

Selenium Status of Very Low Birth Weight Infants

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ABSTRACT. The selenium (Se) intake and status of 82 very low birth weight infants (birth weight 1110 ± 286 g, gestational age 29.2 ± 3 wk, mean \pm SD) was assessed at 36.3 ± 3 postconceptional wk, at 40.1 ± 4 wk (hospital discharge), and at 3, 6, 9, and 12 ± 0.75 mo corrected for gestational age. Infants were fed formula containing $0.13 \mu\text{mol/L}$ ($10 \mu\text{g/L}$) Se. Se-dependent glutathione peroxidase activity in red blood cells declined corresponding to low Se intakes ($\mu\text{g/kd/d}$) for the first 6 mo. With increased consumption of solid foods, intakes of dietary Se and Se-dependent glutathione peroxidase activity increased at 9 mo, suggesting that the earlier supply of Se was suboptimal. Se-dependent glutathione peroxidase activity and intakes of Se were lower in males than in females ($p < 0.05$). We suggest that infant formulas should probably contain $0.26\text{--}0.33 \mu\text{mol/L}$ ($20\text{--}25 \mu\text{g/L}$) Se, particularly those formulas consumed by very low birth weight infants. (*Pediatr Res* 34: 293–296, 1993)

Abbreviations

VLBW, very low birth weight
GSHPx, glutathione peroxidase
SeGSHPx, selenium-dependent glutathione peroxidase
ANOVA, analysis of variance
TPN, total parenteral nutrition

VLBW premature infants (<1500 g birth weight) are at risk for selenium deficiency due to 1) shortened gestation resulting in inadequate stores (1) compared with those of full-term infants, 2) possible diminished selenium absorption during early postnatal development (2), and 3) an extended period of rapid growth (3–5). We (6) and others (7–9) have shown that selenium intakes during unsupplemented TPN are inadequate and can lead to low serum selenium levels. Once TPN is completed, low intakes of selenium may continue during infancy because consumption of formulas based on cow's milk unsupplemented with selenium provide only incidental selenium from the protein source (4, 6, 10). It has been shown that inadequate dietary selenium may lead to unrecognized problems in VLBW infants (7). Infants consuming breast milk will obtain higher levels of selenium (4, 10), and the bioavailability is also superior (11).

Selenium is an essential component of the enzyme SeGSHPx, which is a protective factor against tissue oxidative damage. VLBW infants are known to have lower blood levels of selenium and SeGSHPx than term infants, which may be a contributing

factor in the greater susceptibility of their erythrocytes to oxidative stress (12). Although serum selenium levels have been used to assess selenium status (9, 10), measurement of enzyme activity may be more informative of long-term status because the enzyme is a functional indicator of selenium status (4, 5). Because low intakes of selenium are a risk in this population, we monitored the selenium status of a group of VLBW infants during the first year of life, as part of a larger study involving zinc supplementation (13).

MATERIALS AND METHODS

Subjects. Eighty-two VLBW infants (mean birth weight 1110 ± 286 g; mean gestational age 29.2 ± 3 wk, mean \pm SD) were recruited for this study from the neonatal intensive care units of the Dr. Charles A. Janeway Child Health Centre, the Grace General Hospital, and St. Clare's Mercy Hospital in St. John's, Newfoundland. The study was conducted prospectively in double-blind fashion after approval from the Faculty of Medicine Human Investigations Committee. All infants treated in these centers between June 1, 1984 and June 1, 1988 were eligible for the study if they were <1500 g at birth. Infants were excluded if they had severe bronchopulmonary dysplasia that required more than 2 weeks of oxygen therapy, hydrocephalus, liver dysfunction, any congenital malformations, or were breast fed.

The gestational age of the infants was calculated from the last menstrual period of the mother and was also determined by the Dubowitz method (14). If there was a discrepancy of more than 2 wk between the two assessments, the latter method was used. Size for gestational age was considered appropriate if the birth weight fell within 2 SD of median weight for gestational age according to the growth curves of Lubchenco *et al.* (15).

Approximately 85% of parents of eligible infants consented to enroll their infants. Of the 85 infants, two infants died during their hospital stay and one infant was removed to a hospital in another province, leaving 82 infants for whom data were available at discharge.

All infants received uniform management as established in their respective neonatal intensive care units and were fed a premature special care formula (Special Care, Ross Laboratories, Columbus, OH) containing 24 kcal/fl oz until they could tolerate a formula containing 20 kcal/fl oz. At a mean postconceptional age of 36.3 ± 3 wk, infants received either 1) infant formula with whey and supplemental zinc/copper drops ($n = 29$), 2) infant formula with whey and water drops ($n = 27$), or 3) an experimental low birth weight formula ($n = 26$). All infants were fed *ad libitum*.

Upon discharge from hospital, study formula and drops were provided monthly for 5 mo. After that time, parents were responsible for purchasing their own formula. All formulas were fortified with iron at $233 \mu\text{mol/L}$ (13 mg/L) and contained approximately $0.13 \mu\text{mol/L}$ ($10 \mu\text{g/L}$) selenium (4). All formula was donated by Ross Laboratories in 32-oz ready-to-feed cans;

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who suggest that 2.5 $\mu\text{g}/\text{kg}/\text{d}$ is too low for these infants, who are born with low stores. We suggest that formulas for VLBW infants contain 0.26–0.33 $\mu\text{mol}/\text{L}$ (20–25 $\mu\text{g}/\text{L}$) selenium. Supplementation at this level would provide 3–4 $\mu\text{g}/\text{kg}/\text{d}$, well below the conservative upper limit of 8 $\mu\text{g}/\text{kg}/\text{d}$ suggested by Litov and Combs (4). In addition, these levels are similar to those reported in breast milk consumed by premature infants (10). Further research is needed to establish the optimal level of selenium fortification required to prevent the decline seen in erythrocyte SeGSHPx in the present study. This study suggests that the maintenance of erythrocyte SeGSHPx activity may be the best parameter to determine the optimal level of selenium fortification for these infants.

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