Arsenic, cadmium, lead, and aluminium (along with mercury and manganese) form a suite of toxic metals affecting the brain of exposed children. Although maternal exposure to these toxicants occurs in combination, they are studied mostly per se. Addressing early exposure to them in combination is what makes the study of Chao et al.1 unique and timely. However, in view of recent research, comparatively less attention was given to aluminium than to the other studied elements.

While major sources of maternal exposure were discussed for Pb, As, and Cd, there was no indication as to the sources of aluminium; nevertheless the authors advised preventive strategies that included behavior modification for "women who are at high risk of toxicant exposure". Although they recommended for not switching to formula feeding as a means of abating exposure in general, there was no mention that formulas, based on cow’s milk or soya, are much higher in aluminium than is breast milk.2

They reported that aluminium concentrations were highest in colostrums, at 56.45 ng/mL, decreasing to 36.57 ng/mL in transitional milk, and reaching the lowest concentration in mature milk (13.44 ng/mL) of 60 to 65 days. Quite interestingly, they showed a significant decrease with time of lactation, but did not discuss the fact that formula feeding is constant in concentration and taken in higher quantities than breast milk. In the nursing infant, the exposure to aluminium in colostrum and breast milk is proportional to the amount of milk nursed; it is taken in smaller quantities (proportional to baby’s size) spread out through the course of a day and during the entire lactation.

Another significant route of exposure to aluminium in infants is through immunization with Al-adjuvanted vaccines where the addition to the body burden might be as high as from breast milk or formula feeding.3 Indeed, the first encounter a newborn (at day 0) has with aluminium is not through colostrum but through a large parenteral load of Al(OH)3 from the Hepatitis B vaccine (HBV) as an adjuvant. Indeed, neonates (<24 h and weighing > 2000 g) may receive an Al (250 μg) exposure from HBV that is far in excess of that absorbed from breast milk taken during the entire six months of lactation.3

Rare neurologic adverse events or effects related to adjuvant-Al are unlikely to occur as a result of HBV. However, experimental studies modelling Al exposure relevant to vaccines have shown interesting results. Veiga et al.4 recently showed that young rats are susceptible to aluminium neurotoxicity, while Khan et al.,5 demonstrated the translocation of intramuscular injection of alum-adjuvanted vaccine from muscle to brain. Indeed, adjuvant-Al from vaccines can produce neurological effects.6 Despite this new information on Al exposure during early life, we still do not have reliable measures of Al body loads in babies.2 However, in silico modeling estimated that the Al body burden from feeding (human milk and formulas) during the first year (0.1 mg) was much less than that (4 mg) attributed to vaccines.7

Conflicts of Interest

The author has no conflicts of interest relevant to this article.

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References


