Letter to the Editor

on the Article:

"Prognos in the diagnosis of amalgam hypersensitivity. A diagnostic case-control study."

by Köhler W, Linde K, Halbach S, Zilker T, Kremers L, Saller R, Melchart D.

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Running head: Amalgam and adverse health effects

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Köhler et al. (2007) examine a bioenergetical test machine (Prognos) for the ability to diagnose amalgam sensitivity. They found no differences between “amalgam hypersensitives” and “amalgam insensitives”. Prognos does not seem to differentiate between the two groups. But it is neccessary to mention some other interesting data presented in the study, which are important for clarification of the the real aim of the whole study, which was described in the decision of the court after the trial against a former big amalgam producer: the evaluation of the safety of dental amalgam [1].

The mercury levels in biomarkers of 27 patients who complained of health problems from dental amalgam (“amalgam hypersensitives”), of 27 healthy patients with amalgam (“amalgam insensitives”) and 27 amalgam free volunteers were presented by the authors.

Interestingly, the “amalgam hypersensitives” were 12 years older and had had dental amalgam for 12 years longer than the “amalgam insensitives”.
Because tissue levels of mercury accumulate over time from exposure to amalgam (for review see [2]) and individuals with dental amalgam have over 10-fold more mercury in body tissues [3], the “amalgam-hypersensitives” would have more mercury in their body tissues than the “amalgam insensitives”.
Astonishingly, despite their higher mercury body burden, the “amalgam hypersensitives” showed slightly lower levels of mercury in their urine, even after provocation with the mercury chelator Dimercapto-propan-sulfonate (DMPS). This has to be discussed.
Another study has shown that subjects with highest urine levels after DMPS-challenge showed best recovery rates from complaints [4]. Therefore, individuals with high levels of mercury in biomarkers (like urine) have a better excretion capacity for mercury [5-7]. As a consequence, one may read from the data presented by Köhler et al. (2007) that “amalgam sensitivity” may be partially caused by lower detoxification capacity compared to the “amalgam insensitives” and thus lower excretion of mercury from the body tissues in urine, even after chelation.

We are surprised that Prognos was used to examine “amalgam hypersensitivity”, which is not acknowledged by governmental health authorities. It would have been more appropriate to test proven susceptibility parameters, which differentiate more exactly between “amalgam hypersensitives” and “amalgam insensitives” . Therefore, we wish to ask the authors, whether they have the ability to additionally measure :

A. porphyrine profiles, because aberrant urine porphyrine profiles was described in a relevant portion of individuals with low mercury exposure through dental amalgam [8-10],
B. polymorphism of coproporphyrinoxidase (CPOX4) [11], which leads to increased susceptibility to mercury and impaired production of heme [12] (Heme is critical for several essential biochemical mechanisms (Haemoglobin, all P450-enzymes, oxidative ATP-synthesis, detoxifying of ß-Amyloid from the brain),

C. polymorphism of the brain derived neurotropic factor, which also increases susceptibility to low level mercury exposure [13],
D. apolipoproteine E- genotype, because “amalgam hypersensitives” are more likely to be carriers of the apolipoprotein E4-allele (APO-E4) than “amalgam insensitives” [14, 15] (APO-E4 is the major genetic risk factor for Alzheimer’s disease, perhaps due to his lack in the capacity to remove mercury from the brain [16]),
E. polymorphism of impaired GSH-production, which leads to higher retention of mercury in the body [17, 18]. Glutathione (GSH) is a natural chelator for heavy metals in humans. In fact, only mercury bound to glutathione or other thiols is capable to leave the cells into blood and consequently to urine or bile for excretion.

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Comment: Fehtl da was?

Köhler et al. claim: *“There is no convincing evidence from epidemiological, toxicological or immunological research that “amalgam burden” or “amalgam hypersensitivity” (beyond rare cases of proven allergic reactions like oral lichenoid reactions) are valid pathological concepts.”*
As a proof of this claim they cite two papers, which, unfortunately, are more than 10 years old and were written mainly by dentists and their toxicologists. But more recent data shows that exposure to mercury from amalgam cannot be ruled out as a pathological concept [19, 20].

To mention only “*rare cases of proven allergic reactions*” due to amalgam is misleading. Conventional “proof” of allergic reactions to amalgam involves a positive cutaneous patch test, together with visible mucosal reactions adjacent to the dental filling. But in more than 90% of the cases, these lesions have been found to recover by removal of amalgam, regardless of whether the patch test was positive or not (for review see [3]). Therefore, the cutaneous test for detecting sensitivity or allergy to amalgam has been recently seriously questioned [21].

Köhler et al declared no conflict of interest (2007). One of the authors (S.H.), was referred to by the German Institute for Drug Safety and Medical Products (Bundesinstitut für Arzneimittel und Medizinalprodukte) as a well known expert for the amalgam industry [22]. He was also described by others as an official representative of the German Dentistry Board (Bundeszahnärztekammer) [23, 24]. In reaction to an amalgam critical expertise [25], required by the prosecuting attorney in the above mentioned litigation against an amalgam producer [1], he defended the safety of dental amalgam together with the attorney of the amalgam producer [26]. It was described that they used unscientific statements [27, 28] in order to influence the following political decisions against the prohibitation of dental amalgam in Germany.

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