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Blood Aluminum Levels as a Function of Aluminum Intake from Drinking Water

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Exposure to aluminum occurs mostly via food and water. The average aluminum intake is considered to be about 20-40 mg per day from food and beverages (Greger 1987). Although there is exposure to airborne aluminum, inhalation of aluminum does not contribute significantly to total daily intake (Sorenson et al. 1974).

In general, the absorption of aluminum from the gastrointestinal tract is poor (Greger 1987). However, the human pharmacokinetics of each aluminum species may be different. It is therefore important to characterize the aluminum species found in treated drinking water. If the form or forms of aluminum found in treated drinking water are readily bioavailable, then water may be an important source of the aluminum body burden. Although the aluminum intake from drinking water is small compared to that of food, the absorption of the species found in drinking water may be much higher than that of food species.

The speciation of aluminum in water is complex and is not well understood (LaZerte 1984; Campbell et al. 1983; Driscoll 1984). Alum (aluminum sulphate) is added to drinking water for the purpose of clarification. Theoretically, the addition of alum to water for treatment purposes should not increase the total aluminum concentration of water. However, for many water supplies, the total aluminum is increased after treatment (Schenk et al. 1989).

The presence of aluminum in surface drinking water sources occurs naturally. The levels of aluminum found in groundwater are low and are negligible when compared to surface water concentrations (Sorenson et al. 1974). The levels found naturally in raw surface water range from about 10 to 2000 μg/L (Sorenson et al. 1974). Levels in areas where surface waters have become acidified are in excess of 40,000 μg/L (Hultberg and Wenblad 1980). The levels found in treated water range from 0 to 1029 μg Al/L (Schenk et al. 1989).

Concern over the levels of aluminum found in surface drinking water sources and treated drinking water has arisen for two reasons.

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First, acid rain has caused the aluminum level in many fresh water sources to increase (Driscoll and Schecher 1989). Second, the possibility of an association between aluminum and human disease is frequently hypothesized. However, it is very important to note that to date ecological epidemiological studies have not provided convincing evidence of a cause-effect relationship between waterborne aluminum and neurodegenerative processes (Martyn et al. 1989; Flaten 1987; McLachlan 1989).

Questions regarding the health effects of aluminum are still unanswered. The speciation, pharmacokinetics, and toxicity of aluminum are not well understood. Furthermore, no animal or human studies of aluminum absorption have been reported using drinking water as the source of aluminum. The following experiment attempted to reach a better understanding of the bioavailability of aluminum from drinking water. Its objective was to determine whether or not increased aluminum ingestion from drinking water would be reflected in increased serum and whole blood aluminum levels in the baboon experimental model.

MATERIALS AND METHODS

The water used in the experiment was Chicago treated drinking water collected at one time and stored in six acid washed twenty liter polypropylene containers. Part of the stock was volumetrically reduced to increase the concentration of aluminum in the water. The speciation of aluminum in treated drinking water is not clearly defined at this time. Therefore, addition of a particular species to increase the aluminum concentration of the sample would likely change the true aluminum chemistry of the drinking water. "Concentrated" water refers to the water having the higher concentration of aluminum; "unconcentrated" water refers to Chicago treated drinking water. The aluminum concentration of the water before and after concentration was determined using flameless atomic absorption and a reference solution of aluminum chloride.

The water was condensed using a glass three liter round bottom flask connected to a rotary evaporator. The round bottom flask was placed in a hot water bath to help increase the evaporation rate of the water. The temperature of the water bath was maintained at 50° C in order not to change the species of aluminum found in the water. The concentrated water was collected and stored in an acid washed twenty liter polypropylene container at 5°C.

One adult female (21 kg and 12 years old) olive baboon (Papio anubis) was given food and water ad libitum. The experimental design consisted of the administration of food and unconcentrated water during exposure period 1 (days 1 - 10) and administration of food and concentrated water during exposure period 2 (days 11 - 31). The animal's intake of food and water was measured daily. Food used during the entire experiment came from one production batch with an aluminum concentration of 13.3 mg/kg food. All of the water that the animal received was administered in an acid washed polypropylene one liter container. The nozzle for drinking was

stainless steel and attached by a rubber stopper. Both the container and nozzle were checked for aluminum absorption or release.

Plastic containers, syringes, and labware were used because they neither absorb nor release aluminum. The use of the glass round bottom evaporation flask was necessary due to the unavailability of a plastic substitute. As a precautionary measure, all plasticware and the glass round bottom flask were acid washed for 24 hours using 6 mol/L hydrochloric acid to remove any contamination (Burnatowska-Hledin and Mayor 1985).

Blood samples were drawn using plastic syringes with stainless steel needles. Before each sample was drawn the animal was sedated using an injection of 10 mg Ketamine/kg body weight. A 10 mL sample was taken for each sampling day and divided between a 7 mL plain Vacutainer tube and a 2.5 mL "purple cap" Vacutainer tube containing 7.5% potassium EDTA. The EDTA in the "purple cap" Vacutainer tubes acted as an anticoagulant. The sample in the plain Vacutainer tube was allowed to clot for 20 minutes and transferred to an acid washed 10 mL polyallomar centrifuge tube. Each sample was then low-speed centrifuged and the serum removed. Each serum sample was stored in a 7 mL acid washed polypropylene bottle and stored at 5°C until analyzed by flameless atomic absorption. Each whole blood sample was also transferred to a 7 mL acid washed polypropylene bottle and stored at 5°C until analyzed.

Both types of Vacutainer tubes were tested for contamination by aluminum even though the plain Vacutainer tubes are not considered a significant source of aluminum contamination (Burnatowska-Hledin and Mayor 1985). No significant aluminum contamination of either type of Vacutainer tube was found.

Concentrations of aluminum in the serum, whole blood, and water samples were determined using a Perkin-Elmer Model 5000 Atomic Absorption Spectrophotometer, equipped with a Model 500 graphite furnace (HGA). Pyrolytically coated graphite tubes with L'Vov platforms were used. An aluminum hollow cathode lamp (P-E) with correction was operated at 25 ma, 309.3 nm with a spectral band width of 0.7 nm (Burnatowska-Hledin and Mayor 1985; Perkin-Elmer 1985; Leung and Henderson 1982; van der Voet et al. 1985).

The serum and whole blood aluminum levels for each sample were determined using the method of standard additions (Perkin-Elmer 1985; D'Haese et al. 1985). This method was used to compensate for the inability to prepare perfect standard solutions which would account for the interferences caused by blood components.

RESULTS and DISCUSSION

The unconcentrated and concentrated water had aluminum concentrations of 174 μg Al/L and 631 μg Al/L, respectively. Repeat analyses showed that these Al concentrations were stable over the

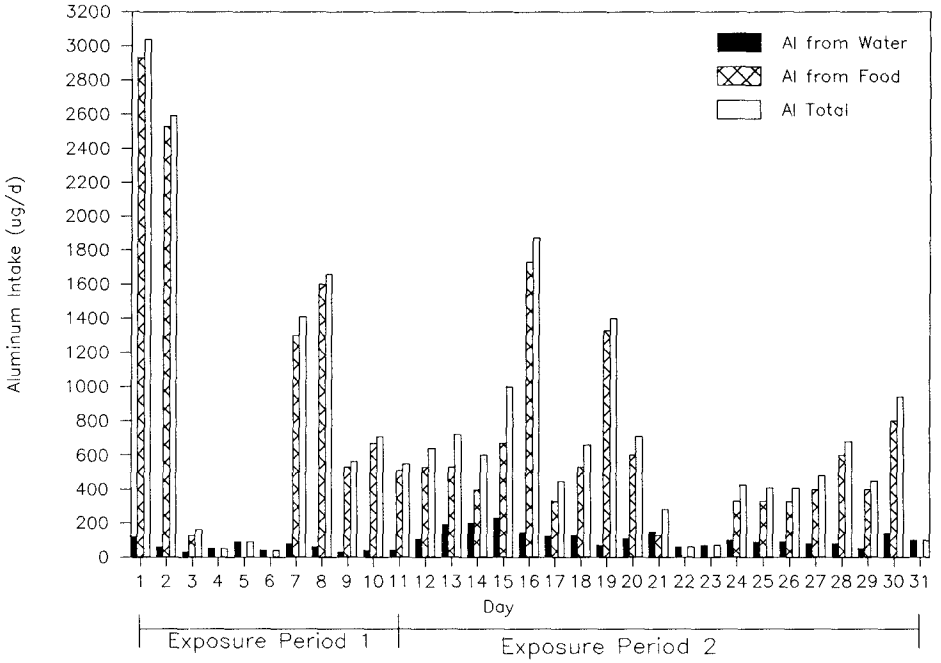

Figure 1. Aluminum intake (μg/d) from food and water.

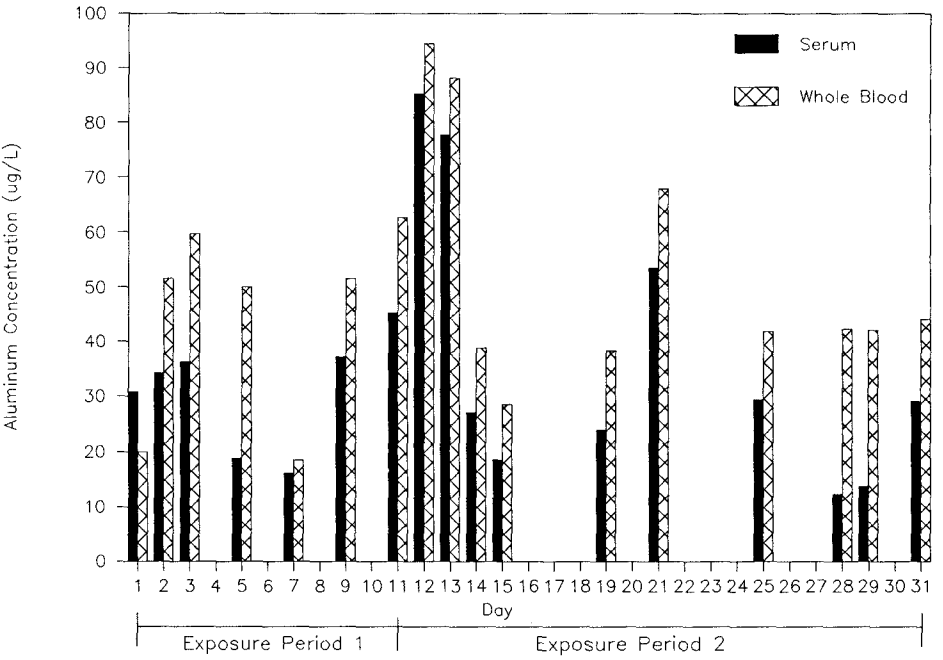

Figure 2. Aluminum concentration (μg/L) in serum and whole blood samples.

Table 1. Al intake and blood Al levels for exposure periods 1 and 2

	Exposure period 1		Exposure period 2		P-value
	Mean	SD	Mean	SD	
Food intake (g/d)	73	82	38	31	.05<P<.1
Al intake, food (μg/d)	972	1086	499	411	.05<P<.1
Water intake (mL/d)	301	127	178	81	P < .01
Al intake, water (μg/d)	60	29	112	50	P < .01
Total Al intake (μg/d)	1032	1104	611	424	P > 0.1
Serum Al (μg/L)	29	9	38	25	P > 0.2
Whole blood Al (μg/L)	42	18	53	23	P > 0.1

Table 2. Correlations between blood Al levels and Al intake from food and water during the 24 hour period preceding the collection of blood samples

	Dependent variable	
	Serum	Whole blood
Independent variable		
Water	0.20	-0.04
Food	-0.16	-0.44
Total intake	-0.15	-0.44
Multiple regression[a]		
Water, food	0.28	-0.45
Water, total	0.28	-0.45

[a] The high correlation (r = 0.998) between food and total intake prevented a three variable analysis.

course of the experiment. Hence, volume reduction increased the concentration of aluminum 3.6 times.

There were two periods (days 4 - 6 and days 22 - 23) during which there was no food intake. The baboon lost about 3 kg over the course of the experiment. Aluminum intake from food and water are shown in Figure 1. Regarding exposure period 1 vs. 2, it can be seen from Table 1 that: (a) the decrease (49%) in Al intake from food was not statistically significant at $p < 0.05$, (b) the increase (87%) in Al intake from water was statistically significant at $p < 0.05$, and (c) the decrease (41%) in total Al intake from food and water was not statistically significant at $p < 0.05$.

The experiment yielded 17 serum and whole blood samples (see Figure 2). Duplicate analyses of serum and whole blood samples were within ± 5% of each other. Regarding exposure period 1 vs. 2, it can be seen from Table 1 that the increases in serum and whole blood levels (31% and 26%, respectively) were not statistically significant at $p < 0.05$. Also, it can be seen from Table 2 that there was no association between serum or whole blood Al levels and Al intake from water and food in the 24 hour period preceding each blood collection.

Overall, data analyses showed that there was no association between Al intake via food or water and Al levels in serum and whole blood. One might be tempted to speculate from Figure 2 that increased Al intake from water resulted in a temporary increase in blood Al levels on days 12 and 13. However, it is equally likely that the increase represents only a random fluctuation in blood Al level. The interpretation of the increase would have been facilitated had it been possible to take blood samples each day. The experiment was originally designed to collect blood samples each day. However, blood samples had to be taken on an irregular schedule in order to prevent loss of appetite.

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