The Effect of Mecury Contamination on Human Health and a Comparative Method of Extracting Hg(II) from Water Solution

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In this paper it has been done an experimental work regarding the optimal conditions of removing mercury from water using ion exchange resins and cellulose xanthate (CX/Na). It were prepared water solution of $HgCl_2$ with very well determined concentrations where it was added different amount of ion exchanger resin and CX/Na. The system was shaking 20, 40, 60 and 80 min. It has been found that the optimal conditions for removing of mercury from contaminated water solutions are 2.5 g/L CX/Na for 60 min shaking time for 100mg/L initial mercury concentration and 2.0 g/L CX/Na for 60 min shaking time for 50mg/L initial mercury concentration.

Keywords: mercury; ion exchange resin; cellulose xanthate, toxicity

Mercury is the only metal that is liquid at room temperature. It exists in three states: elemental/metallic Hg^0, mercurous Hg_2^{2+} and mercuric Hg^{2+}. It forms inorganic (e.g. mercuric chloride) and organic (e.g. methylmercury) compounds [1-3].

The clinical presentation of mercury poisoning is influenced by the chemical form, the amount involved, the route of exposure, and whether the exposure was a single acute episode or repeated. The central nervous system is particularly susceptible to mercury poisoning.

Elemental mercury vapor is the most important form toxicologically because it is absorbed rapidly following inhalation and can cross the blood brain barrier before oxidation to Hg^{2+}, which accumulates in the brain. Acute mercury vapour inhalation causes headache, cough, nausea, a metallic taste, dyspnea and chest pain. Chemical pneumonitis can ensue and, in severe cases, renal and/or liver failure may occur. Repeated exposure to low mercury vapor concentrations presents typically with characteristic neurological features including fine tremor, lethargy, memory loss, insomnia, personality changes and ataxia. Other features include stomatitis, gingivitis, hypersalivation and renal tubular damage. Mixed motor and sensory peripheral neuropathy can develop. Ingestion of metallic mercury is not usually a significant toxicological hazard because less than 1% is absorbed [4-7].

Many inorganic mercury salts are corrosive, and substantial ingestion has led to fatalities from hemorrhagic gastroenteritis. Renal tubular damage predominates in those who survive this initial phase. Neurological features of mercury poisoning can follow chronic exposure.

Organic mercury salts, methylmercury compounds, have been used as fungicides, and poisoning has usually followed ingestion Mercury (Hg) is a well-known contaminant that most people are exposed to in the organic form of methylmercury (MeHg). MeHg exposure can result in diverse negative health effects, including neurobehavioral [9-11], neurodevelopmental [12], immunological [13] and cardiovascular [14] outcomes. However, the mechanism(s) of action underlying MeHg toxicity are not well understood, and multiple processes are thought to play a role. Potential mechanisms of toxicity include the inhibition of protein synthesis and cell division, and interactions with cellular defenses. One mechanism of MeHg toxicity may occur through the glutathione antioxidant system. Glutathione is a major antioxidant in humans and many other organisms and plays a direct role in neurobehavioral, cardiovascular and immunological pathologies, many of which are also known effects of MeHg toxicity. Therefore, MeHg's association with glutathione, and the oxidative stress that may result, may play a direct role in these diverse manifestations of Hg toxicity [21]. Currently, our understanding of MeHg's associations with the glutathione system, specifically the relative balance of reduced glutathione (GSH) and oxidized glutathione (glutathione disulfide, GSSG), is limited.

The retina and gray, but not white, matter of the cerebral and visual cortex are major targets of methylmercury exposure [15]. Methylmercury produces immediate and persistent scotopic vision deficits, type II and III color vision deficits, visual field constriction, and reduced contrast sensitivity Methylmercury, in contrast to inorganic mercury, accumulates in the brain (i.e., cerebral and visual cortex, cerebellar Purkinje cells, and basal nuclei), outer and inner retinal layers, and retinal capillaries, where it is retained for over 10 years [16].

Experimental part

It was prepared water solution of $HgCl_2$ with very well determined concentrations: 50mg/L and 100mg/L [17-20]. To 25 mL from those solution were added different amount of ion exchange resin first and, in the same kind, solution of cellulose xanthate CX/Na second: 0.5g/L resin or CX/Na, 1.0g/L resin or CX/Na, 1.5g/L resin or CX/Na, 2.0g/L resin or CX/Na and 2.5/L g resin or CX/Na The system was shaken 20, 40, 60 and 80 min. The resin used was PUROLITE S920. Main characteristics of the resin are presented in table 1.

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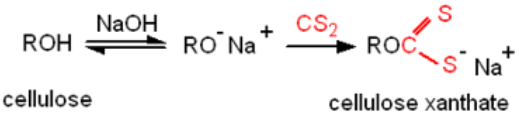
Tabel 1

MAIN CHARACTERISTICS OF THE RESIN PUROLITE S920

Structura polimerului	Stiren-divinilbenzen macroporos
Forma fizică a polimerului	Granule sferice
Grupe funcţionale	Tioronice
Forma ionică	H^+
Capacitatea totală	1.6 eq/L min
Umiditate	48 – 54 %
Mărimea granulelor	0.3 – 1.2 mm
Densitatea	700 – 730 g/L
Temperatura max de lucru	60 °C
Domeniul de pH	1 – 9

Generally the xanthation reaction is carried out according to the reaction:



Residual concentration of mercury was measured by atomic absorption spectrophotometry using Varian AA 110 spectrophotometer. The ion exchange resin was regenerated with HCl solutions. The method used for regeneration and the results are the objects of another study.

Results and discussions

The experimental results concerning the residual concentration of mercury depending on the amount of ion exchange resin and the amount of CX/Na, in solution with initial mercury concentration of 50mgHg/L and 100mgHg/L are presented in figures 1-4.

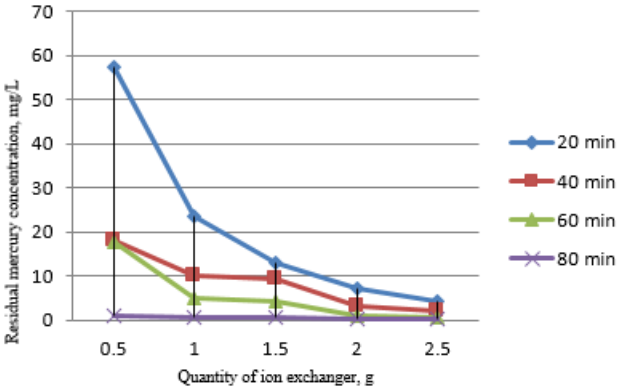


Fig. 1 Residual mercury concentration (mg/L) in solution as a function of quantity ion exchanger (g) (Initial concentration of solution was 100 mgHg/L)

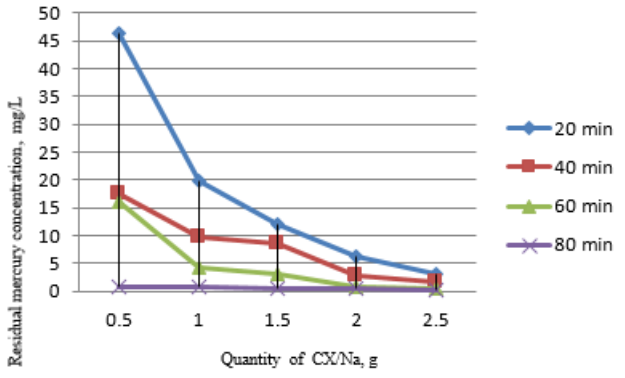


Fig. 2 Residual mercury concentration (mg/L) in solution as a function of quantity CX/Na (g) (Initial concentration of solution was 100 mgHg/L)

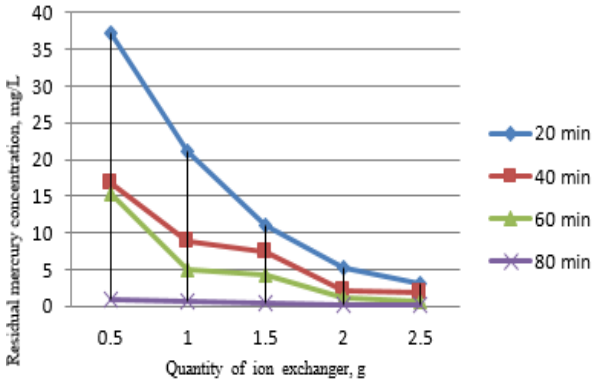


Fig. 3 Residual mercury concentration (mg/L) in solution as a function of quantity ion exchanger (g) (Initial concentration of solution was 50 mgHg/L

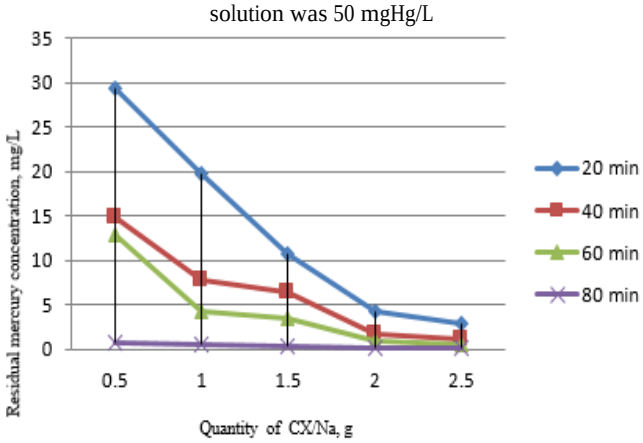


Fig. 4 Residual mercury concentration (mg/L) in solution as a function of quantity CX/Na (g) (Initial concentration of solution was 50 mgHg/L)

The experimental results show a general tendency of decreasing for residual concentration of mercury solution with the increasing of ion exchange resin amount or CX/Na amount and with the increasing of shaking time. Also it shows that with the use of 0.5 g of ion exchange resin the residual concentration of mercury decrease at 57.3 mg/L from 100 mg/L initially and at 37.3 mg/L from 50 mg/L initially at 20 min shaking time. With the use of 2.5 g of ion exchange resin the residual concentration of mercury decrease at 4.3 mg/L from 100 mg/L initially and at 3.2 mg/L from 50 mg/L initially at 20 min shaking time. When the CX/Na is used on the same condition the decrease of the amount of residual mercury is more efficient. From the analyses of the results shown in figure 1 and 2 the optimal condition from the extraction of mercury are with 2.5 g of CX/Na at 60 min shaking time. From the analyses of the results shown in figure 3 and 4 the optimal conditions from the extraction of mercury are with 2.0 g of CX/Na at 60 min shaking time.

Conclusions

It was prepared water solution of $HgCl_2$ with very well determined concentrations where it was added a different amount of ion exchange resin namely PUROLITE S920 and CX/Na. The system was shaking 20, 40, 60 and 80 min. It has been found the optimal parameters of removing mercury from water solutions with initial concentration of mercury very well known, using the ion exchange resin PUROLITE S920 and CX/Na. The ion exchange resin was regenerated by HCl solutions.

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