

Alliance contribution

Question 1: Are mercury releases caused by the use of dental amalgam a risk to the environment? The fate of mercury released from dental clinics as well as the fate of mercury released to air, water and soil from fillings placed in patients should be taken into account

SCHER noticed that nowadays dental amalgams may represent one of the major intentional uses of mercury. A mass balance of mercury emissions, in air, water and soil, from dental amalgam has been proposed by Bio Intelligence Service (2012). This type of mass balance contributes to the understanding of the magnitude and sources of mercury contamination caused by dental applications. However, it does not enable to quantitatively assess the risks of Hg in amalgam, particularly if one considers that a non-negligible risk from mercury in dental amalgam is likely to occur only at a local scale, close to relevant emission sites. For the soil and air compartment SCHER concluded that a quantitative Predicted Environmental Concentration (PEC) cannot be estimated and an assessment of local risk is not possible at the moment. Only for the aquatic environment a more quantitative assessment is considered possible. Exposure in surface water has been calculated considering three possible scenarios (worst, average and best case). The PECs calculated in the three hypotheses have been compared with the Water Framework Directive Environmental Quality Standards (Annual Average EQS and Maximum Allowable Concentration EQS) that have been set for mercury. The comparison shows that only in the worst scenario the PEC is above both AA and MAC EQS.

The most pessimistic estimations must be taken into account in the calculation of fish impregnation. In fact:

- 1) A part of the population (especially heavy consumers of coastal areas, including pregnant women and children) exceeds the TWI (INRA AFSSA, 2006). Yet it is essential in order to protect the entire population.
- 2) Exposure to different types of mercury is cumulative. But the "worst case scenario" takes place in countries where the situation is most critical on dental mercury exposure, such as France and Poland (which both represent half of the EU consumption of dental mercury, as the first source of exposure (BIOIS 2012)). In order to protect every one, the risk assessment should be based on the most worrying data and not on "average" values.

1. INRA, AFSSA. **Etude des Consommations Alimentaires de produits de la mer et Imprégnation aux éléments traces, Polluants et Oméga 3**. 2006.
<http://www.anses.fr/fr/documents/PASER-Ra-Calipso.pdf>
2. Bio Intelligence Service. **Study of the potential for reducing mercury pollution from dental amalgam and batteries**. Final report. European Commission-DG ENV, 11 July 2012.

http://ec.europa.eu/environment/chemicals/mercury/pdf/final_report_110712.pdf

Question 2: Is it scientifically justified to conclude that mercury in dental amalgam could cause serious effects on human health due to mercury releases into the environment?

Mercury coming from dental amalgam as well as from many other sources, ubiquitously distributed in the environment, can be taken up by the general human population via food, water and air. Regarding the contribution of environmental mercury coming from dental amalgam use, it can be concluded that emissions of Hg to soil are not considered as a concern regarding human health. Regarding inhalation, amalgam use will also make only a limited contribution to the overall human inhalation exposure. The contribution of amalgam use to the concentrations of methyl mercury found in fish is not known and consequently no clear conclusion on possible health risks is possible. However SCHER estimated three scenarios in fish based on five hypothetical values for the methylation rate of mercury. SCHER also noted that all additional sources which add to the methyl mercury burden in humans may increase the number of people at risk, thus respecting the more conservative WFD threshold would contribute to the prevention of human health effects.

The SCHER report ignores the many publications that have shown insufficient protection afforded by the current TWI.

The TWI must protect the most vulnerable organisms.

- This is first of the embryo, fetus and child, the developing nervous system is extremely sensitive to the effects of mercury, even at very low doses. Some studies have demonstrated an inverse relationship between the concentration of mercury in cord and psychomotor development, verbal and performance IQ of young children [Lederman 2008] , and between the concentration of mercury in maternal erythrocytes and performance of vocabulary as well as visuomotor abilities of the child [Oken 2008], in moderately intensive fish populations.

- Second, a significant proportion of the population is particularly vulnerable to very low levels of mercury exposure because of its genetic susceptibility and thus its inability to eliminate mercury [Wang 2012, Goodrich 2011, Schläwicke 2008, Godfrey 2003, Heyer 2004, Heyer 2008, Heyer 2009 , Echeverria 2010, Lee 2010, Woods 2012].

In addition, assessments used to determine the toxicological reference values do not take into account the multi-shot (mixture effects): yet it is shown that mercury toxicity is greatly enhanced by the lead [Schubert 1978] , the hydroxide aluminum or antibiotics [Haley 2005]. It also demonstrated that the capacity of urinary mercury disposal to reduce as exposure [DeRouen 2006; Mutter 2011].

Finally, the European people do not undergo comparable mercury exposure: the French and the Poles are on average much more exposed to dental mercury, while the Spanish, French (still more people in Guyana) and all coastal residents are more exposed to methylmercury in fish. It would be unacceptable to consider an average exposure, which would leave millions of Europeans exposed beyond the TWI. Given the foregoing, and having established that mercury (metal- and organic form) is a neurotoxic, immunotoxic and endocrine disruptor, it is impossible to determine a threshold below which adverse effects would be excluded [WHO 2005] : the current TWI is not sufficiently protective. This indisputable fact should be

mentioned by the SCHER must conclude that all unnecessary uses of mercury should be banned as soon as possible.

THUS, IT IS THE "WORST CASE SCENARIO" TO BE CHOSEN BY EXPERTS TO PROTECT THE EUROPEAN POPULATION.

3. Lederman SA, Jones RL, Caldwell KL, Rauh V, Sheets SE, Tang D, Viswanathan S, Becker M, Stein JL, Wang RY, Perera FP. **Relation between cord blood mercury levels and early child development in a World Trade Center cohort.** *Environ Health Perspect.* 2008 Aug;116(8):1085-91.
4. Oken E, Bellinger DC. (2008). **Fish consumption, methylmercury and child neurodevelopment.** *Curr Opin Pediatr.* 2008 Apr;20(2):178-83.
5. Wang Y, Goodrich JM, Gillespie B, Werner R, Basu N, Franzblau A. **An investigation of modifying effects of metallothionein single-nucleotide polymorphisms on the association between mercury exposure and biomarker levels.** *Environ Health Perspect.* 2012 Apr;120(4):530-4.
6. Goodrich JM, Wang Y, Gillespie B, Werner R, Franzblau A, Basu N. **Glutathione enzyme and selenoprotein polymorphisms associate with mercury biomarker levels in Michigan dental professionals.** *Toxicol Appl Pharmacol.* 2011 Dec 1;257(2):301-8.
7. Schläwicke Engström K, Strömberg U, Lundh T, Johansson I, Vessby B, Hallmans G, Skerfving S, Broberg K. **Genetic variation in glutathione-related genes and body burden of methylmercury.** *Environ Health Perspect.* 2008 Jun;116(6):734-9.
8. Godfrey ME, Wojcik DP, Krone CA. **Apolipoprotein E genotyping as a potential biomarker for mercury neurotoxicity.** *J Alzheimers Dis.* 2003 Jun;5(3):189-95.
9. Heyer NJ, Echeverria D, Farin FM, Woods JS. **The association between serotonin transporter gene promoter polymorphism (5-HTTLPR), self-reported symptoms, and dental mercury exposure.** *J Toxicol Environ Health A.* 2008;71(19):1318-26.
10. Heyer NJ, Echeverria D, Bittner AC Jr, Farin FM, Garabedian CC, Woods JS. **Chronic low-level mercury exposure, BDNF polymorphism, and associations with self-reported symptoms and mood.** *Toxicol Sci.* 2004 Oct;81(2):354-63.
11. Heyer NJ, Echeverria D, Martin MD, Farin FM, Woods JS. **Catechol O-methyltransferase (COMT) VAL158MET functional polymorphism, dental mercury exposure, and self-reported symptoms and mood.** *J Toxicol Environ Health A.* 2009;72(9):599-609.
12. Echeverria D, Woods JS, Heyer NJ, Martin MD, Rohlman DS, Farin FM, Li T. **The association between serotonin transporter gene promoter polymorphism (5-HTTLPR) and elemental mercury exposure on mood and behavior in humans.** *J Toxicol Environ Health A.* 2010;73(15):1003-20.
13. Lee BE, Hong YC, Park H, Ha M, Koo BS, Chang N, Roh YM, Kim BN, Kim YJ, Kim BM, Jo SJ, Ha EH. **Interaction between GSTM1/GSTT1 polymorphism and blood mercury on birth weight.** *Environ Health Perspect.* 2010 Mar;118(3):437-43.
14. Woods JS, Heyer NJ, Echeverria D, Russo JE, Martin MD, Bernardo MF, Luis HS, Vaz L, Farin FM. **Modification of neurobehavioral effects of mercury by a genetic polymorphism of coproporphyrinogen oxidase in children.** *Neurotoxicol Teratol.* 2012 Sep-Oct;34(5):513-21.
15. Schubert J, Riley EJ, Tyler SA: **Combined effects in toxicology - a rapid systematic testing procedure: cadmium, mercury, and lead.** *J Toxicol Environ Health* 1978 , 4:763-776.
16. Haley B: **Mercury toxicity: Genetic susceptibilities and synergistic effects.** *Medical Veritas* 2005, 2:535-542.
17. DeRouen TA, Martin MD, Leroux BG, et al. **Neurobehavioral effects of dental amalgam in children: a randomized clinical trial.** *JAMA.* 2006;295(Suppl 15):1784-1792.

18. Mutter J. **Is dental amalgam safe for humans?** The opinion of the scientific committee of the European Commission. *J Occup Med Toxicol*. 2011 Jan 13;6(1):2.
19. WHO: **Mercury in Health Care**. Policy Paper 2005.

Question 3: Comparison of environmental risk from the use of mercury in dental amalgam and the use of alternatives without mercury

The information available on the Hg-free alternatives does not allow for a sound risk assessment to be performed. For the human health, SCHER is of the opinion that the conclusions of the 2008-opinion are still valid, except for alternative materials containing bisphenol A-glycidyl methacrylate (Bis-GMA). For these materials SCHER recommends to refer to an on-going SCENIHR mandate on the use of bisphenol A in medical devices, as soon as this becomes available. For the environment, considering the probably low level of emissions and the relatively low toxicity of the chemicals involved, it is reasonable to suppose that the ecological risk should be low. However, it is the opinion of the SCHER that, at present, there is no scientific evidence for supporting and endorsing these statements. Therefore, more research on alternative materials is recommended.

Bisphenol A (BPA) is this only danger that has been identified in alternative dental materials. However, the environmental footprint of this substance remains much lower than the one of mercury because BPA is neither biopersistent nor bioaccumulative. Several resins and all glass ionomer cements do not contain BPA. Even though scientific datas confirming their safety are scarce, the use of these materials should be preferred to the use of materials for which hazards have been clearly demonstrated.