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EFFECT OF DIETARY ZINC ON THE ABSORPTION OF ORALLY ADMINISTERED Zn^{65}*

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Abstract—An attempt to modify the absorption of oral Zn^{65} in rats was made by the addition of natural zinc acetate to the diet of male Sprague-Dawley rats. After the rats were maintained on a zinc-supplemented diet for 4 weeks, an oral dose of $Zn^{65}Cl$ was administered to the rats. The retention of Zn^{65} was determined by whole-body assay techniques. Differences in per cent Zn^{65} retained were attributed to differences in the zinc acetate level in the diet.

Data from 1 human subject suggest that intestinal absorption may be decreased by increasing dietary zinc levels.

INTRODUCTION

WHEN carrier-free $Zn^{65}Cl$ was administered to four human subjects in tracer amounts (0.6–1.0 μc), that portion of the dose eliminated with a biological half-time of more than 400 days was between 25 and 70 per cent (Fig. 1). BALLOU[1] reported that a thousandfold variation in the specific activity of an orally administered dose of $Zn^{65}Cl$ did *not* influence the amount of Zn^{65} absorbed by rats. MAGER *et al.*[2] state that "saturation" of the gut, with reference to zinc, may influence the absorption of orally administered zinc. The following experiment was designed to test the effect of increased dietary concentrations of zinc on the absorption of a single orally administered dose of Zn^{65}.

METHODS

Male Sprague–Dawley rats about 70 days old (average weight 280 g) were randomly assigned to six groups of nine rats each. They were allowed free access to water and pulverized Purina Laboratory Chow for a period of 2 weeks to permit the animals to become accustomed to the pulverized food. At the end of the 2-week period, powdered zinc acetate was added to the food in quantities sufficient to make the concentration of zinc 58, 117, 175, 293, 410 and 644 p.p.m. The Ralston–Purina Company gives an assay of 58 p.p.m. of zinc in Purina Lab Chow, so that the increments of zinc added were 0, 1, 2, 4, 6 and 10 times the normal concentration. Twenty-eight days after the initiation of the zinc-supplemented dietary regimen, 1.2 μc of $Zn^{62}Cl$ was administered by gastric gavage while the animals were under light ether anaesthesia. The animals were not fasted. Whole-body activity was determined at 30 min and at 1, 2, 4, 7 and 11 days after injection in the Los Alamos Small Animal Counter (LASAC-III). A diagrammatic cross-section showing the relation of an animal to detector and shielding is shown in Fig. 2. Two of the seven photomultiplier tubes are shown. Except for the 3-in. lead door, the detector assembly is surrounded by 7¼-in. steel armor plate weighing approximately 3 tons. The photomultiplier tubes are individually balanced for pulse-height output and their signals are connected in parallel. The output of the photomultipliers is fed into standard transistorized circuitry consisting of preamplifier, 5-step amplifier and a single-channel analyzer of the threshold-plus-slit type. The information is presented on a scaler which is controlled by a preset timer. Additional information on this counting system may be found in Ref. 3. Periodic weighings of the animals showed that diets supplemented with zinc had no effect on weight or growth.

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Table 1. Effect of dietary zinc on the retention of orally administered Zn^{65} *in rats*

Group No.	Stable zinc in diet (p.p.m.)	Day				
		1	2	4	7	11
1	58	20.22 ± 3.30	14.86 ± 2.44	12.73 ± 2.17	10.74 ± 1.90	9.22 ± 1.70
2	117	15.86 ± 3.50	10.78 ± 2.28	9.04 ± 1.90	7.63 ± 1.57	6.47 ± 1.28
3	175	16.68 ± 6.00	9.52 ± 1.21	7.80 ± 1.09	6.57 ± 0.88	5.56 ± 0.78
4	293	14.26 ± 7.76	9.36 ± 6.69	7.77 ± 5.67	6.44 ± 4.71	5.42 ± 3.85
5	410	12.53 ± 3.97	5.96 ± 1.64	4.73 ± 1.17	3.95 ± 0.92	3.30 ± 0.79
6	644	13.15 ± 10.98	4.69 ± 2.55	3.45 ± 1.59	2.88 ± 1.23	2.32 ± 0.97

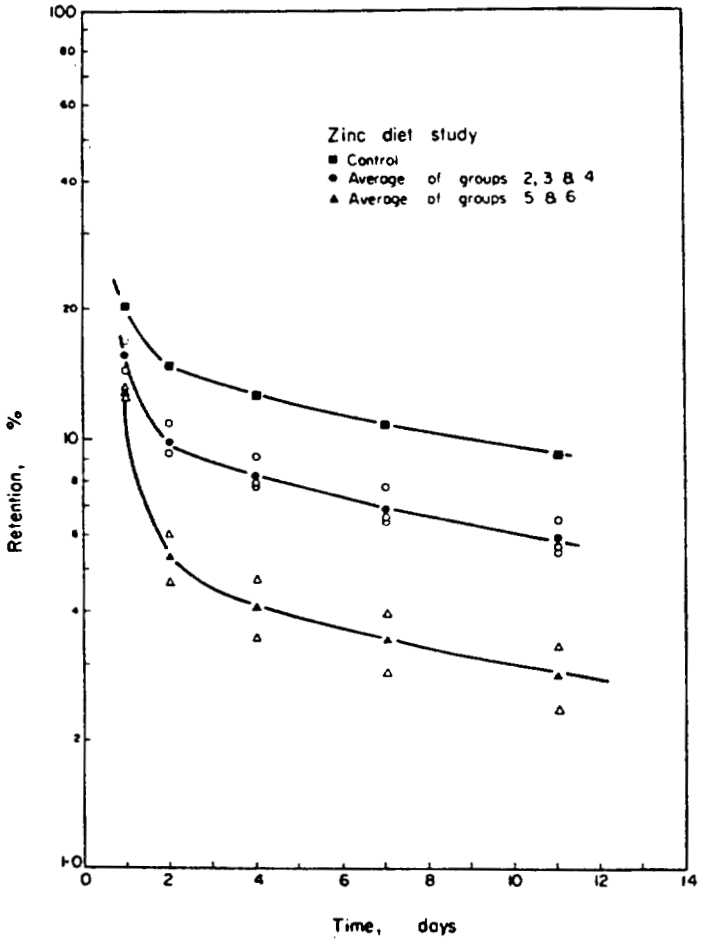

FIG. 3. Retention of Zn^{65} in rats maintained on differing concentrations of stable zinc in the diet.

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Inspection of Table 1 and Figs. 3 and 4 shows that dietary zinc levels are effective in altering the absorption of zinc by the gut or, perhaps, in altering mechanisms affecting early elimination of zinc. On day 1, differences in the amount of zinc retained by the various groups are not as great as at later times, and the coefficient of variation ($\sigma/\bar{X}$) is large, perhaps as a result of differences in gut passage time. On day 2 and thereafter, the differences are greater and maintain a constant ratio, as shown by the parallel lines in Figs. 3 and 4. Thus, it can be seen that although different amounts are excreted from the "compartment" with the longest half-time, they are all excreted at approximately the same rate (i.e. the dietary intake does not affect the rate of excretion of this compartment). The same observation applies to the human data (Fig. 1).

It can be seen in Fig. 4, by extrapolation from normal zinc concentration (1, on the abscissa) toward 0, that zinc-deficient diets result in increased retention of ingested Zn^{65}. Experiments conducted at this Laboratory show that rats maintained on diets deficient in sodium and potassium lose these elements less rapidly than rats maintained on "normal" diets. These results were sufficiently encouraging to lead to a test on a human subject. Zn^{65} retention, in the same subject, was measured on two occasions. The retention pattern after the first ingestion of Zn^{65} is given by the uppermost line in Figs. 1 and 5. The retention pattern after the second ingestion of Zn^{65} is given by the large open circles in Fig. 5. The second ingestion took place 700 days after the first, when the residual Zn^{65} activity was less than the natural body activity (K^{40}). The second ingestion of Zn^{65} was preceded by a period of 30 days during which 10 mg of zinc (as zinc acetate) was added to the daily diet of this individual. The estimated daily consumption (typical North American diet) is 12 mg, according to EGGLETON[5]. Fig. 5 indicates that, at 10 days, excretion has been increased by more than a factor of 3. Radioassay of fecal samples showed that, after the first ingestion, 11 per cent of the Zn^{65} was excreted during the first 4 days post ingestion and that, after the second ingestion, 44 per cent was excreted during the first 3 days post ingestion. While it is possible that equal fractions of the ingested dose were absorbed on each occasion and that the difference in the amount retained at 5–10 days was due to an accelerated excretion *rate* through the pancreas, it seems more reasonable to attribute the difference to an inhibited absorption because rats showed some differences in the amount retained on the first day post ingestion and because the *rate* of the long component of the retention function was unchanged by differences in dietary zinc concentration. Moreover, McCANCE and WIDDOWSON[6] showed that

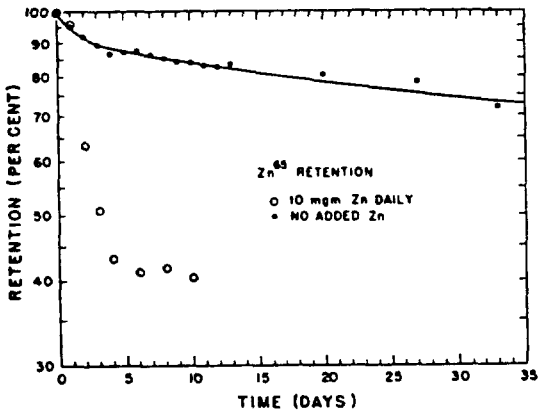

FIG. 5. Retention of Zn^{65} in the same human subject with and without the addition of stable zinc to the diet.

increased dietary intake of zinc in humans resulted in increased fecal excretion but that injected zinc was eliminated more slowly.

SUMMARY

(1) The per cent of orally administered Zn^{65} excreted with a biological half-time greater than 400 days in humans varied by a factor of more than 2.

(2) Rats maintained on diets supplemented with zinc acetate retained less Zn^{65} than rats on normal diets. Addition of 6–10 times the normal zinc intake reduced retention by a factor of 3.

(3) Data from one human suggest that dietary zinc is a factor regulating the amount of zinc retained in humans.

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