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Tissue Mineral Levels in Victims of Sudden Infant Death Syndrome II. Essential Minerals: Copper, Zinc, Calcium, and Magnesium[45]

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Summary

Deficiencies of various vitamin and minerals *per se* have been suggested as possible causes of sudden infant death syndrome (SIDS). Further, a deficiency of essential minerals may lead to enhanced toxicity of toxic elements, in particular, lead and cadmium. To explore the possibility of mineral deficiencies or interactions with the toxic metals, lead and cadmium, lung, liver, kidney, and rib specimens were obtained at autopsy from 66 SIDS infants and 23 infants who died suddenly from other causes. Tissue copper, zinc, calcium, and magnesium were measured by atomic absorption spectroscopy. No differences were found between SIDS and non-SIDS for any element in any tissue except for more magnesium in the liver ($P < 0.0001$) and less copper in the lungs ($P < 0.02$) in the SIDS group. Only sporadic interactions between toxic and essential elements could be found. We found no evidence of any essential mineral deficiencies *per se* or significant interactions of essential and toxic minerals that might potentiate the effects of toxic metals. The physiologic significance, if any, of the higher liver magnesium and lower lung copper found in SIDS is unclear.

Abbreviations

AAS, atomic absorption spectrophotometry
CaBP, calcium-binding protein
SIDS, sudden infant death syndrome

Because of their wide ranging effects, deficiencies of various vitamins and minerals have been suggested as a possible cause of SIDS including biotin (22), thiamine (11, 37), vitamin D (21) vitamin E and/or selenium (29, 38), and magnesium (5). Lapin, *et al.* (25) measured selenium, magnesium, copper, and zinc concentrations in liver samples from 13 SIDS cases ranging from 1–10 months of age and 14 non-SIDS cases, age 5 days to 13 years. They found no significant differences between the two groups; however, because different deficiencies are manifest in different tissues and because some tissue concentrations vary with age, mineral deficiencies were not totally excluded.

Mineral interactions are known to occur both among the essential elements and between toxic and essential elements (20, 35, 42). Copper and zinc are antagonistic to each other (10), while normal calcium metabolism, in some instances, depends on the presence of normal amounts of magnesium (1). A deficiency of copper or zinc will lead to enhanced toxicity of lead or cadmium whereas an excessive dietary intake of the essential minerals will be protective (9, 23, 35). Tissue lead concentrations are greatly elevated when dietary calcium is low (27, 28, 36). Magnesium supplementation has been shown to effect increased excretion of lead in rats (41).

The determination of copper, zinc, calcium, and magnesium concentrations in tissues of SIDS victims was undertaken to see if any relationships existed between these elements and the cadmium and lead levels previously reported (14) and at the same time with a larger number of cases and with appropriately age-matched controls to further examine the possibility of the presence of deficiencies or excesses of these elements in multiple tissues.

MATERIALS AND METHODS

Fresh lung, liver, right kidney, and right 5th rib specimens were obtained from infants autopsied at the St. Louis City and County Medical Examiners' Offices. The ages at death ranged from 4–26 wk. There were 66 SIDS and 23 non-SIDS infants. Each tissue was packaged separately in plastic bags and stored at −20°C until the analyses were run. The tissues were processed as previously described. (14). The tissue acid digests were analyzed for copper using electrothermal AAS and for zinc, calcium, and magnesium using air-acetylene flame AAS. A Varian Model 1200 AAS was used for rib zinc measurements and a Perkin-Elmer Model 403 equipped with an HGA 2200 graphite furnace was used for all other mineral measurements. Deuterium background correction was used for copper and zinc. Lanthanum was used to suppress

phosphate interference in the calcium analyses. National Bureau of Standards SRM 1577 bovine liver was used as a tissue reference. The results are reported in micrograms per gram of dried tissue.

Log transformations of the raw data were used to calculate Pearson's correlation coefficients (r). Significance levels (P) were taken from a table (12) using r values and degrees of freedom. To avoid the possibility of errors resulting from changes with age in mineral concentrations, all values were corrected to the age of 13 wk. The age-corrected values in the two groups then were compared. Tissue mineral levels that were more than four standard deviations from the mean were eliminated from the subsequent calculations.

Table 1. *Tissue mineral concentrations*[1]

Mineral/ tissue	SIDS[2]		non-SIDS[2]	
	n	Mean ± S.D.	n	Mean ± S.D.
Copper				
Lung	66	5.84[3] ± 1.55	23	6.94 ± 2.18
Liver	66	146.9 ± 107.0	23	125.0 ± 94.6
Kidney[4]	66	10.36 ± 1.61	23	10.53 ± 1.67
Rib	57	0.913 ± 0.341	21	0.941 ± 0.241
Zinc				
Lung	66	66.4 ± 12.2	23	69.8 ± 10.0
Liver	66	155.8 ± 97.1	23	138.6 ± 56.4
Kidney[4]	66	101.7 ± 15.8	22	104.0 ± 11.1
Rib	57	109.6 ± 21.9	21	105.3 ± 20.9
Calcium				
Lung	66	555.7 ± 176.9	23	541.0 ± 131.7
Liver	66	237.8 ± 101.3	20	212.0 ± 63.9
Kidney[4]	66	405.9 ± 84.9	23	447.0 ± 140.9
Rib[1]	57	202.6 ± 18.6	21	206.9 ± 15.6
Magnesium				
Lung	66	555.0 ± 61.2	23	569.3 ± 45.6
Liver	66	601.6[3] ± 53.6	23	543.1 ± 87.5
Kidney[4]	66	749.7 ± 80.8	23	758.8 ± 95.3
Rib	56	3856 ± 254	21	3870 ± 198

[1] Mineral concentrations are expressed in μg/g dry weight except rib calcium which is expressed in mg/g dry weight.
[2] Mean SIDS age = 11.9 wk, mean non-SIDS age = 13.4 wk.
[3] Significant diffference, lung Cu $P < 0.02$, liver Mg $P < 0.0001$.
[4] Cortex.

RESULTS

The mean and standard deviation of the mineral concentrations for each of the four tissues are shown in Table 1. No differences were found except for significantly more magnesium in the liver ($P < 0.0001$) and less copper in the lungs ($P < 0.02$) in the SIDS group than was found in the non-SIDS group. Age correction did not produce any significant changes in mean ± S.D. mineral concentrations and thus only non-age-corrected means are shown in the Table.

The only mineral concentration changes with age: a decrease in liver copper in both the SIDS and non-SIDS ($P < 0.0001$), a decrease in liver calcium in SIDS ($P < 0.0005$) but not in non-SIDS, a decrease in lung magnesium ($P < 0.0005$), lung zinc ($P < 0.05$), and rib zinc ($P < 0.005$) in non-SIDS but not in SIDS. Only the slopes of liver calcium *versus* age were different between SIDS and non-SIDS ($P < 0.01$). The intercept for lung magnesium was lower ($P < 0.01$) in SIDS and the intercepts for liver magnesium ($P < 0.05$) and liver calcium ($P < 0.01$) were higher in SIDS.

Correlation between essential elements and rib or same tissue lead and kidney or same tissue cadmium (14) can be seen in Table 2. There was no significant differences in mineral levels between black and white infants or between males and females.

DISCUSSION

A search of the literature has revealed no previous studies of tissue minerals in infants as a distinct group. In some studies infants were grouped with older children and even with adults; however, our findings are all within expected ranges.

Copper. Copper is an element essential for bone growth and maintenance, for hematopoiesis and as a component of several key metalloenzymes including lysyl oxidase, tyrosinase, superox-

Table 2. *Tissue mineral correlations (r)*

	Lead				Cadmium			
	SIDS		non-SIDS		SIDS		non-SIDS	
	Rib	Same tissue	Rib	Same tissue	Kidney	Same tissue	Kidney	Same tissue
Copper								
Lung	0.1054	0.0804	−0.2223	−0.1447	0.1040	0.2419	0.3954	0.5220[1]
Liver	−0.2406	−0.2371	−0.3063	−0.3084	−0.4537[3]	−0.0543	−0.4291[1]	−0.0520
Kidney	−0.2344	−0.0120	−0.3081	0.0511	−0.0452	0.0452	0.0543	0.0543
Rib	−0.0580	−0.0580	0.2517	0.2517	−0.3604[2]	0.1010	−0.4843[1]	−0.0563
Zinc								
Lung	−0.0019	−0.2322	0.0028	−0.3369	−0.2200	−0.0698	−0.2646	−0.1999
Liver	−0.0260	0.2066	−0.1216	0.3366	0.0246	−0.0073	0.0531	−0.0754
Kidney	0.0272	0.1461	0.0751	−0.0850	−0.0794	−0.0794	−0.3264	−0.3264
Rib	0.0124	0.0124	0.1902	0.1902	−0.2865[1]	0.0451	−0.4569[1]	0.1677
Calcium								
Lung	−0.0993	0.1629	0.1255	0.0561	−0.0182	−0.0359	−0.1447	0.0584
Liver	−0.0914	−0.2036	−0.2032	−0.0353	−0.4009[3]	−0.1830	0.2442	0.2918
Kidney	0.0337	−0.3023	−0.1046	−0.2994	−0.1527	−0.1527	−0.3552	−0.3552
Rib	−0.0731	−0.0731	−0.1304	−0.1304	0.2946[1]	−0.0372	0.2042	0.0829
Magnesium								
Lung	0.0361	−0.3219[2]	−0.3004	−0.4742[1]	−0.1706	−0.0877	−0.4937[1]	−0.0277
Liver	0.1169	−0.0079	−0.3599	0.2536	0.0469	0.2509[1]	0.2321	0.0476[1]
Kidney	−0.0150	0.0281	−0.2569	−0.2159	−0.2538[1]	−0.2538[1]	−0.0745	−0.0745
Rib	−0.0383	−0.0383	−0.3366	−0.3366	−0.0365	−0.0564	−0.0406	0.0149

[1] $P < 0.05$.
[2] $P < 0.01$.
[3] $P < 0.001$.

ide dismutase, ceruloplasmin, uricase, dopamine β-hydroxylase, cytochrome oxidase, diamine oxidase, and histaminase. The average daily copper requirement for infants is 80 μg/kg (32). Sudden death has been reported in copper-deficient cattle (43). Anemia, neutropenia, and impaired bone mineralization have been reported in humans (18).

Cadmium is known to be a metabolic antagonist of copper (42). Rat studies have shown that relatively low levels of dietary cadmium supplementation along with a copper-sufficient diet caused significant reductions in plasma ceruloplasmin activities and kidney copper concentrations. Higher cadmium intake caused marked reduction in liver copper reserves. These effects were reversed by increasing dietary copper (4, 8). Lead also interferes with copper metabolism (23, 30, 31, 33, 39). When rabbits were given a single intraperitoneal injection of tetraethyllead, 100 mg/kg, brain copper was reduced by about 40% within 24 h (33). Murthy, *et al.* (30) found that there were additive or greater effects of the combination of cadmium and lead on copper metabolism. In both SIDS and non-SIDS kidney cadmium was inversely correlated with liver and rib copper. No relationship to lead was seen.

The present study found no significant differences in liver, kidney, and rib copper concentrations between the two groups thus ruling out a systemic copper deficiency. The physiologic significance, if any, of the lower lung copper found in the SIDS group is not known although potential relationships with superoxide dismutase could stimulate speculation.

Zinc. Zinc is another essential trace element. More than 70 zinc proteins have been described and most of these are enzymes. The average daily zinc requirement for infants up to 6 months of age is 3 mg (32). Among the features of zinc deficiency in infants are hypogeusia, anorexia, growth retardation, and failure to thrive.

Several investigators have demonstrated zinc:lead (9, 13) and zinc:cadmium (10, 15, 16, 34) interactions and have shown that diets deficient in zinc result in increased absorption and retention of lead and cadmium. An increased intake of zinc reduced or eliminated the toxic effects of the two metals.

None of the four tissues showed a significant difference in zinc concentration between the SIDS and non-SIDS groups. In both SIDS and non-SIDS kidney cadmium was inversely correlated with rib zinc. No relationship to lead was seen.

Calcium. About 99% of the total body calcium is in the skeleton. The remaining 1% performs a variety of vital functions relating to cellular adhesiveness, transmission of nerve impulses, neuromuscular excitability, maintenance, and function of cell membranes, blood coagulation, and activation of enzyme reactions and hormone secretion (2).

Animal studies have shown that dietary calcium interferes with the absorption of cadmium (17, 24, 26). A low calcium diet results in increased absorption of cadmium. The same effect has been found on the absorption of lead (3, 27, 28). In a study of *in vivo* binding of lead and calcium by duodenal mucosal proteins, Barton, *et al.* (3) found that both cations bound not only to the vitamin D-induced CaBP but also to another protein of higher molecular weight. CaBP had a greater affinity for calcium whereas the other protein had a greater affinity for lead. They concluded that both vitamin D-dependent CaBP and the higher molecular weight protein are of physiologic significance in lead absorption and that calcium inhibits absorption, at least in part, by successfully competing for binding sites on the two proteins.

No correlation between tissue calcium and tissue lead was seen in this study. Kidney cadmium was inversely correlated with liver calcium in the SIDS but not the non-SIDS group; however, the significance of this is unclear. At death the tissue calcium concentrations are not significantly different in the two groups. Liver calcium decreased significantly with age in the SIDS group ($P < 0.005$) but not in the non-SIDS group and these two regression lines were significantly different. If one assumes the intercept value to be the mean concentration at birth, then the SIDS group had significantly (about 50%) more liver calcium at birth than the non-SIDS group. The physiologic significance, if any, of this finding is unknown, however the lung magnesium intercept was also significantly higher and the calculated liver lead at birth also was suggestively higher in SIDS (14). This might be consistent with an increased maternal bone turnover resulting in an increased *in utero* lead exposure.

Magnesium. Magnesium is an activator for a number of enzyme systems that are responsible for optimal cellular metabolism. A major group of enzymes are those that hydrolyze and transfer phosphate groups including the phosphatases and the enzymes that participate in reactions involving ATP. Because ATP is required for protein, fat, nucleic acid and coenzyme synthesis, muscle contraction, glucose utilization, methyl group transfer and acetate, formate, and sulfate activation, magnesium also is required (44). Its involvement in protein synthesis is through its action of ribosomal aggregation, in binding messenger RNA to 70S ribosomes, and in the synthesis and degradation of DNA. The recommended daily allowance of magnesium for infants is 50–70 mg (32). Magnesium deficiency is manifested by hyperirritability, neuromuscular dysfunction, and psychotic behavior (40, 44). Tetany may occur in the absence of hypocalcemia or acid-base changes. Cardiovascular and renal disease and hypertension may also be associated with hypomagnesemia.

In a study of the effect of magnesium on the absorption and excretion of lead, Singh *et al.* (41) found that magnesium administration to lead-fed rats increased blood lead levels and urinary excretion of lead. Bone and brain lead levels were significantly lower in the magnesium-fed group. The authors conclude that magnesium intake decreased the deposition of lead in bone and helped mobilize lead from bone, thus increasing the blood level and subsequent urinary excretion.

Magnesium deprivation has been proposed as the cause of sudden infant death syndrome (5). A study of magnesium deficient rats indicated that bone was the most reliable tissue for determining magnesium status (7). In a study of 64 infants from 1–6 months of age, using a magnesium load test, 38 infants retained more than 60% of the magnesium. Seven infants of 8.2 ± 1 wk who had had one or more sudden transient episodes that included the following apnea, gasping, tonic cyanosis, sweating, eye signs or tearing were also studied. Despite preload magnesium treatment in two infants, the group retained 88% of the load (6). These investigators stated that the signs are nonspecific but resemble premonitory signs and the type of episode that may occur in sudden infant death syndrome, suggesting a possible link between magnesium depletion and SIDS.

The present study provides no support for a theory of magnesium deficiency in SIDS because there were no significant differences in lung, kidney, or rib magnesium concentrations between SIDS and non-SIDS groups. Indeed, the liver magnesium was higher in the SIDS group ($P < 0.0001$) but the physiologic significance of this is unknown. In both SIDS and non-SIDS, lung lead and magnesium were inversely correlated and liver cadmium and magnesium were directly correlated. Again, however, no clear pattern of interaction or specificity to SIDS was seen.

Previous reports of conditions accompanying deficiencies of these minerals had suggested the possibility of a relationship with SIDS. Other reports have shown that deficiencies of these minerals would increase absorption and retention of the toxic elements, lead and cadmium. We have found no evidence of a deficiency of any of these essential minerals nor were any of these minerals present at excessively high levels. No consistent interactions of any of the elements with lead or cadmium were found. The physiologic significance, if any, of the isolated interrelationships between a toxic and an essential mineral is osbscure. To assign any significance to the lower lung copper or elevated liver magnesium in SIDS will require further study.

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