Journal of Physical Science, Vol. 16(2), 1–14, 2005

MODELING MERCURY UPTAKE IN THE EVERGLADES ALGAE

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Abstract: *The presence of mercury in the aquatic food chain has always been a concern due to its accumulative effects in the chain. Over the last 100 years the average global atmospheric mercury concentration has increased by five fold from 0.3 ng m^{-3} to 1.5 ng m^{-3}. The two main sources of atmospheric mercury emissions are natural sources such as volcanoes and soils, and anthropogenic sources that include combustion and waste incineration. The Florida Department of Health recorded high mercury burdens in the largemouth bass in the Everglades, with average concentration of 2.5 mg/kg mercury. The high level of mercury found in the largemouth bass exceeded all health-based standards in the USA. Mercury traces its pathways into the Everglades via runoffs from the watershed and atmospheric deposition. However, over 90% of the annual budget of mercury in the Everglades Protection Area is due to contribution from atmospheric deposition, and that amounted to 35.3 $\mu g/m^2$ per year according to a recent study. Furthermore, it is noted that local emissions contributed some 52% of the total. Once mercury is deposited into the Everglades wetlands, bacteria will convert the mercury from its inorganic form to the highly toxic and readily available bioorganic methylmercury. This paper presents an approach to modeling mercury concentration in the Everglades ecosystems. It will present an analysis on the bioaccumulation of methyl-mercury in the algae.*

Keywords: mercury contamination, Everglades, algae, mathematical model

1. INTRODUCTION

Methylmercury which is a highly toxic, lipophilic, organic form of mercury [1], has been observed to accumulate to dangerously high levels in many types of organisms in numerous aquatic food chains [2–8], beginning with the first trophic level organisms such as algae [9]. It is observed that methylmercury is first synthesized by sulfate-reducing bacteria (SRB) found in periphyton through an anaerobic process [10]. However, how do concentrations of parts per trillion of mercury in water yield concentrations of parts per million in the individual fishes [11]? The problem of high concentration of mercury in fishes is further compounded by the fact that mercury concentrates in high levels only in the muscle tissue of the fish. Hence, mercury cannot be filleted or cooked out of consumable game fish [12]. The most well-documented cases of severe methylmercury poisoning among humans were recorded in Minamata Bay, Japan

in 1956 and in Iraq in 1971. The occurrence of methylmercury in Minamata was through the industrial release of methylmercury into the bay [12,13], while the case in Iraq is due to the consumption of wheat treated with a methylmercury fungicide. In each of the aforementioned case, hundreds of people died and thousands more were afflicted, with many of them suffering permanent neurological disorders [12] due to mercury poisoning.

Over the last 100 years, the global atmospheric mercury concentration has increased from an average of 0.3 ng m^{-3} to an estimated 1.5 ng m^{-3} [14]. According to Lin and Pehkonen [15], approximately 6000 and 10800 tons of mercury are present in the troposphere and the various water bodies in the world respectively. It is further noted that an estimated 66% of mercury found in the environment comes from man-made sources [5]. In this paper we try to assess and quantify the bioaccumulation factor of methylmercury in the first level of the food chain, the algae. This is an important step in aiding us to understand the mechanics of transfer and bioaccumulation process of methylmercury in higher levels of the food chain in most aquatic systems.

2. MERCURY PROBLEM IN THE EVERGLADES

The Everglades is one of the biggest natural freshwater wonder of the world. It is located in the southern region of the Florida panhandle and is bounded in the north and west by Lake Okeechobee and the Big Cypress Swamp respectively. The Atlantic Ocean and the Florida Bay form the eastern and southern borders of the Everglades respectively [16]. Monitored by the Florida Fish and Wildlife Conservation (FWC), the Florida Department of Environmental Protection (FDEP) and the Florida Department of Health (FDOH) first noted the high levels of mercury concentration detected in fishes from Everglades in 1989. In the early 1990s, an extensive sampling exercise was then carried out in the Everglades. The sampling exercise subsequently revealed high mercury burdens in largemouth bass, with the average ratio of nearly 2.5 mg/kg of mercury [3]. This high concentration of mercury is known to exceed all health-based standards in the USA [17]. It is further observed that the mercury problem in the Everglades is due primarily to the conversion of inorganic mercury, Hg(II) to organic methylmercury (MeHg), which is more toxic and which has the potential to be biomagnified. As an example, Atkeson and Axelard [18] have recorded fish bioaccumulation factors ranging up to 10 million for MeHg.

3. THE PRODUCTION AND TRANSFER OF MEHG IN THE AQUATIC SYSTEM

The main sources of mercury in the Everglades are atmospheric deposition and runoffs from watersheds. Mercury loads from the wet deposition through rainfall are about 50 times more than mercury loads from surface water inflows. Dry mercury deposition adds another 30% to the total rainfall deposition [2,9]. The FDEP estimated the total deposition from June 1995 to June 1996 to be 35.3 $\mu g/m^2$ per year. The Florida Atmospheric Mercury Study (FAMS) recorded 23 $\mu g/m^2$ of mercury per year as wet deposition, while the remaining 12.2 $\mu g/m^2$ of mercury per year was modeled as dry deposition [2,3].

Upon entering the water, the mercury is subjected to a transformation process. During the transformation process, inorganic mercury in the form of Hg(II) is converted into organic mercury in the form of methylmercury (MeHg). This transformation process is known as methylation [8,19]. Methylation may occur in two different ways, either biological methylation or chemical methylation [20]. However, in this paper we will only discuss biological methylation, as it is the main methylation process reported in the Everglades. Furthermore, it should be noted that only small amounts of mercury found in the sediments undergo this process [2,21]. In addition, Krabbenhoft et al. [10] has contended that methylation only occurs in periphyton "mats", through SRB when oxygen is absent and sulfate is present [2]. SRB are a component of periphyton. The periphyton mats cover most submerged plants and form thick mats on the sediment surface in many locations in the Everglades [22]. Subsequently, the next consumer in the food chain may eat the periphyton mats which contains the bacteria with high methylmercury concentration. Moreover, the bacteria themselves in these mats may excrete the methylmercury into the water where it is taken up by the planktons [7]. Methylmercury can also be converted back by bacteria into inorganic Hg(II) [13,23]. This process is known as demethylation [19] but it is not well understood. USGS [7] has suggested that sunlight may also cause the breakdown of organic methylmercury into inorganic Hg(II) as shown in Figure 1.

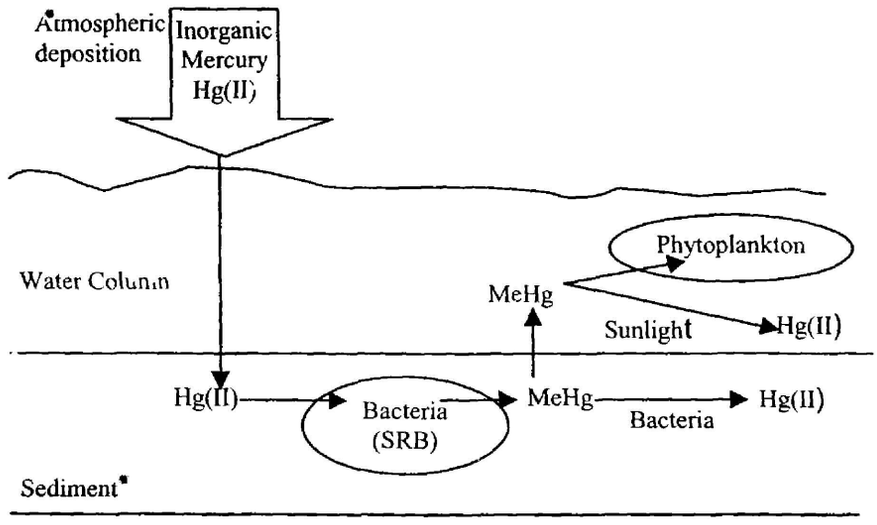

Figure 1: The cycle of methylmercury MeHg and Hg(II) in the Everglades

4. UPTAKE OF MERCURY BY ALGAE AND BACTERIA

Algae are made up of phytoplankton and other multicellular plants, which are either suspended in water or attached to rocks and other substrates such as periphyton [24,25]. Algae are an essential component of the lake ecosystem and are the basic food base for most lake organisms, including fish [25]. SRB form a distinctive and ubiquitous group of anaerobic prokaryotes. These bacteria are unified by a shared ability to carry out sulfate reduction as a principle component of their bioenergetics process [26]. SRB in the Everglades use organic carbon as a source of energy for growth and reproduction in the sediments or other anoxic microhabitats. Sulfate, which is required by SRB as a substrate for their metabolism is derived from the Everglades Agriculture Area (EAA). The transport of substances through the cell membranes of algae and bacteria is dependent on the properties of the cell and the transporting substances [27]. The cell membrane controls the movement of substances that enter or leave the cell. All cell membranes are made up mostly of lipids and proteins. Some substances can pass through the cell membranes easily without any input of energy from the cell. This type of movement is called passive transport [28]. Passive transport includes passive diffusion and facilitated diffusion [28]. Diffusion is a process by which matter is transported from one part of the system to another through random molecular motions [29] with a net direction towards lower concentration regions in order to achieve equilibrium. Passive diffusion occurs when small molecules pass through the lipid bi-layer of a cell membrane. Facilitated diffusion however, depends on carrier proteins imbedded in the membrane to allow specific substances to pass through the cell membrane [27].

5. UPTAKE MECHANISM

Methylmercury primarily accumulates in phytoplankton [8]. Hence, it is important to understand the uptake process of methylmercury by phytoplankton. However, the mechanism of mercury uptake by phytoplankton still remains uncertain. Hudson et al. [30] and Powell [31] have concluded that it is still undetermined whether passive diffusion or facilitated diffusion controls the process. Since methylmercury is a lipophilic, organic form of mercury, we may assume that the uptake of MeHg by phytoplankton is primarily controlled by passive diffusion [32]. Landing et al. [33] and Glimour et al. [34] have observed that only neutral inorganic Hg complexes, such as HgS may enter bacteria cells. In the absence of a specific uptake mechanism, organic compounds are probably transported into bacteria cells by passive diffusion through the cells' lipid membrane [35]. Since Hg(II) is the inorganic form of mercury, we may assume that the uptake of Hg(II) by SRB is controlled by facilitated diffusion. As was mentioned earlier, bacteria and algae in the first trophic level of the food chain initiate the accumulation of high levels of mercury in the food chain. Thus, the bioaccumulation of mercury in bacteria and algae is the key to understanding the bioaccumulation process of mercury in higher trophic levels (e.g. fish). Hence, in this paper we will attempt to model the bioaccumulation of mercury in algae (phytoplankton and periphyton). Since bacteria is a component of periphyton and since methylmercury is produced by bacteria, we will consider the bacteria as a component of periphyton in the modeling process. In order to further understand the bioaccumulation of mercury in algae, we will need to determine the uptake rate of mercury by phytoplankton and bacteria. Thus, we will divide our computer model into two components, the first component to compute the bioaccumulation of methylmercury by the phytoplankton, and the second component to determine the bioaccumulation of methylmercury by bacteria.

6. MODEL DESCRIPTION

According to Newman and Jagoe [36], contaminant bioaccumulation models begin with a single compartment model with first order uptake and elimination kinetics. The model can be described by the following differential equation for the change in contaminant concentration in the organism with time:

$$\frac{dC}{dt} = k_u C_s - k_e C \tag{1}$$

where

C = the contaminant concentration in the organism (mass.mass^{-1})
C_s = concentration in the source (mass.mass^{-1} or mass.volume^{-1})
k_u = the uptake rate (time^{-1}, volume.mass^{-1}.time^{-1})
k_e = the elimination rate (time^{-1})

In Thomann [37], the mass balance equation for the bioaccumulation of chemical concentration in Phytoplankton is written as:

$$\frac{dC_p}{dt} = k_u C_w - k_d C_p \tag{2}$$

where

C_p = the chemical concentration in the phytoplankton (μg/g(w))
C_w = the dissolved chemical concentration in water (μg/L)
k_u = the uptake sorption rate (L/d.g(w))
k_d = the desorption rate (d^{-1})

Since methylmercury crosses the cell membrane of phytoplankton by passive diffusion as we had mentioned earlier, while the cells of phytoplankton are periodically being subjected to cell division process [31], then Eq. (2) can be rewritten as:

$$\frac{dC_{mp}}{dt} = k_p C_{mw} - (\mu_p + k_d).C_{mp} \tag{3}$$

where

C_{mp} = the concentration of methylmercury in phytoplankton (μg kg-cell^{-1})
C_{mw} = the concentration of dissolved methylmercury in water (μg/L)
k_p = rate of passive uptake (L kg-cell^{-1} d^{-1})
μ_p = the instantaneous rate of cell division for phytoplankton (d^{-1})
k_d = depuration rate of MeHg (d^{-1})

At steady-state condition, Eq. (3) can be written as:

$$C_{mp} = \frac{k_p}{\mu_p + k_d}.C_{mw} \tag{4}$$

From Hudson et al. [30], we know that:

$$k_p = P_m . A_{cell} \tag{5}$$

where

P_m = the permeability coefficient for MeHg (dm/day)
A_{cell} = the specific surface area of the cells (dm^2 kg-$cell^{-1}$)

Substitute Eq. (5) into Eq. (4), and we derive:

$$C_{mp} = \frac{P_m \, A_{cell}}{\mu_p + k_d} . C_{mw} \tag{6}$$

In the case of SRB, we have to take into consideration that inorganic mercury, Hg(II) that enters the bacteria cell is different from the organic mercury, MeHg that leaves the bacteria cell. Furthermore, we need to note as well that only a small fraction of MeHg is reconverted back to Hg(II) by the bacteria. The source of this fraction of MeHg may come from either of the two cases.

SRB Model will consist of two differential equations, one equation for Hg(II) with the uptake of Hg(II) into bacteria cell occurring via facilitated diffusion and the second equation for MeHg produced inside bacteria cell.

Based on Eq. (1) we can write the following equations:

$$\frac{dC_{hb}}{dt} = k_f . C_{hw} - (\mu_b + k_{d1}) . C_{hb} \tag{7}$$

$$\frac{dC_{mb}}{dt} = k_m . C_{hb} - (\mu_b + k_{d2} + k_{dm}) . C_{mb} \tag{8}$$

where

C_{hb} = the concentration of Hg(II) in bacteria (μg/kg)
C_{hw} = the concentration of Hg(II) in water (μg/L)
k_f = rate of facilitated uptake (L kg^{-1} d^{-1})
k_{d1} = depuration rate of Hg(II) (d^{-1})
μ_b = the instantaneous rate of cell division for bacteria (d^{-1})
C_{mb} = the concentration of methylmercury in bacteria (μg/kg)
k_m = methylation rate (d^{-1})

k_{dm} = demethylation rate (d^{-1})
k_{d2} = depuration rate of MeHg (d^{-1}).

At steady-state condition, we can simplify Eq. (7) and Eq. (8) to:

$$C_{mb} = \frac{k_m.k_f}{(\mu_b + k_{d1}).(\mu_b + k_{d2} + k_{dm})}.C_{hw} \quad (9)$$

From Powell [31], we know that:

$$k_f = \frac{k_x}{1 + \beta_{HX}.[H^+]} \quad (10)$$

where

k = mass specific mercury uptake rate (L $kg^{-1}.d^{-1}$)
β_{HX} = stability constant for the first order protonation reaction with membrane transport ligand.

Hence we derive:

$$C_{mb} = k_m.\frac{k_x}{1 + \beta_{HX}.[H^+]}.\frac{1}{(\mu_b + k_{d1})(\mu_b + k_{d2} + k_{dm})}.C_{hw} \quad (11)$$

Table 1: Parameter values used in phytoplankton model

Parameter description	Unit	Value	References
Permeability coefficients for MeHg	cm/s	0.00032 0.0052	Petri [38] Hudson et al. [30]
Phytoplankton cell radius	μm	10	Gorm [39]
Phytoplankton cell density	kg dry wt L-$cell^{-1}$	0.2	Hudson et al. [30]
Phytoplankton cell division rate	day^{-1}	1	Hudson et al. [30] Powell [31]
Depuration rate	day^{-1}	0.01	Hudson et al. [30]

7. APPLICATION

The bioconcentration factor (BCF) of a chemical is the ratio of its concentrations in the organism to that in water during steady state or equilibrium [40]. Partition coefficient is the ratio of a chemical's concentration in one phase to its concentration in the other phase [41]. Our application will focus only on phytoplankton model, because there is insufficient data regarding some of the parameters of SRB model (k_{d2}, μ_b). Here, we consider the spherical shape of phytoplankton cell.

From Eq. (6), we have:

$$C_{mp} = \frac{P_m A_{cell}}{\mu_p + k_d} . C_{mw} \quad \Rightarrow \quad \frac{C_{mp}}{C_{mw}} = \frac{P_m A_{cell}}{\mu_p + k_d}$$

we know that:

$$A_{cell} = \frac{surface\ area}{mass} = \frac{surface\ area}{density.volume} = \frac{4\pi.R^2}{density.\frac{4}{3}\pi.R^3} = \frac{3}{density.R}$$

Then we obtain:

$$\frac{C_{mp}}{C_{mw}} = \frac{3.P_m}{density.R.(\mu_p + k_d)} \tag{12}$$

Using the parameters listed in Table 1 in phytoplankton model [Eq. (12)], we have two cases, case (i) for $P_m = 0.00032$ cm/s and case (ii) for $P_m = 0.0052$ cm/s. We had discovered two values for methylmercury BCF for phytoplankton $10^{5.6}$ and $10^{6.8}$ L/kg (dw) for case (i) and case (ii) respectively, for one specific species of green algae (Cosmarium botrytis). The first value $10^{5.6}$ differ slightly from methylmercury BCF $10^{5.5}$ for phytoplankton which was mentioned in USEPA [42], while the second value $10^{6.8}$ is between two values of phytoplankton-water partition constants for MeHg $10^{6.6}$ and $10^{7.3}$ for Cosmarium botrytis, which obtained by Moye et al. [32] by using three methods in the laboratory. Figure 2 [solid curve for case (i) and dotted curve for case (ii)] shows that BCF increases as cell radius decreases. This means that small phytoplankton cells accumulate more MeHg than larger cells. This conform to what was mentioned in Moye et al. [32]. Table 2 shows the predicted phytoplankton MeHg concentrations for three sites in the Everglades.

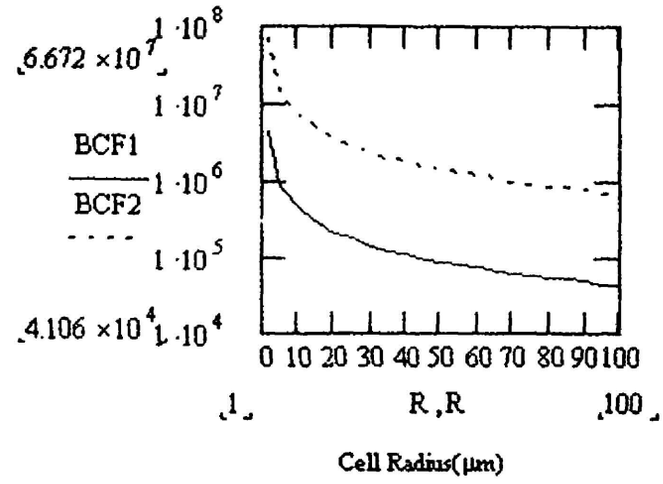

Figure 2: Size-dependence of MeHg bioconcentration factor for phytoplankton calculated for passive diffusion

Table 2: Predicted phytoplankton MeHg concentration for case (i)

Site	Observed Mean (C_{mw})*	Predicted Mean (C_{mp}), (n = 3)	Observed (C_{mp})
WCA-2A-F_1	0.19 ng/L (ppt)	0.04 mg/kg (dw) (ppm)	NA
WCA-2A-U_3	0.54 ng/L (ppt)	0.12 mg/kg (dw) (ppm)	NA
WCA-34-15	0.40 ng/L (ppt)	0.09 mg/kg (dw) (ppm)	0.05–0.1 mg/kg (dw) – Estimated

*Observed mean methylmercury concentrations in surface waters (dissolved) for the period of January 1, 1995 through December 31, 1999 [41].

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