6 for statistical analysis. Descriptive and inferential statistics such as percentage calculation of cocaine content, correlation of concentration of adulterants present concentrations with cocaine content and Analysis of variance (ANOVA) to determine significance difference among the concentration of different year groups were employed.

CHAPTER FOUR

RESULTS

Table 4.1: Cocaine concentration and purity at the time of arrest in 2010

	2010	2010		
Sample ID	Concentration (mg/g)	Purity (%)		
9 _B	9.29	79.12		
4	9.23	78.6		
76	10.23	87.1		
14 ₈	9.24	78.7		
6 x	9.90	84.3		
13 ₆	8.88	75.6		
13 ₁	8.88	75.6		
71	10.23	87.1		
14 ₁₀	9.24	78.7		
12 ₂	10.51	89.5		
7 8	10.23	87.1		
6у	9.90	84.3		
9 _A	9.01	76.7		

	2013			
Sample ID	Concentration (mg/g)	Purity (%)		
15A10	9.53	81.2		
10A6	10.54	89.8		
5	7.03	59.9		
10A10	10.54	89.8		
15A6	9.53	81.2		

 Table 4.2: Cocaine concentration and purity at the time of arrest in 2013

 Table 4.3: Cocaine concentration and purity at the time of arrest in 2014

-	2014	
Sample ID	Concentration (mg/g)	Purity (%)
184	3.16	26.9
324	3.80	32.4
258	8.78	74.8
20	3.16	26.9
235	7.62	64.9
326	5.67	48.3
195	6.75	57.5
298	7.96	67.8

215	7.21	61.4
232	7.62	64.9
246	11.48	97.8
251	8.78	74.8
2110	7.21	61.4
29	7.96	67.8
318	9.35	79.6
22f	0.08	0.7
244	11.48	97.8
248	11.48	97.8
316	9.35	79.6
198	6.75	57.5
282	9.07	77.2
238	7.62	64.9
263A	8.21	69.9
183	3.16	26.9
283	9.07	77.2
265B	8.95	76.2
252	8.78	74.8

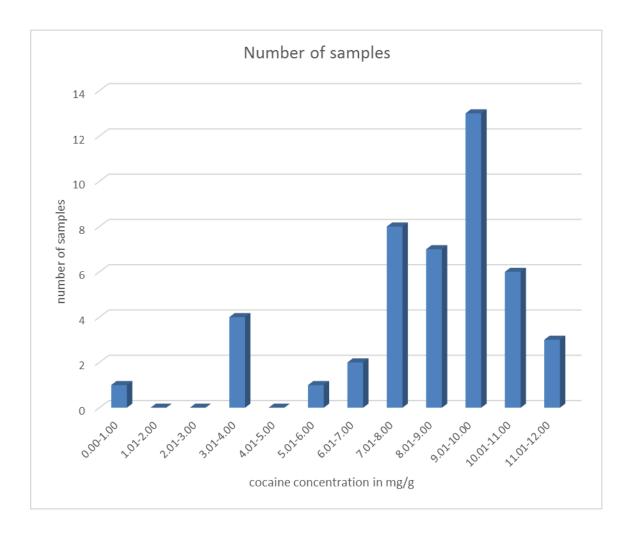


Fig 4.1. Statistical distribution of cocaine content (mg/g of powder) in 45 samples seized in Ghana analyzed at the time of arrest

_	2010	
Sample ID	Concentration (mg/g)	Purity (%)
9B	2.62	22.3
4	2.05	17.46
76	2.27	19.29
148	2.76	23.49
6X	5.02	42.75
136	4.71	40.15
131	4.50	38.34
71	2.07	17.59
1410	2.20	18.73
122	1.39	11.8
78	2.01	17.13
6Y	0.00056	0.0048
9A	5.17	44.03

Table 4.4: Cocaine concentration and purity of 2010 seized samples analyzed in2016

-	2013	
Sample ID	Concentration (mg/g)	Purity (%)
15A10	3.53	30.09
10A6	1.99	17.02
5	3.78	32.22
10A10	1.56	13.26
15A6	1.72	14.62

Table 4.5: Cocaine concentration and purity of 2013 seized samples analyzed in2016

Table 4.6: Cocaine concentration and purity of 2014 seized samples analyzed in2016

	2014		
Sample ID	Concentration (mg/g)	Purity (%)	
184	1.88	16.02	
324	1.70	14.49	
258	4.90	41.76	
20	1.96	16.67	
235	0.01	0.04	
326	1.77	15.03	
195	1.98	16.86	
298	2.10	17.91	

215	3.68	31.36
232	0.00	0.00
246	3.08	26.24
251	1.75	14.87
2110	2.31	19.67
29	1.52	12.93
318	5.10	43.42
22f	0.00113	0.0096
244	2.32	19.72
248	2.23	18.96
316	4.51	38.37
198	0.007991	0.07
282	2.19	18.64
238	0.00	0.00
263A	5.52	46.99
183	3.64	30.96
283	6.66	56.75
265B	2.70	22.99
252	1.94	16.56

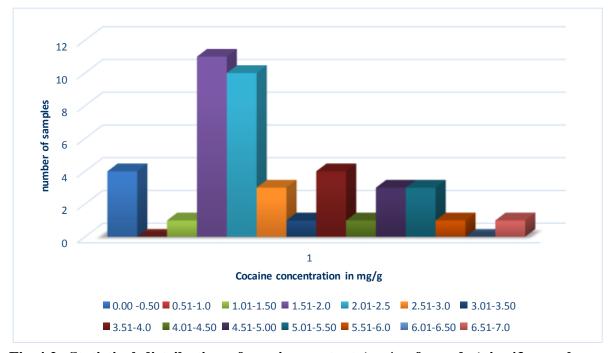


Fig 4.2: Statistical distribution of cocaine content (mg/g of powder) in 43 samples seized in Ghana analyzed in 2016

Year	At the time of arrest	At the time of study	p-value
2010	81.72 ± 4.96	24.08 ± 13.32	< 0.0001
2013	80.38 ± 12.23	21.44±9.00	0.0027
2014	65.65±20.23	20.64 ±15.07	<0.0001

Table 4.7: Paired T-Test for Purity

	2010	2013	2014	p-value	
Mean	81.72 ± 4.96 *	80.38 ±12.23	65.65±20.23 *	0.0129	

Table 4.9: ANOVA for samples analyzed in 2016

	2010	2013	2014	p-value	
Mean	24.08 ±13.32	21.44±9.00	20.64 ±15.07	0.9337	

Table 4.10: Samples containing Caffeine Adulterant

Sample ID	Caffeine (mg/g)	
5	0.071	
20	0.0075	
22F	5.77	
258	0.0087	
326	1.496	

Sample ID	Lidocaine (mg/g)
5	0.000011
10A10	0.000039
15A10	0.000006
20	0.000013
22F	0.00017
235	1.94E-05

Table 4.11: Samples containing Lidocaine adulterant

 Table 4.12: Summary of seized cocaine content analyzed in 2016

Content	Number of Samples (N)	Mean conc. (%)	Standard Deviation	Min conc. (%)	Max. Conc. (%)
Cocaine	43	0.266972	0.156594	0.000056	0.666
Lidocaine	6	4.30667E-05	6.32375E-05	0.000006	0.00017
Caffeine	5	0.147064	0.248607	0.00075	0.577
Procaine	0	0	0	0	0

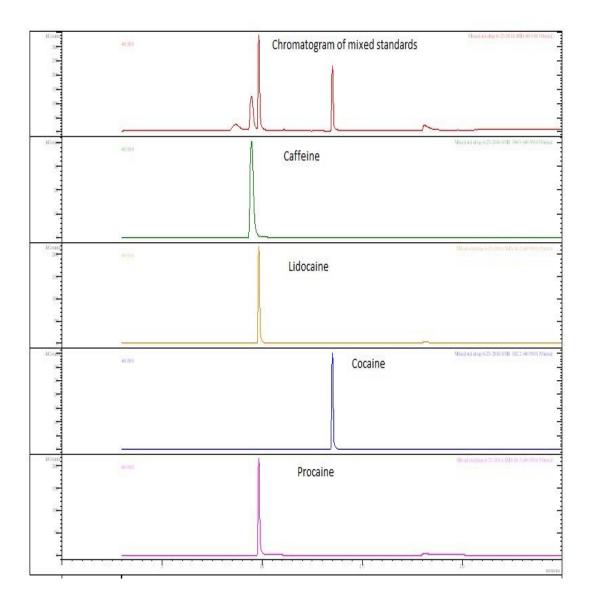


Fig. 4.3: GC Chromatogram of standards

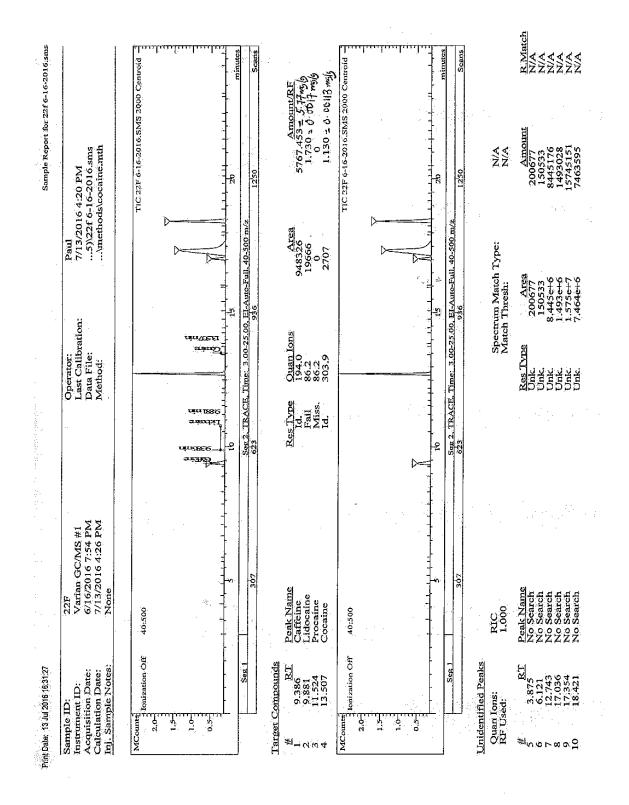


Fig 4.4: Chromatogram of cocaine analysis

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Fig 4.5: Chromatogram of cocaine analysis

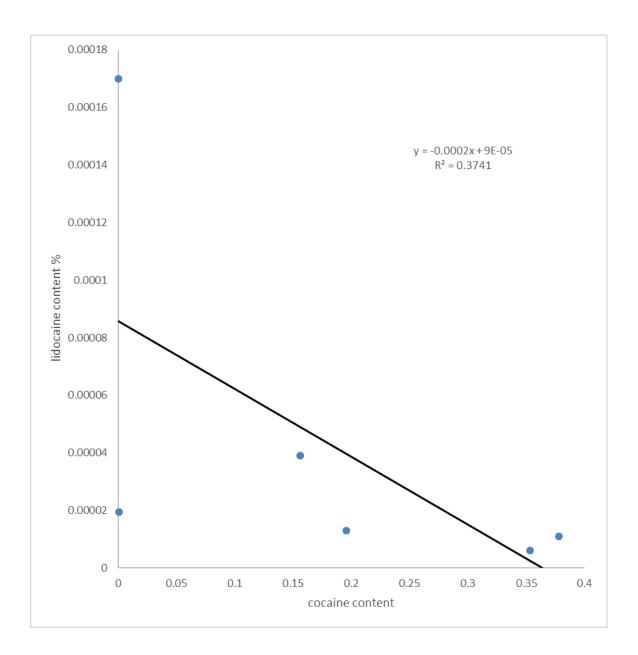


Fig 4.6: Graph of linear relationship between lidocaine and cocaine content

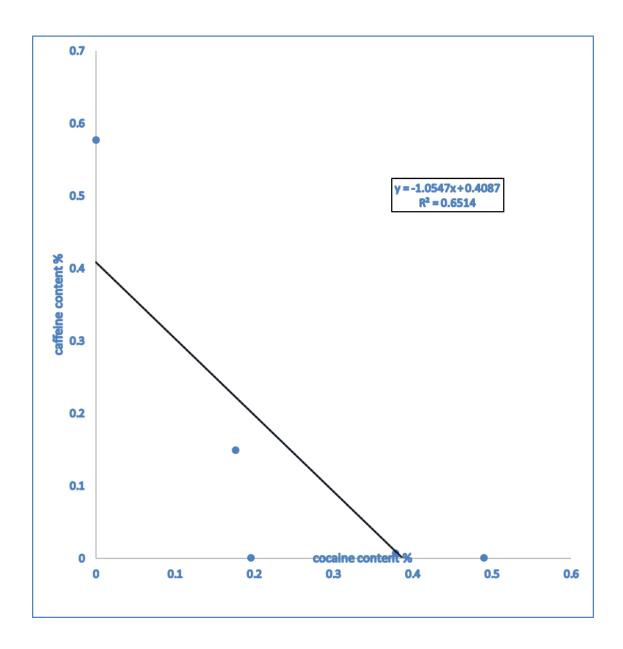


Fig 4.7: Graph of linear relationship between caffeine and cocaine content

CHAPTER FIVE

DISCUSSION

As a result of high demands for illicit drugs, the production of more complicated products which includes the use of several and agents in secret laboratories have increased. Determining the content of seized cocaine samples is not only essential for clinical purposes but also because high purity of cocaine poses a greater risk of addiction. The profiling of illicit drugs such as cocaine can help provide a signature for seized drug samples which can be used for intelligence purposes by law enforcement agencies.

In this study 45 samples seized by the Law enforcement agencies in 2010, 2013 and 2014 were analyzed. The sample size used was based on the fact that out of the 123 samples seized and provided by the Narcotic Control Board to the testing unit, majority had melted and unsuitable for analysis at the time of this study. It must be noted that the cocaine purity and concentration of these samples were previously determined at the time of arrest for their respective years. The purity and concentration of the samples analyzed during this study in 2016 was compared to the purity and concentration of the same samples analyzed at the time of arrest in 2010, 2013 and 2014.

Results of analysis for cocaine concentration and purity as at the time of arrest are presented in table 4 and results for cocaine purity, concentration and adulterants present as at the time of study are presented in table 5. The mean purity of the cocaine samples previously determined was compared to the mean purity of cocaine samples in this present study. This comparison was done using a paired t-test. The results showed a significant difference in the mean cocaine purity for the previously determined samples at the time of arrest and the samples at the time of this present study for each of the corresponding year groups that is 2010, 2013 and 2014. The p-values are <0.0001, = 0.0027 and <0.0001 respectively. The reason for the differences in mean cocaine purity can be attributed to the breakdown of cocaine into its metabolite as a result of storage conditions and length of storage, (Nielsen *et al.*, 2016). This has been reported by Nielsen *et al.*, (2016) who clearly demonstrate that cocaine alkaloid profiles change over time and are most susceptible to sample purity and storage temperature. Two samples (23₂ and 23₈) out of the 45 samples analyzed (~4 %) were found not to contain cocaine in this present study, however these same samples when previously determined both had a cocaine purity of 64.9%. This shows the severity of cocaine breakdown during storage.

The mean cocaine purity of the samples previously analyzed at the time of arrest was 70.49% with a minimum purity of 0.7% and maximum purity of 97.8 %. This mean purity is similar to that of Fukushima *et al.*, 2014 who recorded a mean purity of 71.3%. However the mean purity in this present study after years of storage was 22.73% similar to that of Evrard *et al.*, (2010), who reported a mean purity value of 22% for cocaine detected in adulterated cocaine. The high cocaine purity determined at the time of arrest confirms the assertion by the UNODC (2015) that Ghana is used as a transit point to export cocaine.

			Evrard <i>e</i> (2010)	t al.	Fukushima <i>et al.</i> (2014)		
			N =	: 343	Ν	=404	
	F	Mean	F	Mean	F	Mean	
Cocaine (previously determined)	45	70.49%	343	22	403	71.3	
Lidocaine	6	4.31	36	11	25	0.7	
Caffeine	0.15	0.25	62	17	22	0.4	
Procaine	0	0	0	0	9	0.02	

 Table 5.1: Comparison of seized drug composition with previous studies (mean of purity).

Analysis of variance for the mean cocaine purity of the previously analyzed samples showed a significant difference for the various year groups with p-value of 0.0129. The intergroup difference was identified using a Tukey test with the difference observed between 2010 and 2014 samples. This significant difference is as a result of the high variation in purity levels recorded for samples in 2014 resulting in a low mean value.

The average cocaine content was 8.28mg/g with a minimum content of 0.08 mg/g and maximum content of 11.48. Analytical results obtained in the 45 samples were gathered in intervals of 1.0 mg of cocaine in a gram of powder (Fig.4) and the statistical distribution was studied. The analysis of the graph shows that 62 % of the samples

presented cocaine contents in a range of 7.01 to 10.0 mg/g of powder. The modal interval that corresponds to the maximum frequency was between 9.01 mg/g - 10.00 mg/g. This results contradicts that of Goncalves de Carvalho and Midio, (2003) who had a modal interval of 501-600 mg/g.

Out of the forty five analyzed samples, lidocaine was detected in six (6) whereas caffeine was present in five (5). None of the samples contained procaine. Fewer test samples had the adulterants which are similar to results of Fukushima 2014, who analyzed 403 samples and identified lidocaine in twenty five and caffeine in twenty two. Rodrigues, in 2013, reported the results of analyzing ninety-one cocaine samples seized in 2008-2010 in Brazil. He identified 4 adulterants-caffeine, lidocaine, benzocaine, and boric acid, but in twenty-two out of their ninety-one samples only.

In this study, lidocaine was found to be dominant adulterant detected in our samples. This is consistent with findings by Goncalves de Carvalho and Midio, 2003 who reported lidocaine as the most frequent adulterant in cocaine samples analyzed in Brazil. Lidocaine is the most frequent adulterant used probably in order to simulate cocaine organoleptic characteristics in spite of its high toxicity (Gomez and Rodriguez, 1989).

Analytical results of our samples show a lidocaine concentration ranging from of 0.00006 – 0.00039 mg/g. This gives an average of 0.00431mg/g representing 0.004% lidocaine content in our study. Fukishima *et al.*, (2014) reported a mean concentration of 0.7% for the adulterant lidocaine in cocaine samples analyzed in Brazil. Also Shneidr and Meys (2011) reported a mean of 1.9% for lidocaine in samples seized in Germany. These values are higher than the mean value of 0.004% reported for lidocaine in our samples analyzed.

The amount of caffeine present in our samples ranged from 0.0087 - 5.77 mg/g with a mean concentration of 1.47 mg/g (std = 0.25). This amount which represents 13.55% is higher than 0.4% and 5.1% reported for caffeine presence in adulterated cocaine by Fukishima *et al.*, (2014) and Shneider and Meys (2011) respectively. Fig 5 and 6 show the relationship between each detected adulterant and cocaine content. There was no correlation between the adulterants found in the samples and the cocaine content. All of this increases the need to establish a database to track the changes and materials that are identified in different areas, not only to look for the distribution channels but also to inform the users and health care workers about what they may expect in emergency cases due to overdose or toxic substances.

CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

Purity of cocaine seized by law enforcement agencies in Ghana between 2010 and 2014 showed very high purity levels similar to those seized in other countries. The average cocaine content determined were 9.60 mg/g, 9.44mg/g and 7.43mg/g for 2010, 2013 and 2014 respectively with corresponding purity levels of 81.72 ± 4.96 , 80.38 ± 12.23 and

 63.25 ± 20.23 . This is testament to the assertion that Ghana is usually a transit point for

the movement of cocaine further into European countries. Adulterants detected in the cocaine samples were lidocaine and caffeine with mean concentrations of 4.30667×10^{-05} and 0.147064 respectively. No procaine was identified in any of the samples analyzed. Though the values of the adulterants may seem low to elicit any toxicological effect, it is possible that the adulterants may have undergone degradation owing to the length of time under storage and prevailing storage conditions. My recommendation is for seized cocaine samples to be tested for presence of adulterants and quantified as part of routine analysis on cocaine to help monitor changes in cutting agents over time. Looking at the cocaine breakdown that has occurred since the time of arrest till the time of this study, it is my recommendation that storage conditions be significantly improved by custodians of seized cocaine samples so that if ever a case has to be recalled after a number of years, results may not differ too significantly from initial results obtained.

Further studies can be carried out on the presence of diluents in seized cocaine samples in Ghana.

REFERENCES

Amato P. R., Previti D. (2003). People's reasons for divorce in: gender, social class, the life course, and adjustment. J Fam Issues; 24: 602–26.

Ambre, J.J., Ruo, J.H.; Smith, G.L. Backer, D.; and Smith, C.M. (1982). Fegonine methyl ester, a major metabolite of cocaine. J Analytical Toxicol, 6:26-29.

Ames B. N., Magaw R., and Gold L. S. (1987). Ranking possible carcinogenic hazards. *Science* Vol. **236**: 271–80.

Apostolakos M. J. and Varon M. E. (1996). Antiarrhythmic and anti-ischemic properties of calcium-channel antagonists. *New Horiz* 1996; **4**: 45–57.

Barrio, G., Saavedra, P., De la Fuente, L., Royuela, L., Villanueva, M. D., Balseiro, A., Cordoba, C., Dominguez, C., Esquerra, J. L., Garrido, D., Gutierrez, H., Dominguez, L., Menendez, M., Repetto, M., Senra, L., Servera, J., Vicente, A. and Yeregui, C. (1997) 'Purity of cocaine seized in Spain, 1985-1993: Variations by weight, province and year of seizure', *Forensic Science International*, 85(1), pp. 15–28.

Behrman A. D. (2008) Luck of the draw: common adulterants found in illicit drugs. *J Emerg Nurs* Vol. **34**: 80–2.

Bermejo-Barrera, P., Moreda-Piñero, A., Moreda-Piñero, J., Bermejo-Barrera, A., Bermejo-Barrera, A.M., (1999). A study of illicit cocaine seizure classification by pattern recognition techniques applied to metal data. *J. Forensic Sci.*, 44, pp 270-275.

Best, D., Beswick, T., Gosssop, M., Rees, S., Coomber, R., Witton, J. and Strang, J. (2004). From the deal to the needle: Drug purchasing and preparation among heroin users in drug treatment in south London. *Addiction Research and Theory*, 12(6), pp 539-548.

Boghdadi, M. S. and Henning, R. J. (1997), 'Cocaine: athophysiology and clinical toxicology', *Heart and Lung* 26, pp. 466–483.

Brownlow, H. A. and Pappachan J. (2002), 'Pathophysiology of cocaine abuse', *European Journal of Anaesthesiology* 19, pp. 395–414.

Brunt, T.M., Rigter, S., Hoek, J., Vogels, N., van Dijk, P., Niesink, R.J.M. (2009). An analysis of cocaine powder in the Netherlands: Content and health hazards due to adulterants. *Addiction*, *104*(*5*), 798-805.

Buttner A., Mall G., Penning R., Sachs H. and Weis S (2003). The neuropathology of cocaine abuse. *Leg Med (Tokyo)* Vol. **5**: S240–2.

Casale, J.F. and Waggoner Junior, R.J., (1991). A chromatographic impurity signature profile analysis for cocaine using capillary gas chromatography. *J. Forensic Sci.*, 36, pp 1312-1330.

Chambers, H. F., Morris D. L., Tauber, M. G. et al. (1987), 'Cocaine use and the risk for endocarditis in intravenous drug users', *Annals of Internal Medicine* 106, pp. 833–836.

Chang, A., Osterloh, J. and Thomas, J. (2010) 'Levamisole : A Dangerous New Cocaine Adulterant', *Clinical Pharmacology & Therapeutics*. Nature Publishing Group, 88(3), pp. 408–411. doi: 10.1038/clpt.2010.156.

Cole C., Jones L., McVeigh J., Kicman A., Syed Q., Bellis M. (2010). Centre for Public Health Engagement Liverpool: Liverpool John Moores University;. Cut: A Guide to Adulterants, Bulking Agents and Other Contaminants Found in Illicit Drugs.

Cole, C., Jones, L., McVeigh, J., Kicman, A., Syed, Q. and Bellis, M.A., (2010). Cut: A guide to adulterants, bulking agents and other contaminants found in illicit drugs, pp 9-10.

Cone E., J., Tsadik, A., Oyler, J., Darwin, W., D. (1998). Cocaine metabolism and urinary excretion after different routes of administration. Ther drug Monit. 20(5): 556 - 560.

Darke, S., Kaye, S. and Duflou, J. (2005), 'Cocaine related fatalities in New South Wales, Australia 1993–2002', *Drug and Alcohol Dependence* 77(2), pp. 107–114.

Devous, M. D., Ph, D., Ruiz, P. and Ait-daoud, N. (2000) 'Reviews and Overviews Treatment Advances for Cocaine-Induced Ischemic Stroke : Focus on Dihydropyridine-Class Calcium Channel Antagonists', (15), pp. 1191–1198.

Egred, M. and Davis, G. K. (2005), 'Cocaine and the heart', *Postgraduate Medical Journal* 81(959), pp. 568–571.

European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) (2006a), Annual report 2006: the state of the drugs problem in Europe, Lisbon. **European Monitoring Center for Drugs and Drug Addiction**, (2007). Cocaine Related Offences, Office of Official Publications of the European Communities, Luxemburg, pp 30.

Foldes, F.F. (1978). Enzymes in anesthesiology. In: Foldes, F.F., ed. Enzymes in Anesthesiology. New York: Springer-Verlag, pp.91-168.

Foltin RW, Christiansen I, Levin FR, Fischman MW (1995). Effects of single and multiple intravenous cocaine injections in humans maintained on methadone. J Pharmacol Exp Ther. Vol. 275:38–47.

Frishman W. H., Del Vecchio A., Sanal S. and Ismail A. (2003). Cardiovascular manifestations of substance abuse part 1: cocaine. *Heart Dis* Vol. **5**: 187–201.

Fucci, N. and De Giovanni, N. (1998) 'Adulterants encountered in the illicit cocaine market', *Forensic Science International*, 95(3), pp. 247–252.

Fukushima A.R., Carvalho VM, Carvalho D.G., Diaz E, Bustillos J.O. and Spinosa Hde S, Chasin A.A. (2014). Purity and adulterant analysis of crack seizures in Brazil. Forensic Science Int. Vol. 243: pp 95-8.

Geyermek L. (1998). Pharmacology of Antimuscarinic Agents. In: Kane H., editor. *Pharmacology and Toxicology: Basic and Clinical Aspect Series* (Hollinger, M. A., editor). Boca Raton, FL: CRC Press; p. 1–477.

Ghuran, A. and Nolan, J. (2000), 'Recreational drug misuse: issues for the cardiologist', *Heart* 83, pp. 627–633.

Gold M., S. (1997). In: Clinical aspects of substance abuse: A comprehensive text book. 3rd Edition Lewinson, editor. Wilkinson and Willey; Baltimore.

Inaba, T.; Stewart, D.J.; and Kalow, W. (1978) Metabolism of cocaine in man. Clin Pharmacol Ther, 23:547-552.

Jaffe J. H. (1992), 'Drug addiction and drug abuse', in A. Goodman Gilman, T. W. Rall, A. S. Nies and P. Taylor (eds), *Goodman and Gilman's: the Pharmacological Basis of Therapeutics*, eighth edition, Mc Graw-Hill International, New York, p. 542.

Jatlow, P., and Bailey, D. (1975). Gas chromatographic analysis for cocaine in human plasma with use of a nitrogen detector. Clin Chem, 21:1918-1921.

Jatlow, P.; Barash, P.G.; Van Dyke, C.; Radding, J.; and Byck, R. (1979). Cocaine and succinylcholine sensitivity: A new caution. Anesth Analg Curr Res, 58(3):235-238.

Johnson, Emanuel. Alkaloid content in *Erythroxylum coca* tissue during reproductive Development. Phytochemistry. V. 42 1996. P. 35-38.

Kaye, S. and Darke, S. (2004), 'Non-fatal cocaine overdose among injecting and noninjecting cocaine users in Sydney, Australia', *Addiction* 99, pp. 1315–1322.

Krol, C. (1998). The Coca Plant. Ethnobotanical Leaflets, 2, p.5.

Maietti, S, Castagna, F., Molin, L., Ferrara, S. D. and Traldi, P. (2009). JMS Letters: Cocaine Adulterants Used as Marker Compounds. *Journal of Mass Spectrometry*; 44: 1124-1126.

Maldaner, A. O., Zacca, J. J., Melo, R. C. A., Zancanaro, I., Oliveira, C. S. L. and Kasakoff, L. B. (2016) 'Chemical Profiling of Street Cocaine from Different Brazilian Regions', 27(4), pp. 719–726.

McKinney, C, D¹, Postiglione K. F. and Herold D.A (1992). Benzocaine-Adultered Street Cocaine in Association with Methemoglobinemia. *Clinical Chemistry*; 36 (4): 596-597.

Mittleman, M. A., Mintzer, D., Maclure, M. et al. (1999), 'Triggering of myocardial infarction by cocaine', *Circulation* 99, pp. 2737–2741.

Mouhaffel, A. H., Madu, E. C., Satmary, W. A. and Fraker, T. D. (1995), 'Cardiovascular complication of cocaine', *Chest* 107, pp. 1426–1434.

Perry, D.C., (1975). Heroin and cocaine adulteration. *Clinical Toxicology*, 8(2), pp 239-243.

Plowman, T. (1982) 'The identification of coca (Erpthroxylum species): 186M910', pp. 329–353.

Pozner C. N., Levine M. and Zane R. (2005). The cardiovascular effects of cocaine. *J Emerg Med* Vol. **29**: 173–8

Pozner, C. N., Levine, M. and Zane, R. (2005), 'The cardiovascular effects of cocaine', *Journal of Emergency Medicine* 29, pp. 173–178.

Preble, E. and Casey, J.J. (1969). Taking care of business - The heroin user's life on the street. *Substance Use & Misuse*, 4(1), pp 1-24.

Shesser, R., Jotte, R. and Olshaker, J. (1991). The contribution of impurities to the acute morbidity of illicit drug use. *American Journal of Emergency Medicine*, 9(4), pp 336-342.

Staack R. F., Paul L. D., Schmid D., Roider G., Rolf B (2007). Proof of a 1-(3-chlorophenyl) piperazine (mCPP) intake: use as adulterant of cocaine resulting in drug–drug interactions? *J Chromatogr B Analyt Technol Biomed Life Sci V.* **855**: 127–33.

Steven, B. and Karch, M. D. (1999), 'Cocaine: history, use, abuse', *Journal of the Royal* Society of Medicine 2, pp. 393–397

Stewart, D.J.; Inbaba, T.; Lucassen, M.; and Kalow, W. (1979). Cocaine metabolism: Cocaine and norcocaine hydrolysis by liver and serum esterases. Clin Pharmac Ther, 25:464-468.

Sweetman S. C. (2006). *Martindale: the Complete Drug Reference*, 35th edn. London: Pharmaceutical Press pp 20- 79.

Sztajnkrycer, M. D. and Baez, A. A. (2005), 'Cocaine, excited delirium and sudden unexpected death', *Emergency Medical Services* 34(4), pp. 77–81.

The Analytical Sanitary Commission, (1854). Records of the results of microscopical and chemical analyses of the solids and fluids consumed by all classes of the public. Drugs and pharmaceutical preparations. "To attack vice in the abstract, without attacking persons, may be safe fighting indeed, but it is fighting with shadows. "Opium and its adulterations. *Lancet*, 63(1587), pp 107-109.

Tseng, C. C., Derlet, R. W. and Albertson, T. E. (1991), 'Cocaine induced respiratory depression in urethane-anesthetized rats: a possible mechanism of cocaine-induced death', *Pharmacology, Biochemistry Behaviour* 39, pp. 625–633.

United Nations Office on Drugs and Crime, (2006). World Drug Report, Vol.2 p. 368

United Nations Office on Drugs and Crime, (2011). World Drug Report.

United Nations Office on Drugs and Crime, (2015). World Drug Report.

UNODC, (2014). Colombia-Coca cultivation survey 2013. United Nations Office on Drugs and Crime.

Vasica, G. and Tenant, C. C. (2002), 'Cocaine use and cardiovascular complications', *Medical Journal of Australia* 177, pp. 260–262.

Weiner A. L., Bayer M. J., McKay C. A. Jr, DeMeo M., Starr E. (1998). Anticholinergic poisoning with adulterated intranasal cocaine. *Am J Emerg Med* Vol. **16**: 517–20.

Wolford A., McDonald T.S., Eng H., Hansel S., Chen Y., Bauman J., Sharma R., Kalgutkar A.S. (2012).Immune-mediated agranulocytosis caused by the cocaine adulterant levamisole: a case for reactive metabolite(s) involvement. Drug Metab. Dispos. Vol.40:1067–1075.