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Received 24 May 1986

High aluminium content of infant milk formulas

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SUMMARY The aluminium content of several commercially available infant milk formulas was measured by electrothermal atomic absorption spectrometry. Results were compared with those for fresh breast milk, cow's milk, and local tap water. Differences in aluminium concentration of greater than 150-fold were found, with the lowest concentrations in breast milk.

Because of its ubiquitous nature, aluminium has not traditionally been regarded as an essential trace element.[1] No cases of aluminium deficiency have been reported and minimum daily requirements for different ages are unknown. Fluids yielding as little as 7·5–15 μg of aluminium per day have been used recently without detriment, however, in infants receiving long term total parenteral nutrition.[2]

Aluminium toxicity in patients with chronic renal insufficiency undergoing haemodialysis or ingesting large quantities of phosphate binding gels is well recognised.[3] Recently, the high aluminium content of a proprietary milk formula was implicated as the cause of aluminium toxicity in two infants with neonatal uraemia.[4] In this paper we report the results of the measurement of aluminium in a variety of infant feeds.

Methods

Sample collection and preparation. Preprepared liquid and powdered formulas were sampled either directly from their glass containers or, in the case of preparations in cans, by collecting aliquots into acid washed polystyrene tubes. Each feed was sampled on two occasions and, where possible, from different batches. Breast milk was collected by lactating mothers directly into acid washed polystyrene tubes, using a no touch technique.

The powders were reconstituted in the laboratory by adding distilled, double deionised (aluminium free) water, using acid washed volumetric apparatus, to portions of accurately weighed milk powder.

Aluminium analysis. Aluminium was assayed by graphite furnace atomic absorption spectrometry employing a Varian Techtron AA975 and GTA 95 with autosampler.

All samples were initially diluted in the ratio of 1 volume of sample to 14·2 volumes of 0·3% analytical grade hydrochloric acid on an automatic diluter.

The aluminium content of samples was quantitated by the method of standard additions to allow for the variation in instrument response caused by the different sample matrices. Typically, a single random representative sample was used to generate a standard additions calibration curve for each analytical run. Subsequent samples in the run were quantitated by comparison with this curve. Samples yielding a mean peak height absorbance greater than the highest calibration point were further diluted with 0·3% hydrochloric acid until they fell within the calibration range.

The analytical technique was controlled with 'in house' aqueous and serum based control materials (method coefficient of variation at 80 μg/l was roughly 10%, run to run) and by participation in a national aluminium analysis quality control survey (Department of Applied Biology, Royal Melbourne Institute of Technology).

The spectrometer was operated in peak height

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Table *Comparative aluminium content of infant feeds*

Sample	*Type of feed†*		*Aluminium content (μg/litre of feed)‡*	*Country of origin*
	Powder (μg/g)	*Liquid (μg/ml)*		
Breast milk		0·03	30	
5% glucose		0·04	35	
Tap water (four readings)		0·04	40	
Glucose-electrolyte mixture		0·04	40	
'Locasol'	0·23		85	Holland
Pasteurised cow's milk		0·09	95	Australia
'De-Lact Infant'*	0·50		105	Australia
'Lactogen'		0·17	105	Australia
'S26'*		0·14	125	United States
'Digestelact'*	1·01		165	Australia
'Similac'		0·20	200	Australia
'Alfare'	1·85		315	Switzerland
'Enfalac Premature'	2·17		335	Canada
'Nan'*		0·34	345	Australia
'Portagen'*	6·25		935	United States
'Infasoy'*	9·68		1335	Australia
'Glucose Nutramigen'*	11·49		1725	Australia
'Pregestimil'	11·80		1780	United States
'Isomil'		3·74	1890	United States
'Prosobee'		10·02	5030	Australia

*Samples taken from different batches.
†Aluminium content in μg/g (powder) or μg/ml (liquid) of feed. Mean of two measurements taken on separate occasions.
‡The calculated aluminium content per litre of feed diluted according to the manufacturers' recommendations (to 20 kcal/30 ml) with water assumed to contain 40 μg/litre of aluminium (using the measurements in column 1).

mode with no background correction required. Pyrolytic furnace tubes were used.

Contamination was controlled by ensuring that the peak height absorbance of a blank firing of 0·3% hydrochloric acid did not exceed 0·02 units relative to an air firing yielding a visually flat baseline.

Results

The results are shown in the Table. Fresh breast milk, 5% glucose, and a glucose-electrolyte mixture (both prepared in the formula room of our hospital) were found to have similarly low concentrations of aluminium comparable with known hospital tap water concentration.

Among the milk feeds, the mean concentration of aluminium ranged from 0·09 to 10·02 μg/ml for liquid feeds and 0·23 to 11·80 μg/g for powdered feeds. Compared with a litre of breast milk there was an increase of up to 165-fold in aluminium content per litre of reconstituted feed. Depending on the formula used, infants drinking a litre of milk daily would be exposed to between 30 and 5000 μg of aluminium per day.

Batch to batch variation and country of origin of the formulas did not seem to influence our results.

Discussion

Aluminium intoxication is now recognised as the cause of a progressive encephalopathy, vitamin D resistant osteomalacia, and one form of anaemia in the presence of chronic renal insufficiency.[3] Although these manifestations of toxicity were initially recognised in adults with uraemia undergoing haemodialysis, they were subsequently described in children who were receiving large doses of aluminium containing phosphate binding agents but who did not yet require dialysis.[5]

Less severe forms of neurological and intellectual dysfunction in infants developing chronic renal failure in the first year of life are well known. In fact, in one major study 20 of 23 such children were found to have developmental delay, microcephaly, hypotonia, dyskinesia, seizures, and electroencephalographic abnormalities on subsequent follow up.[6] Poor nutrition, raised serum parathyroid hormone concentration, subsceptibility of the infant brain to the uraemic environment, and aluminium toxicity were suggested as causes;[6] the possibility that the aluminium was derived from infant formulas was not considered.

More recently, cerebral aluminium accumulation has been documented in two infants with neonatal uraemia who developed a progressive and fatal encephalopathy in the absence of phosphate binding gels or dialysis with water appreciably contaminated with aluminium.[4] Both infants were fed with a proprietary milk formula that was found to contain high concentrations of aluminium relative to breast milk.

Our results confirm that infants may be exposed

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to appreciable amounts of aluminium from a variety of proprietary milk formulas, some of which contain more than 15 times the concentration of aluminium already implicated in cerebral toxicity.[4] This source of exogenous aluminium may contribute to other recognised forms of neurological and intellectual dysfunction in infants with chronic renal insufficiency. The effects of exposure to large quantities of ingested aluminium in infants with lesser degrees of renal impairment or normal renal function remain to be documented.

By comparison with phosphate binding gels, the total aluminium content of infant milk formulas is only modest. In the absence of other commonly recognised risk factors cerebral toxicity may therefore reflect a greater bioavailability of aluminium in milk formulas or an increased uptake of aluminium by the immature brain.

The authors gratefully acknowledge the cooperation of Sister Barton-Bishop for her help with the feeds and Mrs L Beer for the preparation of the manuscript.

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Received 29 April 1986

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Arch Dis Child 1986 61: 914-916
doi: 10.1136/adc.61.9.914

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