

Mercury in Human Maternal and Cord Blood, Placenta, and Milk¹ (39259)ROY M. PITKIN,² JOHN A. BAHNS, L. J. FILER, JR., AND W. ANN REYNOLDS*Departments of Obstetrics and Gynecology and Pediatrics, University of Iowa Hospitals, Iowa City, Iowa 52242; and Department of Anatomy, University of Illinois at the Medical Center, Chicago, Illinois 60612*

Environmental pollution by toxic agents is a matter of considerable current concern. Much of the interest has centered on mercury, an agent long known to exert toxic effects on a variety of tissues, especially the central nervous system. Observations from outbreaks of mercury poisoning in Japan, Iraq, and southwestern United States have indicated that the fetus and newborn infant appear particularly susceptible to lethal and sublethal mercury toxicity, often exhibiting evidence of damage in the absence of maternal symptoms (1).

In order to assess the risk of mercury toxicity in the population, information regarding levels in individuals without known abnormal exposure is necessary. This report concerns total mercury concentrations in maternal and cord blood pairs and placentae from women at delivery and in breast milk samples from nursing mothers.

Methods. Heparinized blood was collected from the antecubital vein of 100 consecutive parturient women within 1 hr of vaginal delivery and from cut end of the umbilical cord immediately after birth of the same 100 infants. Following alternate freeze-thawing to promote cellular lysis, the total mercury concentration in whole blood was measured on a Perkin-Elmer 303 atomic absorption spectrophotometer, using the method of Magos and Cernik (2) modified by injecting the sample into the system after (rather than before) a constant absorption reading was obtained on the spectrophotometer.

Mercury content was measured in 38 placentae (from patients other than those who had maternal-cord studies) obtained from normal vaginal deliveries at term. The fetal

membranes and cord were trimmed from each placenta and adherent blood clots removed prior to analysis. A 6-g aliquant of the ground and homogenized whole placenta was oxidized with concentrated nitric acid for 48 hr and then neutralized to pH 7 with 45% sodium hydroxide. The mercury concentration of the neutralized sample was then measured by atomic absorption spectrophotometry.

Samples of breast milk were obtained by manual expression from 32 women with established lactation of 56 or more days duration. Milk samples were homogenized and their mercury concentration measured by atomic absorption spectrophotometry.

Containers and syringes of polycarbonate plastic were used in all collection and analytical procedures. Reagent-grade chemicals were employed. The results were expressed in terms of total mercury concentration in parts per billion (ppb). Using sample volumes of 2 ml, the limit of detection of the method was 1 ppb. Values below the limit of detection were considered, for statistical purposes, to be equivalent to zero. Statistical evaluation of the data was by Student's *t* test.

Results. The concentration of total mercury ranged from 0 to 8 ppb in maternal blood and from 0 to 6 ppb in cord blood. Assuming levels below the limit of detection equivalent to zero, the maternal mean ($\pm$ SEM) was 1.01 ($\pm$ 0.12) ppb, and the cord mean was 1.24 ($\pm$ 0.14) ppb. While cord levels averaged more than maternal, the difference was not statistically significant ($P > 0.05$). However, maternal and cord levels were significantly related with each other ($r = 0.31$, $P < 0.01$).

Additional data regarding the relationships between mercury levels in maternal and cord blood are listed in Table I. Slightly more than a third of the maternal-cord pairs had no detectable mercury, and a similar proportion exhibited detectable levels in

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both mother and fetus. In the remainder, mercury was found in one, but not in the other, circulation. A tendency to higher levels in cord than in maternal blood was reflected in the findings that of the total group of 100 maternal-cord pairs, maternal levels exceeded cord in 17%, cord exceeded maternal in 35%, and the two were equal in 48%.

The influence of various clinical features on the results was examined by calculating correlation coefficients for maternal and cord levels with maternal age, parity, duration of pregnancy, and birthweight. As indicated in Table II, the only significant correlation among these variables was that between maternal age and maternal mercury level.

Mercury was detected in 34 to 38 placentae analyzed, and the mean ($\pm$ SEM) concentration was 2.28 ($\pm$ 0.29) ppb. The mean ($\pm$ SEM) mercury content per placenta was 1.38 ($\pm$ 0.15) μ g. No significant correlation between placental mercury concentration and maternal age, parity, or birthweight was found.

Detectable mercury levels were found in 14 of 32 milk samples examined. Assuming values below the limit of detection to be equivalent to zero, the mean ($\pm$ SEM) level was 0.93 ($\pm$ 0.23) ppb. No significant correlation between milk mercury concentration and age or duration of lactation was found.

Discussion. Total mercury was measured in the present study since the various forms in which mercury exists could not be differentiated at such low levels. However, considerable inferential evidence suggests that the principal form of mercury in fetus is methylmercury. At least in the mouse, placental permeability is 10 to 20 times greater for methylmercury than for inorganic mer-

TABLE II. CORRELATION COEFFICIENTS BETWEEN MATERNAL AND CORD MERCURY LEVELS AND CLINICAL PARAMETERS.

	Maternal	Cord
Maternal age	0.21*	0.13
Parity	0.07	0.03
Duration of pregnancy	0.11	-0.11
Birthweight	0.08	0.13

* $P < 0.05$.

cury salts or phenylmercury (3). Moreover, acute placental transfer studies in the monkey have shown that methylmercury is transported more rapidly to the fetus than from the fetus (4).

Table III compares the present data with previous reports of total mercury levels in maternal and cord blood and placentae from populations not known to have particular exposure. In all studies, cord levels have been found to exceed maternal by 20 to 30% and placental levels to be two- to three-fold greater than cord. The values found in this study are substantially lower than those reported previously. Part of the explanation of this apparent discrepancy may lie in methodology. While each of the studies listed in Table III employed atomic absorption spectrophotometry, our method involved injection of each sample into the system *after* a constant reading of the spectrophotometer was obtained, in order to correct for mercury contamination in reagents and glassware. If injection is done *before* a constant reading, as described in the original method of Magos and Cernik (1), elevated levels may occur as a result of contamination. (Preliminary studies indicated that as much as 10 ng of mercury may be present in the reagent grade chemicals and acid-rinsed utensils used for mercury analysis.) However, the major reason for the difference probably results from varying degrees of mercury exposure of the populations studied. Contaminated fish represents the principal food source of mercury, and in Japan and Sweden fish constitutes a major portion of the usual diet. The only previous study from the United States involved subjects in Nashville, Tennessee, whose mercury levels could well reflect industrial pollution in such an urban area. By contrast, our subjects were from a predominately rural state in which fish represents a minor

TABLE I. MERCURY CONTENT IN MATERNAL AND CORD BLOOD.

Relationships in 100 pairs	
Nondetectable in either maternal or cord	38
Detectable in maternal but not in cord	9
Detectable in cord but not in maternal	17
Detectable in both maternal and cord	36
Maternal > cord	8
Maternal = cord	10
Cord > maternal	18
	100

TABLE III. REPORTS OF MATERNAL AND CORD BLOOD AND PLACENTAL MERCURY LEVELS IN NORMALLY EXPOSED POPULATIONS.

Author	Population	Mean total mercury concentration (ppb)		
		Maternal blood	Cord blood	Placenta
Suzuki <i>et al.</i> (5)	Japan: 9 paired placental, maternal blood, and cord blood specimens	16.8*	20.0*	71.5
Tejning (6)	Sweden: 4 paired maternal and cord blood specimens	6.0*	7.7*	
Baglan <i>et al.</i> (7)	USA (Nashville, Tennessee): 1061 placentae, 929 maternal bloods, and 872 cord bloods	8.7	11.5	21.5
Present	USA (Iowa): 38 placentae and 100 maternal-cord blood pairs	1.0	1.2	2.3

* Values calculated from graphs assuming maternal hematocrit 33% and cord hematocrit 45%.

component of the diet and in which heavy industry is not particularly prevalent.

The results of this study suggest that the danger of mercury-related damage in pregnancy and lactation need not be an area for major concern in a midwestern rural population at the present time. More than one-third of our subjects had levels below the limit of detectability in both maternal and cord blood and more than half of the breast milk samples did not contain detectable mercury. An expert committee has recommended that the maximum allowable concentration of mercury should be 0.1 $\mu\text{g}/\text{ml}$ of whole blood (8). This value is equivalent to 100 ppb, or 12 times the maximal blood level found in our studies. Nevertheless, the possibility of increasing environmental contamination coupled with the well-established propensity to mercury toxicity during the perinatal period indicates a need for continuing surveillance.

Summary. Total mercury concentration was measured by atomic absorption spectrophotometry in 100 maternal-umbilical cord blood pairs, 38 placentae, and 32 breast milk samples, all from patients at the University of Iowa Hospitals. The mean maternal and cord blood levels were 1.01 and 1.24 parts per billion (ppb), respectively. Though the difference between maternal and cord values was not statistically significant,

the two were significantly correlated with each other, and maternal blood level increased significantly with maternal age. Mean placental and milk concentrations were 2.28 and 0.93 ppb, respectively. The mercury levels in this study were lower than those reported from elsewhere, probably reflecting a combination of methodological refinement and relatively low mercury exposure in a rural population whose diet is low in fish consumption. These low levels suggest that the risk of perinatal mercury damage is small in such a population.

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