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## Mercury Levels in Newborns and Infants After Receipt of Thimerosal-Containing Vaccines

To the Editor.—

Magos et al<sup>1</sup> found that in Porton-Wistar rats the overall brain mercury concentration was lower after exposure to ethyl mercury than to methyl mercury. However, it is significant to note that this study also demonstrated that a higher proportion of inorganic mercury was retained in the brain after exposure to ethyl mercury than to methyl mercury, in each case the inorganic mercury likely being formed as a result of the dealkylation of the ethyl or methyl mercury in the brain. In fact, the absolute concentration of inorganic mercury found in the brain was higher after exposure to ethyl mercury. Furthermore, in addition to the findings commented on by Pichichero et al<sup>2</sup> in the February 2008 *Pediatrics Electronic Pages*, thorough examination of the data from Burbacher et al<sup>3</sup> demonstrates that in infant *Macaca fascicularis* monkeys a higher proportion of mercury from thimerosal-containing vaccines was retained in the brain as inorganic mercury than from oral dosing of methyl mercury; this time, approximately the same level of inorganic mercury was found in both cases.

As previously discussed,<sup>4</sup> once inorganic mercury has found its way into the brain, it has a half-life therein considerably longer than that of ethyl mercury or methyl mercury and has potential to accumulate in cases of repeated or prolonged exposure. Thus, although ethyl mercury and methyl mercury may exhibit more acute neurotoxicity than inorganic mercury, they do not share the same ability to accumulate over time that inorganic mercury exhibits (Vahter et al<sup>5</sup>). Thus, when one considers whether ethyl mercury from thimerosal might play an etiologic role in the development of autism, surely one should also consider that thimerosal may exert toxic effects by contributing to an inorganic mercury load of long half-life in the brain that originates from multiple sources of mercury (including but not limited to elemental mercury from dental amalgam; methyl mercury from fish consumption; elemental mercury and methyl mercury that may have crossed the placenta in utero; methyl mercury in breast milk; and other environmental exposures).

Although the Pichichero et al<sup>2</sup> study provided data regarding the toxicokinetics of ethyl mercury in new-

borns, it did not provide any insight into the toxicity or toxicodynamics of the small quantities of long half-life inorganic mercury that likely resulted from the dealkylation of ethyl mercury in the central nervous systems of these children. Furthermore, they did not consider that these small quantities of long half-life inorganic mercury may be additive to a preexisting central nervous system load of inorganic mercury of indeterminate amount.

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In Reply.—

Our study was not a toxicology evaluation of ethyl mercury in children; it was a pharmacokinetic study. We are aware of the works cited by Rooney and interpret the findings as he does in the animals evaluated; their applicability to humans is unknown. We certainly could not perform brain biopsies on the normal children we studied to determine if any mercury was present. Furthermore, some mercury in the children's blood was clearly methyl mercury, so even if we performed a brain biopsy it would remain unknown whether the source was methyl or ethyl mercury. We did study the kidneys for evidence of toxicity, because we could obtain urine