Journal of Veterinary Medicine and Health

Ayub, J Vet Med Health 2021, 5:2

Review Article Open Access

Arsenic Induced Alternation in Animals by its Toxicity

Hina Ayub*

Department of Zoology, University of Narowal, Narowal, Pakistan

***Corresponding author:** Dr. Hina Ayub, Department of Zoology, University of Narowal, Narowal, Pakistan, E-mail: ayubhina@gmail.com

Received date: February 18, 2021; **Accepted date:** March 04, 2021; **Published date:** March 11, 2021

Abstract

This study assessed the developmental and behavioral effects of chronic arsenic exposure in animals. Therefore, the current research is to study the effects of arsenic trioxide on nephrotoxicity, hepatoxicity, and nephrotoxicity. Attempts to monitor the effect of arsenic are based on extensive literary research, with special emphasis on the latest works. Arsenic is a carcinogen to both humans and animals. Arsenicals have been associated with skin, lung, and bladder cancers of the skin, lung, and bladder. Arsenic (As) is classified as a metalloid and exhibits both metallic and nonmetallic properties. It is found in ore and crust rocks with average density. It exhibits a complex chemistry, occurring in four different valences (-III, O, III and V), and in many different chemical forms and it has these being used for a wide variety of industrial and agricultural purposes. Arsenic oxide is the most important arsenic compound used in industry and is used to synthesize other inorganic and biological weapons. Arsenic trioxide is now used to treat acute promonocytic leukemia. Absorption occurs primarily through ingestion in the small intestine, although less absorption occurs through skin contact and respiration. Acute arsenic poisoning was initially associated with nausea, vomiting, abdominal pain, and severe diarrhea, and peripheral neuropathy was reported. Chronic arsenic poisoning can cause multisystem disease. Affects signaling pathways by being able to activate proteins such as ERK2, p38, and JNK as shown in mammals. A comparison between the phosphorylation sites of these proteins is performed to determine whether they are observed in water and in mammals.

Introduction

Arsenic is a metalloid found mainly in water, soil, and air from natural and man-made sources. It is volatile in organic forms and in various oxidation states (−3, 0, +3, +5). In terms of environmental exposure, toxicologists are primarily concerned with arsenic in the mild pentavalent oxidation state. Arsenic ionic form, arsenic acid ionic form, and arsenic compounds. Monomethylargonic acid (MMAV) and dimethylarcinic acid (DMAV) are volatile arsenic-stable methylated mammalian metabolites that are mainly excreted in the urine. One type of interest is that DMAV and sodium salts of MMAV have been used as herbicides.

Literature Review

Occurs extensively in animal and vegetable cells, arsenic has been extensively studied for its toxicity and therapeutic use. It is found in rocks and soils ranging from one million (ppm) to hundreds of ppm, with an average value of 2 ppm. Arsenic is used as an agricultural wood preservative, desiccant, and herbicide and as a finishing agent for the glass industry and for copper and lead base alloys. Human emissions of arsenic are estimated at 41,000 metric tons per year. Global emissions from natural resources are estimated at 7800 metric tons per year). Further input of marine arsenic encouraged the study of the accumulation and toxic effects of marine organisms [1].

In regard to arsenic, most people think of it as a dead toxin that has been used for centuries. However, most human exposure to arsenic comes from sources such as drinking water, food, dust, and soil. Although it is believed that low levels of arsenic are essential nutrients (US Environmental) has adverse health effects, although the concentration of exposure in drinking water depends on the determination of the current maximum container level. Some studies have reported that arsenic leads to gene enhancement, chromosomal abnormalities, inhibition of DNA repair, global DNA hypomythylation, decreased DNA methyl transferase activity, and protonogen activation. Previously, we reported that V79-Cl3 Chinese hamster cells underwent early genetic instability or apoptosis when exposed to sodium arsenic (SA) [2].

In observations made during and after treatment, genetic instability was evident in the presence of aniploid and morphologically abnormal cells, but not chromosome abnormal cells. As mitotic cells became more sensitive to SN exposure due to the direct action of arsenic in the mitotic spindle assembly, we later discovered the movement of genetically unstable cells that escape apoptosis by cutting mitotic round-up. Cells at the end of 24-hour treatment. The cell population was still not genetically stable when tested after 2 months of cell culture (120 cell generations) [3].

Although there has been much progress in the field of synthetic drugs in recent years, they have had some or other side effects, but it has been suggested that naturopathy with phytochemicals may still be beneficial in many situations. Arsenic is formed in the blood-brain barrier, where it has neurotoxic effects on many structures, such as the basal ganglia. Although information on the effects of arsenic on the central nervous system is limited, the basal ganglia appear to be particularly sensitive to the effects of toxins such as 3-nitroprionic acid, succinic dihydroxydip; For example, manganese globus causes selective neuronal damage in palidas and iron is abnormally deposited in the form of Parkinsonism.

Sodium arsenic exposure in rats increased levels of strytal dopamine, 3,4-dihydroxyphenyl acetic acid (dopak), and 5-hydroxindol acetic acid (5-HIOA). Exposure to arsenic trioxide in the striatum reduced DOPAC and homovanolic acid (HVA.) In mice exposed to sodium arsenate, serum serotonin content increased and

dopamine levels in the nucleus accumbens decreased. Dopamine and its metabolic disorders (dopamine, HVA) have been described in the striatum of mice contaminated with high arsenic content. Dopamine is involved in many functions, including movement control, learning and memory, cognition, and emotion. If the monoamine content of the basal ganglia can be modified by arsenic exposure, it affects the behavior. Studies of locomotor activity in rats have been reported to reduce the levels of arsenic trioxide used. Defects in functional study work have also been reported. The inefficiency of the data and the fact that the results of the dose and exposure time have not been examined in previous studies make it difficult to come to a conclusion about the effects of arsenic on the nervous system in the in vivo model. To study the neurotoxic effects of arsenic exposure and to assess the ance of basal ganglia for functions such as learning, memory, and movement, it is important to assess whether arsenic exposure alters these complex functions [4].

PubMed has conducted a literary search using the Google Scholar and Science-Hub database to identify relevant studies from the past to the present. Summary of studies reported on exposure to arsenic-contaminated drinking water.

Using a cross-checked reference list from previous review articles, I searched manually for more relevant studies to identify studies that could not be retrieved through an electronic database. We only consider peer-review journals and articles published in English. Each selected article critically assessed and summarized the range of relationships between position, design, gestational outcome, organ impact, sample size, exposure assessment, and arsenic exposure and its outcome. Arsenic was used as an attenuating agent after Greek physicians such as Hippocrates and Galen popularized its use. Arsenic compounds are found in solutions, tablets, pastes, and injection forms. Fowler's solution of 1% arsenic trioxide was widely used in the 19th century [5]. Most recently, in 1958, the British Pharmaceutical and Therapeutic Products Handbook, revised by Martindale, listed indications for Fowler's solution: leukemia, skin conditions (psoriasis, dermatitis, herpetiformis, eczema and stomatitis in infants). Fowler's solution is also indicated as a health tonic. Overdose can lead to severe reactions such as diarrhea, vomiting, pain, dehydration, and weakness. Nowadays, severe intoxication is very rare in Western European countries; if that happens, it is usually the result of intentional (suicide or homicide) or accidental poisoning. Professional exposure to As is very rare and usually occurs in the form of arsenic gas. It causes different symptoms due to eating It is associated with gastrointestinal disorders such as nausea, vomiting, abdominal pain, and severe diarrhea. Cardiac and pulmonary symptoms include hypotension, shock, pulmonary edema, and cardiac arrest. Arsenic exposure is caused by inhalation, absorption through the skin and, first, the consumption of contaminated drinking water. Arsenic in the diet occurs as relatively nontoxic organic compounds (arsenobentin, arsenicoline). Seafood, fish, and algae are rich biological resources. Arsenic poisoning changes with its oxidation conditions. Organic species are generally more toxic than organic matter, and arsenic (AsIII) is 60 times more toxic than arsenate. This latter species is 70 times more toxic than the methylated species, with monomethylargonic acid (MMA) and dimethylarcyanic acid (DMA), the last two forms being considered only moderately toxic due to the role of phytoplankton in defining the fundamental properties of seawater, very little has been published about the total arsenic in plankton. The total arsenic concentration of three species of suplankton from seawater in the Northeast Atlantic Ocean [6]. The methods by which arsenic exposure adversely affects pregnancy are not fully understood, however, several possible approaches have been suggested based on experimental evidence. Arsenic occurs in elemental form as part of organic compounds or volatile compounds. In water, volatile organisms predominate. The main target of the toxic effects of many metals is the nervous system, especially heavy metals such as mercury, lead, and arsenic. Nervously effects are many and varied. Peripheral neuropathy is the most common finding that mimics Gullein-Barre syndrome with similar electromyographic results. Improved oxidative stress is an important component in arsenic-induced neurotoxicity. Arsenic exposure has been found to cause oxidative damage to ecosystems by increasing the production of free radical species [7-10]. Arsenic treatment involves histological changes in hepatic tissues, including cytoplasmic vacuuming, cell degeneration, and focal necrosis. Accumulation of neutrophils and lymphocytes leads to degeneration of liver tissue and necrosis of the central vein. Similar results in liver pathology in mice confirm our results. Biochemical and morphological evidence of arsenite-induced hepatotoxicity in rat liver development has been found. Evidence includes GSH levels (leading to increased oxidative stress), size of hepatocyte nuclei, intermittent vaculation of hepatocytes, and sinusoidal dilatation in arsenic-fed animals. To understand the exact mechanisms underlying arsenic-induced toxicity at the cellular and molecular levels, extensive studies using different doses of sodium arsenite and different lengths of exposure to different developmental periods should be considered. Kidneys are prone to arsenic-induced damage due to large perfusion and increased concentration of excretory compounds in renal tubular cells (Verma et al., 2004). Our results confirm that there are increased serum urea, uric acid, and creatinine levels in the excretion of high levels of arsenic metabolism by renal tubular cells, which are more prone to damage and kidney problems.

Conclusion

Arsenic compounds are toxic substances that can greatly affect the health of animals. Arsenic poisoning has been confirmed to be related to its chemical forms. Although most are known to affect signaling pathways in mammals, in this review we compared phosphorylation sites in different mammalian and nonmammalian species.

As a result, we can assume that the same effects found in mammalian specimens of aquatic life are actually likely to occur. In addition, arsenic has been shown to cause oxidative stress in mammals and some aquatic organisms. Current work shows that animals intoxicated with arsenic trioxide show significant impairment of liver function and kidney function. There are reports that the effects of AS exposure on the development of sensory systems are minimal. In addition, it is likely to cause significant changes in certain biochemical parameters, including the liver and kidneys. There is consistent and reliable evidence to support a positive relationship between high levels of inorganic arsenic exposure (>50 ppm) to drinking water, miscarriage, stillbirth, and low birth weight.

References

1. Brodziak A, Kołat E, Wolińska A. Conclusions from the applications of transcranial magnetic stimulation on the psychopathology of depressive syndromes and the essence of self-consciousness. Psychiatria Polska. 2018;52: 767-772.
2. Hofmann SG, Gómez AF. Mindfulness-Based Interventions for Anxiety and Depression. Psychiatr Clin North Am. 2017;40: 739-749.

3. Eisendrath SJ, Gillung E, Delucchi KL, Segal ZV, Nelson JC, et al. A Randomized Controlled Trial of Mindfulness-Based Cognitive Therapy for Treatment-Resistant Depression. Psychother Psychosom. 2016;85: 99-110.
4. Cladder-Micus MB, Speckens AEM, Vrijsen JN, T Donders AR, Becker ES, Spijker J. Mindfulness-based cognitive therapy for patients with chronic, treatment-resistant depression: A pragmatic randomized controlled trial. Depress Anxiety. 2018;35: 914-924.
5. Kuyken W, Warren FC, Taylor RS, Whalley B, Crane C, Bondolfi G, et al. Efficacy of Mindfulness-Based Cognitive Therapy in Prevention of Depressive Relapse: An Individual Patient Data Meta-analysis From Randomized Trials. JAMA Psychiatry. 2016;73: 565-574.
6. Gross CG, Rocha-Miranda CE, Bender DB. Visual properties of neuron in inferotemporal cortex of the macaque. J Neurophysiol. 1972;35: 96-111.
7. Mishkin M. A memory system in the monkey. 1982;298: 83-95.
8. Galus W, Starzyk J. Reductive Model of the Conscious Mind. IGI Global publication. 2020.
9. Devue C, Brédart S. The neural correlates of visual self-recognition. Conscious Cogn. 2011;20: 40-51.
10. Uddin LQ, Kaplan JT, Molnar-Szakacs I, Zaidel E, Iacoboni M. Self-face recognition activates a frontoparietal "mirror" network in the right hemisphere: an event-related fMRI study. Neuroimage. 2005;25: 926-935.