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ORIGINAL RESEARCH

Methyl Mercury in Dental Amalgams in the Human Mouth

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We report the presence of methyl mercury from the analysis of three samples of restorations associated with dental amalgam. We believe this to be the first finding of methyl mercury in the human mouth. Although the amounts found are small (4.0, 5.3 and 37.3 ng per sample), any measurable amount of methyl mercury contributes to the total body's burden of mercury. Despite the long history of apparent safe use of dental amalgams, this finding obviously warrants further investigation and confirmation. A possible mechanism to explain the formation and distribution of methyl mercury from dental amalgams is proposed.

Keywords: methyl mercury, dental amalgams.

INTRODUCTION

Inorganic mercury compounds have been well documented as present in, and emitted from, dental amalgams. To our knowledge, methyl mercury has not been documented in this context. The history of methyl mercury as a highly toxic material is extensive over the past 160 years [1–3]. The reported effects range all the way from mild subclinical behavioral disturbances through birth defects to death [4–6]. Recent advances in the speciation methods of mercury analysis, allowing high accuracy in the picogram level, have made this work possible.

MATERIALS AND METHODS

Experimental Determination of Mercury Species in Dental Mercury Amalgam

The speciations method of analysis used was by gas chromatographic/cold vapor atomic fluorescence spectrometry (GC/CVAFS) as described by Lian Liang *et al.* [7–10]. The detection limits were about 0.01 ng/sample for methyl mercury and 0.04 ng/sample for inorganic mercury.

Two porcelain veneer specimens ((a three-unit porcelain veneer crown bridge) (PVCB) and a single porcelain veneer crown (PVC)) were removed from one patient in Burleson County, Texas by R. Sellars, Jr DDS. Because of pain and periodontal disease, both specimens were removed. Both emitted a foul odor when removed. Each had been in contact with the underlying amalgam restoration build-ups for more than 8 years. The PVCB included an attached coping of gold alloy. The PVC did not have any metal attached.

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TABLE 1. Forms of mercury in porcelain veneer crowns and amalgam

Sample (aliquot) description	Methyl mercury	Inorganic mercury
PVCB DDW 30-h extract	< 0.001 ng/sample	0.2 ± 0.05 ng/sample
PVCB 2-h extract I	5.2 ± 0.3 ng/sample	3400 ± 300 ng/sample
PVCB 20-h extract II	5.3 ± 0.4 ng/sample	3502 ± 290 ng/sample
PVC 2-h alkaline extract	37.3 ± 0.15 ng/sample	7886 ± 367 ng/sample
Amalgam 2-h extract	4.0 ± 0.2 ng/sample	3200 ± 290 ng/sample
Amalgam 2-h control	< 0.001 ng/sample	469 ± 24 ng/sample

There was no visible amalgam attached to either of these two initial specimens. The specimens were express mailed in ultra clean Teflon vials to Lian Liang PhD, Brooks Rand Ltd, Seattle, Washington for analysis for methyl mercury and inorganic mercury.

The whole sample of the PVCB was rinsed with double deionized water (DDW) to remove any blood from the surface. It was then shaken in a Teflon vial with DDW for 30 h and analyzed (see PVCB DDW 30-h extract, Table 1). Then the PVCB specimen was placed in another vial containing 2 ml of 25% KOH/CH_3OH alkaline solution extract and shaken in the vial for 2 h (PVCB 2-h extract I, Table 1). The solution was divided into two aliquots, one of which was shaken for an additional 18 h (PVCB 20-h extract II, Table 1).

Methyl mercury and inorganic mercury were measured by aqueous phase ethylation, room temperature pre-collection, GC separation and CVAFS detection. The PVC specimen was extracted for 2 h in the same manner. After Dr Lian Liang found methyl mercury in these two initial specimens in April 1993, a third specimen was submitted by Dr R. Sellars, consisting of a second molar with a large amalgam restoration. The amalgam filling was separated from the tooth and analyzed by alkaline extraction in May 1993, (see Amalgam 2-h extract, Table 1). As a control, a small specimen (1.258 g) of amalgam (see Amalgam blank control, Table 1) was freshly titurated, and analyzed in the same manner for methyl mercury and inorganic mercury for 2 h.

RESULTS

A certified reference material obtained from the National Research Council of Canada (Dorm-I) which consists of fish with a high level of methyl mercury was analyzed (729 ± 2.4 ng g^{-1}). The known standard reference value (732 ± 60 ng g^{-1}) compared to the results obtained indicates that the results of analysis for methyl mercury are reliable.

Very low quantities (< 0.001 ng/sample for methyl mercury and 0.2 ng/sample for inorganic mercury) were found in the PVC DDW 30-h extract, indicating that both methyl mercury and inorganic mercury are tightly bound to the PVC metal, and cannot be separated by aqueous extraction.

Methyl mercury and inorganic mercury were demonstrated in both the 2-h and 20-h PVCB extracts in similar amounts, indicating that both methyl mercury and inorganic mercury are present when infection is present, and that 2 h is sufficient time for alkaline extraction of both. The PVC was extracted for 2 h, and again both methyl mercury and inorganic mercury were demonstrated. Methyl mercury was found again on the amalgam 2-h extract. This finding indicates that methyl mercury is being generated in the mouth in association with amalgam fillings and infection, and some of it is tightly bound to the amalgam filling itself.

No methyl mercury was found in the amalgam blank control (Table 1) although inorganic mercury was demonstrated. This control specimen had never been used as a filling, indicating that methyl mercury was not detected on freshly titurated amalgam.

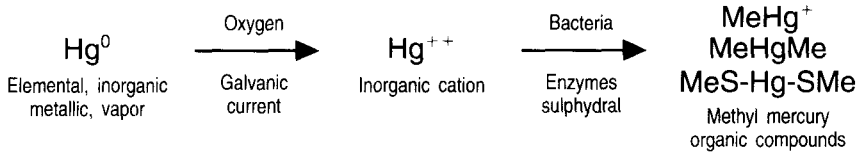
DISCUSSION

"Methyl mercury readily passes through physiological barriers such as the blood–brain barrier, blood–testes barrier and placenta, in contrast to the inorganic forms. Thus, methyl mercury is much more likely to target the nervous system, testes and developing embryo/fetus. The principal target organ for methyl mercury is the brain. It causes less dramatic lesions in the liver and kidneys" [2]. Possible mechanisms for the formation of methyl mercury include salty foods and acid foods such as citrus. The soaking by solutions of sodium chloride and/or weak acids provides a mechanism for corrosion of amalgam, releasing metallic mercury in the oral cavity [11–15]. Moreover, the influence of microbes in the mouth may convert metallic mercury to organic mercury. Dental infectious foci seem to be a factor that enhances the degree of mercury toxicity [16–19]. Galvanic current owing to the presence of dental amalgams could be the driving force for the oxidation of metallic mercury according to the classical Nernst equation [20].

Our findings indicate that a small amount of methyl mercury is bound tightly to the nearby metal (bridge alloy or amalgam) surfaces and porcelain veneer. How much additional methyl mercury is being absorbed directly through the mucous membranes of the mouth or swallowed and absorbed through the gastrointestinal tract to be subsequently taken into somatic cells is unknown. The purpose of this paper is to report the finding. Further investigation is needed to determine if these small amounts of methyl mercury are significant. So far, nobody knows the relationship.

A possible mechanism for the generation of methyl mercury species is as follows.

Mercury has three stable oxidation states: metallic mercury is the zero oxidation state. The mercurous ion is the $+1$ oxidation state and the mercuric ion is the $+2$ oxidation state. In addition to these inorganic states, mercury can exist as organometallic compounds. Of interest in this paper are the monomethyl mercury ion with a $+1$ charge and dimethyl mercury. Because of the many species (forms) of mercury, speciation is necessary. Elemental mercury is released from the dental amalgam, then oxidized by oxygen and/or galvanic currents in the mouth, then the ionic mercury is methylated by bacterial action, enzymes and/or sulphydral groups.



CONCLUSIONS

Under the conditions of infections associated with dental amalgams, methyl mercury is generated in the mouth and very small amounts of it are tightly bound to the surface structure of metal in the PVCB and amalgam, and to the PVC itself. How much additional methyl mercury is being absorbed directly through the mucous membranes and accumulating systemically is unknown, but it appears that at least some of it must contribute to the total body burden of mercury. Further study and confirmation of these observations are clearly indicated.

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