Lead and Zinc Poisoning and the Interaction Between Pb and Zn Poisoning in the Foal

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ABSTRACT

Groups of young growing horses were fed toxic amounts of lead only, zinc only and the same amounts of lead and zinc together. Those fed Pb only developed pharyngeal and laryngeal paralysis ("roaring") whereas those fed Zn only and Pb and Zn together developed the same clinical syndrome which included swelling at the epiphyseal region of the long bones, stiffness and lameness. Anemia and decreased weight gains were most pronounced in animals fed Zn for the longest periods. Animals fed Pb only did not become anemic and weight loss did not occur until after there was an interference in swallowing.

The clinical signs and tissue Pb values from animals fed toxic amounts of both Pb and Zn continuously, differed markedly from those present in animals fed comparable amounts of Pb only. Th clinical signs were similar to those caused by Zn poisoning. The hepatic and renal tissue Pb values were approximately twice as high and the epiphyseal and cancellous bone sample results were one half as high as the comparable Pb values from animals fed toxic amounts of Pb only.

It appeared that toxic amounts of Zn prevented the development of clinical signs of Pb poisoning in the young growing horse.

RÉSUMÉ

On a donné à des groupes de poulains des doses toxiques de plomb (Pb) et de zinc (Zn), séparément et simultanément. Les sujets qui ne reçurent que du Pb développèrent une paralysie du pharynx et du larynx (cornage). Par ailleurs, ceux à qui on n'avait donné que du Zn, ou un mélange de Pb et de Zn, développèrent un syndrome identique incluant des tuméfactions dans la région épiphysaire des os longs, de la raideur et de la boiterie. L'anémie et la diminution du gain de poids étaient le plus marquées chez les sujets ayant reçu du Zn le plus longtemps. Les poulains à qui on n'avait donné que du Pb ne devinrent pas anémiques et ne subirent pas de perte de poids, jusqu'à ce qu'ils éprouvent de la difficulté à avaler.

Les signes cliniques et la teneur des tissus en Pb des poulains auxquels on avait continuellement donné des doses toxiques de Pb et de Zn différaient beaucoup de ce qu'on observa chez les sujets n'ayant reçu que des doses comparables de Pb. Les signes cliniques manifestés par ces poulains ressemblaient à ceux que provoqua l'empoisonnement par le Zn. La teneur en Pb du foie et des reins était environ deux fois plus élevée tandis que celle des os spongieux et de la région épiphysaire des os longs n'atteignait qu'environ la moitié des concentrations comparables de Pb recouvrées chez les sujets n'ayant reçu que des doses toxiques de ce métal.

Il semble que les quantités toxiques de Zn empêchèrent l'apparition des signes cliniques de l'empoisonnement par le Pb, chez ces jeunes poulains.

INTRODUCTION

Lead and zinc coexist in many ores and manufactured goods. For this reason, the two elements could both be present in toxic amounts in one environment and animals reared in such environments could be exposed simultaneously to toxic amounts of both elements.

The body burden of Pb has been shown to increase when the intake of Ca, P and vitamin D are not optimal (22, 23, 24). There are well known nutritional interactions between Ca and Zn (25, 28). Zinc

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toxicity has produced lameness and arthritic-type changes in young growing swine (4, 11, 20). Considering the above, we reasoned that any element, nutrient, or biologically active material capable of altering the absorption and deposition of minerals in bone could significantly influence the metabolism of lead. An interaction between toxic amounts of Pb and Zn was thus predicted.

The present study was further prompted by reports of lameness and deaths in foals reared near lead-zinc smelters (13, 21). When adult horses were affected in these areas, they did not become lame but had the predominant clinical syndrome of lead poisoning for the horse; pharyngeal and laryngeal paralysis. Lameness and "arthritis-like lesions" were described in two foals that had been raised next to a zinc smelter that was emitting large amounts of Zn but little Pb (10). An attempt to reproduce the disorder by feeding mares up to 0.057% Zn for as long as 400 days on the assumption that their nursing foals would develop the condition was unsuccessful (10).

The purpose of the present study was to produce zinc toxicity and lead toxicity in the young foal and to determine any interaction effect that might result from feeding toxic amounts of the two elements together.

MATERIALS AND METHODS

Ten pinto and standard-bred type fillies were weaned at two to three weeks of age. After weaning, the animals were fed a reconstituted calf milk replacer, horse pellets and a small amount of freshly cut grass. The feed was gradually changed over seven to water, horse pellets and a small amount of an alfalfa-grass hay mixture.

The animals were housed in three 10′ by 15′ box stalls and weighed at weekly intervals. They were tied twice daily while feeding, and water was available, on a free choice basis in a rubber trough in each pen. The animals were exercised twice daily, weather permitting, by being led and exercised on a lunge line in a grass paddock.

The animals were randomly assigned to treatment groups and to box stalls as shown in Table I. The experiments commenced seven days after the animals arrived, i.e. when they were three to four weeks of age.

TABLE I. The Assignment of Animals to Treatments and Box Stalls

	Animal Number	Treatment
Stall 1	4	Zn
	6	Pb
	14	Pb + Zn
Stall 2	2	Zn
	5	Pb
	7	Control
	9	Pb + Zn
Stall 3	1	Zn
	8	Control
	13	Pb + Zn

TABLE II. The Formulated Amounts and Chemical Analysis of the Experimental Feeds

	Formulated Amount Added Per Ton	Chemical Analysis in ppm	
		Pb	Zn
Control Basal Ration	—	4.0	180
Pb Containing Ration	1170.6 gm $PbCO_3$	800	170
Zn Containing Ration	6778.5 gm ZnO	18	5400

All animals received a basal ration[1]. Two batches of the basal ration had formulated amounts of Pb, as lead carbonate, and Zn as zinc oxide added prior to being pelleted. The formulated and determined amounts added are in Table II. Each animal was fed a total of 15.5 gms of pelleted feed per kg body weight daily. Those animals destined to receive Pb only, Zn only or both Pb and Zn received the amounts in Fig. 1. The intake of Pb and Zn was increased when signs of toxicity failed to appear by adding more treated pelleted feed and reducing the portion of untreated feed. After the 15th week of the experiment, it was necessary to place the Pb and Zn compounds onto the feed because more was required than was contained in the Pb and Zn treated feeds. Each animal received between one and three pounds of an alfalfa-grass hay mixture daily.

Clinical examinations were conducted daily. Blood samples were collected at weekly intervals and urine samples at irregular intervals. The determinations conducted thereon included: serum Ca and Mg (30), and whole blood Pb by atomic absorption

[1]"Frisky Foal Ration", Master Feeds Ltd., Toronto, Canada.

spectrophotometry (7); serum delta-amino-le-vulenic acid (ALA) by the modified method of Davis and Andelman (5) serum P by the Goldenberg and Fernandez method (8); serum iron and serum iron binding capacity by the Highland Ferro-Chek spectrophotometric method[2]; and the hemogram according to standard laboratory prodcedures.

As animals became ill, the syndrome was allowed to develop for varying periods before the animals were euthanized by the intravenous injection of a saturated barbiturate solution. At necropsy, sections of liver, kidney, metacarpal epiphysis and diaphysis, vertebrae, rib, skeletal muscle, brain and lung were removed and a sample of feces was collected at necropsy for Pb and Zn determination. The dithizone method was used for tissue and fecal Pb (29) and atomic absorption spectrophotometry for all Zn determinations (14).

[2]Hyland Division Travenol Laboratories, Inc., Costa Mesa, California, 92626, U.S.A.

RESULTS

In the first two weeks of the experiment some of the foals had intermittent diarrhea. During the fourth week, some had a mild upper respiratory infection that responded rapidly to antibacterial therapy. Ova of gastro-intestinal parasites were occasionally found in the feces, but were not found following appropriate anthelmintic therapy

The amount of Pb, Zn or both fed and the duration of feeding before severe illness developed are in Fig. 1. The order in which severe illness appeared is in Table III.

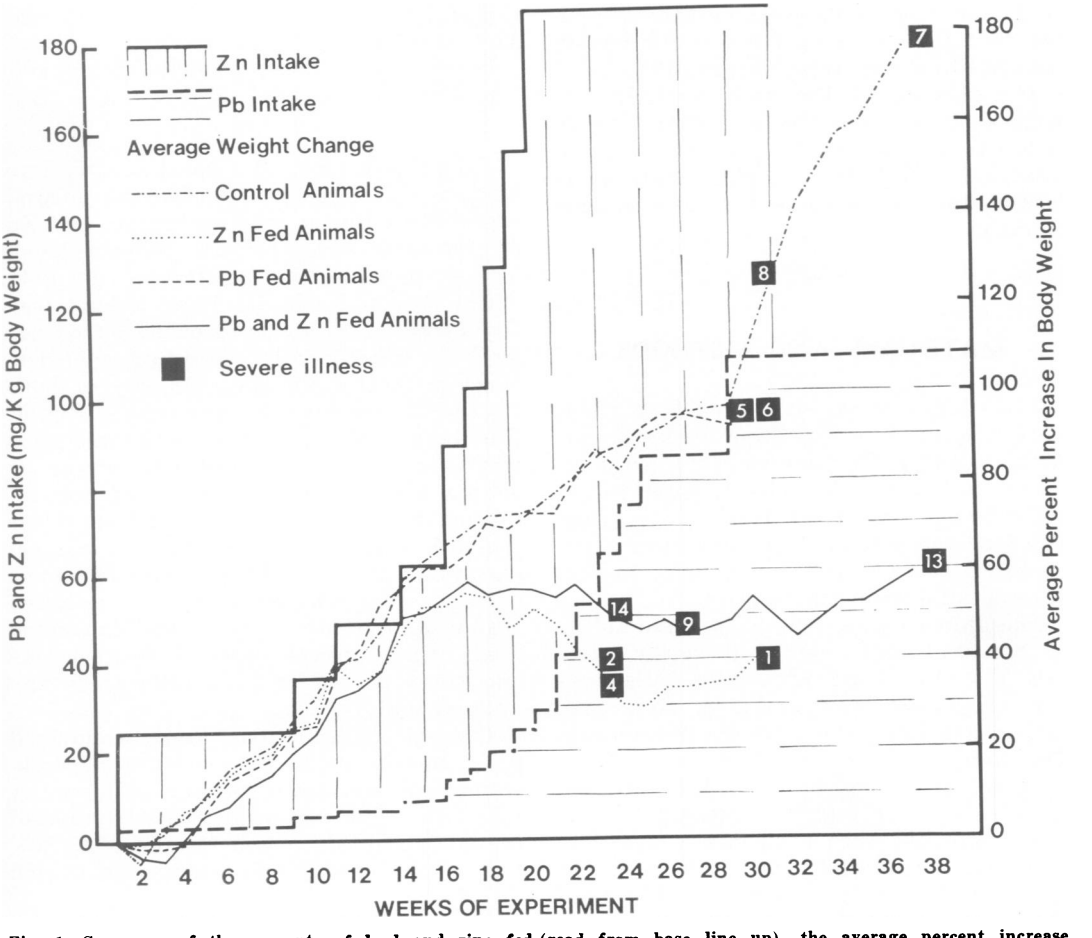

Fig. 1. Summary of the amounts of lead and zinc fed (read from base line up), the average percent increase in body weight of the foals in each group and the times when tissue samples were taken at necropsy.

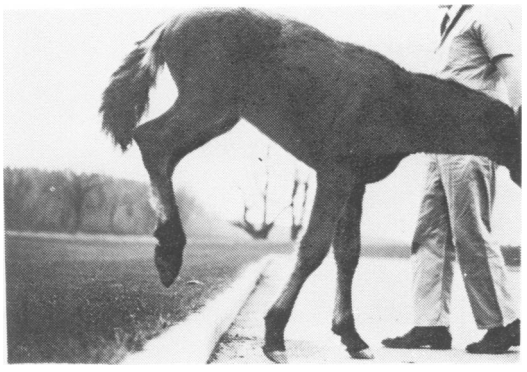

Fig. 2. The typical locomotion of foals with zinc toxicity while stepping over a curb (Animal Number 4).

The growth rates of all animals fed Zn decreased after the intake exceeded 90 mg Zn per kg body weight per day. The growth rates of the animals that received Pb only were similar to that of the control animals until signs of illness occurred in the Pb fed animals (Fig. 1).

The clinical syndromes that developed were uniform within groups and are described below, according to groups.

ZINC TOXICITY GROUP

The animals fed toxic amounts of zinc (Numbers 1, 2 and 4) developed a similar clinical syndrome.

The epiphyseal areas of the long bones were visibly enlarged (details to be published later) in these animals seven to 21 days before evidence of lameness or pain due to the enlargements could with certainty be identified.

The first evidence of pain was a tendency to lie in complete lateral recumbency and a reluctance to rise. When exercised, the animals moved with a stiff gait and were disinclined to curve the spine laterally when turned sharply to the right or left.

Three or four days after these signs became apparent, the animals would make three or more efforts to stand before succeeding. When exercised, the stiffness was more severe and the animals were reluctant to move faster than a walk. When the animals were required to step over a curb approximity eight inches high, they would stop in front of the curb, shift the body centre of gravity forward, step over the curb by alternately placing the fore limbs over the curb and then in one movement thrust the neck ventrad and move both hind limbs over the curb together (Fig. 2). Three to four days before necropsy, the stiffness had worsened and assistance was required to help the animals stand. The

TABLE III. Onset of Illness and Time of Death

Animal Number	Experimental Group	Time of Death (Weeks)	Duration of Illness Prior to Death
4	Zn only	23	10 days
2	Zn only	23	10 days
14	Pb and Zn	24	10 days
9	Pb and Zn	27	14 days
5	Pb only	30	6 weeks
6	Pb only	31	3 weeks
8	Control animal	31	Normal
1	Zn only	31	7 weeks
13	Pb and Zn	38	10 weeks

TABLE IV. Fecal Analysis of Lead and Zinc[a]

Treatment	Animal Number	Week of Euthanization	Pb Value	Zn Value
Pb Fed	5	30	4500	580
	6	31	4700	640
Pb and Zn Fed	14	24	2100	7600
	9	27	2090	3400
	13	38	2450	390
Zn Fed	2	23	160	7500
	4	23	N.D.	N.D.
	1	31	20	2300
Control	8	31	30	350

[a]Values in ppm, dry weight basis

feces were of normal consistency until the animals were recumbent, at which time they became more fluid in nature.

As the amount of joint fluid and the degree of pain produced by palpation of the fetlock, carpal, hock, elbow and stifle regions increased, the Zn fed animals had greater difficulty in rising (Fig. 3). Aspirated joint fluid samples contained slightly increased numbers of cells (macrophages containing engulfed red blood cells, free red blood cells and neutrophil leukocytes). Bacterial cultures were negative.

The first two animals that became ill (Numbers 4 and 2) retained a reasonably normal appetite until studies on them were terminated. The other animal (Number 1) was maintained for eight weeks on the same Zn intake (184 mg per kg body weight daily) as Numbers 4 and 2 were receiving when studies were terminated. Its appetite decreased over that period, bony changes developed more slowly than in the first two that became ill, and it became progressively anemic (Figs. 4 and 5).

Serum Ca, P and Mg values (Fig. 6) remained relatively constant and were within

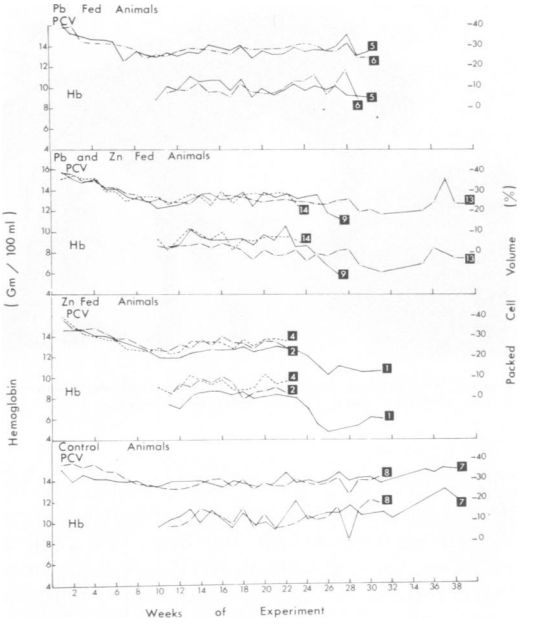

Fig. 4. The packed cell volumes and hemoglobin values, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

Fig. 3. Swelling at the hock and fetlock regions typical of foals with zinc toxicity (Animal Number 4).

normal limits as were the whole blood Pb (Fig. 7) and serum ALA values (Fig. 8). The whole blood Zn values increased after the daily Zn intake exceeded 60 mg per kg body weight (Fig. 9).

Lead Toxicity Group

A similar clinical syndrome developed in both animals (Numbers 5 and 6) that compared to "roaring" in adult horses with Pb poisoning.

The first abnormality noted was an increased guttural sound while swallowing pelleted feed (Week 25, Animal Number 5 and Week 28, Animal Number 6) or when the animals were startled ("roaring"). Over a period of three weeks (Weeks 25-28) Animal Number 5 consumed its pelleted feed but the feed could be palpated within the esophagus before the animal, through continued swallowing movements, was able to move the material to its stomach. During the next two weeks, (Weeks 29-30), the mastication movements while eating the pelleted feed changed from that typical of a horse eating coarse feed to those of a horse drinking a fluid. During the three week period prior to the termination of the study at Week 30, Animal Num-

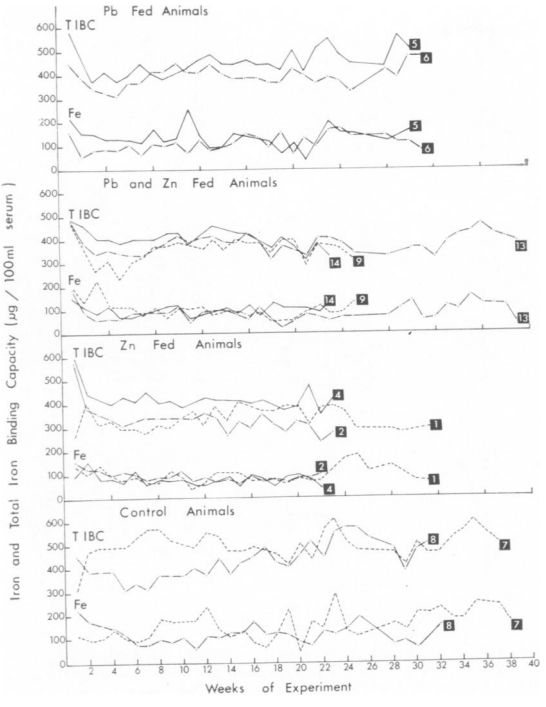

Fig. 5. Summary of the serum iron and the total serum iron binding capacity values, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

ber 5 salivated profusely while eating and ingesta was persistently noted at the nostrils. Two days prior to being euthanized, the respiratory rate increased to 50 per minute. On lung auscultation, crepitant sounds were evident over the base of the heart. The clinical syndrome in the other animal (Number 6) was similar in all respects except that it developed more rapidly (three weeks versus six weeks).

The clinical signs were allowed to progress to establish whether stiffness as reported (21) in young horses would occur. Both animals became slightly ataxic within three hours of euthanasia. At that time, the respiratory rates exceeded 60 per minute and one animal had commenced oral breathing.

There was no evidence of a "lead line" on the gums, lameness, blindness, swelling at the epiphyseal ends of the long bones, severe or progressive anemia (Figs. 4 and 5) or increase in the amount of joint fluid or the cells contained therein in either animal.

The increases in whole blood Pb values corresponded to the increased intake of Pb (Fig. 7) and clinical signs of Pb poisoning were not evident until the values had exceeded and remained above 60 µg per 100 ml blood for two to four weeks. The animal that developed the syndrome over the shortest period of time had higher blood Pb values before the onset of the signs.

There was no distinct pattern in serum ALA values (Fig. 8). Serum iron and iron binding capacity (Fig. 5), serum Ca, P and Mg values (Fig. 6) and whole blood Zn values (Fig. 9) were similar to values obtained from the control animals.

Combined Lead and Zinc Toxicity Group

The clinical syndrome that developed in these animals (Numbers 9, 13 and 14) was indistinguishable from that observed in the group with Zn toxicity.

The first animal (Number 14) to develop the syndrome, like the first which developed the Zn toxicity syndrome (Numbers 4 and 2), became stiff and lame (Fig. 10) but not anemic (Figs. 4 and 5). The others (Numbers 9 and 13) developed a progressively severe anemia that was similar in all respects to that of the animal (Number 1) that received Zn only for the longest period (Figs. 4 and 5).

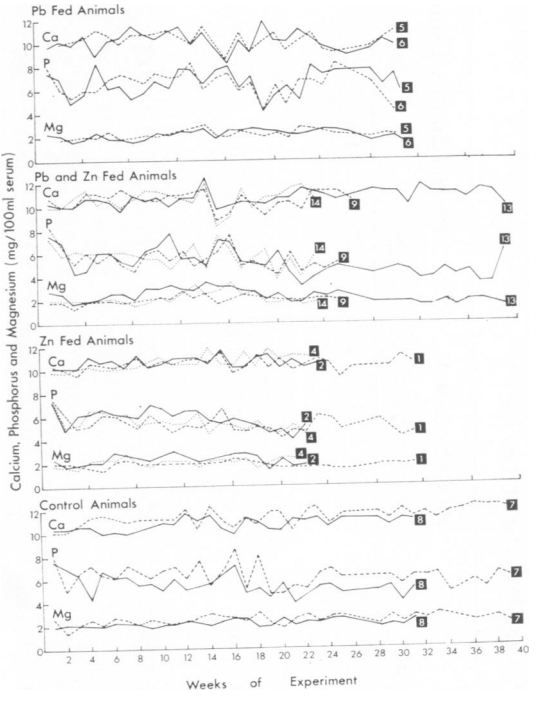

Fig. 6. Summary of the serum calcium, phosphorus and magnesium values, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

TABLE V. Lead Content of Tissues[a]

Tissue	Pb Fed Animals			Pb and Zn Fed Animals				Zn Fed Animals				Control	Control[c] X + S.D. (of 6 Animals)
	5[b]	6	$\bar{X}$	9	13	14	$\bar{X}$	1	2	4	$\bar{X}$	8	
Liver	20	33	26.5	70	41.5	48	53	0.9	4.5	7.0	4.1	1.0	0.83 ± 0.35
Kidney — Cortex	55	86	70.5	185	300	150	212	1.9	5.0	6.5	4.5	1.0	
Kidney — Medulla	20	25	22.5	75	47	45	56	0.9	2.5	4.0	2.5	0.5	0.23 ± 0.08
Metacarpal — Epiphyseal	340	410	375	140	445	200	262	8.2	4.0	19.0	10.4	8.0	7.33 ± 3.01
Metacarpal — Mid-Shaft	42	52	47	35	70	49	52	3.7	4.5	7.7	5.3	4.5	4.50 ± 2.07
Lumbar Vertebrae	440	360	400	180	380	215	258	13	17	22	17.3	8.0	7.00 ± 3.29
Rib	200	210	205	130	260	110	167	12	10	12	11.3	8.0	7.17 ± 3.76
Skeletal Muscle	<0.5	<0.5	<0.5	0.1	1.3	0.2	0.5	0.2	0.1	0.1	0.1	<0.5	<0.2
Brain	1.5	3.0	2.25	1.0	2.9	1.0	1.6	0.2	<0.5	<0.5	<0.4	<0.5	<0.2
Lung	1.0	1.0	1.0	1.5	1.0	1.5	1.3	0.1	<0.5	<0.5	<0.4	<0.5	<0.2

[a]Values in ppm, wet weight basis
[b]Number refers to the identification of each horse
[c]Results from previous experiment

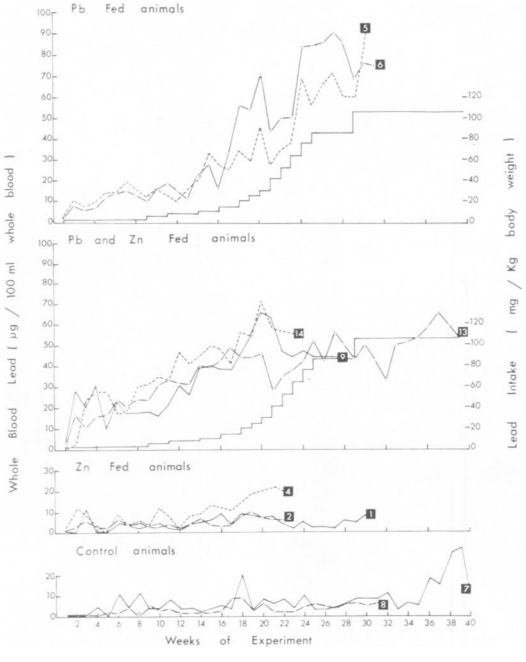

Fig. 7. Summary of whole blood lead values and the amount of lead fed, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

There was no evidence of laryngeal paralysis or difficulty in swallowing food.

The appetites of animals receiving both Pb and Zn decreased after the intake of Zn reached 90 mg per kg body weight per day. One animal (Number 13) eventually (Week 33) refused to eat any feed that contained ZnO and following an attempt to administer the compound orally with a dose syringe, it refused to eat the basal ration. To determine whether the signs of Pb poisoning could be induced in Animal Number 13, Zn administration was discontinued and the animal was dosed orally with Pb (as Pb acetate), for the last five weeks of the study (Fig. 1). It did not develop signs of pharyngeal or laryngeal paralysis during that period. It did, however, remain lame for this extended period.

The whole blood Pb values from animals fed both Pb and Zn increased initially at a faster rate than from animals fed Pb only (Fig. 7). However, despite comparable intake and longer duration of intake, the blood Pb values seldom exceeded 50 μg per 100 ml whole blood. The serum ALA values from animals fed both Pb and Zn, like the values from animals fed Pb only or Zn only, were variable (Fig. 8). The whole blood Zn values were increased in two animals (Number 9 and 14) (Fig. 9). There was no appreciable change in serum Ca, P or Mg values (Fig. 6).

Control Animals

Since no clinical evidence of disease oc-

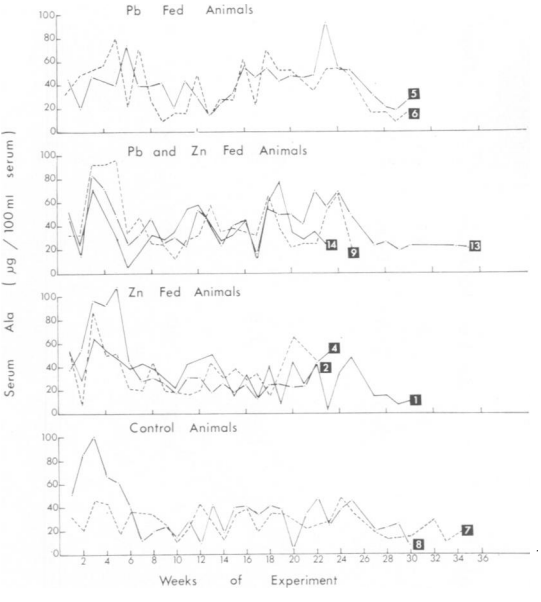

Fig. 8. Summary of serum delta-aminolevulenic acid (ALA) values, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

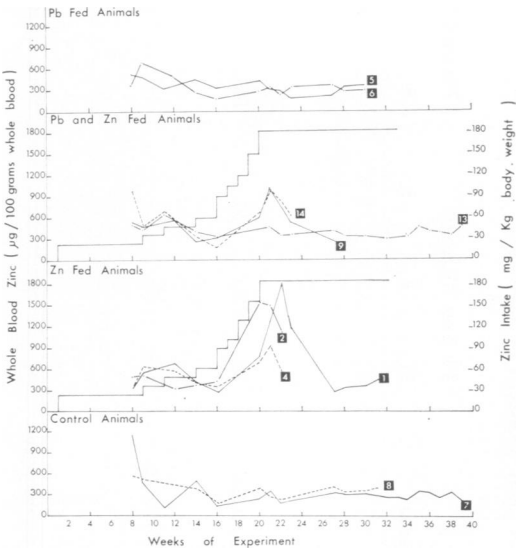

Fig. 9. Summary of whole blood zinc values and the amount of zinc fed, in descending order, from the groups of foals fed toxic amounts of lead only, lead and zinc together, zinc only and the control group.

TABLE VI. Zinc Content of Tissues[a]

Tissue	Pb Fed Animals			Pb and Zn Fed Animals				Zn Fed Animals				Control	Control[c] X + S.D. (of 6 Animals)
	5[b]	6	$\bar{X}$	9	13	14	$\bar{X}$	1	2	4	$\bar{X}$	8	
Liver	42	55	48.5	785	102	1200	696	1370	1300	1900	1523	74	34.67 ± 7.17
Kidney — Cortex	20	20	20	115	26	490	210	295	710	580	528	27	
Kidney — Medulla	15	15	15	65	10	260	112	120	490	350	320	12	20.67 ± 5.78
Metacarpal — Epiphyseal	87	75	81	220	150	185	185	190	130	220	180	105	82.50 ± 12.94
Metacarpal — Mid-Shaft	100	90	95	150	115	147	137	127	120	130	126	95	83.33 ± 9.83
Lumbar Vertebrae	90	75	82.5	175	160	270	202	215	300	250	255	90	78.33 ± 9.31
Rib	75	65	70	210	165	140	172	340	230	160	243	75	82.50 ± 15.08
Skeletal Muscle	14	11	12.5	11	20	20	17	15	21	21	19	12	10.33 ± 1.21
Brain	4	8	6	6	5	7	6	12	14	10	12	13	13.00 ± 1.55
Lung	10	8	9	9	12	12	11	10	12	10	11	12	17.00 ± 4.05

[a]Values in ppm, wet weight basis
[b]Number refers to the identification of each horse
[c]Results from previous experiment

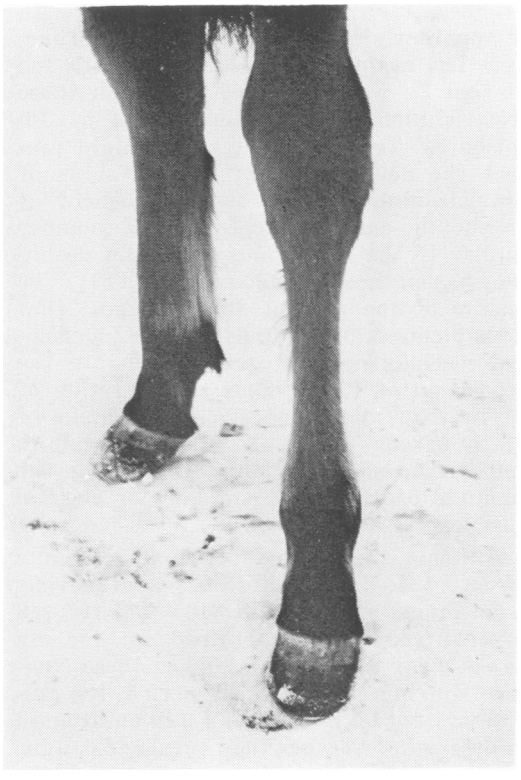

Fig. 10. Swelling at the hock and fetlock regions typical of foals fed toxic amounts of lead and zinc together (Animal Number 13).

curred, the values obtained (Figs. 4 to 9) indicate normal values and serve to monitor the uptake of Pb or Zn the animals acquired through pica.

Tissue Analysis

The results of Pb and Zn tissue analysis for the different groups of animals are given in Tables V and VI respectively. Control animal Number 7 was not euthanized since data from six normal animals of comparable age and breeding was available (26) and is included in both tables for control data purposes.

Fecal Analysis

The results of fecal Pb and Zn analysis conducted on samples collected from the rectum at necropsy are given in Table IV. The values are comparable to the oral intakes of Pb and Zn prior to necropsy (Fig. 1).

DISCUSSION

A clinical syndrome of stiffness, lameness and enlargement at the ends of the long bones of growing horses was produced by feeding toxic amounts of Zn only, Zn and Pb together, but not Pb alone. The Zn poisoning syndrome produced was similar to one described in two foals reared adjacent to a Zn emitting smelter (10) and in foals reared adjacent to smelters emitting both Pb and Zn (13, 21). Since this syndrome did not occur in the animals fed toxic amounts of Pb only, there appears to be no justification for attributing clinical signs resulting from changes in bone to Pb poisoning in the growing horse. The results further indicate that pharyngeal and laryngeal paralysis in the young growing horse, like adult horses, is the major clinical syndrome of Pb poisoning for the species.

The clinical signs of Zn poisoning that occurred in the young growing horse compared closely to those described in Zn poisoning of young growing swine (4, 11, 20). The growth rate of the horses fed toxic amounts of Zn decreased markedly after the intake exceeded 90 mg Zn per kg body weight per day. Similar reductions in body weight have been shown in Zn poisoning in other species (4, 15, 16, 17). Further studies will be required to determine if the horse, like swine, becomes less susceptible to Zn poisoning with increasing age.

The amounts of Pb only and Zn only fed before evidence of illness occurred reached 86 mg Pb and 90 mg Zn per kg body weight per day. These amounts approximate 3,400 ppm Pb and 3,600 ppm Zn in the feed on a dry matter basis when the daily dry matter intake from both the pelleted feed and the hay are considered in the calculations. More Pb is required to produce Pb poisoning under experimental conditions than is apparently necessary to cause Pb poisoning when animals are exposed while on pasture and consuming contaminated vegetation (1, 2, 3). The same conclusion would appear valid for the horse when the results of field studies (6, 12) are compared with the results of the present study. It is difficult to compare the dosage of Zn that caused Zn toxicity in the present study with other reports because of differences in methods of feeding and compounds used. The young growing horse seems to be as susceptible to Zn poisoning as are young growing swine

(4, 11, 20) and more susceptible than lambs (16) and feeder cattle (17). Other evidence that supports this conclusion is contained in a report (21) where young growing horses developed a clinical syndrome similar if not identical to that of Zn poisoning produced in the present study. Only young growing horses raised in the area were affected whereas young and older cattle were unaffected.

The whole blood Pb and Zn values closely paralleled the intake of these elements. Clinical signs of Pb poisoning were not evident until after the values reached and remained above 60 μg Pb per 100 ml whole blood for three or more weeks. These values are within the same range as those diagnostic of Pb poisoning in man (9). The terminal decline in whole blood Zn values appeared to reflect the reduced appetites caused by painful locomotion or unpalatability of feed. Although an appreciable variation in values from normal animals was evident, values greater than 600 μg Zn per 100 gm whole blood would be indicative of Zn poisoning.

The only changes of importance in the packed cell volumes and hemoglobin values were declines that occurred in animals fed toxic amounts of Zn for the longest periods. There was a relatively parallel decline in serum iron values but no distinct change in serum total iron binding capacity. It has been reported (15, 19, 27) that chronic Zn toxicity in the rat caused a hypochromic, microcytic, iron deficient type anemia. The changes observed in the present study were in some respects of a similar type. There was no evidence of anemia in animals fed toxic amounts of Pb only.

The results of tissue Pb and Zn analysis indicate some aspects of an apparent interaction between toxic amounts of Pb and Zn. The two animals that continued to consume toxic amounts of Pb and Zn (Numbers 9 and 14) had hepatic, renal cortical and medullary Pb values that were approximately twice as high as comparable tissue values from animals fed toxic amounts of Pb only. In contrast, the metacarpal epiphyseal, lumbar vertebrae and rib Pb values from the Pb and Zn fed animals were approximately one half as high as the comparable tissue values from animals fed toxic amounts of Pb only. These results indicate that toxic amounts of Zn interfere with the uptake of Pb in areas of active bone formation and, in some manner, result in higher hepatic and renal Pb values without these high levels being associated with or apparently causing neurological disfunction. The brain Pb values from animals fed Pb and Zn were slightly lower than those from animals with clinical signs of Pb poisoning. Toxic amounts of Zn might prevent the development of clinical signs of Pb poisoning through the impairment of Zn metallo-enzyme function in a manner similar to the deleterious effect Zn deficiency has on those metallo-enzymes (19). The results of the present study suggest that toxic amounts of Zn impaired the biochemical mechanisms that are involved in the deposition of Pb in bone, the excretion of Pb by liver and kidney, and the uptake of Pb by nervous tissue. Toxic amounts of Pb and Zn had no appreciable influence on the amounts of Pb and Zn in lung or skeletal muscle.

Animals rapidly excrete toxic amounts of Zn (18). The results of tissue analysis from the animal (Number 13) that refused to eat feed which contained Zn for six weeks prior to necropsy support these findings. During that six week period, the animal was fed high levels of Pb in an attempt to determine whether the neurological signs of Pb poisoning would occur. Although the bone changes characteristic of Zn poisoning persisted, signs of Pb poisoning did not occur. The tissue Zn values were similar to those from the control animals and those fed toxic amounts of Pb only, thus indicating a relatively rapid excretion of Zn. The liver and kidney Pb values were as high as those from animals fed Pb and Zn and considerably higher than the values from animals fed Pb only. The bone Pb values, however, compared closely with those from animals fed Pb only which were approximately twice as high as those from animals fed both Pb and Zn. These results indicated that bone deposition of Pb resumed after Zn feeding ceased. Valid comparison of liver Pb values cannot be made since this animal was consuming more PB prior to necropsy than any other in the study.

The results of the present study indicate that a high intake of Zn is not well tolerated by the young horse. If a high intake of both Pb and Zn occurred concomitantly, clinical signs of Zn poisoning would occur in the young animal and the clinical signs of Pb poisoning, despite extremely high hepatic and renal Pb values, would not occur. As the present report appears to be the first where an interaction between toxic

amounts of Pb and Zn has been demonstrated, studies designed to more fully describe the nature of this interaction and to determine if it occurs in a similar manner in adult horses and other species are indicated.

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