

Letter to the Editor

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Dr. Mizuta and his colleagues examined the thyroid hormone content of breast milk from three normal women and eighteen women with thyroid disease. From this group of largely abnormal individuals, they concluded that low concentrations of T₃ (about one-third that of serum) and little or no T₄ is present in human milk. They conclude that no correlation between serum and milk T₃ levels exists. Last, they find no significant differences in thyroid hormone or thyrotropin levels of breast- versus bottle-fed infants.

Although it is interesting to examine individuals with thyroid disease in such studies in order to achieve a wide scatter of serum hormone levels, it seems questionable to extrapolate the results obtained from these women's milk to the normal population. This may be especially true in the case of Graves' disease, with its myriad physiologic and biochemical abnormalities of peripheral thyroid hormone metabolism. Only three euthyroid women were included in this study, and their milks were obtained at widely disparate times of lactation. Actual data for serum and milk T₃ levels from these normal individuals are not, in fact, provided at all in the manuscript, and T₄ levels are at or below levels of detectability for their assay. Thus, it is impossible to determine from the data whether or not thyroid disease influences the secretion of thyroid hormone into breast milk.

The second part of this manuscript deals with a comparison between hormone levels of 24 breast- and 10 formula-fed infants. This aspect of the article would have been more meaningful if the breast milks which fed these same infants had been analyzed, because apparently these were not the offspring of the thyroid-diseased mothers who comprised the bulk of the study. Critical details such as % total nutrition as breast milk, duration of breast feeding, and gestational age of the infants can influence the outcome of such statistical analyses. Furthermore, it is desirable for such comparisons to include approximately equal populations for both study groups if possible.

In a recent publication by Hahn *et al.* (1) these same issues of thyroid function were examined in younger infants (16–18-day-old) either breast- ($n = 22$) or formula-fed ($n = 25$). Although significant differences were found in both T₃ and T₄ concentrations, the elevation of T₃ levels in breast-fed infants was most pronounced. The fact that these younger breast-fed infants displayed significantly higher T₃ levels than their formula-fed counterparts, vis à vis those in the study by Mizuta *et al.*, is not surprising in light of our preliminary data which indicate an inverse relationship between thyroid hormone concentrations in milk and time of lactation. This is particularly true within the first month postpartum.

Hahn's data are also consistent with our recently published findings of low to undetectable ($1.21 \pm 0.48 \mu\text{g}/\text{dl}$, $n = 7$) T₄ levels (also in agreement with the present study), but physiologic or higher concentrations of T₃ ($275 \pm 132 \text{ ng T}_3/\text{dl}$, $n = 9$) in mature human milk from euthyroid mothers (2). Our data were obtained using an extraction procedure very similar to that of Mizuta *et al.* except that we incorporated a chromatographic step which assured elimination of interfering or inhibitory substances in the assay.

For the sake of accuracy, this article might better have been titled, "Thyroid Hormone Concentrations in Breast Milk of Women with Thyroid Disease." In that case, consistency would strongly suggest the infants examined be offspring of those same women.

REFERENCES AND NOTES

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Response

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We think that Dr. Oberkotter's suggestions are certainly very important. In our study, patients with thyroid disease were included to obtain a wide scatter of serum hormone levels for the comparison of T₃ concentrations between serum and milk. We had also examined 11 samples of milk obtained from normal women within 1 year postpartum, although the detailed data were not described. The results were almost the same as those described in our paper: thyroxine (T₄) concentrations in milk of these normal subjects were under the detectable level ($<0.7 \mu\text{g}/\text{dl}$). The more sensitive methods such as Mallol's report (1) would provide more detailed results on T₄ concentrations. Triiodothyronine (T₃) concentrations in milk of these normal subjects were $53 \pm 24 \text{ ng}/\text{dl}$. The serum concentrations of T₃ were $123 \pm 26 \text{ ng}/\text{dl}$ and the ratio of serum T₃ to milk T₃ was 2.3. The mean concentrations of T₃ in the milk within 1 month postpartum ($34 \pm 19 \text{ ng}/\text{dl}$) were significantly lower than those after 1 month postpartum ($70 \pm 11 \text{ ng}/\text{dl}$, $P < 0.05$); thus we believe that T₃ concentrations are low and little or no T₄ is present in human milk obtained from normal women.

The neonatal subjects in our study were completely breast or bottle-fed for at least 3 wk and examined 1 month after birth. There were no significant differences in gestational age (full-term), sex, birthweight, and increasing rate of bodyweight between the two groups.

Iodine is well-known to influence thyroid hormone economics (2, 3), and the difference in the iodine contents in milk may also result in the difference in T₃ concentration in newborns. The discrepancy of serum thyroid hormones in neonates between our results and Hahn's report (4) may be explained by the possible difference in iodide contents between milk of Japanese women and women in the United States; however, the exact reasons are unknown at present.

Our results on low T₃ concentrations in milk are consistent with Sato's report (5) but not with Oberkotter's results. The reason for the discrepancy is obscure but it may be due partly to the difference in the methodology. In any event we don't believe that the level of thyroid hormones in milk is sufficient to supplement the thyroid economy of the thyroid-deficient suckling infant.

REFERENCES AND NOTES

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