Chap. 4

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4. BIOLOGIC EFFECTS IN DOMESTIC AND WILD ANIMALS

Domestic Animals

Several outbreaks of lead poisoning in domestic animals have been recorded in North America where the apparent source of metal was contamination of pasture or crops by industrial lead operations. Some of these areas include St. Paul, Minnesota (Hammond and Aronson, 1964), Trail, British Columbia (Larsen et al., 1969) and Benecia, California (Ottoboni and Kahn, 1970). Deaths in horses attributable to lead poisoning have been noted in the Benecia area since the early 1900's. The presence of lead and arsenic have made it impossible to raise horses near smelters located in East Helena, Montana for several decades (Gordon, 1968).

Pastures and crops are contaminated by fumes and dusts emitted from lead industries settling out on the surrounding countryside. Animals eating this vegetation can accumulate amounts of lead sufficient to produce clinical signs of lead poisoning. A number of studies have been made to determine if the lead found in vegetation is the result of direct airborne origin or due to translocation from soil. These studies have been reviewed recently by Mueller and Stanley (1970). They conclude that translocation from soil does not contribute more than 15 μg Pb/gm dry weight of forage even when plants are grown in soil containing up to 700 - 3000 μg/gm. Thus, amounts of lead in excess of 15 μg Pb/gm most likely are due to direct aerial fallout. The extent to which contamination can occur is illustrated by concentrations of 3200 μg Pb/gm dry weight found in corn leaves located 75 yards from a lead smelter (Hammond and Aronson, 1964).

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It has been possible to estimate that 6 - 7 mg Pb/kg/day constitutes a minimum cumulative fatal dose of lead for cattle (Hammond and Aronson, 1964). These cattle were located approximately 2 miles from the smelter, but were fed lead-contaminated hay and corn silage grown in fields adjacent to the smelter. A fatal case of lead poisoning occurred following approximately 2 months on this diet. An intake of approximately half this daily dose had no observed effect on cows at another farm the previous winter. In this connection it is of interest that daily doses of 5 - 6 mg Pb/kg/day have been fed to cattle for a period of 2 years with no observable clinical effects (Allcroft, 1950). Continued intake at this rate may be fatal or may be continued almost indefinately without toxic effects (Allcroft, 1951).

There is some evidence suggesting that horses may be more susceptible than cattle to the chronic ingestion of lead. Whereas horses contracted lead poisoning on pastures adjacent to a lead smelter in the Trail area, (cattle grazing in the same area appeared healthy (Larsen et al., 1969). At one farm adjacent to the St. Paul smelter, horses succumbed to lead poisoning in March following a winter intake in their hay of 2.4 mg Pb/kg/day (Hammond and Aronson, 1964). It was not possible to determine possible lead intake from pasture grazing the previous summer. However, since cows and horses had similar pasture that summer, and since the winter ration for the horses contained appreciably less lead than that for the cows, it would seem that cumulative toxicity occurred

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somewhat more readily in horses. It is of interest to consider that pasture grass containing in excess of 80 µg Pb/gm dry weight was toxic to horses in the Benecia area (Mueller and Stanley, 1970). If one assumes the horses weighed 400 kg and ate 10 kg grass per day, a minimally toxic dose could be estimated at 2 mg Pb/kg/day; a figure close to the previous estimate.

Although the evidence above does suggest that horses might be more sensitive to lead than cattle, a consideration of the grazing habits of horses precludes any firm conclusions. Horses occasionally will pull forage out by the roots and eat the roots and attendent soil along with the forage. Cattle rarely, if ever, do this. The concentration of lead in soil near smelters usually contains far greater amounts of lead than the forage. It is apparent that a horse showing a marked tendency toward this habit could be ingesting far greater quantities of lead than would be estimated from the the analysis of forage alone.

It is only natural that human beings residing adjacent to these smelters in chose proximity to animals dying of lead poisoning be concerned about their own health. In many cases these people are eating produce from home gardens. It is noteworthy that analysis of blood and urine of these people by local public health officials have not revealed any evidence of increased lead absorption. It is emphasized that horses and cattle are vegetarians. If raised in the vivinity of a lead industrial operation their entire diet may consist of contaminated vegetation. Probably only a small fraction of the total diet of human beings would consist of food grown in the

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vicinity of the lead operation. Furthermore, it is customary for people to wash garden produce (or husk corn) before consumption. This practice undoubtedly removes appreciable quantities of surface lead deposits. Since the animal and human population are breathing the same air, and since residents in the area did not show evidence of increased lead absorption it may be justified to conclude that animals are receiving virtually all of their lead burden through oral ingestion.

It generally is recognized that lead is the most common cause of self poisoning in domestic animals, including pet dogs.

The natural curiosity and licking habits of cattle make any available lead - containing material a potential source of poisoning. Some of the sources incriminated include lead-base paint (either from discarded paint cans or paint peeling from walls), used motor oil, discarded oil filters, storage batteries, certain types of greases and putty, and linoleum (Hammond et al., 1956; Buck, 1970). It is noteworthy that these sources have not been incriminated in lead poisoning of horses, which are much more selective in their eating habits than cattle. They do not lick old paint cans, storage batteries, peeling paint, nor do they find the taste of motor oil attractive.

Common histories of exposure in dogs include chewing on objects painted with lead-base paints, the home being remodeled with scraping of plaster and/or old paint, eating linoleum or ingesting lead materials such as lead slugs or curtain weights (Zook et al., 1969). Dogs less than 6 months of age are affected more commonly than older dogs, but this may be related to the almost completely indiscriminate eating habits of younger dogs.

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All domestic species exhibit varying degrees of derangement of the central nervous system, gastrointestinal tract, muscular system, and hemopoeitic system. Differences occur clinically, however, in the relative severity of signs referrable to these organs and tissues. The most striking syndrome is presented commonly by young calves. The calf may suddenly begin to bellow and stagger about with rolling eyes and frothing mouth. During this phase the animal often blindly crashes into objects. This phase may last up to 2 hours before sudden collapse. With less severe cases, depression, anorexia and colic may be observed. The animals may grind their teeth, move in a circle, push against objects and be ataxic. Adult cattle present the latter signs most frequently although the syndrome of maniacal excitement is not uncommon.

The syndrome in sheep consists mainly of depression, anorexia, abdominal pain and usually diarrhea. Excitatory phases have never been reported for sheep. Anemia is common during chronic ingestion.

The syndrome in horses consists mainly of depression, stupor, knuckling at the fetlocks, and a laryngeal paralysis causing the horse to "roar." The paralysis interferes with constriction in the air passage. Anemia is commonly associated with lead poisoning in horses. (Clarke and Clarke, 1968).

Gastrointestinal and central nervous system signs are seen with almost equal frequency in dogs. At some time during the course of poisoning approximately 87% of dogs show GI signs consisting of emesis, colic, diarrhea, and anorexia. Approximately 76% of dogs show CNS signs consisting of hysteria and convulsions. Anemia and basophilic stippling are commonly associated with lead poisoning in dogs and are considered to be of diagnostic significance (Dodd and Staples, 1956; Zook et al., 1969). This point will be considered further in a following section.

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Abortions have been reported in ewes grazing in lead-mining areas (Egan and O'Cuill, 1969). A high rate of abortions and failures to conceive were noted in ewes fed finely divided metallic lead at a rate sufficient to induce signs of intoxication (Buck, 1970b). Cattle and horses have given birth to normal offspring following excessive lead exposure (Shupe, 1967; Egan and O'Cuill, 1970) but the numbers of animals involved (five) make it impossible to state that lead has no effect on the fetus in these species.

An elevated concentration of lead in blood and/or tissues, together with clinical signs, is considered to be the best criteria for a diagnosis of lead poisoning. The presence of basophilic stippling of red blood cells, immature (especially nucleated) red blood cells and acid-fast inclusion bodies in the kidneys have been reported (Dodd and Stapleo, 1956; Zook et al, 1969) to be commonly associated with lead poisoning. Unpublished work from 3 different laboratories indicates that elevated urinary ALA concentrations are a good indication of excessive lead exposure in several species of domestic animals.

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Fish

Very little information is available regarding the toxicity of lead to fish. In two recent reviews (Danielson, 1970; anon-summary of the report on air quality criteria for lead - submitted to the national air pollution control adrinistration in partial fulfillment of contract PH 86 - 68 - 35 with the California state department of public health) the following points were made. Fish can be poisoned by lead, but few examples of fish kills have been recorded due to contamination of water. Analysis of lobster for lead reveals concentrations of 0.08 - 2.4 µg Pb/gm depending on the report. In Sweden, concentrations of 12 µg Pb/gm liver have been measured in pike taken from lakes with naturally occurring high concentrations of lead. An outboard motor can emit 140 mg lead into the water for every liter of gasoline burned. Thus, it appears a potential exists for fish contributing a dietary source of lead to man. There appears to be little evidence of lead being a significant toxicologic problem for fish, but there is too little information on the subject to justify a conclusion. Obviously, more work is needed.

Zoo Animals

Chewing on cage bars painted with lead-base paint has been incriminated as a source of lead poisoning in bats (Zook et al., 1970), and in nonhuman primates (Sauer et al, 1970).

Birds

As with mammals, the presence of lead in birds is a constant finding. A study including between 1 to 37 representatives of 28 different bird species with no known "excessive" lead exposure revealed concentrations in fresh liver ranging between 0.3 to 7.0 ppm. (Bagley and Locke, 1967).

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Lead poisoning has been recognized in waterfowl and upland game bird species for at least 100 years. Lead poisoning was considered to be a serious problem of ducks and other waterfowl in the United States by 1919 (Wetmore, 1919).

Recently it was estimated that one million ducks, geese and swans die each year because of lead poisoning in the United States (Andrews and Longcore, 1969). Another estimate places the number at 2 - 3 million dying from lead poisoning with an economic value approximating $1,000,000 (Bond, 1970). These figures become even more impressive considering that most of the birds die following the hunting season and thus represent a loss of breeding stock. The principal, and probably the only significant, source of lead is spent lead shot from hunting. Approximately 6,000 tons of lead shot are deposited on waterfowl habitats each year (Andrews and Longcore, 1969). Cases of lead poisoning occur in upland game birds as well as waterfowl (Bagley and Locke, 1967). As in the case of waterfowl, the primary source of lead appears to be spent lead shot as evidenced by the recovery of the material from the gizzards of affected birds.

Efforts to develop a safer material than lead for shot began as early as 1936 (Green, 1936). Copper or hard iron are safer materials, but ballistic considerations and damage to gun barrels and chokes make the the materials unsuitable. The Sporting Arms and Ammunition Manufacturing Institute (SAAMI) recently supported a $100,000 study by the Illinois Institute of Technology to find a more suitable material than lead. They found soft iron pellets to be the best substitute. Although soft iron pellets compared satisfactorily to lead for killing ducks in flight (Andrews and Longcore, 1969), the cost of manufacturing them is at present prohibitive.

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A lethal amount of lead for a duck can be absorbed from one #6 lead shot. Variable mortality occurs following the ingestion of up to six #6 shot which nearly always is lethal (Wetmore, 1919). A daily dose of 6 mg/kg/day (as lead nitrate) for 137 days did not produce any observable ill effects, but when the dose was increased to 8 and 12 mg/kg/day, the survival periods averaged 28 and 25 days, respectively. (Coburn et al., 1951). The disposition of lead in soft tissues was almost proportional to dosage rate, but no appreciable differences were measured for bone. When the dosage of lead for the 2 birds receiving 6 mg/kg/day for 137 days was increased to 12 mg/kg/day, death occurred about the same time expected for this dosage. The preceding long period on the lower dosage seemed to have little effect on the rate at which clinical signs developed after the dosage was increased or on the deposition of lead in tissues. When excretion was expressed as a percentage of dose an average of only 3% was excreted the first week, but the average increased to 67% and 63% for the second and third weeks, respectively (Coburn et al., 1951).

Concentrations of lead in muscle are very low in mammals, but it has been reported that breast muscle and liver from a pheasant dying of lead poisoning contained 42 and 169 ppm., respectively (Hunter and Rosen, 1965). A total of 29 shot were recovered from the gizzard. Concentrations of lead in muscle of mallards dying of lead poisoning are considerably lower than 42 ppm. (L. Locke, 1970).

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The usual picture of lead poisoning in waterfowl consists of lethargy, weakness, flaccid paralysis, emaciation, anemia, greenish-colored diarrhea, impaction of the proventriculus and distention of the gall bladder. The large pectoral muscles waste away and in some cases the stermum is covered merely with a thin layer of fascia, muscle and skin - the so-called "razor-keel" or "hatched-breast" (Rosen and Bankowski, 1960; Coburn et al., 1951; Trainer and Hunt, 1965; Locke et al., 1967). Geese develop a striking high-pitched call (Bagley et al., 1967). There is a diffuse necrosis of the liver with hemosiderosis (Locke et al., 1967). Acid-fast intranuclear inclusion bodies of the renal tubular cells are commonly, but not always found in cases of poisoning (Locke et al., 1967b). A number of workers have stated that basophilic stippling of red blood cells does not occur.

Several reports have appeared that indicate a possible influence of dietary factors in protection from lead poisoning.

It has been reported that certain green feeds such as coontail (Ceratophyllum demersum) will prevent lead poisoning from occuring in mallards fed levels of lead shot which will kill birds maintained on cracked or whole corn (Jordan and Bellrose, 1951). In another study, ducks maintained on corn and on grain-duck pellet diet developed acid-fast intranuclear inclusions in renal tubular cells when given 1 or 3 #6 lead shot orally. No lesions developed in ducks maintained on a duck pellet ration and fed the same number of shot (Locke et al, 1966). It is not known whether these dietary factors affect the absorption or the biological effects of the lead.

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TREATMENT OF LEAD POISONING IN CATTLE

Arthur L. Aronson

The chelating agent calcium disodium ethylenediaminetetraacetate (CaEDTA) first was introduced as a successful treatment for lead poisoning in man by Bessman and associates (1952). The drug also has been used successfully as an antidote for lead poisoning in cattle (Holm et al., 1953; Lewis and Meikle, 1956, 1958; Hammond and Sorenson, 1957; Todd, 1957). The dosage and schedule of administering CaEDTA to lead poisoned cattle in these reports were based largely upon toxicity and metabolic studies in laboratory animals (Foreman et al., 1953). In order to establish a more definitive basis for the use of CaEDTA in the treatment of lead poisoning, a study was made in which various dosage regimens of CaEDTA were administered to calves previously dosed with lead (Aronson et al., 1968). The purpose of this communication is to summarize these findings.

Previous studies established that the amount of lead mobilized by a single rapid intravenous injection of 110 mg CaEDTA/kg to calves was proportional to the concentration of lead in erythrocytes at the time of treatment (Hammond and Aronson, 1960). This dosage probably produced the maximal amount of lead mobilizable in calves by a single rapid intravenous injection since a range of doses of 11 - 165 mg/kg did not produce significantly greater urinary excretion. This relationship served as the standard base of reference for evaluating other doses and schedules of CaEDTA administration.

The study indicated that optimal conditions of lead mobilization were provided by concentrations of approximately 135 micromoles EDTA/liter plasma and above maintained for 10 - 12 hours (Aronson et al., 1968). These

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concentrations could be achieved by the constant intravenous infusion of CaEDTA at 110 - 220 mg/kg over 12 hours or approximated by 2 rapid intravenous injections 6 hours apart at 110 mg/kg each. Over twice as much lead could be mobilized by these procedures as with a comparable dose given as a single rapid intravenous injection. The administration of CaEDTA as a 12 hour infusion for 3 consecutive days proved to be no more effective in mobilizing lead than a single 12 hour infusion.

Since these experiments were carried out with calves not showing clinical signs of lead poisoning it might be argued that the conclusions would not necessarily apply to clinical cases of bovine lead poisoning. It cannot be stated unequivocally that higher concentrations of lead in the body would not require higher concentrations of EDTA for maximal lead mobilization. However, the concentrations of lead measured in liver (2.3 - 3.2 µg/g wet wt.), kidney cortex (2.8 - 15.0 µg/g wet wt.) and blood (0.27 - 0.58 ug/ml) of untreated animals given lead were of the order of one - half the concentrations commonly encountered in clinical cases of lead poisoning (Hammond et al., 1956). Furthermore, there was no significant difference in lead mobilization when CaEDTA was infused for 12 hours with a 4-fold variation in the rate of administration (110 - 440 mg CaEDTA/kg). This would suggest a ready capacity to cope with the greater amounts of lead that would be involved in actual poisoning.

In regard to the lack of additional benefit attained by repeating 12-hour infusions beyond the first infusion, a clinical situation might yield different results since a much larger reservoir of unabsorbed lead in the gastrointestinal tract might exist, providing a greater continuing absorption of metal into the circulation. However, multiple daily infusions at the level

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of 220 mg/kg over 12 hours would be hazardous because of the toxicity of CaEDTA (Aronson et al., 1968). It would, therefore, seem that even under conditions of tissue concentrations of lead greater than were produced under our conditions, 12 - hour infusions of CaEDTA should not exceed 220 mg/kg and probably should not be instituted more than once every other day. Probably no more than t treatments should be given. A rational basis for an intermittent schedule of therapy also is provided by evidence indicating that EDTA acts to remove lead from the bone and that a subsequent period of time is required for redistribution of lead from the soft tissues to the EDTA - sensitive sites in the bone (Hammond et al., 1967).

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Economic Cost of Lead Poisoning in Cattle

This estimate is presented only as a very crude estimate of what the economic cost of lead poisoning might be in cattle affected with clinical lead poisoning. The key point is the incidence of lead poisoning in the United States which is based upon an extrapolation of the estimated incidence of lead poisoning in Tompkins County, New York; an area served by the Ambulatory Clinic of the New York State Veterinary College.

No estimate is being made for the cost of lead poisoning in other species of animals. No estimate is being made for the cost of a pasture or crops contaminated by lead fallout from a smelting or mining operation.

It is my belief that this estimate probably is conservative. I hope it is not so far off as to be totally misleading. It points out the difficulty of trying to assess the cost of a disease in domestic animals for which no reporting system exists. A knackery survey (an establishment that receives and processes dead animal carcasses) in Northern Ireland estimated that lead poisoning accounted for [redacted] 1.7 % of the deaths of adult cattle and 4.5 % of the deaths of young calves (Todd, 1962). This rate was considerably greater than clinical diagnoses and suggested that cases of lead poisoning were not being diagnosed for a variety of reasons.

The cost estimate of lead poisoning in cattle is based upon the following facts, estimates and assumptions.

Facts

1. Cattle population of the United States.
   a. Cattle other than dairy cattle.. 109,661,000
      Includes heifers and calves not kept for milk and steers in 1969.

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b. Cows and heifers 2 years old and over kept for milk..... 14,123,000

TOTAL..... 123,784,000

These data were obtained from Agricultural Statistics 1969 U.S. Dept. Agric., U.S. Government Printing Office, Washington.

2. Cattle population of Tompkins County, New York.....24,410

Data obtained from the 1964 U.S. Census of Agriculture for the State of New York. Vol. 1 part 7, page 321.

Estimates

The Ambulatory Clinic of the N.Y.S. Veterinary College treats approximately 4 cases of lead poisoning per year.

$\frac{24,410}{4} = 6102$. Thus, based on the incidence in Tompkins County, New York, approximately 1 case of lead poisoning occurs per 6000 cattle per year.

If this rate holds throughout the United States:

$\frac{123,784,000}{6000} = 20,631$

Thus, it is estimated that 20,000 cases of lead poisoning occur in the United States per year.

Assumptions

1. The cost of treatment per animal based on 2 calls plus drugs.....$25.00
2. Mortality. Assume 50% although this figure probably is low.
3. Value of an animal.....$150.00

The average value of a milk cow is $269.00 and cattle other than dairy cattle $158.00 according to

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Agricultural Statistics 1969. Most cases of poisoning, however, are in young animals - probably worth somewhat less than $150.00.

4. Summary:

Treat 20,000 cases at $25.00/case.....	$500,000
10,000 cases die at $150/animal.....	1,500,000
Total cost /yr	$2,000,000

Prognosis of Lead Poisoning.

Lead poisoning of domestic animals is generally considered to be a disease of low morbidity and high mortality. In general, it is felt by clinicians that if an animal/~~xxxxxx~~/ lives, the prognosis for recovery is good. For example, a calf often is blind during acute manifestations of the disease. If the animal lives sight usually returns within a week; at least sufficiently well that the animal can move about with no difficulty.

Reference

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