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The effects of amalgam restorations on plasma mercury levels and total antioxidant activity

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ABSTRACT

Objective: This study evaluated the effects of amalgam restorations on plasma mercury levels and total antioxidant activities (TAA).

Design: The study was comprised of 48 subjects ranging in age from 20 to 32 years. Of these, 33 had dental amalgam restorations and 15 had no dental amalgam restorations. In those patients with amalgams, the total number of amalgam restorations and surfaces were counted, and the total and occlusal areas (mm²) of restorations were measured using a Counting Measurement Machine. Blood samples were collected from all participants. Plasma mercury levels were measured using an Atomic Absorption Spectrometer and Hydride System, and plasma TAA levels were measured using an Antioxidant Assay Kit. Statistical analysis was performed using the SPSS 10.01 software program. Data was evaluated by t test and correlation analysis.

Results: Plasma mercury (P-Hg) levels were found to be significantly higher in subjects with amalgam restorations when compared to subjects without amalgams ($p < 0.01$); the differences in P-TAA levels between subjects with and without amalgams were not found to be statistically significant ($p > 0.05$). No significant correlations were found between P-Hg concentrations and P-TAA levels ($p > 0.05$). Significant positive correlations were found between P-Hg concentrations and the number of amalgam restorations ($p < 0.01$), number of amalgam surfaces ($p < 0.05$), total amalgam surface area ($p < 0.05$) and amalgam occlusal surface area ($p < 0.01$). However, no significant correlations were found between these parameters and P-TAA ($p > 0.05$).

Conclusions: The results of our study showed that dental amalgams are a major source of plasma mercury; however, amalgam restorations were not found to have a significant effect on plasma-total antioxidant activities.

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1. Introduction

Dental amalgam has been used as a restorative material in dentistry for more than 150 years. Amalgam is an alloy containing approximately 50% mercury and another metal or metals, including silver, copper, or tin.¹

Both ionic and elemental mercury have been shown to be released from amalgams into the body.² Whereas ionic mercury is released as a result of corrosion and wear,³ elemental mercury is released by direct vaporisation that can occur during preparation and condensation of the amalgam material and throughout the functional life of the restoration.^{4,5} When ionic

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mercury is swallowed, only a small amount is absorbed by the gastrointestinal system; however, when elemental mercury is inhaled, it is absorbed by the lungs,⁶ and about 80% of the amount absorbed is released into the bloodstream and transported to various tissues.⁷ In view of the known toxicity of mercury and the possibility that the mercury in dental amalgams could affect body mercury levels, arguments have arisen regarding the safety of dental amalgams, and several studies have been conducted on this subject.^{8–21}

Some studies have shown mercury to bind with protein sulphhydryl groups, leading to the release of reactive oxygen species (ROS) such as superoxide, hydrogen peroxide and hydroxyl radicals and to the formation of free radicals.²² ROS may induce peroxidative injury in membrane lipids and proteins, as well as DNA fragmentation, which can result in the disruption of nerve cell function and integrity.²³

The body's antioxidant defense systems are the most important protective mechanisms against ROS and their harmful effects. These systems include enzymes such as superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx); macromolecules such as albumin, ceruloplasmin and ferritin; and small molecules such as α -tocopherol, β -carotene, uric acid and bilirubin. The totality of endogenous antioxidants and dietary antioxidants represents the total antioxidant activity (TAA) of a system.²⁴

In recent years, studies have examined the effects of mercury on TAA and on a number of specific antioxidant enzymes such as SOD, GPx and CAT in various tissues^{25–28} and fluids such as plasma^{20,21} and saliva.^{8,9} Elia et al.²⁵ found decreases in GSH-S-transferase (GST) and GPx and increases in GSH levels in the lungs of *Ictalurus melas* 96 h after they were injected with varying doses of mercury (100, 200 and 400 $\mu\text{g/l}$ Hg^{2+}). Lund et al.²⁶ stated that mercury in the form of Hg(II) (1.5 or 2.25 mg HgCl_2/kg) caused an increase in H_2O_2 , a decrease in glutathione, and lipid peroxidation in the kidney mitochondria of rats. Subcutaneous injection of HgCl_2 in rats has also been shown to cause renal toxicity and acute renal failure.²⁷ Moreover, it has been suggested that mercury can lead to the depletion of glutathione in the renal tubes and a decrease in SOD, CAT and GPx enzyme activity.²² In a study by Rao and Gangadharan,²⁸ after incubating rat epididymal sperm with mercury, SOD, GPx and glutathione reductase activity decreased significantly and H_2O_2 generation increased in a dose-dependent manner.

Only a few studies have looked at the relationship between mercury released from dental amalgams and antioxidant systems.^{8,9,11,12,20,29} Although some of these studies have suggested that there is a relationship between mercury and/or amalgam and antioxidant,^{11,12} others have found no such relationship.²⁰ This study aimed to evaluate the effects of amalgam restorations on plasma mercury and plasma TAA levels.

2. Materials and methods

2.1. Participant selection

Subjects were selected from among patients referred to the Atatürk University Faculty of Dentistry in the city of Erzurum in

northeastern Turkey. All of the subjects gave their written informed consent to participate in the study. Out of a total of 48 participants (24 males, 24 females; age range: 20–32 years), 33 had dental amalgam restorations and 15 had no amalgam restorations. All of the subjects were born in Erzurum and had spent most of their lives there. In order to control for local and systemic factors that might affect body mercury concentrations and antioxidant levels, individuals meeting any of the following criteria were excluded from the study: systemic disorders^{30–33}; medication usage during the previous 3 months³⁴; smoking/alcohol habits (any past or present consumption of tobacco or alcohol)^{35,36}; occupational exposure to mercury^{35,37–40}; placement of new amalgam restorations during the previous year³⁷; consumption of fish/seafood during the previous month³⁷; parafunctional habits (e.g., bruxism)^{10,19}; frequent gum chewing (every day, several times a day)^{10,19,41,42}; periodontal problems^{40,43,44}; and active caries lesions.^{45,46}

2.2. Measurement of amalgam restoration surface area

In subjects with amalgams, the total number of amalgam restorations and amalgam surfaces were counted, impressions were taken and study models were cast from dental stone. Amalgam restoration margins were traced on models, and the total and occlusal areas of the restorations were measured using a Counting Measurement Machine (Renishaw Cyclone Series 2/Renishaw SP 620) in square millimeters (mm^2).

2.3. Blood samples

Blood samples were collected from all participants using heparin as an anticoagulant. Samples were centrifuged at $700\text{--}1000 \times g$ for 10 min at 4°C , the white buffy coat was removed, and the yellow plasma layer was stored at -80°C until analysis.

2.4. Mercury analysis

Plasma samples were prepared for mercury analysis using a microwave digestion system (Milestone ETHOS 1600). Plasma samples of 2 ml each were placed in teflon pans with 6 ml of 65% HNO_3 and 1 ml 30% H_2O_2 , sealed and placed in the microwave and subjected to a 4-stage digestion program. The pressurised containers were removed, and the contents were poured into a 10 ml glass beaker. The emptied containers were rinsed with bidistilled water, and this liquid was added to the glass beaker to produce a total of 10 ml of working solution for the measurement of mercury levels.

Cold vapor atomic absorption spectrometric (CVAAS) analysis is the most common method used to measure mercury levels in biological samples.⁴⁷ In the present study, mercury analysis was performed using an Atomic Absorption Spectrometer with an FIAS 100 Hydride System (PerkinElmer SIMAA 6000), which applies the same procedures as CVAAS, has a sensitivity of 10 $\mu\text{g/l}$ (for 0.07 abs) and makes it possible to analyse other elements in addition to mercury. Mercury analysis was conducted by preparing standard working solutions of 0.5, 1.0, 2.0, 3.0, 4.0 and 5.0 $\mu\text{g/l}$ using a Merck reference solution of 1000 ppm Hg and 1% HCl as a reducer.

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