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Estimation of Lead Level in Blood among South Delhi Population: A Cross Sectional Autopsy Based Study

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Abstract

The widespread use of lead causes accumulation results in poisoning known as plumbism. The acceptable reference range for lead in human beings is 25μg/dl. Present study shows the blood lead levels of South Delhi population. 5 ml of blood was taken and digested followed by analysis using trace metal analyser (TMA). The results obtained were further categorized onthe basis of sex and age. Out of 250 autopsies 77 were females having mean blood lead level 0.41 μg/dl while rest 173 weremales having mean blood lead level 16 μg/dl. According to age-wise distribution the highest mean blood lead level is found in the age group of 41-60 years i.e. 20.73 μg/dl.

Keywords: Lead; TMA; Blood; Microwave Digestor; Poisoning.

Introduction

The widespread use of lead because of its properties leads to its poisoning termed as plumbism, colicapictonum, aturnism, devoncolic, or painters colic. It is a medical condition caused by increased lead levels in the body [1].

Symptoms include headache abdominal pain memory loss kidney failure, male reproductive problems, and weakness, pain, or tingling in the extremities. The symptoms in children's are different which includes loss of appetite, abdominal pain vomiting weight loss constipation anaemia kidney failure irritability lethargy learning disabilities and behaviour problems [2]. Children may also experience hearing loss, delayed growth, drowsiness, clumsiness or loss of new abilities specially speech skills [3].Symptoms in childrens may appear at lower blood lead levels [4].

One of the main causes for the pathology of lead is that it interferes with the activity of an essential enzyme called delta-aminolevulinic acid dehydratase or ALAD, which is important in the biosynthesis of heme, the cofactor found in hemoglobin [5]. Lead also inhibits the enzyme ferrochelatase, another enzyme involved in the formation of heme. Ferrochelatasecatalyzes the joining of protoporphyrin and Fe^{2+} to form heme [6]. Lead's interference with heme synthesis results in production of zinc protoporphyrin and the development of anemia [4]. Another effect of lead's interference with heme synthesis is the buildup of heme precursors, such as aminolevulinic acid, which may be directly or indirectly harmful to neurons. Lead interferes with the release of neurotransmitters, chemicals used by neurons to send signals to other cells [7]. It interferes with the release of glutamate, a neurotransmitter important in many functions including learning, by blocking N- methyl D -aspartic

acid (NMDA) receptors. The targeting of NMDA receptors is thought to be one of the main causes for lead's toxicity to neurons [8]. The current reference range for acceptable blood lead concentrations in healthy persons without excessive exposure to environmental sources of lead is less than 5 µg/dl for children and less than 25 µg/dl for adults [9].

Material & Methods used

In this study all deaths from south Delhi reported at mortuary, AIIMS on whom medico-legal autopsy was conducted, were included. 5 ml of blood was collected and preserved in EDTA vials and brought to the toxicology laboratory to estimate the blood lead level using trace metal analyzer. Subsequently data obtained wasanalyzed by using appropriate statistical methods to estimate the blood lead level.

Exclusion Criteria

- All decomposed dead bodies.
- Unknown dead bodies.
- Dead bodies from other than south Delhi region and from outside of Delhi, although died at AIIMS and not from south Delhi jurisdiction.
- All the non medico-legal cases whose death occurred at AIIMS.

Preparation of Sample and Analysis

Instrumentation/Accessories and Operating Conditions

Voltammetric determination of Pb was performed by Trace Metal Analyzer model 797 VA Computrace from Metrohm AG Ltd, Switzerland (Figure 1). It is a three-electrode system consisting of Multy Mode Electrode MME (mercury) as working electrode, Platinum (Pt) as auxiliary electrode and Ag/AgCl/ 3M KCl as reference electrode. The operating parameters are given in Table 2. Nitrogen gas of high purity was used. Micropipette of Eppendorf make of volume 10-100 µl and 100-1000 µl were used. pH measurements were done with pH meter of model Inolab WTW series at ambient temperature of laboratory. Whatman filter paper 41 Ashless Circles of 125 mm Cat No 1441 125 from Whatman International Ltd Maidstone England were used.

Reagents/Chemicals

Suprapure acetic acid from Merck, Germany, Nitric acid, Liquor ammonia, ammonium oxalate, Lead nitrate from Merck Specialiaties Private Limited, Mumbai, sulphuric acid from Qualigens Fine Chemicals, Mumbai, ultrapure water from Rion India were used.

Preparation of Ammonium Acetate Buffer

Ammonium acetate buffer was prepared by dissolving 5.55 ml of acetic acid in 10 ml of ultrapure water. Then 3.7 ml of Suprapure ammonia was added slowly and pH was adjusted to 4.6 by adding few drops of Suprapure ammonia. Finally the volume was made upto 50 ml with ultrapure water.

Preparation of Standard Solution of Lead

1000 ppm solution of Lead was prepared from Lead Nitrate. 1 ppm standard of lead was prepared by diluting 0.1ml of 1000 ppm stock solution of lead to 100 ml ultrapure water.

Sample Preparation

Vessel of microwave digester was cleaned up by Nitric acid (HNO_3) and H_2O mixture (1:1) and dried. One gram sample was transferred into the linear vessels.15 ml of 35 % HNO_3 was added into each vessel and the mixture was left for few minutes for auto gas. In the reference vessel, 1 ml of water was added along with 15 ml of 35% HNO_3 for sample blank. Vessel carrousel was loaded in the microwave digestion oven and the digestion machine was run according to program given in Table 2.

After completion of run, microwave digestion was kept for cooling. After cooling, the vessels were opened and digested material was completely transferred in 50 ml volumetric flask with the help of ultrapure water and final volume was made upto 50 ml with ultrapure water.

Anodic Stripping Voltammetricmeasurements (Figure 1)

10ml ultrapure water and 1ml of acetate buffer (pH 4.6) was taken in polarographic vessel and then the measurement was started under the given parameters (Table 1). After this voltamogramme of the 'blank' was recorded. 0.1 ml of prepared sample solution was added to polarographic vessel and then voltamogramme of the sample solution was recorded under the same conditions. After the sample voltamogramme was recorded, 0.1 ml of 1 ppm standard of lead was added twice and then voltamogramme of the standard was recorded. Finally the concentration of the metal was calculated

by linear regression method (standard addition) using following formula

$$\text{Final Result} = \text{Concentration} \times \frac{\text{Cell Volume}}{\text{Sample amount}} \times \frac{\text{Multiplier}}{\text{Divisor}}$$

Where, Multiplier = Dilution

Divisor = Sample amount taken for preparation

Results

This study was conducted to estimate the blood lead levels in post-mortem cases of South Delhi area that was brought to the Department of Forensic Medicine and Toxicology, AIIMS, New Delhi. Two hundred and fifty cases were studied as per the inclusion criteria during the period of March 2012 to October 2013. We found that the mean blood lead levels in South Delhi population were 14.28 µg/dl and range was 2.34 to 53.69 µg/dl. 15.2% of south Delhi population were having blood lead level >25µg/dl. This observation is lower when compared to the study conducted by Kumar S et al [10] at Rohtak, on 42 male volunteersin the absence of local industrial sources of lead with varying degrees of exposure to vehicular exhaust. Automobile workers were found to have the highest levels of blood lead (mean value 21.26 µg/dl) followed by roadside population (mean value 14.91 µg/dl).

Sokas RK et al [11] conducted a cross-sectional study on Lead levels in Maryland construction workers. 264 Maryland construction workers had mean values of 8.0µg/dl, ranging from 2 to 30 µg/dl. None were currently engaged in known lead work. Blood lead levels were significantly higher for the 124 who had 'ever' worked in demolition (8.8 µg/dl). The 58 workers who had workplace lead monitoring had higher lead levels (9.7 µg/dl). Blood lead levels increased with age, and cigarette smoking.

Shobha N et al [12] conducted a study at NIMHANS, Banglore described the phenotypic and electrophysiological profile in five male patients working in a battery factory who developed radial nerve neuropathy due to lead exposure. All patients had elevated blood lead levels that were in the toxic range but higher to the study conducted by Reynolds SJ et al [13] that blood lead level for painters and laborers were significantly higher than other occupational group. The probable reason for higher blood lead level in South Delhi is might be due to higher rate of lead level in the environment namely air pollution (Vehicle exhaust), occupation related hazards etc.

Also, we found that mean blood lead level(Table 3) in male (16 µg/dl) was significantly higher than the female (10.41 µg/dl). 19.6 % male and 5.2 % female were having >25 µg/dl blood lead level. This finding is similar to the observation of Medical Surveillance of Blood-Lead Levels in British Workers [14]. The probable reasons for this may be due to male population being exposed more to the lead pollution than female.

Fig. 1: Trace Metal Analyser
(Make- Metrohm AG Ltd, Model- 797 VA Computrace)

Fig. 2: Microwave digestion system
(Make-Aurora Instruments Ltd., Canada, Model- MW 680)

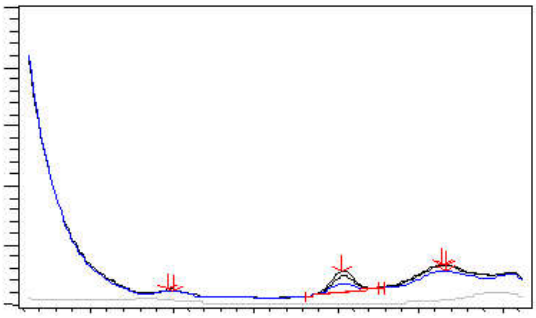

Fig. 3: DPAS Voltamogramme of Pb obtained from standard addition technique with number of replications being 2. A) 0.1 ml sample in 1ml acetate buffer (pH 4.6) + 10 ml distilled water, B) A + 0.1 ml standard solution of Pb (1 ppm), C) B + 0.1 ml standard solution of Pb (1 ppm)

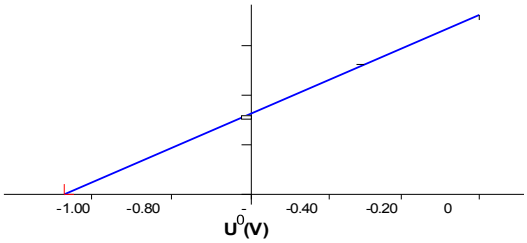

Fig. 4: Extrapolation graph of Pb obtained from standard addition technique

Table 1: Operating parameters for the determination of Lead

Parameters	Description
Working electrode	Hanging Mercury Dropping Electrode
Calibration	Standard addition method
Number of replications	2
Drop size	4
Stirrer speed	2000 rpm
Mode	Differential pulse
Initial purge time	300 s
Addition purge time	10 s
Deposition potential	-1150 mV
Deposition time	90 s
Equilibration time	10 s
Pulse amplitude	50 mV
Start potential	-1150 mV
End potential	-700 mV
Voltage step	6 mV
Voltage step time	0.1 s
Sweep rate	60 mV/s
Peak potential Pb^{2+}	-380 mV

Table 2: Programming conditions for the microwave digester

Step	Time (s)	Starting Temp (°C)	Ending Temp (°C)
1	210	28	100
2	600	100	160
3	600	160	170

Table 3: Mean values according to Sex

Gender	No of Persons	Mean value of Lead in µg/dl
Male	173	16
Female	77	0.41

Table 4: Mean values according to age group

Age Group in years	No of Persons	Mean values of PB in µg/dl
<14	8	7.28 µg/dl
14-17	18	8.07 µg/dl
18-40	172	13.71 µg/dl
41-60	40	20.73 µg/dl
>60	12	14.88 µg/dl

We also found in our study group that the age wise blood lead level (Table 4) was highest in middle age group and lowest in child age group. Mean blood lead level for middle age group was 20.73 µg/dl, range 3.84 – 53.69µg/dl and for child it was 7.28 µg/dl, range 2.34 – 17.42µg/dl. 50% children were having blood lead level >5µg/dl

Conclusion

Maximum number of cases occurred in the age-group of 18 - 40 years, followed by age-group of 40 - 60 years. Males outnumbered females with male to female ratio of 2.25:1. Mean blood lead level for South Delhi population was 14.28 µg/dl. Range for blood lead levels in South Delhi population were 2.34 – 53.69 µg/dl. Highest mean blood lead level was in middle age group 20.73 µg/dl. Lowest mean blood lead level was in child age group 7.28 µg/dl. Blood lead level for males were higher than females.

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Evaluation of the Concentration of Heavy Metals in Sindoor using ICP-OES

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Abstract

Sindoor has been considered as auspicious and is put on the centre parting of the head (*maang*) by married women in India. Presently, most sindoor manufacturers use synthetic materials and dyes to make these products. In the present study an attempt has been made to determine the concentration of heavy metals especially lead and mercury in the sindoor samples. These sindoor samples were purchased from the local markets in two forms, i.e. in liquid and powder forms. The heavy metals were determined using ICP-OES. The results obtained in the study has been alarming specially in case of lead which was found to be as high as 382.3ppm in some powder sindoor brands and 34.6ppm in the liquid sindoor brands. Though meant for topical application this cosmetic product with excessively high content of lead can be taken as a storehouse for heavy metal toxicity which may take a dermal route for entry in to the human system causing serious health hazards.

Keywords: Sindoor; Lead; ICP-OES; Toxicity.

Introduction

Sindoor, also known as kumkum or vemillion in India, is meant for use as a forehead mark, referred to as tilak, bindi or pottu, put on the spot between the brows or put on the centre parting of the head which is considered as a sign of a married woman in India. In earlier times, sindoor was made with a special type of red marble stone, covered with turmeric and a little oil and left undisturbed for a few days, after which it turned into red powder. Today most modern cosmetic product manufacturers produce sindoor from synthetic materials, lead, zinc and industrial dyes. It is also called red lead (Pb_3O_4). Manufacturers follow no single method. Some mix oxidized metals and substandard oil to bring about the texture (Kapoor, 2007). Now sindoor is also available in liquid form. Red is also being derived from mercury sulphite, which can cause skin cancer. All these toxic substances can trigger hair loss, edema and erythema. Branded sindoor and kumkum, even the liquid sindoor marketed by some reputed cosmetic company, does not carry the mandatory label of ingredients. The toxicity problems are manifold as a flood of unbranded products are available in the local markets (Alkhwajah, 1992). Studies have shown that sindoor can cause local irritation and skin toxicity. The nature of sindoor or kumkum can change with exposure to the environment over time and this can result in blisters, itching, rashes, pigmentation and, at times, serious dermatological disorders.

Material and Methods

Objective

Analysis and estimation of Lead and Mercury in

sindoor samples (powder & liquid form) available in local market.

Hypothesis

1. Sindoor samples collected from the local market would contain lead & mercury that can cause skin diseases.
2. Quantitative estimation of lead and mercury can be done using ICP-OES.

Sample Type & Size

Sindoor samples in liquid & powder form were collected from the local markets for the study. In all 11 vermillion samples (7 liquid and 4 powder forms) were taken for analysis of lead and mercury.

Sample Preparation

All plastic and glassware were washed, rinsed many times with tap water and then soaked in 5% HNO_3 solution for a minimum of 24 h and were followed rinsing with deionized water before use.

Lead

1. 1 gm/ 1 ml of sindoor was taken in a beaker.
2. The beaker was then heated in muffle furnace at 450°c.
3. After the sample was turned to ash, the digestion was done.
4. For acid digestion, hydrochloric acid and nitric acid was taken in a ratio of 1:3.
5. 25 ml of acid digestion was added to the beaker and heated on a tripod stand till the solution was clear.

Mercury

1. 1 gm/ 1 ml of sindoor was taken in a beaker.
2. The beaker was then heated in muffle furnace at 450°C.
3. After the sample was turned to ash, digestion was done.
4. For acid digestion, 25 ml of nitric acid was added to it.
5. The solution was heated on a tripod stand till the solution became clear.

Instrument Used

Vista-MPX simultaneous ICP-OES with axially viewed plasma was used for this work. The instrument was fitted with the 3-channel peristaltic pump option for easy introduction of ionization buffer to the sample via a post-pump Y-piece. Instrument operating conditions were as follows:

Power	1.4 kW
Plasma gas flow	15.0 L/min
Auxiliary gas flow	1.5 L/min
Nebulizer type	Glass concentric
Sample tubing	Grey/grey
Internal standard tubing	Orange/white
Drain tubing	Purple/black Pump
Speed	15 rpm
Sample delay	35 s
Stabilization time	15 s
Rinse time	60 s
Replicate time	60 s
Replicates	2

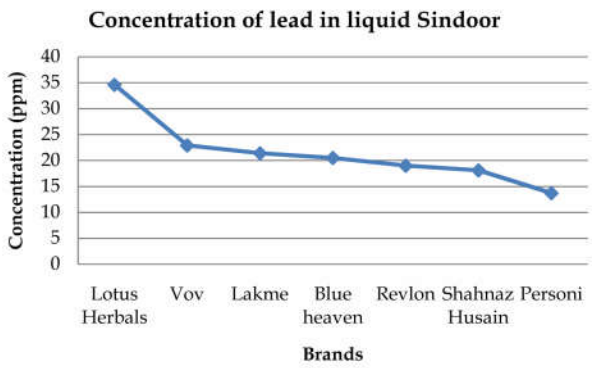
Result and Data Analysis

This research was performed in triplicate analysis. The number of selected sindoor was seven in case of liquid form and four in powder form collected from the cosmetic shops and local market. The data presented in Table 1 shows remarkably high concentration of lead in all the brands, the least being in Personi estimated at 13.7ppm. The maximum was observed in Lotus Herbals at 34.6ppm. However, Mercury was found to be less than 1 ppm in all these products.

In the four powdered forms of sindoor that was taken up for the study, the concentration of lead was found to be the highest at 382.3 ppm in the Suhag Shingar brand and the least was found in the brand

Table 1: Shows the concentration of lead and mercury in liquid sindoor

Brand	Lead	Mercury
Lotus Herbals	34.6 ppm	< 1.0 ppm
Vov	22.9 ppm	< 1.0 ppm
Lakme	21.4 ppm	< 1.0 ppm
Blue heaven	20.5 ppm	< 1.0 ppm
Revlon	19.0 ppm	< 1.0 ppm
Shahnaz Husain	18.1 ppm	< 1.0 ppm
Personi	13.7 ppm	< 1.0 ppm



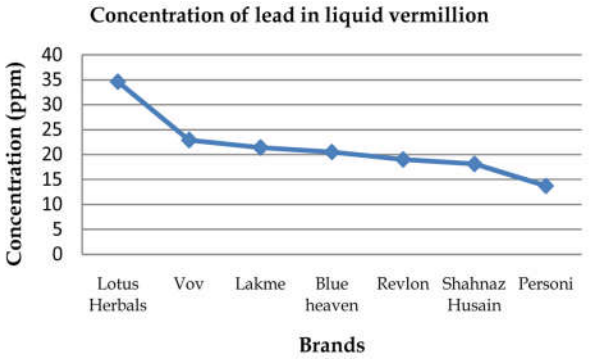


Table 2: Shows the concentration of lead and mercury in powder sindoor

Brand	Lead	Mercury
Suhag shingar	382.3 ppm	< 1.0 ppm
Milap	32.6 ppm	< 1.0 ppm
Drolias	31.7 ppm	< 1.0 ppm
Shingar	27.2 ppm	< 1.0 ppm

Shingar at 27.2 ppm. Mercury in this case as well was found to be less than 1 ppm. An overall assessment of these two forms of sindoor shows that the liquid form is comparatively better than the powder form, though the toxicity of bothese forms cannot be ignored and in both cases the lead content is much higher than that is prescribed in the FDA limit.

Conclusion

In earlier times, women preferred to prepare sindoor at home. Now, most of them buy the readymade sindoor from the market. A traditional component of the sindoor is powdered red lead and other ingredients are alum and turmeric. Sindoor is considered to be very auspicious by Indians and thus, used for various purposes on special occasions like wedding and festivals. The study has revealed that very high values of lead among samples may be due to the spurious nature of the samples as there are no proper safety regulations in the production and distribution of these cosmetic products. However, the possibilities of spuriousness of these products cannot be ignored. The data obtained clearly showed that further studies are also needed of these heavy metals in cosmetic products of daily use. Acceptable limits of potential contaminants in cosmetics such as the sindoor must be enforced. The principle of good manufacturing practice must be followed. There is need for an assessment of human risk from the exposure to cosmetics which are highly contaminated with heavy metals. It was inferred from the result that most of the brands of sindoor were contaminated with very high concentrations lead. Removal of heavy metals from personal care products after manufacture is not possible, however if careful selection of the raw material is made keeping in view the heavy metal contents we can improve the quality of the products and save the beauty of the environment.

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Original Article

Detection and Identification of Xylocaine in Cadaver Material: A Case Study

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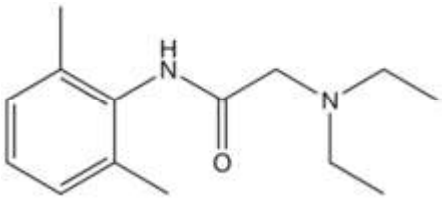
Abstract

Psychotropic substance like Xylocaine used as anesthetic substance in surgical operations were detected and identified in cadaver material of two deceased died during operation. The present work describes the detection and identification of Xylocaine in cadaver materials by chemical colour tests, thin layer chromatographic and U.V. spectroscopy.

Keywords: Lignocaine; Lidocaine; TLC; U.V.

Introduction

Xylocaine [1,2] is 2-Diethylaminoaceto-2'-6'-Xylidide used as local anesthetic which is long acting membrane stabilizing agent against ventricular arrhythmia having white to light yellow crystalline substance with molecular formula $C_{14}H_{22}N_2O$ the chemical structure of Xylocaine is



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The present work describes the detection and identification of Xylocaine, in cadaver material by chemical colour test, thin layer chromatography and U.V. spectroscopy.

Case History

An old lady was admitted in hospital for eye operation on the day of operation doctor, before operation, gave her injection. The old lady immediately became unconscious and died. After thorough examination she was suspected to die due to drug reaction, which was given to her. Similarly in another case a person admitted to hospital for elbow surgery he also died due to an injection given before surgery.

The postmortem of the deceased was conducted and different autopsy material was sent to R.F.S.L. Gwalior for chemical analysis in order to establish the cause of death in these surgical procedures.

Material Received for Examination

1. Stomach, pieces of small intestine, liver, spleen, kidney, lung and brain.

2. Piece of skin and soft tissue from injection site.

Method of Isolation from Different Cadaver Material

Visceral tissues were subjected to standard Stas-Otto procedure of deproeination and acid as well alkaline; chloroform and ether (1:4) extracts were taken for analysis.

Pieces of skin, soft tissues from injection site were separately macerated and homogenized in 50ml absolute alcohol, each along with 1 ml of glacial acetic acid.

The tissues were refluxed over boiling water bath for about four hours. Filtrates were taken, evaporated and subjected to identification procedures for further examination.

Methods of Identification

All chemicals such as Benzene, Methanol, Ammonia, Bismuth sub nitrate, Potassium nitrate and Acetic acid were of analytical reagent grade. Distilled water was used throughout the study. Control drug sample Xylocaine were purchased from local medical shop.

Thin Layer Chromatographic Analysis

A standard glass TLC plates was coated with slurry of silica gel G in water to a uniform thickness of 0.25 mm. Heating in an oven at 110°C for about one hour activated the plate. An aliquots of standard Xylocaine and extract obtained from autopsy material were spotted on to the plate, which was developed with Cyclohexane: toluene: diethyl amine (75:15:10) in a pre saturated TLC chamber, to a height of 10 cm. The plate was removed from the chamber dried in air and sprayed by dragendroffs reagent at which gave orange coloured spots. The R*f* value of Xylocaine 0.35 can be compared with the obtained spots of visceral extract. The Rf value of Xylocaine in different solvent systems are given in following table

Colour Tests [3,4][4]

The extracted residue were dissolved in 2 ml alcohol 1 ml of each extract was taken for colour test.

S. No.	Solvent system	RF value
1.	Methanol/ strong Ammonia (100:1.5)	0.70
2.	Benzene/Acetone/ Methanol (80:10:10)	0.66
3.	Chloroform/Methanol (90:10)	0.73
4.	Chloroform/Acetone (90:10)	0.12
5.	Cyclohexane/Toluene/Diethylamine (75:15:10)	0.35

Observations

a. To an extracted residue, add 1 ml of dil nitric acid and 3 ml of mercuric nitrate solution and heat to boiling yellow green colour appears.

b. A conspicuous pink colour was observed in extractives from visceral material and skin piece when Marquise reagent (formaldehyde/ sulphuric acid) is added. This colour was well tallying with the colour given by control standard xylocaine using same spot test reagent.

UV Spectroscopy

The UV spectra were taken as Shimadzu UV spectrophotometer model 2550. The tissue and visceral extractives showed ë max in aqueous acid at 263nm and 272 nm tallied with the control sample.

Results and Discussions

The isolation and identification of drug by TLC technique, colour test and concordantly confirmed by U.V. spectral studies showed that drug were isolated.

TLC coupled with U.V. spectroscopy between 200nm to 400nm provided a reliable quick method of detection and identification of Xylocaine drug used as anesthetic agent which is evident from recorded observation a sharp λ max in aqueous acid at 263nm and 272 nm in skin as well as visceral extracts tallied with standard sample, concluded that Xylocaine drug would have been given by doctors to old lady and person underwent for surgery leading to the probable drug reaction fatality case by this local anesthetic.

Such rapid resolution by TLC, colour tests and

U.V. spectroscopy will undoubtly be of great value to the analytical toxicologist who may be confronted with the analysis of this drug in cadaver materials referred in emergency cases of fatal drug reaction.

Acknowledgement

Authors are grateful to Ex Joint Director, Mr. S. R. Patidar R.F.S.L. Gwalior and Director, FSL Sagar (M.P.) for providing necessary facilities and encouragement.

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Review Article

Polynuclear Aromatic Hydrocarbons (PNHS)- Toxic Air Pollutants (TAPS): A Review

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Abstract

In the environment by various processes such as incomplete combustion of recent and fossil organic matter at higher temperatures, slow maturation of organic matter under geochemical gradient conditions. Most PNH inputs in the environment are linked to the anthropogenic activity that is generally considered to be the major source of these compounds.

PNHs have different distribution patterns according to their production sources. In addition, physical–chemical properties of some PNHs, like chemical reactivity, can contribute to modify the original distribution pattern of the emission sources. PNHs in general are toxic, carcinogenic in many animals, act as anti-estrogens in mammals, and induce reproductive toxicity in women. Degradation is the most common way to remove PNHs in the environment. PNHs partially dissolved in water tend to be adsorbed in suspended solids PNHs in water bodies can accumulate in fatty tissues of organisms through the food chain. Thus, it is imperative to understand the relative importance of pollutant emission sources for coastal air and watersheds.

Keywords: PNHS; Taps; Cops; Toxic; Carcinogenic.

Toxic Air Pollutants

Toxic air pollutants are known or suspected to cause cancer or other serious health effects, which is found in gasoline; perchlorethylene, which is emitted from some dry cleaning facilities and methylene chloride, which is used as a solvent and paint stripper by a number of industries. Other listed air toxics include polycyclic aromatic hydrocarbons.

Environmental Protection Agency (USA) identifies a list of the 33 air toxics that present the greatest threat to public health in the largest number of urban areas. Of these 33 urban air toxics, EPA has identified the 30 with the greatest contribution from smaller commercial and industrial operations or so-called "area" sources.

Principal Pollutants and Air Toxics

Air pollutants can be divided into two categories: principal pollutants and air toxics. Principal pollutants are often considered a group of 'traditional' air pollutants. These have a demonstrable health toxic. The principal pollutants constitute mainly carbon monoxide, sulphur dioxide, photochemical oxidants (as ozone), oxides of nitrogen, lead and particles. Air toxics are pollutants present in the environment in low concentration (other than principal pollutants) that are known, or suspected to be as toxic and persistent. The term 'air toxics' and 'toxic air pollutants' (TAP) are used interchangeably. Some PNHs are semi volatile in nature & therefore, these can be present in particulate as well as vapour phase while others are mostly adsorbed onto particles in the environment (IARC, 1983).

Conscious Organic Pollutants (COPs)

Conscious organic pollutants (COPs) are amongst the most dangerous substances released by humans into the environment. COPs include a wide range of substances, including organochlorine conscious. They possess toxic characteristics and are likely to causesignificant adverse effects on health of exposed wildlife and humans, such as allergy, damaged nervous system and immunity, congenital diseases, cancer. These substances are conscious, resist degradation under natural conditions and remain unchanged in the environment for a long period. The upper tropic levels of food chains (fish, predatory birds, mammals and humans) can bio accumulate the highest concentrations of COPs. Very often the alternatives to conscious organic pollutants are available. But the high costs, poor public awareness, lack of appropriate infrastructure and technologies are the reasons why these alternatives are not being used widely enough.

Possible solutions to these problems should be adjusted to the properties and possible use of each substance, as well as to the climatic and socio-economic circumstances of each country (AMAP, 2002).

Polynuclear Aromatic Hydro-Carbons (PNHs)

Chemicals can be transported through the environment by several mechanisms. Even pristine areas are subjected to the deposition of chemicals carried thousands of kilometres through the atmosphere. Polynuclear Aromatic Hydrocarbons (PNHs) are chemicals containing two or more fused benzene rings in a linear, angular or cluster arrangement

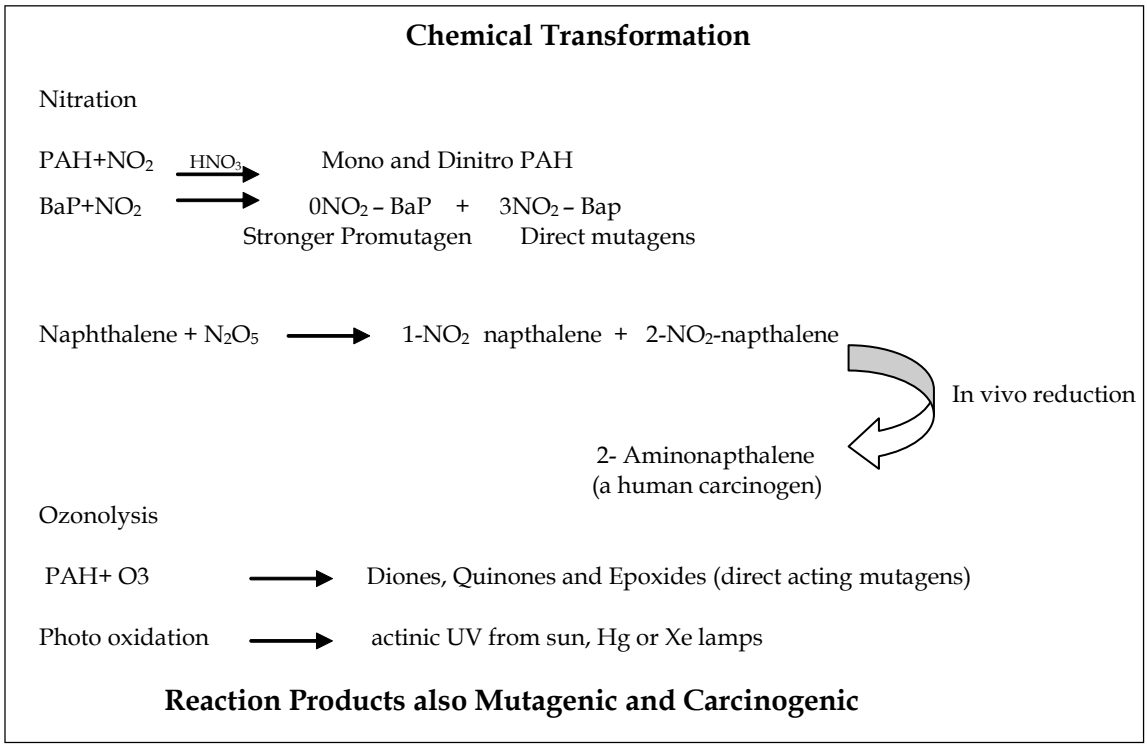

Fig. 1: Reaction of PNHs with atmospheric pollutants viz. NO2& O3

(Masih and Taneja, 2006). PNH contain only carbon and hydrogen. They belong to the group – Conscious Organic Pollutants (COPs) known for their chemical Carcinogenicity (EPA, 1986; Masih et al, 2008). They are members of a unique class of air pollutants relevant to many scientific and societal issues having a variety of aspects: chemical, toxicological, engineering, technological, public health, economic, regulatory and legislative (Pitts & Pitts, 1996). PNHs are released into ambient air as constitutes of highly complex mixtures of Polynuclear Organic Matter (POM). As defined in the U.S. Clean Air Act Amendments of 1990 (CAAA, 1990), POM "includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 212oF (100°C)". The ubiquitous nature of these airborne PNHs is evident from the fact that the 16 U.S. Environmental Protection

Agency "Priority Polynuclear Aromatic Hydrocarbons Pollutants" shown in Table 1 are found in urban air sheds throughout the world (U.S.EPA, 1988). Polynuclear aromatic hydrocarbons (PNH's) are an important group of compounds which are transported through the atmosphere. Their widespread presence is due to their emissions from a wide range of combustion sources, including diesel and gasoline engines, biomass burning of agricultural and forest fuels (Jenkins et al 1996; Masih et al, 2010), and outdoor wood smoke (Schauer et al, 1999). PNHs are also common constituents of air indoors, arising from coal and wood combustion (Mumford et al, 1990) and environmental tobacco smoke, ETS (California EPA, 1997).

Formation of PNHs

PNH are usually generated under insufficient combustion (pyrolysis) conditions, such as insufficient oxygen (Sorensen, 1994; Nam et al, 2003) or high temperature pyrolytic process during combustion of fossil fuels/ organic materials, as well as in natural processes such as carbonization(pyro synthesis). Thus, PNHs are the constituents of the products of incomplete combustion (PIC). PNHs on reaction with atmospheric pollutants viz., NOx, SO2, O3 etc. may form hetro-PNHs. The products formed during the chemical transformation (Figure 1) are more mutagenic and carcinogenic than the parent compounds. The contribution from natural sources of PNHs is limited, being restricted to spontaneous forest burning and volcanic emissions (Bourotte et al, 2005).

Vapour Particle Phase Distrbution of PNHS

According to review and analysis done by white et al, (1980) PNHs and nitro-PNHs may exist in vapour or particle phase .The lower molecular weight PNHs and nitro-PNHs with a ring number of 2and 3 tend to be associated with the vapour phase. The larger molecular weight PANs tend to be associated with particulate in the atmosphere. PNHs species with a molecular weight below that of pyrene exist to a larger extent in the gas phase. On an average 47% of the total PNH were reported in gas phase. Three ring PAN are predominantly gaseous, four ring PNH mixture of both phases and five-six ring PHN primarily particulate. Pierce and Katz (1975) also studied size distribution of PHN containing particulates, which showed approximately a log-normal relationship for suburban and rural sampling sites with majority of PNH content (50-78%) associated with particles below 3.0 µm diameter. Total PNHs were higher period by a factor of 65-75% and concentration range vary between lowest 2 µg gparticulate (anthracene) to 20 µg/g (BghiP).

Air-Soil Exchange/Interaction of PNHS

Polynuclear aromatic hydrocarbons are often found in air, precipitation, lakes, estuaries, soil, sediments and groundwater systems. Once released into the atmosphere PNHs are highly persistent in the environment and migrate within and between the atmosphere, terrestrial and aquatic compartments. Atmospheric deposition constitutes the main input of semi-volatile organic compounds to soil (Tremolada et al., 1996). Once entered in the soil they accumulate in horizons rich in organic matter where they are likely to be retained for many years due to their persistence and hydrophobicity. (Krauss et al., 2000). Soil is the primary terrestrial reservoir of persistent organic pollutants such as PANs (Ribes et al., 2002; Wileke et al., 1996) and the atmosphere is their main transport vector (Drooge, 2002), so, it is important to determine the amounts of PNHs in soil as their concentration in soil correlates significantly with the corresponding levels in air (Nam et al, 2003; Massei et al, 2004) and is a good indicator of the surrounding air pollution and the proximity of sources. Soil air exchange is therefore a key process governing the environmental fate of these compounds on a regional and global scale. Air to soil transport may occur through dry depositions of aerosols, wet deposition or sorption to soil constituents (Cousins, 1999). Soil to air diffusion is

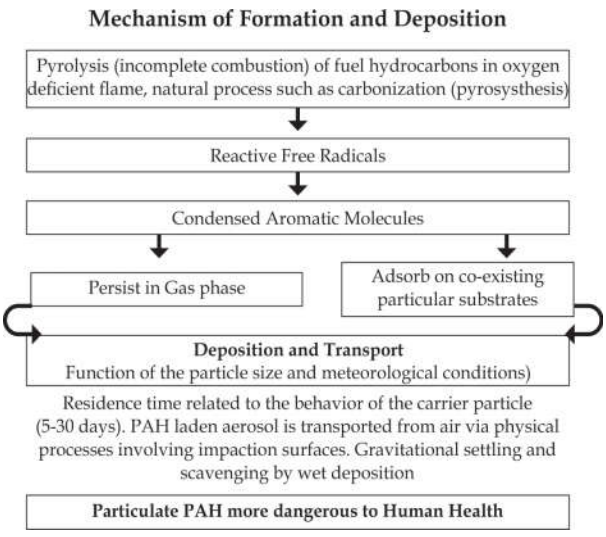

Fig. 2: Mechanism of Formation & Deposition of PNHs.

driven by the chemical potential gradient between the soil and the atmosphere (Masih et al, 2012). Consequently, soils are an important reservoir for these compounds (Ockenden et al., 2003) andexchanges between soils, water and the atmosphere is a widely studied process (Bidleman and McConnell,1995; Wania and Mackay, 1996).

Structure of PNHs

In general, hydrocarbons that display benzene like properties are called aromatic; those which contain fused benzene rings are called polynuclear or (polynuclear) aromatic hydrocarbons or PNHs for short. Like benzene itself many PNHs possess unusually great stability and a planar geometry (Colin Baird, 1999). There is a series of benzene like hydrocarbons that contains several six membered rings connected together by the sharing of a pair of adjacent carbon atoms between adjoining fused rings. The simplest example is naphthalene C10H8 as shown in Figure. Notice that there are ten, not twelve, carbon atoms in total and that there are only eight hydrogen atoms, since the shared carbons have no attached hydrogen's. As compound naphthalene is a volatile solid whose vapour is toxic to some insects. It has found use as one form of "moth balls", the other being 1, 4-dicholorobenzene. Other than naphthalene, PHNs are not manufactured commercially, since they have no uses. Conceptually, there are two ways to fuse a third benzene ring to two carbons in naphthalene, one result in a "linear" arrangement for the centers ('the nuclei') of the rings while the other is a "branched" arrangement (Figure 2). Both anthracene and phenanthrene are pollutants associated with incomplete combustion, especially of wood and coal, and are also released in to the environment from the dump sites of industrial plants that converts coal into gaseous fuel, and fro petroleum and refineries. In rivers and lakes, they are found mainly attached to sediments rather than dissolved in the water, both are found to be subsequently partially incorporated by fresh water mussels (Masih et al, 2014).

Sources of PNHs

Individual sources of PNHs are characterized by combustion processes with PNH-containing compounds, e.g. processing of coal, crude oil, creosote, coal-tar, bitumen and vehicular exhausts (Gasoline and Diesel). The sources considered are industrial, domestic, mobile, agricultural and natural (PNH Position Paper, 2001).

Industrial Sources

In general industrial sources are comparatively well understood and are increasingly being regulated. Currently these include: Petrochemical and related industries, cement manufacture, rubber tyre manufacturing, bitumen and asphalt industries, commercial heat and power, waste incineration, creosote and wood preservation, coke production. Overall PNH emissions are believed to be decreasing; improved energy management is leading to improved combustion which, in turn, leads to lower emissions.

Domestic Sources

The domestic sources of PNH which can influence ambient air quality are:

Cooking, use of diesel generators, incineration, smoking cigarettes, cigar and tobacco. The usage of heavy diesel generators to generate electricity because of erratic electricity supply is predominant. Modern gas and oil burners used for cooking have relatively low PNH emissions.

Mobile Sources

Mobile sources are modes of transport reliant on a combustion engine. This includes aircraft, shipping, railways, automobiles and motor vehicles including off-road vehicles and machinery. Automobile internal combustion engines are generally fuelled by gasoline (petrol) or diesel fuels. There are a relatively small proportion of vehicles which run on LPG or LNG. Two-Stroke engines are relevant in the motor scooter and motor cycle sector of transportation. Two-stroke fuel is a mixture of gasoline and oil. The engines are generally small and not equipped with additional emission control systems. Recent search has estimated that unabated PANemission whilst performing the ECE R40 simulation are 1.6 mg km-1 for the sum of 29 PANs with 2 to 6 rings and 20.8 µg kg-1 for six carcinogenic PNHs (BaP, B[b+k]F, BaA, DBahA). One of the major influences on the production of PNH from gasoline automobiles is the air-to-fuel ratio; it has been found that the amount of PNH in engine exhausts decreases with a leaner (thin) mixture. The use of catalytic converters has also been shown to have a significant effect on the reduction of the PNH concentration in the exhaust gases. All internal combustion engines have varying PNH emission characteristics dependent on engine temperature (particularly cold-start), load, fuel

quality and speed. Urban areas with congested traffic conditions and with vehicles often only traveling short journeys promote the emission of PNH. Engine deterioration and high mileage also increases emissions. Studies have shown that for all PNH compounds studied the reduction achieved due to catalytic converters was between 80 and 90% (for BaP 94%). Catalytic converters for diesel engines also reduce total PNH emissions, however the reductions are not as high as for gasoline engines. Diesel fuelled vehicles have higher particulate emissions than gasoline fuelled vehicles. The particles consist of combustion-generated soot, a solvent extractable hydrocarbon fraction. Other control technologies are currently being developed and improved (trap oxidizers and filters for example) for heavy-duty diesel engines. Such devices will be necessary to meet emission limit values set within EU regulations ('EURO 4'). As for gasoline vehicles, an additional source of PNH in the exhaust of diesel fuelled vehicles is the PNH content in the fuel i.e. by reducing fuel PNH content a reduction of exhaust PNH can be achieved. The popularity of the diesel engine in heavy-duty applications in trucking, railroad, marinetransport, DG sets and construction industry is due to both its fuel efficiency and long service life relative to the gasoline engine. Compared with gasoline engine, diesel emissions are lower in carbon monoxide (CO), hydrocarbon (HC), and carbon dioxide (CO_2), but higher in oxides of nitrogen (NOx) and particulate matter (PM). Diesel exhaust is a complex mixture of both particulate and gaseous phase. Diesel exhaust has particulate with mass median diameter of 0.05 to 1.00 micrometer, a size rendering them easily respirable and capable of depositing in the airways and alveoli. The particles consist of a carbonaceous core with a large surface area to which various hydrocarbons are absorbed, including carcinogenic poly nuclear aromatic hydrocarbons [Benzo(a)Pyrene] and Nitro-PNHs that have elicited the most concern with respect to human health. If only the most harmful of the exhaust emissions that is particulate emission is considered, the carcinogenic effect of one new diesel car is equivalent to 24 petrol cars and 84 new CNG cars on the road. The honorable Supreme Court of India has restricted the use of commercial diesel driven vehicles in Delhi due to its harmful effects (CPCB, 2003). The main source of PNH emissions in rail transport is the use of diesel and diesel-electric locomotives. As some locomotives are old, and produce large amount of black smoke, they may be a significant source of PNH but no measurement data are available. There have been very few studies carried out on PNH emissions of aircraft, and of those carried out, most have been for military aircraft. However, the results show that PNH emissions are dependent on fuel composition (volatility). Average emission factors for an aircraft gas turbine engine have been given as 1.24 mg/LTO (Landing-Take Off cycle) for BaP. As air travel increases the proportion of total PNH emissions, which are attributable to, air transport could increase, though it is unlikely that it will become a major contributor to total PNH emissions. Particulate emissions from shipping are not currently regulated. There are a limited number of publications focusing on PNH emissions from shipping. PNH emissions from an onboard marine diesel engine (6600 kW, maximum continuous rating) burning marine diesel fuel with a Sulphur content of 1.9% (w/w). Within the Lloyds Marine Exhaust Emissions Research Programme, individual PNH from several different ships using marine distillates and heavy bunkers were measured.

Agricultural Sources

Activities like stubble burning, openburning of moorland heather for regenerationpurposes, open burning of brushwood, strawand use of diesel generators for irrigationpurposes, involve the burning of organicmaterials under sub-optimum combustionconditions. Thus it can be expected that a significant amount of PNH are produced. Insome countries there are regulations in placeregulating these activities. Due touncertainties in emission factors and theoccurrence of these activities, the emissions of PNH from agricultural sources are difficult toquantify. Nevertheless, they may contributesignificantly to PNH level at certain locations.

Natural SourcesNatural sources of PNH include theaccidental burning of forests, woodland,moorland etc. Meteorological conditions (suchas wind, temperature, humidity) and fuel type(moisture content, green vs. seasoned wood,etc.) may play an important role in the degreeof PNH production. Another natural source ofPNH are volcanic eruptions. No data are available regarding these emissions and theircontribution to the overall PNH profile.

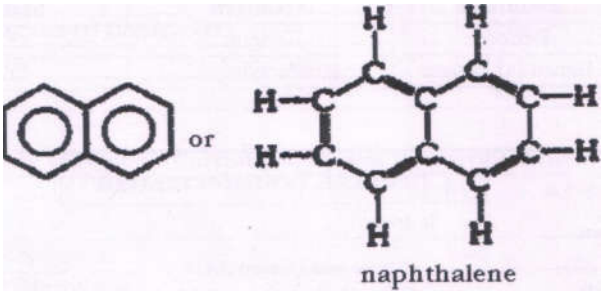

Fig. 3: Structure of Naphthalene

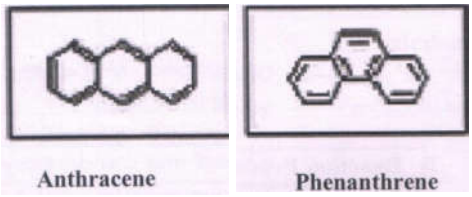

Fig. 4: Linear & Branched Arrangement of Benzene Molecule Selected PNHs

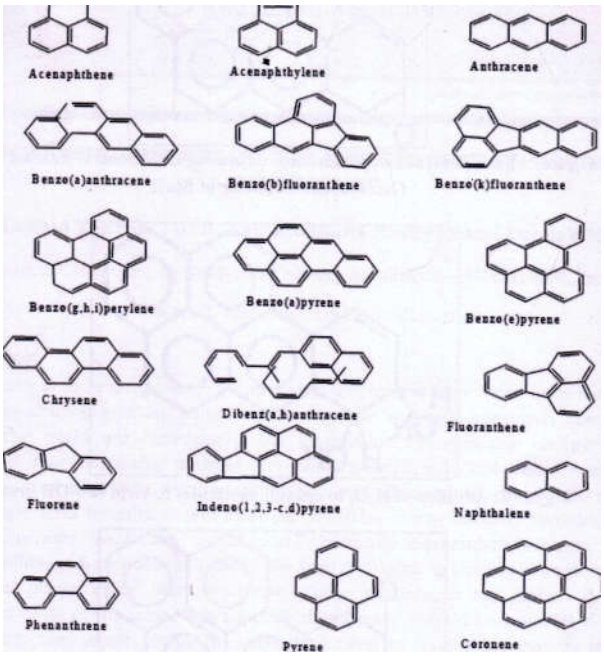

Fig. 5: Structure of selected PNHs

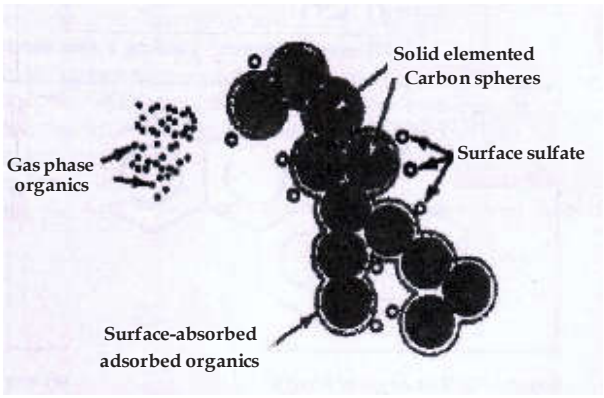

Fig. 6: Physio-chemical Complexity of Primary Combustion-generated POM

Oxy-PNHs

Oxy-PNHs are semi-volatile compounds, with high molecular weights and lower vapour pressures than their parent PNHs. Their sources can be both natural and anthropogenic. These compounds also could be originated from reactions between PNHs and other compounds founded in ambient air. Allen et al. (1997) mentioned that these compounds could originate from phoNto-oxidation of PNHs. There are experiments which suggest that several of the PNHs that are emitted in sizable amounts from various natural and anthropogenic sources are degraded in the atmosphere by sunlight or by interactions with other reactive airborne species as O_3, NO_x, and from these reactions products founded were quinones, ketones, coumarines, aldehydes and acids (Pitts et al., 1980). Oxy-PNHs may befounded in gas phase or may be adsorbed onto particles (Pankow et al., 1993). Pierce and Katz (1975), investigated that benzanthrone and phenalen-1-one concentrations increased in respirable particulate matter during winter. Oxy-PNHs were founded to be mutagenic in bacterial and human cell mutation assays (McDonald et al., 2004). PNHs are oxidized into oxygenated derivatives (Oxy-PNHs) by UV-light, O_3, NO_x and reactiveoxygen species. There are studies which show us that particulate matter emitted from vehicles contain higher amounts of Oxy-PNHs than particulate matter found in the atmospheric air (Oda et al., 1998).

Physio-Chemical Properties of PNHs

PNHs are Colourless, white or pale yellow-green solids. They are planar, relatively inert and volatile in nature. Their Melting Point is generally at room temperature while their Boiling Point is above than 100°C. The physical and chemical complexity of primary combustion-generated POM is illustrated in Figure 3 (Johnson et al, 1994), a schematic diagram of a diesel exhaust particle and associated co-pollutants. The gas-phase regime contains volatile (2-ring) PNHs and a fraction of the semivolatile (3- and 4-ring) PNHs. The particle phase contains the remainder of the semivolatile PNHs (particle associated) along with the 5- and 6-ring heavy PNHs adsorbed/absorbed to the surface of the elemental carbon spheres that constitute the backbone of overall diesel soot particles. PAHs have low solubilities in water as expected from their non-polar character. These decrease dramatically in going from the 2- and 3- ring compounds (e.g., naphthalene, with a solubility of 31 mg L-1) N0.26 mgL-1) to 5-ring B(a)P, with a solubility of only 0.0038 mgL-1) (Mackay et al, 1992). However reactions of PNHs in ambient air to form more polar species (e.g., nitro-PAHs, ketones, quinones, lactones and dicarboxylic acids) greatly enhance their solubilities in aqueous systems. This has major implications when one considers the distribution of PAHs and their atmospherically formed PAC derivatives, through air, water and soil environments. These increases in solubility upon

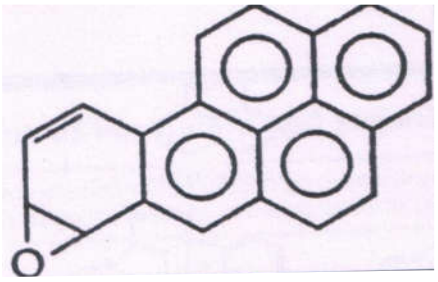

Fig. 7: Formation of an epoxide ring one C=C bond of interest to the Carcinogenic behaviour of B(a)P

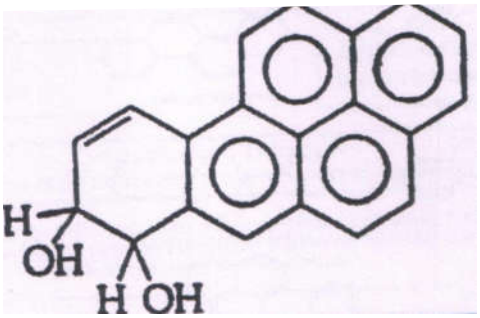

Fig. 8: Addition of H_2O, to epoxide molecule to yield two-OH groups.

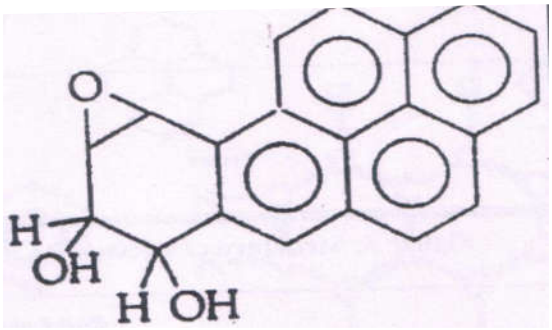

Fig. 9: Epoxidation of two-OH groups

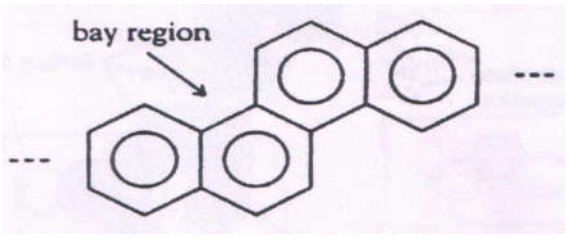

Fig. 10: The Organisation of carbon atoms at a bay region.

reaction are important not only from an environmental chemistry perspective but also in terms of possible impacts on public health and ecosystems, e.g., in both the exposure and the health effect portions of risk assessments of PNHs.

Uses of PNHs

There is no known use for acenaphthylene, benz[a]anthracene, benzo[b] fluoranthene, benzo[e]pyrene, benzo[j] fluoranthene, benzo[k] fluoranthene, benzo[g,h,i] perylene, benzo[a]pyrene, chrysene, dibenz [a,h]anthracene, indeno [1,2,3-c,d] pyrene, or pyrene except as research chemicals (Holloway 1987; HSDB 1994). Anthracene is used as an intermediate in dye production, in the manufacture of synthetic fibre, and as a diluent for wood preservatives. It is also used in smoke screens, as scintillation counter crystals, and in organic semiconductor research (Holloway 1987). Anthracene is used to synthesize the chemotherapeutic agent, Amsacrine (Wadler et al. 1986). Acenaphthene is used as a dye intermediate, in the manufacture of pharmaceuticals and plastics,

Table 1: Common Names, Empirical Formulas, Molecular Weights, Melting Points, Boiling Points, and CAS Numbers for 16 EPA "Priority PAH Pollutants"[a,b]

Common Names	MW(g mol^{-1})	Empirical Formula	Mp(^{o}C)	Bp (^{o}C)	CAS Number
Naphthalene	128	$C_{10}H_8$	80.5	218	91-20-3
Acenaphthylene	152	$C_{12}H_8$	92	265-275	208-96-8
Acenaphthene	154	$C_{12}H_{10}$	96.2	277.5	83-32-9
Fluorene	166	$C_{13}H_{10}$	116	295	86-73-7
Anthracene	178	$C_{14}H_{10}$	216.2	340	120-12-7
Phenanthrene	178	$C_{14}H_{10}$	101	339	85-01-8
Fluoranthene	202	$C_{16}H_{10}$	111	375	206-44-0
Pyrene	202	$C_{16}H_{10}$	156	360	129-00-0
Benz(a)anthracence	228	$C_{18}H_{12}$	160	435	56-55-3
Chrysene	228	$C_{10}H_{12}$	255	448	218-01-9
Benz(b)fluoranthene	252	$C_{10}H_{12}$	168	481	205-99-2
Benz(k)fluoranthene	252	$C_{10}H_{12}$	217	481	207-08-9
Benzo(a)pyrene	252	$C_{10}H_{12}$	175	495	50-32-8
Benzo(ghi)perylene	276	$C_{10}H_{12}$	277	525	191-24-2
Indeno(1,2,3-cd)pyrene	276	$C_{10}H_{12}$	163	---	193-39-5
Dibenz(a,h)anthenracene		$C_{10}H_{14}$	267	524	53-70-3

Adapted from MacKay et al, (1992); data on Indeno(1,2,3-cd)pyrene from Harvey (1997). Structures and their numbering are based on IUPAC recommendations as described by Loening et al. (1990).b USEPA (1998) designation.

Table 2: Half Lives of PNH under Simulated Atmosphere Conditions (Hrs)

PNH	Simulated Sunlight	Simulated Sunlight+Ozone(0.2ppm)	Dark reaction Ozone (0.2ppm)
Anthenracene	0.20	0.15	1.23
Benzo(a) Anthenracene	4.20	1.35	2.88
Dibenz(a,h)anthenracene	9.60	4.80	2.71
Pyrene	4.20	2.75	15.72
Benzo(a)pyrene	5.30	0.58	0.62
Benzo(a)pyrene	21.10	5.38	7.60
Benzo(b)fluoranthene	8.70	4.20	52.70
Benzo(b)fluoranthene	14.10	3.90	34.90

source: Katz,et al, 1979

Table 3: Intake of Potential Carcinogenic PNHs by non smoker & smokers

Source of PNH	Intake			
	Non-Smoker		Smoker	
	UG day- I	Total %	U day- I	Toyal %
Food	3	9.3	3	44.5
Air	0.16	4.9	0.16	2.4
Water	0.006	0.2	0.006	0.01
Soil(accidental Ingestion)	0.06	1.9	0.06	1.0
Cigrette	-	-	3.5	52
Total	3.22	100	6.72	100

Table 4: Potential Carcinogenicity and Bioactivity of the PNH

S. No.	PNH	Cracinogenicity	Bioactivity
1	Anthenracene	0	0
2	Fluoranthene	0	CC
3	Pyrene	0	
4	Benzo(a)pyrene	+	TI
5	Chrysene	+	Ti
6	Benzo(b)fluoranthene	++	C,TI
7	Benzo(k)fluoranthene	0	0
8	Benzo(a)pyrene	+++	C,TI
9	Dibenz(a,h)anthenracene	+++	C,TI
10	Benzo(ghi)pyrene	0	CC
11	Indeno(1,2,3-cd)Pyrene	+	TI

Note: + to +++ Active, CC= Carcinogen with BaP, TI= Tumour initiator, C= Complete Carcinogen, 0= Inactive

Table 5: Portion of carcinogenicity effect of BaP

Source of BAP	Carcinogenicity of BAP Portion (%)
Automobile exhaust condensate(Gasoline engines)	9.6
Automobile exhaust condensate(Diesel engines)	16.7
Domestic hard coal heating[a]	6
Domestic brown coal heating (briquets)	9
Lubricating oil of cars (used)	18
Sewage sludge (extracted)	22.9
Cigarette smoke condensate	1

(Source: A. Bjorseth, 1979)

Table 6: Prescribed Ambient Air Quality Guidelines (AQG) for the PNHs by WHO and the Netherlands

Country	Limit value	Guide value	Measuring Period
Netherlands	5ng/m^3	0.5 ng/m^3	Year as ng BAP/m^3
WHO-AOG	-		Year as ng BAP/m^3

Table 7: Proposed Ambient Limits for PNH in India

S .No.	Air Toxic Pollutents	Ambient Air Quality Standard	Ambient Air Quality Standard up
1	Benzene	10 ng/m^3	5 ng/m^3
2	Benzo(a) Pyrene	5 ng/m^3	1 ng/m^3

and as an insecticide and fungicide (HSDB 1994; Windholz 1983). Fluorene is used as a chemical intermediate in many chemical processes, in the formation of polyradicals for resins, and in the manufacture of dyestuffs. Phenanthrene is used in the manufacture of dyestuffs and explosives and in biological research (Holloway 1987; HSDB 1994). Fluoranthene is used as a lining material to protect the interior of steel and ductile-iron drinking water pipes and storage tanks (NRC 1983).

Photolysis and Half Life of PNHS in the Environment

Submicron aerosol has a half life of about 5-30 days in the atmosphere thus PNH may be transported and deposited at other surface in very remote region at highly reduced concentration as a result of the effects of atmospheric dispersion and chemical reaction. PNH laden aerosol is transported from air to soil and water via physical processes involving impaction surfaces, gravitational setting and scavenging by rain and snow. Transfer rates are also highly sensitive to particle size. The physical removal or transport of airborne particles is a function of the particles size and meteorological conditions. The occurrence of some PNH in remote areas such as Arctic and marine atmospheres (Mc Veety et. al. 1998) was mainly by aerial transport from distant anthropogenic sources. Half lives of PNH under simulated atmosphere conditions (expressed in hours) are given in Table 2. A number of research workers have demonstrated that may PNH are susceptible to photo chemical and or chemical transformation of PNH by gas-particle interactions in emission plumes, exhaust systems or even during atmospheric transport according to Atkinson, (1987).

Fate of PNHs in the Environment

- PNHs enter the air mostly as releases from burning coal, coke oven plant, automobile exhaust and wood fire.
- PNHs remains in air attached to dust particles.
- Most PNHs do not dissolve easily in water. They stick to solid particles and settle to the bottom of lakes, rivers or soil.
- Microorganisms break down PNHs in soil or water after a period of weeks to months.
- In soil, PNHs are most likely to stick tightly to particles. Certain PAHs move through soil to contaminate ground water.
- PNHs in remote areas such as arctic & marine atmosphere are result of long range transport.

Mode of Exposure and Daily Intake of PNHs

Human exposure to PNH can occur through several environmental pathways due to their numerous sources. However, the occurrence of PNH in urban air has caused particular concern because of the continuous nature of the exposure and the size of the population at risk. The urban atmosphere is a very complex and dynamic system containing a large variety of interacting chemical species in both the gas and particulate phases. PNHs compounds can reach to the human body by four different mode of exposure:

Tobacco/Cigarette Smoking

Tobacco alone accounts for 30% of total morality due to cancer every year. More than 70 PNH compounds have been analyzed in cigarette smoke. Smokers have eight times more probability of cancer attack than non-smoker. In developing countries approximately 30% smokers are young in the age group to 10 – 29 year. About 30-40% of them fall victim of premature death than expected life. The average total BaP content in the main stream smoke of 1 cigarette was 35 ng before 1960 and 18 ng in 1978-1979. Modern low tar cigarettes deliver 10 ng BaP. The concentration of BaP in a room extremely polluted with cigarette smoke was found to be 22 ng/m^3 (WHO 1987). Drinking Water – Examination of number of drinking water has been performed. The concentration of BaP ranges from 0.1 to 23.4 µg L-1, while for other PNHs the concentrations were between 0.001 to 0.01 µg L-1.

Food

American source indicate an intake of total PAHs from food in order of 1.6 – 16 g per day. The contents of BaP in various processed food was repeatedly found to measure up to 50 µg kg-1. The potential doses of carcinogenic PNHs by inhalation range between about 0.02 µg day-1 and 3 µg day-1 with median value of 0.16 µg day-1. This is nearly 20 times less than calculated food dose and about 25 times more than the potential dose with drinkable water. The intake of potential carcinogenic PNHs by non smokers and smokers is given in Table 3. Research up to now has shown that air contributes 3 – 20 % of total human exposure to PAH and comes in second after food.

Human Health Effects of PNHS

As discussed above, PNHs are hydrophobic compounds and their persistence in the environment is mainly due to lower water solubility and electro-chemical stability (CPCB, 2003). Evidence suggest that the lipophilicity, environmental persistence and genotoxicity of PHNs increase as the molecular size of the PNHs increases up to four or five fused benzene rings. More than 200 compounds are tested as possible carcinogens. Among these 25% have been found tumerogenic and about 30% of these were PNH. Lamb et al (1980) reported that BaP is a definite carcinogen with an LD50 of 24 micrograms. In causing health effect, these compounds are primarily activated through an oxidative metabolic pathway to form electrophilic intermediates. Such intermediates can develop covalent bonding with cellular DNA to form DNA adducts. This adducts, if not repaired, may initiate gene mutation and lead to adverse health effect ultimately manifesting in cancer, birth defects and genetic changes. PNHs being highly lipid soluble are absorbed by the lungs and gut of the mammals.PNH also penetrates in to the bronchial epithelium cells where metabolism takes places. International Agency for Research on Caner has classified PNHs according to carcinogenicit (IARC, 1987) as shown in Table 4.

Mechanism of PNH Carcinogenesis

Research has established that the PNH molecules themselves are not carcinogenic agents; rather they must be transformed by several metabolism reactions in the body before the actual cancer causing species is produced (Colin Baird, 1999). The first chemical transformation that occurs in the body is the formation of an epoxide ring across one C=C bond in the PNH. The specific epoxide of interest to the carcinogenic Behavior of B(a)P is shown in Figure 7. A fraction of these epoxide molecules subsequently add H2O, to yield two- OH groups on adjacent carbons as shown in Figure 8. The double bond (shown in the structure above) that remains in the same ring as the two-OH groups subsequently undergo epoxidation, thereby yielding the molecule that is the active carcinogen (Figure 9). By adding H+, this molecule can form a particularly stable cation that can bind to molecules such as DNA, thereby inducing mutations and cancer. The metabolic reactions of epoxide formation and H_2O addition are part of the bodies attempt to introduce-OH groups into hydrophobic molecules like PNHs and thereby make them more capable of becoming water soluble and eliminated. For B(a)P and other PNHs that possess a bay region, one of the intermediate products in this multi step process can be diverted instead into the formation of a very stable cation that induce cancer. The PNHs that are the most potent carcinogens each possess a bay region formed by the branching in the benzene ring sequence, the organization of carbon atoms at a bay region (As shown in Figure 10) imparts a high degree of biochemical reactivity to the PNH. The portion of the total carcinogenic effect of BaP in various particulate emissions is shown in Table 5.

Environmental Monitoring of PAHS and Ambient Limits

USEPA, [1997] has classified PNHs with B(a)P indicator species as a B-2 pollutant that means a probable human carcinogen with sufficient evidence from animal studies and inadequate evidence from human studies. In most of the OECD countries theToxic air pollutants (TAPs) monitoring (Particularly the PNHs, BAP as an indicator species) and the risk assessment has become a regular feature (CPCB, 2003). Integrated and long term monitoring is being carried out in the Netherlands, Sweden and the United States. The world health organization (WHO) has already added PNHs in to the list of the priority pollutants in both air and water. France, Japan, Germany, Netherlands, Sweden and Switzerland have prescribed emission standards for most of TAPs including PNH. The WHO and the Netherlands (WHO, 1987) have even prescribed ambient air quality guidelines (AQG) for the PNHs (Table 6). This will help in the development of data banks of PNH levels in air, formulation and development of standards for ambient air quality, source emissions and effluents, granting consent based on PNHs to the relevant of abatement and control strategies of PNHs in the environment.

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Review Article

Role of Antifouling Paints in Marine Coating

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Abstract

The main objective of this paper is to provide a brief view on the merits, demerits, effects and uses of antifouling paints. The imminent ban of environmentally harmful tributyltin (TBT) and Tin based paint products has been the cause of a major change in the antifouling industry. Fouling organisms such as barnacles, tube worms and algae, which accumulate on any submerged surface, greatly increase drag, and reduce speed and fuel consumption of the vessel other mechanical damages have also been reported due to fouling. A variety of paints were developed in mid 1800s, which were generally based on copper oxide arsenic and mercuric oxide as popular antifoulants. Antifoulants are one of the many additives usually incorporated within the topcoat paint of marine protective coating system. Today's marine coatings must provide protection and performance under extremely harsh conditions.

Keywords: Antifouling; Coating; Marine Coating; Environment; Paint; Toxic Substance.

Introduction

Antifouling coatings are used to control aquatic fouling pest organisms (alga, barnacles, mussels and molluscs) on ships, small boats and other and surfaces found in fresh water and marine environment. The requirement for the registration of antifouling coatings for ships hulls were developed primarily to address an absence of information on the environmental affects of currently registered active ingredients from antifouling coatings have been shown to have some potentially adverse effects on the non-target aquatic organisms. The toxic antifoulants on ship hulls has been a historic method of controlling fouling but biocides such as lead; arsenic, mercury and their organic derivatives have been banned due to the environmental risks that they posed. *(L.D Chambers 2006)* Marine biofouling can be defined as the undesirable accumulation of microorganisms, plants, and animals on artificial surfaces immersed in seawater. In case of ships, the adverse effects caused by the biological settlement on hulls marine coatings provide protection to both ships and the aquatic environment. The process of biological fouling is often grouped in the literature into key growth stages, which include an initial accumulation of absorbed organics, the settlement and growth of pioneering bacteria creating a biofilm matrix and the subsequent succession of micro and macrofoulers (Figure 1). *(L.D.Chambers et.al 2006)*

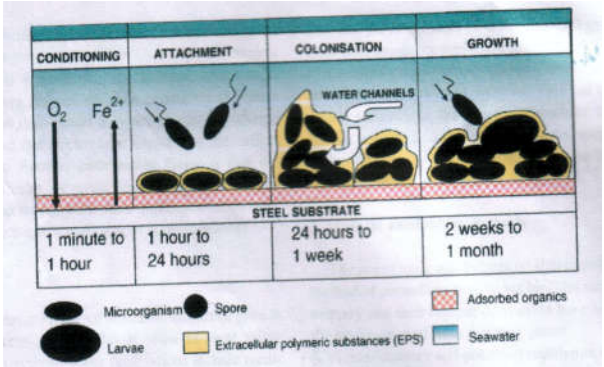


Fig. 1: Schematic representation of Biofouling Stages

Antifouling paints however are generally known for their harmful effects to the living organism of marine water resulting in legislation that culminated in the global ban of tributyltin (TBT) (Figure 2). *(L.D.Chambers et.al 2006)*

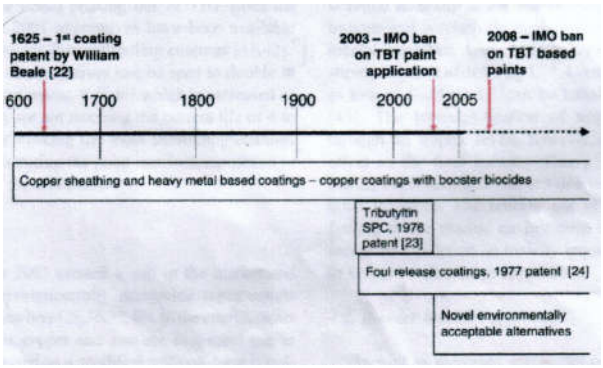


Fig. 2: Timeline for key Antifouling generations

The metallic pollutants present in such kind of paints however unsuitable to aquatic life. The suspended particles or the dissolved compounds of such metals taint the water resources more seriously. Marine coatings are meant to provide complete protection and performance against extremely odd conditions and should meet the challenges of today's physical environment i.e. they can give best performance without disturbing and affecting the environment. In yesteryears toxic ingredients were the main key to the antifouling paints but now these toxic substance have been fully replaced by non-toxic fouling release coatings.

Ships are under constant attack from marine environment and need to be protected from the influences of the key elements of the marine environment such as seawater, biological attack and temperature fluctuations. Methods of protecting marine structures i.e. ships must be capable of resisting such changes in marine environment. Protective organic coatings can offer these functions (*C.G. Munger, 1984)* and consequently are largely used in the shipping industry to increase the working life of systems and improve its reliability. Paint coatings provides resistance towards corrosion, helps in easing maintenance of ships, its appearance, prevents the accumulation of fouling on hull by unwanted marine organisms. Such accumulation of marine organisms on the substrate can cause large penalties to ships, biofouling can clog systems and on ship hulls it can increase the hydrodynamic drag, lower the manoeuvrability of the vessel and increase the fuel consumption. This leads to increased costs within the shipping industry through the increased use of manpower, fuel, material and dry docking time.

Marine-Fouling Organisms

Marine fouling organisms include members of both the plant and the animal kingdoms. Some 2000 species have been reported to cause fouling, many of which are unique to marine environment. Representative include algae, bacteria, fungi, protozoan, barnacles and other arthropods, mollusks, tunicates, hydroids and annelids. Antifouling systems are required wherever unwanted growth of biological organisms occur. Extensive damage, direct or indirect, to wood pilings, hulls, buoys, other immersed materials, devices and organic coatings are caused by fouling organisms. *(Walter, H. 1971, Walters,H. and Elphick,J 1968, Bikales,N.M. and Segal, L.1971, United states Naval Inst. Marine fouling and its Prevention 1952)*

What is Marine Fouling

When a ship is immersed in marine water for a longer duration, the animal mass, algae or other vegetative growing's of sea stick to base, diverse species of hard and soft fouling form colonies on hulls because each requires a permanent anchorage in order to mature or reproduce. This process of attachment of the above objects to the base or "hull" of the ship is termed as fouling.

Occurance of Fouling

Marine water contains different types of vegetables and animal organisms, these organisms required certain substrates to grow on, it may be the bed of sea, the rock or only the saline water of sea. They can also grow on other substrates also like hulls or base of ships. When the ships are suspended in water for longer duration the organisms settles upon the submerged part of the ship and start growing on it. They go on multiplying on the hulls and settles permanently on it. Mainly two types of the following organisms are there *(Gale,G.E 1953).*

Animal Fouling

It includes the barnacle, selfish such as oysters, tubeworms, sea – anemones and hydroids. Mainly the larva of such animals sticks to the base of the ships and complete their life cycle there thus becoming a permanent inhabitant of it.

Vegetable Fouling

It mainly consists of seaweeds, these weeds sometimes grows very densely, and can attain good

height. This growth of weeds greatly hinders the speed of ship. Other vegetable fouling is algae, fungi, diatoms and various species of bacteria.

Fouling Effects

Fouling is said to be the greatest evil of ships. Biofouling of ships increases fuel consumption, increases drag resistance, decreases maximum attainable speed and promotes corrosion. Fouling of power plant intake bays necessitates frequent shut downs and measures such as chlorination. Fouling by calcareous organisms contributes the greatest penalty because of their profile, and their tenacious adhesion to surfaces. Each of calcareous organisms attaches in a slightly different way using different glues.

Following are some ill effects of fouling.

Reduces Speed

A layer of both the animal and vegetable organism greatly reduces the speed of ship. It restricts the further movement of ships there by results in slower speed.

Fuel Consumption

It has been estimated that fouling of hulls can create such turbulence as a ship moves through the water that fuel consumption is increased by as much as 30 percent (*Perez, M. et al. 200, Brady, R.F 2000)* Known antifouling coatings are based upon kinds of mechanical cleaning as well as the release of highly toxic biocides from matrix coatings or upon either combinations.

Corrosion

Corrosion is an electro-chemical process requiring moisture and oxygen having a difference in electrochemical potential. Heavy accumulation of fouling also promotes corrosion of underwater components of ships. *(Brady,R.F.,Griffith,J.R. et al.1978)*

Mechanical Damage

Fouling causes mechanical damage to coatings, moving parts of equipments goes inoperative.

Antifouling systems are required wherever unwanted growth of biological organisms occurs. This generally occurs in saline aqueous environment. Marine Engineered systems have been categorized into seven key types of submerged structures of which ship hulls account for 24% of the total objects fouled (Raikin, *A.I. 2004)* Even though Steel and Aluminum are the materials for the construction of ship hulls they undergo fouling due to constant exposure to a diverse range of environments. Although coatings are used for hull protection, they fail due to the build up of inorganic salts (*Clare A.S., Rittschof, D., Gerhart, J., Maki, J.S 1992)* expolymeric secretions, and the calcium carbonate skeletal structures that from the fouling organisms. There are penalties associated with the unwanted colonization of a hull surface by marine organism *(Townsin, R.L.,2003)* they undergo hull roughness as well as wall shear stress. The effects of antifouling coatings, such as self-polishing copolymer and fouling release coating, on the hydrodynamic boundary layer have been shown little influence on either its thickness or shape factor, although friction velocity was increased *(Candries M., Atlar, J. (2005)*. The negative effects of biofilm roughness on drag was studied by Shultz and Swain *(Schultz M.P., Swain G.W.,2000)*

Biological fouling exploits ship's hull by the settlement of microorganism, which in turn degrade the ship's performance.

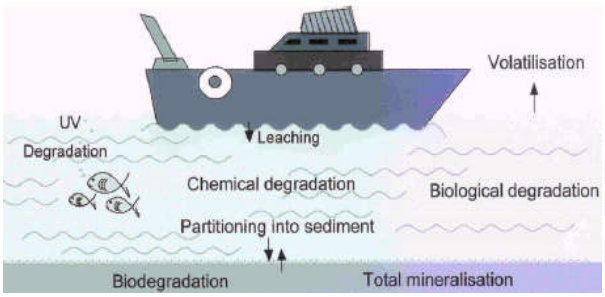


Fig. 3: Fate of active ingredients of antifouling paints in sea water

Historic Antifouling Methods

Ships were historically constructed by wood. The decay from bacteriological and animal attacks was in general mitigated by using hard tight wood, and by treating the wood with "poisoned" tar or oil paint. Later the ships was constructed from steel other forms of "decay" became dominating, and other solutions to prevent such decay (rust) was employed.

Fig: 4: Wooden Constructed Ships

Fouling was first reported on a papyrus dated around 412 BC in which is mentioned they used arsenic and sulfur mixed with Chian oil to help mitigate the problem. Christopher Columbus wrote "All ships were covered with a mixture of tallow and pitch in hope of discouraging barnacles and teredo, and every few months a vessel had to be hoed down and graven on some convenient beach."

In 1625, William Beale filed the first patent for an antifouling recipe that was based on iron powder, copper and cement. Fouling was reported to be up to ½ m long, and giving off odorous and aggressive gases, turning the white lead oxide pigmented paint on the topside darker on a sailing ship anchored in the Indian Ocean. Lord Nelson reportedly employed copper plates attached to the ship's hull to prevent fouling, greatly increasing his ships maneuverability in combat. Steel ships cannot use copperplates due to the galvanic corrosion induced by such bi-metallic couples.

The most common method of prevention of fouling on ship hulls and other underwater structures uses copper or organotin containing paints. Although organotin containing coatings are highly effective, they are also dangerous to the marine environment in which they are used because the tin leachates can poison non-target organisms such as fish, vegetation, and marine mammals.

The use of antifouling coatings for protection from the marine environment has long history. Earlier sailors have used toxic compounds to keep fouling creatures away from hulls. These were like Copper, Arsenic and Mercury. For example, Copper sheathing was earlier in use and was first used by British Noval Ships in 1779. *(Callow, M., 1990,Pain, S, 1999)*

Now a day organotin based antifouling paints is much in use around the world due to their effectiveness even at low concentration against most forms of fouling. However these too have some harmful effects over sea life and the use of toxic organotin derivatives in antifouling paints are fully prohibited.

Organotin compounds do not prevent the accumulation of algae on hull, so small amount of cuprous oxide is used in organotin coatings for control of algae and grasser. *(Ghanem 1980)*

The effectiveness of antifouling paints are generally based upon their toxicity i.e. how much amount of toxic ingredients do these paints possess in their formulations. e.g. Cuprous oxide, Triphenyl or Tributyl derivatives, as cuprous oxide prevents diatoms, algae, sponges and other hard fouling like mollusca from sticking on the surface of hull, it is cheap and easily soluble in sea water. In early 80's antifouling paints have achieved their effectiveness by releasing biocides at their surface. Most of them have been metallic or organometallic substances because these compounds are effective against the broad range of organisms encountered in the marine environment *(Phillip. AT 1973)*. It is clear that toxic compounds used previously and today in marine paints are responsible for some of the present marine pollution problems in coastal waters.

Antifouling paints are the source of most of the contamination of organotin compounds in harbor basins. Large amounts of copper and to some extent lead and mercury found in the sediments originate from these paints as well.

Need for Fouling Release Coatings

Antifouling coatings prevent the growth of marine organisms on hulls, for this growth decrease the speed, maneuverability and range of ships and raises propulsive fuel consumption by as much as 30%. Ultimately ships must be taken from the water and mechanically cleaned to remove fouling Earlier sailors have used poison to keep these creatures off their hulls. Such poisons are as arsenic, cadmium, lead and mercury have been long prohibited by most nations but copper and tin containing toxins continue to be used.

Modern Alternative to Antifouling Paints

Because of the increased evidence of ecosystem damage in areas close to concentrated use of tin-containing paints, application of these antifouling paints is being restricted and in some cases prohibited.

Fouling release-coating technologies are currently under development in response to the need for a nontoxic coating alternative to antifouling paints.

Thermoplastic, non-convertible surface organic coatings, which dry due to simple solvent evaporation, are today readily available although volatile organic compound (VOC) controls are limited in antifouling applications.

Many traditional antifouling systems are paints, which is a comprehensive term covering a variety of materials, enamels, Lacquers varnishes, undercoats, surfacers, primers, sealers, filleres, stoppers and many others *(Turner, G.P.A.,1967.)* Most antifouling coatings are organic and consists of a primer and a topcoat both of which can include anticorrosive

functions, however, the topcoat is often porous. Since the initial phasing out TBT from the antifouling industry in 2001 alternatives have been available (*Omae, I 2003, Watermann, B 1999,Omae, I, 2003)* including biocide-free antifouling coatings *(Watermann, B et.al. 2005, Watermann, B, et.al. 2003)*Fouling organisms may grow on the surfaces of these coatings but adhere poorly and can be removed by light brushing, water spray or by hydrodynamic self-cleaning. Silicone polymers have show better fouling release capability than fluoropolymers and other coatings.

This has been attributed to their being within an optimum range of critical surface tension, which is related (but not equal) to surface energy.

Other factors thought to contribute to silicones superior fouling release ability are their surface structure; extremely low glass transition temperature and low modulus. All of the current coating technology employs condensation cure chemistry.

The coatings are prepared by the reaction of a crosslinker with a silanol polymer in the presence of a condensation cure catalyst such as dibutyltindiacetate.

Types of Antifouling Coatings

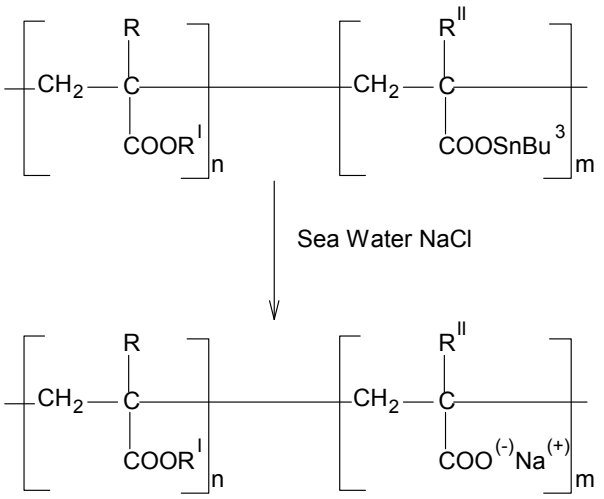
Self Polishing Type

These are Biocidal coatings. A revolutionary self polishing co-polymer technique employing a similar heavy metal toxic action to determine organisms was used with antifoulant Tributyltin (TBT) *(Milne, A. Hails, G., 1976)*

The self-polishing co-polymer technique uses both hydrolysis and erosion to control the antifouling activity. It is an organotin based compound reacted with acrylic polymers (Scheme-1).

The biocide is released following hydrolysis with water. The outer layer of such coating is water-soluble; once the biocide is released the polymer become water-soluble due to the formation of sodium and potassium salts and dissolves slowly, this results in smooth surface.

For example Tributyltin hydrolyzed off, it reacts with chloride ions from seawater to form tributyltin chloride. This type of coating is very effective and can be used for longer duration. (*Takahashi,K. 1991,Takahashi,K and Ohyagi,Y 1990, Atherton, D., Verborgt, J and Winkeler 1979)* They help save fuel.



Scheme 1: Chemical reaction of Organotin polymers in sea water

Thin linear release rate is responsible for the excellent antifouling performance observed with self-polishing paints.*(Khanolka,,R.R 2001, Kajer, E.B 1992).*

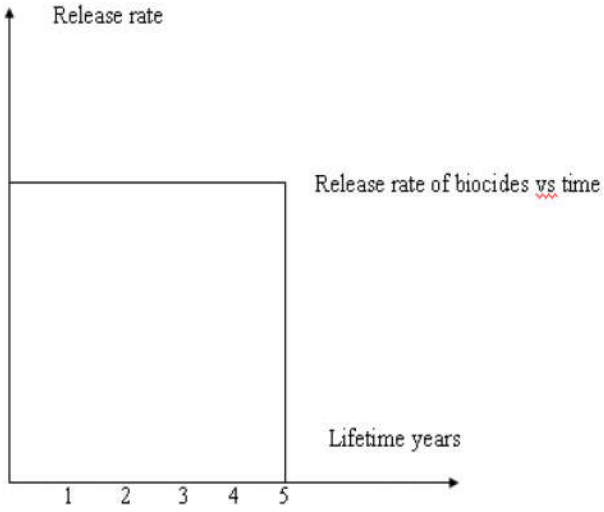


Fig. 5: Self polishing antifouling

Conventional, Soluble Matrix Type Antifouling

Based on cuprous oxide dispersed in gum rosin. Its mechanism based on the partial solubility of the binder, which provides adequate contact between seawater and cuprous oxide. Conventional antifouling work by the dissolution of the acidic rosin in seawater. In principal, the release of biocides remains constant until the paint has completely dissolved. These coatings are mostly used with most of the biocides being metallic or organometallic substances because the compounds are effective

against the broad range of organisms encountered in marine environment, *(Philip, A.T. 1973)*

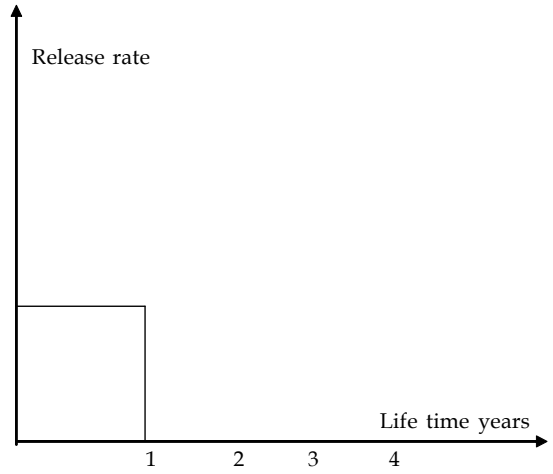


Fig. 6: Conventional antifouling

Advanced, Insoluble Matrix Antifouling

This coating release biocide and other water soluble ingredients, leaving an insoluble binder skeleton. As the thickness of the porous binder skeleton increases, the release of biocide decreases. Commonly used binders are vinyl resin and chlorinated rubber resin *(Upadhya, Shivpujan C. 2002).*

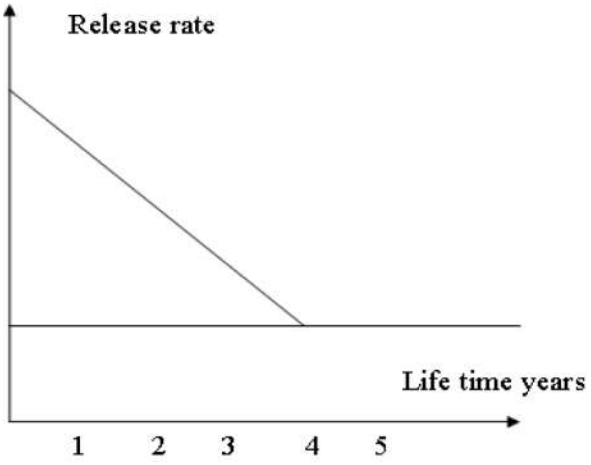
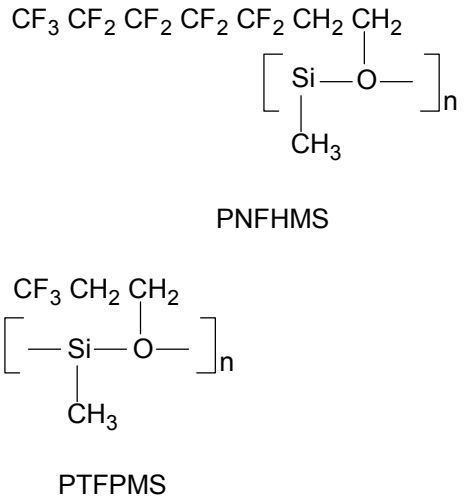


Fig. 7: Insoluble matrix antifouling

Flourinated Coatings

Polymers containing triflouromethyl groups and fluorinated coatings were developed, as flourochemicals *(Upadhya Shivpujan C., 2002)* wettability of the substrates, interatomic attractive forces and molecular interdiffusion and allows coating surface with low intrinsic adhesion.



Scheme 2: Flourinated Silicones

Fluorinated coatings are yet to occupy market. These coatings have excellent resistance properties towards soiling and staining, they give fine color and gloss retention. These coatings are used for other marine uses.

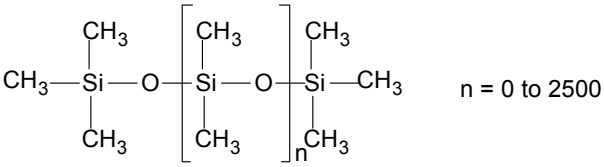
Silicone Coatings

Silicone are fouling release coatings, were first reported in 1972, Polydimethylsiloxane (PDMS) was used as elastomers. Silicones are soft elastomeric materials, having surface energy of above 25mJ/m^2. Since silicone coatings are non-sticky hence did not give good adhesion property therefore it is necessary to develop a coating system that couples hard metal hull to the soft elastomeric silicone coating and ensure adhesion that can adjust marine environment for example: -

Polyurethane – silicone – hydrocarbon coatings,

Polybutadiene – silicone coatings,

Silicone urethane coatings etc.



Scheme-3 Structure of PDMS

Formulation Technique

Different types of Binders and Pigments are used

in antifouling coating *(Borse, Hemant. R. 2003).* Some are listed below:

1. Vinyl coating
2. Epoxy coating
3. Epoxy – polyamide coating
4. Epoxy – ester coating
5. Epoxy – coal – tar – coating
6. Chlorinated – rubber
7. Polyester – glass coatings
8. Urethane coatings

Vinyl Formulation (anticorrosive coat) by %

Read lead	25.18%
Polyvinyl chloride (Alcohol modified)	16.62%
Tricresyl phosphate	1.71%
Aluminum stearate	0.1%
Methyl isobutyl ketone	29.20%
Toluene	27.19%
	100%

Antifouling Coat

Cuprous oxide	80.83
Vinyl resin	8.08
Rosin	8.08
Tricresyl phosphate	3.01
	100 %

Fluorinated – Rubber Antifouling

Red iron oxide	15.2
Rosin	3.73
Talc	7.08
Mlk	20.31
Vinyl	11.16
Xylene	18.84
Bentone	270.51
Menthol	0.17
(Tributyl tin fluoride) TBT	11.86
	100 %

Due to strict environmental laws non – toxic fouling coatings have been prepared. Today's antifouling paints are effective because they contain heavy metals, which are toxic to fouling organisms. Compounds of arsenic, copper, lead, mercury and tin are now forbidden or almost restricted. Research

Table 1: Major Reviews on Antifouling Coatings

Authors	Theme	Year
Abarzua,S. and Jakubwski.S	Biogenic agents to prevent biofouling	1995
Montermosso,J.C. et. al	The polymers of trialkylin acrylates obtained by random or co (or ter) polymerization of tributyltin acrylate with methylmethacrylate	1958
Subramanium, R.V., et.al	Synthesis and properties of thermosett antifouling polymer systems containing tin by crosslinking cycolaliphatic epoxides with free carboxylic groups present on base polymer partially esterified with tributyltin oxide	1977
Pitman,C.U	Chemical anchoring of wild weeds to paints	1976
Videla,H.A	A general review of biofilm	1996
Ghanem,N.A.,et.al	New routes to attach covalently organotin moieties	1975
Van Londen,A.M.,et.al	Showed that the period of fouling protection by the antifouling paints can be substantially extended	1975
Miller,G.A and Lovegrove,T	Antifouling toxicants having high microbial activity against wide range of microorganisms based on isothiazolone derivatives.	1980
Herbert,P.A., et. Al	Described antifouling coatings based on chlorinated rubber	1975
Rascio,V.I.D and Carprar.J.J	Showed that some extenders can also be added to the paint compositions to obtain antifouling properties	1978
Giudice,C.A., et.al	Investigated the bioactivity levels of several antifouling coatings based on gum rosin	1983
Brady,R.F, et.al	Investigated antifouling coatings containing no toxicant but which derive their effectiveness from a surface that weakens or eliminates the adhesive bond between marine fouling and the surface of coating	1987
Cologer,C.P., et.al	Various underwater cleaning methods have been reviewed	1977
Morson,F.	Described a simplified quality control method applicable to Cu (I) oxide based antifouling paints	180
Konstantinou,J.K and Albanist,J.A	Reviewed the worldwide effects of the key booster biocides in antifouling	2004
Champ,M.A and Terlizzi	Reviewed the legislation that culminated in global ban of TBT	2000
Woods Hole	Catalogue of fouling organisms and historic antifouling technology	1952
Fischer et. Al	Technology for control marine biofouling	1984
Wahl.M	Discussed about some basic aspects of fouling andbiofouling	189
Clare,A.S	Discussed the chemical structures, sources and mechanism of testing the efficiency of Antifouling paints	1996
Omae,I	Review of TBT ban and the alternatives focissing on the environmental issue of species	2003
Yebre et. Al	Antifouling technology was reviewed with particular emphasis on commercial products and the development of environmentally benign system	2004

Table 2: Shows the Types of Marine Paint, their Basic Properties and Common uses

Characteristics	Conventional coatings	Bituminous coatings	Vinyl coatings	Chlorinated rubber coatings	Zinc silicate coastings	Pure epoxy coatings	Coal tar epoxy coatings
Number of components	1	1	1	1	2	2	2
Mode of drying	Solvent evaporation and oxidation	Solvent evaporation	Solvent evaporation	Solvent evaporation	Solvent evaporation	Chemical curing	Chemical curing
Application (preferred methods)	Airless Spray, Brush, Roller	Airless Spray	Airless Spray, Conventional Spray	Airless Spray	Conventional Spray, Airless Spray	Airless Spray, Brush, Roller	Airless Spray, Brush, Roller
Qualities (anticorrosive)	***	****	****	****	*****	*****	*****
Antiabrasive	-	*	***	***	*****	*****	*****
Chemical Resistance	*	**	*** (Unmodified coatings)	***	**** (Limited by pH value)	*****	**
Solvent Resistance	*	*	**	**	***** A	***** B	**
Sea Water Resistance	**	**	**	***	** B	****	*****
Antifouling	**	-	***	***	-	-	-
Special qualities	Relatively inexpensive Wide range of colors Good gloss versatile coatings	Excellent water resistance	Good inter-coat adhesion Will dry at low temperature When modified with Coal Tar shows excellent anti corrosive properties	Excellent inter coal adhesion Will dry at low temperature	Excellent anti corrosive and abrasion resistant coatings	Hard, chemically resistant Particularly resistant to alkaline cargoes	Excellent sea water resistant
Additional data			Requires good surface preparation Sa2½ (Swedish standard SIS 05 5600-1967) thermoplastic (softens with heat)	Requires good surface preparation Sa2½ (Swedish standard SIS 05 5600-1967) Thermoplastic	Requires good surface preparation Sa2½ (Swedish standard SIS 05 5600-1967) Critical overcoating parameters and limited pot life	Requires good surface preparation Sa2½ (Swedish standard SIS 05 5600-1967) Critical overcoating parameters and limited pot life does not cure at low temp. (< 5^0C)	Requires good surface preparation Sa2½ (Swedish standard SIS 05 5600-1967) Critical overcoating parameters and limited pot life does not cure at low temp. (< 5^0C)
Common uses	Decks, accommodation, engine rooms, superstructures topsides	Internal under-water areas, Ballast Tanks, and void spaces	Bottom topsides, superstructures Coal Tar modified types are used on	All underwater areas, bootop, topsides, superstructures, decks	Decks cargo tanks shop primers	Internal tanks coatings cargo tanks superstructures	Internal tanks coatings underwater hulls, ballast

work is in progress for deriving alternatives to toxic coatings around the world *(Hittinger, K.J., Kluwer 1988).*

The last ten years has shown an increase in the focus on environmentally acceptable alternatives.

Non – toxic coating works by weakening or eliminating an adhesive bond between marine life and the coating. The fouling organisms dislodge by their own weight or by the motion of the ship through the water *(James D.Adkins, Ann.E.Mera Roe-short et al. 1996).*

Usually antifouling paint contains biocides or toxins held within its structure. The coating is designed to leach biocide slowly into the marine environment. Preventing any organism adhering to the point by poisoning the settling organism. The biocides generally have harmful effects both on fouling organisms for which it is designed to deter but also on marine life unconnected with fouling activity.

It is the potential impact of these points on marine life *(www.marineare. Org.uk/activities/recreation/r03-03.htm).* Today's antifouling paints are based on metals, among the natural substances, metals are most ubiquitous of ultimate persistence. Amongst heavy metals lead, mercury, cadmium and arsenic are the ones, which severely effect the marine environment. Picies are being badly hitten by these heavy metals, studies on certain species of fishes reveled that these fishes exposed to such metals undergo severe damage of their body parts as well as cellular changes, results in their mortality. *(Habib,F.Bajpai.M 2004).* The use of organotins was banned due to sever selfish deformities and the bioaccumulation of tin in some seals and fish *(Strand, J et al. 2005, Evans, S.M., et al. 1995).*Since the service life of antifouling coating is dependent on the dissolution rate and the concentration of the biocide in the coating film, the antifouling coatings based on organotins polymer systems are very efficient because the biocide is covalently bonded to the coating instead of being an external addition to such additives *(GiltizM.H, 1981).*

Qualities of Antifouling Paints

Antifouling paints should possess following qualities:

- Such coatings must be durable enough, so that it can withstand the harsh and unfavorable conditions of marine water.
- Should provide good adhesion i.e. It should properly adhere to base on which it is applied on even under most adverse conditions.
- It is essential for the paint to dry rapidly so the antifouling paint coating must give quick drying after the application on the ship.
- An antifouling coating should be designed in such a manner that it can be easily applied to the substrates, may be by means of spraying, brushing or rolling.
- The last and the most important quality of antifouling paint should by its price i.e. it should be economical. Expensive ingredients should be substituted too less expensive ingredients.

Advantages of Antifouling Paints

- These coatings are eco-friendly, as they do not posses any toxic material.
- Fouling can be easily cleaned as antifouling paint provides low surface energy
- Paint provide smooth surface to the hull thus saving fuel.
- Provide resistance to corrosion and chemical.
- Hinders the stocking of fouling due to non-stick characteristic.

Conclusion

With modern techniques of achieving good surface finish, the advent of high performance coatings, the system of applying coat on ship hull, the recent advances in the antifouling technology with introduction of self-polishing co-polymer compositions makes it possible for offering excellent protection to various segments of ships. The application of antifouling coatings however require careful inspection in order to gain good results in field performance of the products.

The new developments to antifouling coatings must be aimed at

- Excellent surface finish.
- Providing better application techniques so that the paint system can be applied on one or two coats.
- Coatings having excellent exterior durability.
- Superior antifouling, which can be straightaway applied on the anticorrosive paint.
- Antifouling paints should be non-toxic to avoid degrading marine environment.

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Review Article

Screening/Spot Colour Test of Analgesics

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Abstract

Analgesics give relief pain without blocking the conduction of nerve impulse or altering the function of the sensory apparatus. In forensic autopsy case, the forensic pathologist may require a complete toxicological investigation for different poisons including analgesics. The samples have to be analyzed by the Forensic Toxicologist/Chemists/ Scientist. This article deals with the screening/spot test for analgesics. It attempts to simplify the standard procedures in a step-wise manner, which can be of handy reference for the forensic toxicologist.This article is in continuation of Screening/spot/colour test of anti-depresents, Journal of Forensic Chemistry and Toxicology. Volume 2 Number 1,January – june 2016.

Keywords: Analgesics; Screening; Spot Test; Color Test; etc.

Introduction

Analgesia simply means the absence of pain without losing consciousness. Analgesics are the drugs that selectively inhibit the perception (sensation) of pain. There are basically two kinds of analgesics: non-narcotics and narcotics (Table 1). The pain relief induced by analgesics occurs by blocking pain signals going to the brain or by interfering with the brain's interpretation of the signals, without producing anesthesia or loss of consciousness. The common side effects of analgesics are nausea, vomiting, drowsiness, dry mouth, orthostatic hypotension urinary retention and constipation [1-5]. WHO analgesic ladder are

- Patients with mild to moderate pain should be treated with non-opioid analgesic.
- Patients who have limited opioid exposure and present with moderate to severe pain or who fail to achieve adequate relief after a trial of a non-opioid analgesic should be treated with an opioid conventionally used for moderate pain.
- Patients who present with severe pain or who fail to achieve adequate relief following appropriate administration of drugs on the second step of the analgesic ladder should receive an opioid conventionally used for severe pain.

We have tried to set out standard procedures for screening/spot test for analgesics poisons. This article covers different analgesics which include acetanilide, aletamine, alphaprodine, aminophenazone, aspirin, azapropazone, benorilate, butorphanol, cinchnophen, dextromoramide, dextropropoxyphene, diclofenac, diflunisal, dipyrone, etenzamide, fenbufe, fenoprofen, feprazone, ibuprofen, indometacin, ketoprofen, mefenamic acid, marazone, naproxen, nofopam, nifenazone, paracetamol, phenazone, piroxican, salicylamide, sulindac, tolmetin, zomepirac [6-10] etc.

Table 1: Classification of analgesics

S. No.	Narcotic (Opioids)	Non-narcotic (non-opioids)
1.	It relieves pain by acting directly on CNS.	They are principally analgesic and also has anti-inflammatory actions.
2.	They bind to the opioid receptors which are present in many regions of the nervous system and are involved in pain and are classified under controlled substance.	They do not bind to opioids receptors and are not classified under controlled substance.
3.	It can cause addiction to an individual.	It does not cause addiction to an individual.
4.	It produces CNS effect.	It does not produce CNS effect.
5.	It has no anti-inflammatory effect.	It has anti-inflammatory effect.
6.	Examples are butorphanol, dextropropoxyphene etc	Examples are Aspirin, ibuprofen, acetaminophen etc

- **Acetanilide**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish orange colour is observed which indicates the presence of Acetanilide.

- **Aletamine**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of Libermann's reagent are added to it.
3. Orange colour is observed which indicates the presence of Aletamine.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Orange colour is observed which indicates the presence of Aletamine.

- **Alphaprodine**

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Colour changes from brown to red are observed which indicates the presence of Alphaprodine.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Red colour is observed which indicates the presence of Alphaprodine.

- **Aminophenazone**

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Bluish violet colour is observed which indicates the presence of aminophenazone.

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Blue colour is observed which indicates the presence of aminophenazone.

Nitrous Acid Test

1. One to two ml of extract is taken in test tube.
2. Solid sodium nitrite is added in one to two ml of

water.

3. Few drops of hydrochloric acid are added to it.
4. Violet colour is observed which indicates the presence of aminophenazone.

- **Aspirin**

McNally's Test

1. One to two ml of extract in acetone is taken in test tube.
2. One to two ml of water is added to it.
3. Two to three drops of copper sulfate solution is added.
4. Few drops of sodium nitrite solution are added.
5. Red colour is observed which indicates the presence of aspirin.

Jorrisen's Test

1. One to two ml of extract is taken in a test tube.
2. Two ml of distilled water is added to it.
3. Few drops of 10 % potassium nitrite followed by few drops of glacial acetic acid are added to it.
4. Few drops of 10 % copper sulphate solution are added.
5. Red colour is observed which indicates the presence of aspirin.

- **Azapropazone**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Greyish green colour is observed which indicates the presence of azapropazone.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Greyish green colour is observed which indicates the presence of azapropazone.

- **Benorilate**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Black colour is observed which indicates the presence of benorilate.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Green colour is observed which indicates the presence of benorilate.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Violet colour is observed which indicates the presence of benorilate.

- **Butorphanol**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Black colour is observed which indicates the presence of butorphanol.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Grey colour is observed which indicates the presence of butorphanol.

- **Cinchnophen**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish orange colour is observed which indicates the presence of cinchnophen.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Yellow colour is observed which indicates the presence of cinchnophen.

Sulphuric Acid TEST

1. One to two ml of extract is taken in test tube.
2. Few drops of sulphuric acid are added to it.
3. Yellow colour is observed which indicates the presence of cinchnophen.

- **Dextromoramide**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Green colour is observed which indicates the presence of dextromoramide.

- **Dextropropoxyphene**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Brown colour is observed which indicates the presence of dextropropoxyphene.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Grey colour is observed which indicates the presence of dextropropoxyphene.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Colour changes to black to violet and finally to green colour is observed which indicates the presence of dextropropoxyphene.

- **Diclofenac**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish brown colour is observed which indicates the presence of diclofenac.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Reddish brown colour is observed which indicates the presence of diclofenac.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. A colour change slowly to green is observed which indicates the presence of diclofenac.

- **Diflunisal**

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Violet colour is observed which indicates the presence of diflunisal.

McNally's Test

1. One to two ml of extract in acetone is taken in test tube.
2. One to two ml of water is added to it.
3. Two to three drops of copper sulfate solution is added.
4. Few drops of sodium nitrite solution are added.
5. Violet colour is observed which indicates the presence of diflunisal.

Folin-Ciocalteau Test

1. Two ml of extract is taken in a test tube.
2. Few drops Folin-ciocalteau reagent followed by purified water are added to it.
3. One ml of sodium hydroxide solution is added to it.
4. The solution is vortex for 5 seconds.
5. Blue colour is observed which indicates the presence of diflunisal.

- **Dipyrone**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.

3. The solution is heated at 100°C for few minutes.
4. Blue colour is observed which indicates the presence of dipyrone.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Brown colour is observed which indicates the presence of dipyrone.

Nitrous Acid Test

1. One to two ml of extract is taken in test tube.
2. Solid sodium nitrite is added in one to two ml of water.
3. Few drops of hydrochloric acid are added to it.
4. Transient blue colour is observed which indicates the presence of dipyrone.

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Violet colour is observed which indicates the presence of dipyrone.

Folin-Ciocalteau Test

1. Two ml of extract is taken in a test tube.
2. Few drops Folin-ciocalteau reagent followed by purified water are added to it.
3. One ml of sodium hydroxide solution is added to it.
4. The solution is vortex for 5 seconds.
5. Blue colour is observed which indicates the presence of dipyrone.

- **Etenzamide**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Brown colour is observed which indicates the presence of etenzamide.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Green colour is observed which indicates the presence of etenzamide.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. Red colour is observed which indicates the presence of etenzamide.

Nessler's Test

1. Two to three drops of extract is taken in a porcelain basin.
2. Two to three drops of nessler's reagent is added to it.
3. Agitate & heat the mixture to 100! in water bath.
4. Orange to brown colour is observed which indicates the presence of etenzamide.

- **Fenbufen**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish brown colour is observed which indicates the presence of fenbufen.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.
2. Few drops of sulphuric acid are added to it.
3. Yellow colour is observed which indicates the presence of fenbufen.

- **Fenoprofen**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish brown colour is observed which indicates the presence of fenoprofen.

Marquis Test

1. One to two ml of extract is taken in test tube.

2. Few drops of marquis reagent are added to it.
3. Pink colour is observed which indicates the presence of fenoprofen.

- **Feprazone**

P-Dimethylaminobenzaldehyde

1. Two ml of extract is taken in test tube.
2. Few drops of p-dimethylaminobenzaldehyde reagent are added to it.
3. Red colour is observed which indicates the presence of feprazone.

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Brown colour is observed which indicates the presence of feprazone.

- **Ibuprofen**

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Orange colour is observed which indicates the presence of ibuprofen.

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Orangish brown colour is observed which indicates the presence of ibuprofen.

- **Indometacin**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Black colour is observed which indicates the presence of indometacin.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.
2. Few drops of sulphuric acid are added to it.
3. Orange colour is observed which indicates the presence of indometacin.

Mandelin's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of mandelin's reagent are added to it.
3. Grey colour is observed which indicates the presence of indometacin.

- **Ketoprofen**

Koppanyi - Zwikker Test

1. The residue is extracted in 1 ml ethanol in a test tube.
2. One drop of 1% solution of cobalt nitrate in ethanol is added to it.
3. 10 µl of pyrrolidine is added to it.
4. Mixture is agitated for 2 mins.
5. Violet colour is observed which shows the presence of ketoprofen.

- **Mefenamic Acid**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Blue colour is observed which indicates the presence of mefenamic acid.

- **Morazone**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Reddish brown colour is observed which indicates the presence of morazone.

- **Naproxen**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Black colour is observed which indicates the presence of naproxen.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.

2. Few drops of sulphuric acid are added to it.
3. Orange colour is observed which indicates the presence of naproxen

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Brown colour is observed which indicates the presence of naproxen.

- **Nefopam**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Brown colour is observed which indicates the presence of nefopam.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.
2. Few drops of sulphuric acid are added to it.
3. Orange colour is observed which indicates the presence of nefopam.

- **Nifenazone**

Cyanogen Bromide Test

1. One to two ml of extract is taken in test tube.
2. Few drops of cyanogens bromide reagent are added to it.
3. Orange colour is observed which indicates the presence of nifenazone.

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Reddish brown colour is observed which indicates the presence of nifenazone.

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Yellow colour is observed which indicates the presence of nifenazone.

- **Paracetamol**

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Blue colour is observed which indicates the presence of paracetamol.

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Violet colour is observed which indicates the presence of paracetamol.

Nessler's Test

1. Two to three drops of extract is taken in a porcelain basin.
2. Two to three drops of nessler's reagent is added to it.
3. Agitate & heat the mixture to 100! in water bath.
4. Orange to brown colour is observed which indicates the presence of paracetamol.

Folin-Ciocalteau Test

1. Two ml of extract is taken in a test tube.
2. Few drops Folin-ciocalteau reagent followed by purified water are added to it.
3. One ml of sodium hydroxide solution is added to it.
4. The solution is vortex for 5 seconds.
5. Blue colour is observed which indicates the presence of paracetamol.

- **Phenazone**

P-Dimethylaminobenzaldehyde

1. Two ml of extract is taken in test tube.
2. Few drops of p-dimethylaminobenzaldehyde reagent are added to it.
3. Red colour is observed which indicates the presence of phenazone.

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.

2. Few drops of ferric chloride are added to it.
3. Red colour is observed which indicates the presence of phenazone.

Nitrous Acid Test

1. One to two ml of extract is taken in test tube.
2. Solid sodium nitrite is added in one to two ml of water.
3. Few drops of hydrochloric acid are added to it.
4. Green colour is observed which indicates the presence of phenazone.

- **Piroxican**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Yellow colour is observed which indicates the presence of piroxican.

Koppanyi - Zwikker Test

1. The residue is extracted in 1 ml ethanol in a test tube.
2. One drop of 1% solution of cobalt nitrate in ethanol is added to it.
3. 10 μl of pyrrolidine is added to it.
4. Mixture is agitated for 2 mins.
5. Orange colour is observed which shows the presence of piroxican.

- **Salicylamide**

McNally's Test

1. One to two ml of extract in acetone is taken in test tube.
2. One to two ml of water is added to it.
3. Two to three drops of copper sulfate solution is added.
4. Few drops of sodium nitrite solution are added.
5. Orange colour is observed which indicates the presence of salicylamide.

Nessler's Test

1. Two to three drops of extract is taken in a porcelain basin.
2. Two to three drops of nessler's reagent is added to it.
3. Agitate & heat the mixture to 100! in water bath.
4. Brown to orange colour is observed which indicates the presence of salicylamide.

Ferric Chloride Test

1. One to two ml of extract is taken in a test tube.
2. Few drops of ferric chloride are added to it.
3. Violet colour is observed which indicates the presence of salicylamide.

- **Sulindac**

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Brown colour is observed which indicates the presence of sulindac.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.
2. Few drops of sulphuric acid are added to it.
3. Brown colour is observed which indicates the presence of sulindac.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Green colour is observed which indicates the presence of sulindac.

- **Tolmetin**

Formaldehyde-Sulphuric Acid Test

1. Two ml of extract is taken in test tube.
2. Few drops of Formaldehyde-sulphuric acid (6 drops: 4 drops) reagent are added to it.
3. Brown colour is observed which changes to red indicate the presence of tolmetin.

Libermann's Test

1. One to two ml of extract is taken in test tube.

2. Few drops of libermann's reagent are added to it.
3. Red colour is observed which indicates the presence of tolmetin.

- **Zomepirac**

Formaldehyde-Sulphuric Acid Test

1. Two ml of extract is taken in test tube.
2. Few drops of Formaldehyde-sulphuric acid (6 drops: 4 drops) reagent are added to it.
3. Red colour is observed which indicates the presence of zomepirac

Libermann's Test

1. One to two ml of extract is taken in test tube.
2. Few drops of libermann's reagent are added to it.
3. Orange colour is observed which indicates the presence of zomepirac.

Marquis Test

1. One to two ml of extract is taken in test tube.
2. Few drops of marquis reagent are added to it.
3. The solution is heated at 100°C for few minutes.
4. Yellow colour is observed which indicates the presence of zomepirac.

Sulphuric Acid Test

1. Few drops of extract are taken on a white tile.
2. Few drops of sulphuric acid are added to it.
3. Yellow colour is observed which indicates the presence of zomepirac.

Conclusion

In any analysis of poison, screening/spot test is very useful for knowing the presence of the anlagesics which can be confirmed by confirmatory tests. It saves time for the toxicologist in ruling out the poisons. The result of the analytical methods depends on the amount and purity of the sample extracted. Screening/spot test has been developed after repeated trial and testing. The techniques are being improved every time. It is important for the forensic toxicologists to know the best available method so as to help them in detecting the type of poison administered in the process of crime investigations.

- *Preparation of Solutions/ Reagents*

Cyanogen bromide

Solution I: Decolourisation of bromine water is done by the addition of solid potassium cyanide and then more bromine solution is added until the solution becomes pale yellow. Solution II: Saturated solution of aniline in water. Solution (I) and (II) are mixed.

Ferric Chloride Solution

10 gm of ferric chloride is dissolved in 100 ml distilled water.

Folin-Ciocalteau Reagent

100 gm of sodium tungstate and 25 gm of sodium molybdate are mixed in 800 ml of water. 50 ml of concentrated orthophosphoric acid and 100 ml of concentrated hydrochloric acid are added and refluxed for 10 hours. After cooling 150 g of lithium sulfate, 50 ml of water and 0.5 ml of elemental bromine are added and allowed to stand for 2 hours and then boiled for 15 minutes to remove excess bromine. Content is cooled and filtered if necessary, and diluted to 1 litre with water. This solution is yellow and could be stable for 4 months if stored at 4°C.

Formaldehyde-Sulphuric Acid

Four volumes of sulphuric acid and six volumes of formaldehyde solution are mixed.

Koppanyi- Zwikker reagent

A 1% (w/v) solution of cobalt nitrate in ethanol.

Libermann's Reagent

1 gm of sodium or potassium nitrite is mixed in 10 ml of sulphuric acid with cooling and swirling to absorb the brown fumes.

Mandelin's Reagent

1 gm of ammonium vanadate is dissolved in 1.5 ml of water and diluted to 100 ml with concentrated sulphuric acid.

Marquis Reagent

100 ml of concentrated sulphuric acid is mixed

with 1 ml of 40% (v/v) formaldehyde solution.

McNally's Reagent

Solution (I) (0.5 % solution of copper sulphate in 10 % acetic acid. Solution

Solution(II) Freshly prepared 2% solution of sodium nitrite.

Solution (I) is mixed with solution (II).

Nessler's Reagent

Solution (I) 50 gm of mercuric chloride and 35 g of potassium iodide are dissolved in 200 ml of water and cool.

Solution (II) 50 gm of sodium hydroxide is dissolved in 250 ml of water and cool.

Cold solution (II) is mixed with cold solution (I) and made up to 500 ml with water. Mixture is allowed to stand and decanted the clear supernatant for use. Stored in dark brown bottles away from light.

Nitrous Acid

Small amount of solid sodium nitrite is added to 2M hydrochloric acid.

P-Dimethylaminobenzaldehyde

1 g of p-dimethylaminobenzaldehyde is dissolved in 100 ml of ethanol. The solution is acidified with 10 ml of dilute hydrochloric acid.

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Case Report

Copper Sulphate Poisoning Mimicking Smothering: A Case Report with Review of Literature

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Abstract

Copper sulphate is an inhibitor of many enzymes. Its poisoning can cause death because of hemolysis, shock and renal failure. We present a case of a brought dead person where the external appearance of body was giving the picture of assault and smothering but after detailed external and internal examination the cause of death turned out to be copper sulphate poisoning. The authors intend to add to Medical literature this important presentation, which should be kept in consideration while examining cases of copper sulphate poisoning to prevent misinterpretation of external findings as traumatic injuries. One such wrong opinion by an autopsy surgeon can lead to harassment of an innocent person leading to miscarriage of justice.

Keywords: Copper Sulphate; Poisoning; Smothering.

Introduction

An autopsy surgeon should be well aware of the normal as well as abnormal presentations of the autopsy findings so as to interpret the injuries correctly. Interpretation of injuries is a confounding factor that can result in giving of incorrect opinion which can result in miscarriage of justice. To an inexperienced pathologist a lacerated wound over scalp can resemble an incised wound, distant gunshot wounds can be confused with drainage wounds, ant bites may be confused with antemortem abrasions, artificial bruise may be created by chemicals or poisons etc. Post mortem artefacts can resemble antemortem injuries for example undertakers fracture can occur because of mishandling of body [1]. Precautions should be taken while doing post-mortem examination in those cases where no reliable history is available and no characteristic appearance is present. It is possible that the autopsy surgeon may misinterpret some findings as traumatic injuries. We present a case of a young adult male whose external post-mortem appearance gave the picture of assault and smothering, but after detailed external and internal examination, the case turned out to be a case of copper sulphate poisoning.

Case Report

A dead body of 29 year old man was found at his home. The case was then brought for post mortem examination at VMMC and Safdarjung Hospital, New Delhi. On post mortem examination, bluish discoloration over the face of the deceased was present at multiple places (Figure 1). It appeared that there were number of contusions present on the

face of deceased, leading to the suspicion of assault. The lower lip and tip of tongue also had bluish discoloration giving an appearance of smothering. (Figure 2) However on further dissection, no extravasation of blood was found in the underlying tissues. On internal examination, it was seen that there was bluish discoloration of tongue, esophagus and stomach wall, with powder like substance adherent to mucosa. About 200 ml of bluish green fluid was found in stomach of deceased with bluish material adherent to stomach wall (Figure 3). All internal organs were congested. Brain was congested and edematous. Rest of the findings were unremarkable. Toxicological analysis revealed the presence of copper sulphate.

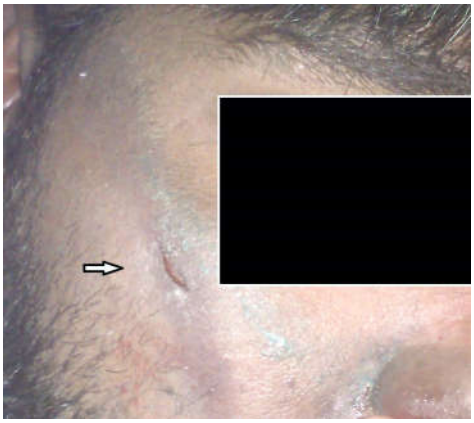

Fig. 1: Bluish discoloration mimicking contusion over right malar eminence with no extravasation in underlying tissue

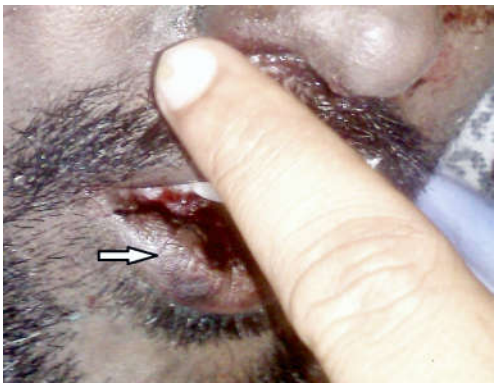

Fig. 2: Bluish discoloration on inner aspect of lower lip and tongue mimicking smothering

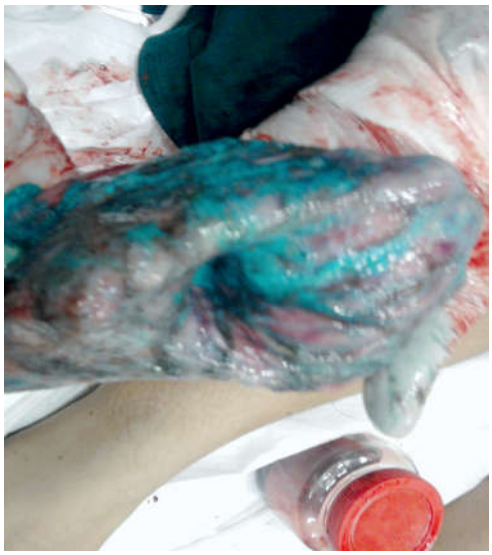

Fig. 3: Bluish green powdery substance found in stomach with hemorrhagic stomach wall

Discussion

Copper is used in manufacture of insecticide and fungicide, as pigments, in making utensils and can be added in medicine [2]. Copper as a metal is not poisonous. In fact, it is an essential trace element with daily dietary requirement of 2 mg and normal body concentration of 50-150 mg [3]. The compounds of copper are poisonous. They copper sulphate is an irritant are powerful inhibitor of enzymes. Copper sulphate an irritant, a corrosive and causes hemolysis. Copper inhibits the enzyme glucose-6-phosphate dehydrogenase and glutathione resulting in intravascular haemolysis [4]. Copper ions inhibit the pyruvate dehydrogenase system causing liver damage [5,6]. Copper increases the permeability of cell membranes by inhibiting the sodium potassium ATPase pump [4]. It can also cause rhabdomyolysis by damaging skeletal muscle cells [7].

The symptoms of acute copper poisoning can start in 15-30 minutes. These include metallic taste, increased salivation, burning pain in stomach, thirst, eructations, repeated vomiting (bluish green), diarrhoea, oliguria, hematuria, albuminuria, acidosis and uraemia. It can also cause pancreatitis, coma, hepatic or renal failure. Subcutaneous administration of copper compounds can cause skin necrosis [1,2,7-11]. The normal reference range for serum copper in healthy adults is 0.7-1.6 microgm/mL [12]. The fatal dose of copper sulphate is 30 grams and of copper subacetate is 15 grams [1]. Sudden deaths are rare as death usually occurs in 1-3 days [7].

Copper sulphate poisoning though rare worldwide, is common in India [13]. Most cases are suicides but accidental deaths have also been reported [14-16]. Cases have been reported where children have consumed it because they became attracted to its colour or when the wound was irrigated with copper sulphate or as a complication of hemodialysis [17-19].

Gulliver JM reports a case of fatal copper sulphate poisoning where a girl child died within 2 hours of consumption of poison. She complained of stomach

pain and vomited blue coloured vomitus [12]. Chuttani et al in 1965, concluded that deaths occurring within 24 hours of copper poisoning is because of shock while that occurring at a later stage is because of renal or hepatic failure [14].

The postmortem findings in a case of copper poisoning are bluish or greenish discoloration of the gastric mucosa with congestion and erosion, which were typically present in our case [7,8]. The authors found it important to report this case because of the fact that deceased was found dead at his home with no history of consumption of copper sulphate poisoning or any other poisoning and was brought for autopsy with no lead whatsoever to the autopsy surgeon. If the bluish discolorations over the face would have been interpreted as contusion due to assault and smothering, the police would have been misguided into a totally wrong direction of investigation. The authors intend to add to Medical literature these abnormal findings in a case of copper sulphate poisoning to act as a reference for the autopsy surgeons.

Conclusion

Copper sulphate poisoning can gave the appearance of multiple contusions over face, lips and tongue mimicking assault and smothering. Care should be taken by the autopsy surgeon while analysing such cases to avoid misinterpretation of the post mortem findings as their wrong opinion about the manner of death could lead to miscarriage of justice and harassment of an innocent person in investigation.

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Journal of Forensic Chemistry and Toxicology [JFCT]
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Title	Frequency	Rate (Rs): India	Rate ($):ROW
Dermatology International	2	5000	500
Gastroenterology International	2	5500	550
Indian Journal of Agriculture Business	2	5000	500
Indian Journal of Anatomy	3	8000	800
Indian Journal of Ancient Medicine and Yoga	4	7500	750
Indian Journal of Anesthesia and Analgesia	3	7000	700
Indian Journal of Anthropology	2	12000	1200
Indian Journal of Biology	2	4000	400
Indian Journal of Cancer Education and Research	2	8500	850
Indian Journal of Communicable Diseases	2	8000	800
Indian Journal of Dental Education	4	4500	450
Indian Journal of Forensic Medicine and Pathology	4	15500	1550
Indian Journal of Forensic Odontology	2	4500	450
Indian Journal of Genetics and Molecular Research	2	6500	650
Indian Journal of Law and Human Behavior	2	5500	550
Indian Journal of Library and Information Science	3	9000	900
Indian Journal of Maternal-Fetal & Neonatal Medicine	2	9000	900
Indian Journal of Medical & Health Sciences	2	6500	650
Indian Journal of Obstetrics and Gynecology	3	9000	900
Indian Journal of Pathology: Research and Practice	3	11500	1150
Indian Journal of Plant and Soil	2	5500	550
Indian Journal of Preventive Medicine	2	6500	650
International Journal of Food, Nutrition & Dietetics	3	5000	500
International Journal of History	2	6500	650
International Journal of Neurology and Neurosurgery	2	10000	1000
International Journal of Political Science	2	5500	550
International Journal of Practical Nursing	3	5000	500
International Physiology	2	7000	700
Journal of Animal Feed Science and Technology	2	4100	410
Journal of Cardiovascular Medicine and Surgery	2	9100	910
Journal of Forensic Chemistry and Toxicology	2	9000	900
Journal of Microbiology and Related Research	2	8000	800
Journal of Orthopaedic Education	2	5000	500
Journal of Pharmaceutical and Medicinal Chemistry	2	16000	1600
Journal of Practical Biochemistry and Biophysics	2	5500	550
Journal of Social Welfare and Management	3	7500	750
New Indian Journal of Surgery	3	7100	710
Ophthalmology and Allied Sciences	2	5500	550
Otolaryngology International	2	5000	500
Pediatric Education and Research	3	7000	700
Physiotherapy and Occupational Therapy Journal	4	8500	850
Urology, Nephrology and Andrology International	2	7000	700
SUPER SPECIALITY JOURNALS			
Indian Journal of Emergency Medicine	2	12000	1200
Indian Journal of Surgical Nursing	3	5000	500
Indian Journal of Trauma & Emergency Pediatrics	3	9000	900
International Journal of Pediatric Nursing	3	5000	500
Journal of Community and Public Health Nurisng	2	5000	500
Journal of Geriatric Nursing	2	5000	500
Journal of Medical Images and Case Reports	2	5000	500
Journal of Nurse Midwifery and Maternal Health	3	5000	500
Journal of Organ Transplantation	2	25900	2590
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Psychiatry and Mental Health	2	7500	750

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