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Chemistry, Biochemistry, Toxicity and Pollution of Molybdenum: A Mini Review

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Abstract

Molybdenum is used in various industries. Its pollution has been recorded globally and is an emerging pollutant. Molybdenum is poorly represented in the literature as compared to heavy metals such as mercury, chromium, arsenic, lead and cadmium for instance, due to the metal's low toxicity to humans. It has now been reported that molybdenum is very toxic to embryo and spermatogenesis of fish and mice and this worrying trend would place molybdenum at the forefront of toxicology and bioremediation studies in the future. This mini review attempts to summarize what we know on its chemistry, biochemistry, toxicity and pollution with the hope that this knowledge would be useful for future studies on molybdenum's removal from the environment.

INTRODUCTION

A 14th century Japanese sword was found to contain molybdenum. However, it was not until 1778 that the Swedish scientist, Carl Wilhelm Scheele, was able to positively identify molybdenum. He decomposed molybdenite by heating it in air to yield a white oxide powder (molybdenum trioxide). Shortly thereafter, in 1782, Peter Jacob Hjelm reduced the oxide with carbon to obtain a dark metallic powder, which he named "molybdenum" from the Greek word "molybdos" or lead-like [1].

Molybdenum is obtained from minerals such as molybdenite (MoS_2), wulfenite ($PbMoO_4$), ferrimolybdate ($FeMoO_3.xH_2O$), and jordisite (amorphous MoS_2). The largest molybdenum producing country is America, producing nearly half of the world production of molybdenum, estimated at twelve million metric tonnes. In Malaysia, molybdenum is not mined commercially (Perangkaan Industri Perlombongan, 1999). However, several potential sites for future molybdenum mining in the Peninsula of Malaysia have been identified in the 1970's as shown in Figure 1 [2].

Molybdenum is the fourth member of the second transition series and is placed with chromium and tungsten in Group VIB of the Periodic Table. Molybdenum has an atomic number of 42 and an atomic weight of 95.94 g/mole. In its chemical properties, molybdenum resembles tungsten and vanadium, the first member of Group VB, rather than chromium [4]. Chemically, molybdenum is extremely versatile, forming compounds in a range of readily inter-convertible oxidation states. Commonly used molybdenum-oxygen compounds are molybdenum trioxide, MoO_3, sodium molybdate, $Na_2MoO_4.2H_2O$, ammonium dimolybdate, $(NH_4)_2Mo_2O_7$, and ammonium heptamolybdate, $(NH_4)_6Mo_7O_{24}.4H_2O$.

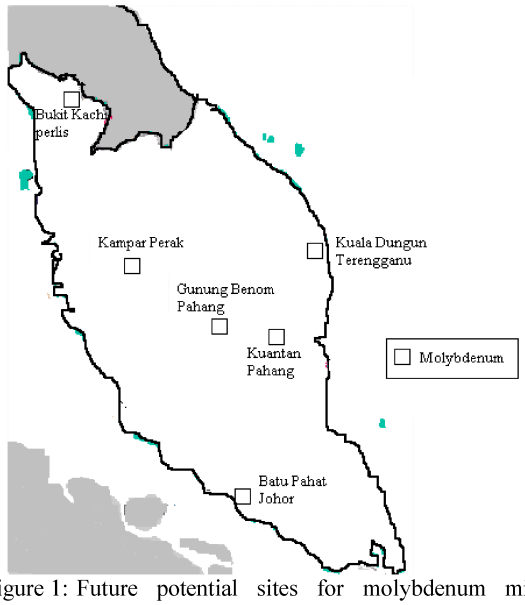

Figure 1: Future potential sites for molybdenum mining in Peninsular Malaysia [3].

Molybdenum has many oxidation states ranging from 2- to 6+. The lowest oxidation states, 2- to 1+, occur in complexes with pi-acceptor ligands, mainly carbon monoxide, cyclopentadiene, nitric oxide, and phosphorus- and arsenic-donor ligands. The low oxidation states of molybdenum (2- to 2+) occur in coordination environments which are unlikely to be encountered in biological systems and so are not expected to arise in enzymatic processes. In the oxidation states of 3+ to 6+, molybdenum forms a large number of complexes with oxygen- and nitrogen-donor ligands and with the halogens. Complexes with sulphur-donor ligands are common but there are few complexes with phosphorus- and arsenic-donor ligands [5].

Molybdenum (6+) or Mo^{6+} ion does not exist in solution. It exists as molybdate ions, $[MoO_4]^{2-}$ [6]. Under acidic conditions molybdate ion would combine and form polyions such as $Mo_7O_{24}^{6-}$, $Mo_8O_{26}^{4-}$ and $Mo_{12}O_{37}^{2-}$ [7]. These polyions can be reduced by reducing agents to form "isopolymolybdenum blue". They could also combine with many heteroatoms such as; phosphate, arsenate, tungstate, sulphate, and silicate forming molybdophosphate, arsenomolybdate, tungstomolybdate, sulphomolybdate, and silicomolybdate respectively.These heteroatoms, which are situated inside "cavities" that are basket-like, consists of several tetrahedral molybdates anions joined to each other at the oxygen atom [1]. These latter compounds are known as heteropolymolybdates, which can be reduced by a variety of reducing agents such as dithionite, ascorbic acid and metal ions into intense blue, colloidal products known as heteropolymolybdenum blue. This phenomenon is a prominent feature of its chemistry [6].

The mechanism of heteropolymolybdate reduction to molybdenum blue or Mo-blue has been extensively studied. According to the electron spin resonance (esr) work, dithionite, a reducing agent, donates two electrons to a heteropolymolybdate, $PMo_{12}O_{40}^{3-}$ (12-molybdophosphate) converting it to Mo-blue. The introduced electrons are uniformly dispersed over the whole polymetallate sphere by a thermally activated hopping process. The electrons in the two-electron reduced forms were shown by ^{17}O nuclear magnetic resonance (nmr) spectroscopy to be very mobile, thus averaging the valence of all 12 molybdenum atoms [8].

This explains the mixed valence (between 5+ and 6+) properties of Mo-blue [4]. The scanning spectrum of the resultant Mo-blue from ascorbic acid-reduced 12-molybdophosphate show a maximum peak of between 860 and 880 nm and a characteristic shoulder approximately at 700 nm [9]. The difference between one heteropolymolybdate species to another can be seen in the scanning spectra of their reduced form (Mo-blue). Figure 2 shows the scanning spectra of various reduced heteropolymolybdates whilst Figure 3 shows the structure of a 12-molybdophosphate.

The Biochemistry of Molybdenum

Molybdenum in Mammals

Molybdenum is an essential constituent of the enzymes xanthine oxidase (XOD) and aldehyde oxidase (AOD), which occur in the liver and intestines, and hepatic sulphite oxidase. XOD has been isolated from cow's milk. The enzyme oxidizes hypoxanthine to xanthine and xanthine to uric acid in purine catabolism (Figure 4).

XOD is a molybdoflavoprotein enzyme with the composition 2FAD:8Fe:2Mo. XOD has a broad spectrum of substrates and will catalyse the oxidation of many purines and aldehydes with very different rates of reaction [10].

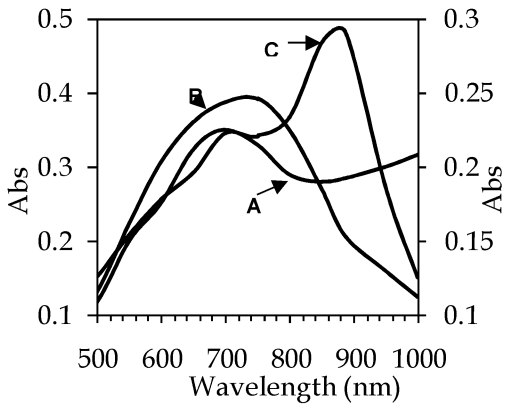

Figure 2: Scanning spectra of Mo-blue from molybdosilicate (A), molybdosulphate (B) and molybdophosphate (C).

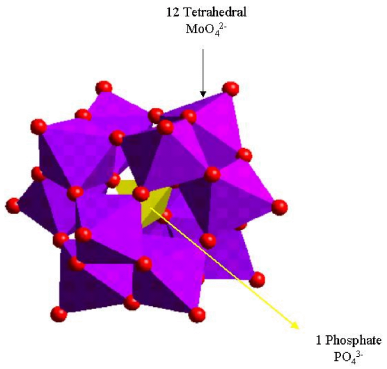

Figure 3: The chemical structure of 12-molybdophosphate (from Molecular Simulations Inc., 2000).

The role of molybdenum in XOD as a cofactor involved in electron transport has been comprehensively studied [10, 11-13]. With the exception of the nitrogenase cofactor, molybdenum is integrated into proteins as the molybdenum cofactor that contains a mononuclear molybdenum atom coordinated to the sulphur atoms of a pterin derivative named molybdopterin. During the enzyme-catalysed reaction, the oxidation state of molybdenum changes and so molybdenum is involved in the electron-transfer pathway. Molybdenum-cofactor-containing enzymes catalyse the transfer of an oxygen atom, ultimately derived from or incorporated into water, to or from a substrate in a two-electron redox reaction [14].

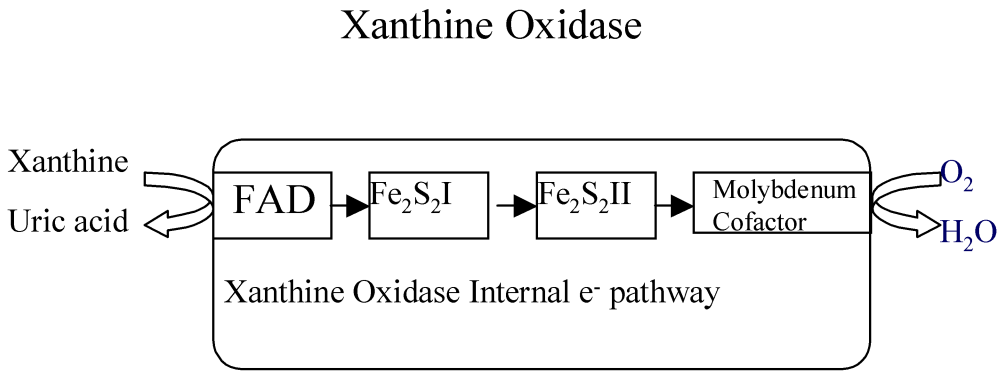

Figure 4. The arrangement of the various cofactors of xanthine oxidase [10].

The cofactor molybdopterin mentioned above are a class of cofactors mostly found in enymes that have the elements molybdenum (Mo) or tungsten (W) either strongly attached (prosthetic group) or not strongly attached (cofactors). The nomenclature for this biomolecule is quite confusing since Molybdopterin by itself (Figure 5) is the name of the ligand that will bind the active metal and contains no molybdenum. After molydopterin binds with molybdenum, the whole complex is called molybdenum cofactor. Synonyms for molydopterin include pyranopterin-dithiolate [15].

The synonym pyranopterin-dithiolate is used because the compound is consisted of a pyranopterin with two thiolates that serve as ligands in molybdo- and tungsto. In certain cases, an alkyl diphosphate nucleotide replaces the alkyl phosphate group. Currently molybdopterin is required as cofactor or prostecthic groups of enzymes such as xanthine oxidase, DMSO reductase, sulfite oxidase, ethylbenzene dehydrogenase, glyceraldehyde-3-phosphate ferredoxin oxidoreductase, nitrate reductase, carbon monoxide dehydrogenase, respiratory arsenate reductase, formate dehydrogenase, purine hydroxylase, thiosulfate reductase. The only molybdenum-containing enzymes that do not have molydopterins are the nitrogenises [15, 16].

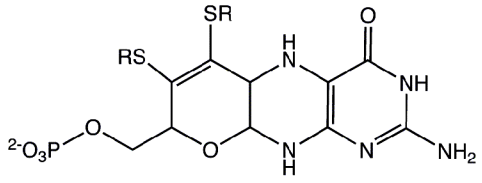

Figure 5. The structure of molybdopterin.

Molybdenum in Plant

Molybdenum is essential to plant growth as a component of the nitrogen fixation enzymes; nitrate reductase, nitrite reductase and nitrogenase, which are found in plant organs called legumes. The mechanism of nitrogen fixation in enzymes and in model systems *in vitro* has been extensively investigated [17]. The microorganisms that fix molecular nitrogen fall into two classes. The first is the symbiotic microorganisms that fix nitrogen in association with plants, e.g., *Rhizobium* and the second is the asymbiotic microorganisms that are free-living and include *Azotobacter vinelandii* and *Clostridium pasteurianum.* From cell-free extracts of *C. pasteurianum*, two metalloproteins have been obtained. One of the two is the hydrogen donating system, azoferredoxin, which contains iron and sulphide and the other is the nitrogen activating system, molybdoferredoxin, which contains molybdenum, iron, and sulphide [18].

In plants, the first stage in the reduction of nitrate is to nitrite. Biological conversion of nitrate to ammonium is an eight-electrons reduction process. There are two enzymes responsible; nitrate reductase and nitrite reductase. The first half of the reduction is catalysed by assimilatory nitrate reductase, a flavoprotein enzyme that has molybdenum as the only metal requirement. Molybdenum acts as an electron acceptor from reduced FAD in the enzyme. The molybdenum cofactor is an oxomolybdenum sulphur species with a pterin ligand [19]. The basic structures of FAD and the Moco pterin ligand in plants involved in nitrate reduction are similar to that of xanthine oxidase.

Molybdenum Toxicity

Molybdenum toxicity, like all compounds, is assessed according to an acute or chronic aspect. It is also important to assess the toxicity of molybdenum compounds on several different species since many chemicals are species specific in their action of toxicity. Also, the existence of many chemical forms of the molybdenum compounds must also be taken into consideration. Thus, toxicity studies of molybdenum compounds such as molybdenum trioxide, ammonium and calcium molybdates and molybdenum disulphide have been carried out with rats and guinea pigs by the U.S. Public Health Service [20].

Molybdenum trioxide, when fed in large daily doses of from 1200 to 6000 mg Mo/kg body weight has been proven to be fatal. Fatalities were few at doses of 120 to 600 mg. Molybdenum disulphide did not produce fatal results. The highest dose in rats and guinea pigs, when extrapolated, corresponds to 420 g for a 70 kg human being. In inhalation experiments (5 mg Mo/cubic feet of air), molybdenum trioxide and ammonium dimolybdate were

injurious but molybdenum disulphide was much less so. Most molybdates compounds also produced toxic effects when administered orally and by intraperitoneal injection in large doses (400 to 800 mg/kg) [21, 22].

Experiments on farm animals supplemented with sodium molybdate ranging from 20 to 1000 mg Mo/kg body weight shows that cows are the least tolerant with drastic scouring at 20-50 mg Mo/kg body weight followed by sheep and pigs. Horses are the most tolerant, with no significant health effect after molybdenum supplementation at 1000 mg Mo/kg body weight for three months. The effects in cows range from hypocupraemia, lameness, osteoporosis and spontaneous bone fractures. It was later found that an excess amount of molybdenum in the diet causes an antagonistic decrease of copper, another important enzyme cofactor and the addition of copper ions can result in a complete recovery from the signs of molybdenum intoxication [23, 24].

Recently, it was discovered that molybdenum shows its toxicity to the embryo of mouse [25] (Bi et al., 2013) and spermatogenesis in catfish and mice at levels as low as several parts per million [26-28].

Bi et al [25] studied the effects of molybdenum on zygotes for 5 days until the mid-blastocyst stage in the Kunming outbred mouse strain. The culture media is composed of potassium simplex optimized medium (KSOM) supplemented with from 0 to 160 mg/L of molybdenum. They discovered that at doses of 40 mg/L and higher, a significant decrease of the average cell number, cleavage, blastocyst and birth rates and significant increased of the proportion of degenerative blastocysts are observed. The process of blastocysts development to birth is arrested at 120 mg/L molybdenum while at 160 mg/L molybdenum a massive degeneration an overall developmental arrest (up to 16-cells) of embryos are observed.

Yamaguchi et al. [26] studied the effect of several metals including molybdenum on the testicular development of fish using in vitro testicular organ culture of Japanese eel (*Anguilla japonica*). They discovered that molybdenum at 10^{-4} M or approximately 9.5 mg/L inhibited the spermatogenesis induced by 11-ketotestosterone (11KT). They infer from this study that the anomaly observed in the testicular organ size of the Mekong Delta catfish male catfish (*Pangasianodon hypophthalmus*) could be linked to molybdenum pollution in the Mekong river.

Zhai et al. [27] studied the effect of sub-acute toxicity of molybdenum on spermatogenesis of ICR strain of adult mice. They discovered that at doses of about 100 mg/L, sperm parameters such as the sperm motility, sperm count, epididymis index and morphology were negatively affected with changes of sperm parameters were correlated with changes of the glutathione peroxidase (GPx) and superoxide dismutase (SOD) activities. In addition malondialdehyde (MDA) level was also affected in testes. They conclude that molybdenum affects sperm quality through regulating the oxidative stress in testis.

Zhang et al. [28] studied a sub-acute toxicity of molybdenum on ovarian function in ICR adult female mice. Mice were exposed to Mo by free access to distilled water containing molybdenum from 5 to 40 mg/L for 14 days. At Mo concentration of 40 mg/L M II oocyte morphology, ovary index and ovulation are strongly affected while ovarian with abnormal morphology are observed. Concomitant changes in levels of glutathione peroxidise (GPx), malondialdehyde (MDA) and superoxide dismutase (SOD) occurred in ovaries. They concluded that molybdenum affects oocyte quality through regulating ovarian oxidative stress in a dose-dependent manner.

Inhalation of dusts of molybdenum trioxide and the more soluble molybdate compounds produced toxic effects in humans [29]. After an 8-hour exposure of workers in a plant producing molybdenum trioxide to respirable dusts of molybdenum trioxide and other soluble oxides of molybdenum to 9.47 mg/m^3, mean serum uric acid levels of 25 male workers increased 1.18 fold and mean serum ceruloplasmin (copper transport protein) levels by 1.65 fold compared with unexposed workers but there was no gout-like syndrome. The maximum permissible concentrations in air recommended by the American Industrial Hygiene Association are 5 mg Mo/m^3 over an eight-hour period [30].

The criterion for the safe level of molybdenum in Malaysia (water supply for drinking, use in agriculture and aquatics) has yet to be worked out. Thus, the safe level from the WHO [20] with a limit of 0.05 mg/L. This safe level is similar to the safe level of several heavy metals included in the Environmental Quality Act, Malaysia such as copper, manganese, nickel, tin, zinc and boron [31]. The product of molybdenum reduction, Mo-blue is often used as pigments for clothing-dye and as pigments they suggest their relative non-toxic nature [1].

Molybdenum as a Pollutant

Molybdenum's diversity has proven precious in industries. Its uses include super alloys, nickel base alloys, lubricants, chemicals, glass workings, ink, pigments, electronics and many other applications. It is from these industries that molybdenum can be found in the discharged effluents [32, 33]. Molybdenum pollution in Malaysia has not been reported before and thus is not a major problem. In the Tokyo Bay and the Black Sea, molybdenum level is in the range of hundreds of ppm (parts per million) making it a significant pollutant [34]. In Tyrol, Austria, molybdenum pollution is caused by industrial effluents and has contaminated large pasture areas, reaching as high as 200 ppm causing scouring in ruminants [24]. In New Mexico, evidences have been documented on molybdenum pollution in a molybdenum mining company, Molycorp. Molybdenum and other toxic metals have been found by the New Mexico Environmental Department at levels exceeding the New Mexico Environmental Standard in the Red River. These elevated levels of molybdenum and metal by-products have polluted the river and caused the extinction of many of the river's floras and faunas [35]. The highest concentrations of molybdate ever reported in soil or water bodies as a pollutant is approximately 2000 ppm or 20.8 Mm [36].

Molybdenum including other heavy metals is a toxicant that needs to be removed from the environment. Like other heavy metals pollution, scientists have turned towards bioremediation, a cheaper alternative using the ability of microbe to remove and resist heavy metals via mechanisms such as sequestration, bioreduction, biosorption, transport mechanisms, bioprecipitation, and/or chelation [37-41].

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