Letter to the Editor

Commentary regarding the article by Drasch et al.: Scientific comment on the German human biological monitoring values (HBM values) for mercury. Int. J. Hyg. Environ. Health 205, 509-512 (2002)

German Human Biomonitoring Commission¹

Sir,

we read the critical points on the HBM values for mercury in blood and/or urine raised by Drasch et al. with interest. However, there are a number of aspects in the commentary that require clarification.

The comments given by Drasch et al. (2002) are derived from the "Mt. Diwata study on the Philippines 1999" (Drasch et al., 2001). The authors have described a highly interesting population in a gold mining area on the Philippines that has been heavily exposed to mercury vapor and probably to organic mercury as well. Based on biological monitoring and local inspection the authors confirmed high mercury exposure in ball-miners and amalgam smelters (n=102). In addition, 3 non-occupationally exposed groups were established (control, n=42; downstream from Mt. Diwata, n=100; Mt. Diwata, n=63). However, as shown by mercury levels in blood and hair, these participants were obviously also exposed to mercury. Median and the maximum mercury blood concentration in the control group were 9.0 µg/l and 31.3 µg/l. The median blood mercury level of 11.4 µg/l of the occupationally exposed group was only slightly higher. For comparison, the median mercury levels in blood as derived from the German Environmental Survey 1998 (GerES III) is much lower (0.3-1.0 µg/l depending on the frequency of fish consumption) (Becker et al., 2002). The reason for the high blood mercury levels in the non-occupationally exposed groups was not evident to the authors. Except locality, criteria applied for defining the individual non-occupationally exposed group(s) are not indicated. The results of biological monitoring, however, were not used for this purpose. No relationship between mercury exposure and clinical symptoms was found. Presence and quality of dental amalgam fillings were not examined.

The first paper by Drasch et al. (2001) was examined critically by the Human Biomonitoring Commission of the German Federal Environmental Agency. In their response to the questions raised, the authors conceded that the control group was also exposed to

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mercury, and that this group was, on average, older in age and of a higher education level. Differences in dietary habits, e. g. fish consumption, were not identified in the 4 groups. The authors also agreed that a single sampling of blood is not sufficient for the evaluation of chronic exposure. In a recurrent investigation one year later significant differences in the mercury levels in blood, were found on an individual basis, whereas the average exposure did not differ from that found in the first investigation.

Because neurological symptoms in particular are strongly influenced by the consumption of alcoholic beverages, and additionally by age, in a second evaluation the entire population including adult men with traditionally high alcohol consumption, were compared with a subgroup of women and children who did not consume alcohol. Although in the (small) group of women and children in general the frequency of symptoms was lower than in the whole cohort, including men, there were no indications of a relationship between mercury levels in blood and urine and symptoms within these groups. On the contrary, to some extent inverse effects were actually observed. Even in the control group (n=20 women and children), pathological symptoms were observed in ca. 20%.

In their commentary in this journal (Drasch et al., 2002), the authors tried to verify the human biological monitoring values (HBM values) (Ewers et al., 1999) recommended by the Commission for mercury levels in blood and urine (Kommission Human-Biomonitoring 1999) by using the results from the Mt. Diwata study. They arrived at statements that are incompatible with the view of the Commission.

The authors criticize that in the HBM values: "No differences are made for inorganic and organic mercury burden, nor for age and gender". However, in the mercury monograph published by the Commission (Kommission Human-Biomonitoring 1999) the following statements are made: The determination of mercury in urine allows evaluation of the internal exposure to inorganic mercury and the determination of mercury in blood allows evaluation of the internal exposure to inorganic and organic (total) mercury. In addition, as far as adequate, different HBM values are derived for children and adults.

In Tables 1 and 2 (Drasch et al., 2002) the authors present data on symptoms diagnosed using neuro-psychological tests in their exposed cohort group (n=261) that are characteristic not only for mercury exposure, e. g. ataxia, tremor and dysdiadochokinesis. For the cohort as a whole, the frequency of symptoms recorded was in the range of ca. 20 to 70 %. Even in the "control" group the corresponding rates were 17 to 22 %. After dividing the cohort into 3 groups of different internal exposure, that is below HBM I, between HBM I and II, and exceeding HBM II, they detected no increase in the frequency of the symptoms under consideration. From this finding they conclude that ".. the mercury concentrations in blood and/or urine alone are not appropriate at all for the establishment of a toxicologically defined threshold limit like the HBM value": The results presented by the authors cannot demonstrate an increase in effects with increasing mercury dose (note: the number of participants in the

group of highest exposure was only 34). Other causes may be responsible for the symptoms observed as well, e. g. alcoholic beverages in the case of men, or drugs taken for the treatment of infectious diseases (tuberculosis) or social factors. Migration within the area investigated was reported to occur frequently and may also have confounded the results. However, a convincing explanation for the results obtained is that mercury is indeed the cause of these symptoms but that the biological monitoring performed by the authors is completely inadequate to illustrate the exposure scenario precisely. It is astonishing that the mercury concentrations analysed in blood and urine are generally in a range in which severe signs of mercury intoxication are not to be expected to such a high degree. Presumably, additional factors may be involved, e. g. short-term peak exposures and highly fluctuating exposures of the individuals, as documented in the second biological monitoring analysis.

Even a short study of the mercury monograph of the Commission (Kommission Human-Biomonitoring 1999) would have revealed that HBM-values should not have been applied to this cohort. In analysing mercury in blood the internal load of inorganic and organic mercury is determined. According to the guidelines of the Commission, in cases in which no occupational exposure to mercury vapor has taken place and no dental amalgam fillings exist, the HBM values in blood can be applied to organic mercury without restriction. The participants of the study by Drasch et al., however, were evidently exposed quite inhomogeneously in dose as well as in the species of mercury compounds.

In conclusion, the study from the Philippines by Drasch et al. (2001) is very helpful to learn more about the adverse effects of mercury compounds. However, the data present a very complex picture. The massive contamination over a large area makes an adequate exposure assessment (past versus present) and selection of controls difficult. Nevertheless, the Commission cannot see a need to revise the HBM values for mercury because of the results published by Drasch et al.(2001).

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