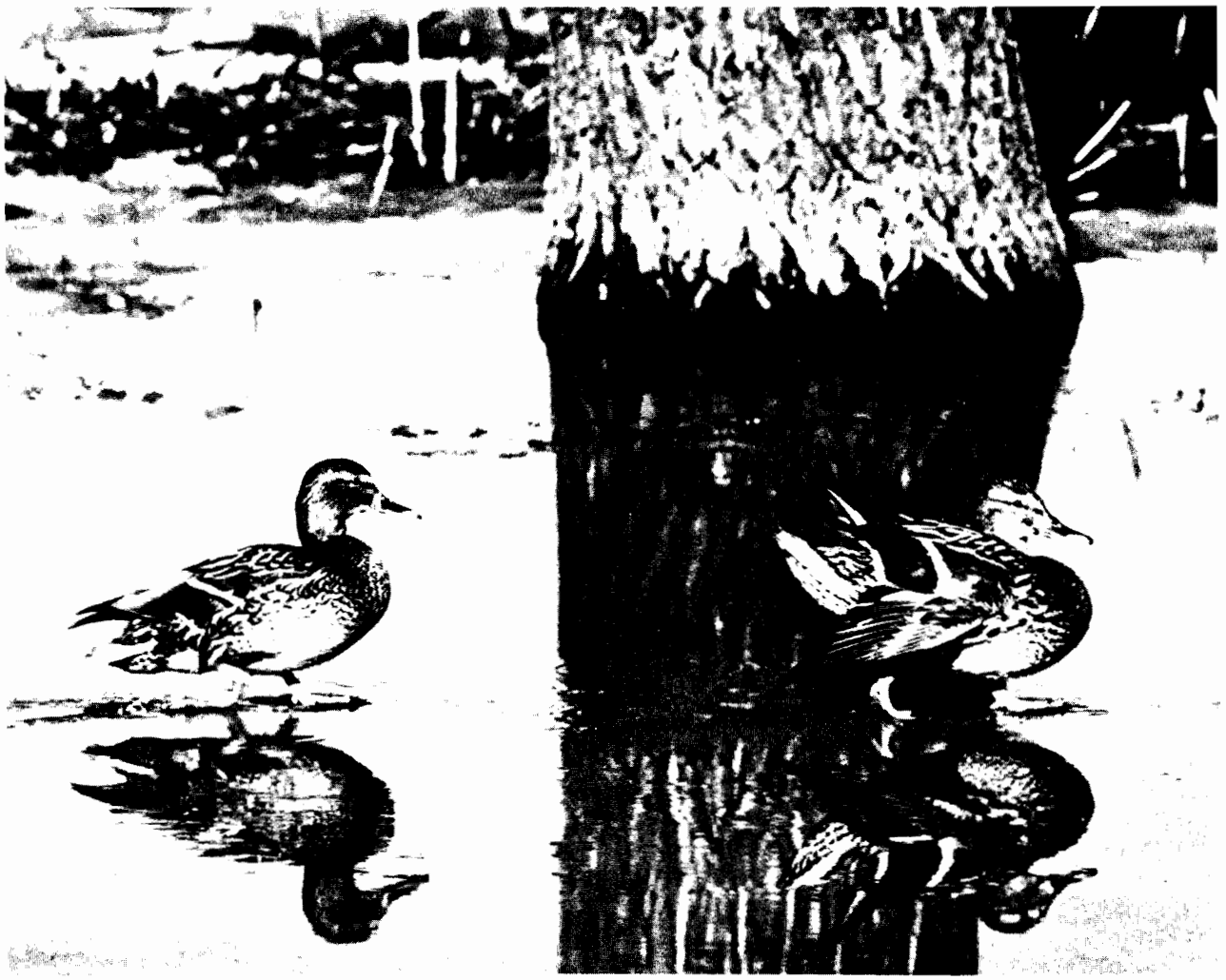
INTERPRETATION OF CRITERIA COMMONLY USED TO DETERMINE LEAD POISONING PROBLEM AREAS



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FISH AND WILDLIFE LEAFLETS

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INTERPRETATION OF CRITERIA COMMONLY USED TO DETERMINE LEAD POISONING PROBLEM AREAS

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Lead Poisoning of waterfowl and other birds continues to be a subject of controversy and confusion. This leaflet summarizes the general characteristics of criteria commonly used by biologists, administrators, and others for evaluating and interpreting data to determine whether lead poisoning is a problem on a site-specific area.

Three categories of criteria were evaluated: (1) determination of mortality, (2) determination of exposure, and (3) environmental indices (Table 1). Each category provides different information, as do different criteria within each category. Failure to appreciate differences in the interpretation of similar, but different, assay systems can result in erroneous conclusions.

Differences between characteristics of lead poisoning, avian cholera, and avian botulism as disease processes were also evaluated. The perception that all three diseases are equally visible is erroneous, and recognition of the differences among them is important for identifying lead poisoning problem areas.

Criteria for Identification of Lead Poisoning Problem Areas

Determination of Mortality

Diagnosis of lead poisoning is accomplished primarily through pathological findings supported by toxicology; however, neither chemistry nor pathology alone can provide a basis for an unequivocal diagnosis. It is beyond the scope of this leaflet to describe the clinical signs and pathology of lead poisoning. Differences in pathology occur among different species, creating the need for experienced diagnosticians to evaluate cases if rigorous determinations are needed. Liver or kidney tissues are preferred for toxicological assays.

The presence or absence of lead shot in the gizzards of dead birds has often been believed to be a reliable indicator of lead poisoning. This is not true, since death is actually caused by lead that has been dissolved, transformed into lead salts, and absorbed into the body.

Determination of Exposure

Three criteria are commonly used to determine whether exposure to lead has occurred: (1) presence of lead shot in gizzards, (2) lead residues in body tissues, and (3) blood enzyme measurements. Interpretation of these criteria represents a major source of confusion.

(1) Examination of gizzards for ingestion of shot pellets is widely used and accepted for obtaining a direct index of exposure rates to lead shot during the period of sampling. The degree of mortality likely to occur is dependent on a number of factors and does not represent a 1:1 relation.

(2) Level of residues in blood, soft tissues, or wing bones is another criterion. Values obtained are a reflection of lead exposure; however, the source of lead (lead shot vs. atmospheric or other sources) is often debated. Tissue lead residues lack the physical evidence of exposure to lead shot provided by gizzard samples. However, tissue residues provide direct evidence of the amount of lead actually taken into the body. Wing bone residues are the least useful of these measurements because they reflect long-term storage of lead rather than recently absorbed lead.

Table 1. *Criteria*[1] *commonly used to identify lead poisoning problems—(1) samples required, (2) examination process, and (3) evaluation of data.*

Mortality
(1) Whole carcasses.
(2) Postmortem examination and laboratory assays, including microbiology and toxicology.
(3) Diagnosis of lead poisoning as a cause of mortality depends on a combination of pathological findings supported by appropriate toxicological results. Liver is most often used for chemical analysis. Lead values of 8 ppm (wet weight) and above are consistent with lead intoxication when supported by pathology.

Lead Concentrations in Body Tissues
(1) Liver, kidney and blood.
(2) Liver and kidney are removed from dead birds. Chemical analysis is usually by atomic absorption spectrophotometry.
(3) Values indicate the amount of lead present. Concentrations of 2 ppm or greater for liver and 0.2 ppm or greater for blood should be considered elevated. These data must be supported by pathological findings before confidence can be placed in a lead poisoning diagnosis.

Lead Shot in Gizzard Samples
(1) Intact gizzards.
(2) Examination of gizzard contents for presence or absence of lead shot.
(3) Presence of lead shot reflects exposure just prior to sampling and constitutes an index for lead poisoning risk. Shot ingestion may be independent of cause of death, particularly if dissolution and absorption have not occurred. One or more ingested shot in 5% or more of gizzards examined is the threshold criteria for potential problem areas.

Measurement of Delta Aminolevulinic Acid Dehydratase (Delta-ALAD) in Soft Tissues
(1) Blood, brain, or liver.
(2) Red blood cells are examined most often. Analysis is usually by spectrophotometry.
(3) Inhibition of delta-ALAD is a sensitive indicator of lead exposure. Suppression below normal values can occur within 24 hours of lead absorption. Recovery of delta-ALAD requires a month or more. The degree of suppression is directly correlated with blood lead levels.

Measurement of Protoporphyrin IX Levels in Blood
(1) Drop of blood.
(2) Fluorometer measurement of red blood cells.
(3) Concentration of protoporphyrin IX results from lead inhibition of heme synthetase, an enzyme responsible for incorporation of iron into protoporphyrin IX. Values above 40 μg/dl indicate exposure to lead. Recovery to normal levels occurs within a month following exposure. High values are correlated with body function impairment and reflect toxicity.

Amount of Lead Shot in Environment
(1) Substrate samples.
(2) Soil and bottom samples are taken at predetermined depths.
(3) The amount of shot present represents the potential for lead shot ingestion, but variables influence its probability of occurrence.

Hunting Pressure
(1) Information on hunter-use days or birds shot per unit area.
(2) Calculation of relative amounts of lead shot deposited.
(3) Provides an index of potential lead shot availability to waterfowl. Many variables impact the probability of lead shot ingestion and subsequent intoxication.

[1]Including levels used for decision criteria in establishing non-toxic shot zones as published in the *Federal Register* 49FR37672, 25 September 1984.

3) Enzyme measurements in blood samples provide yet another index to lead exposure. The two most common test systems in use are the measurement of delta aminolevulinic acid dehydratase (delta-ALAD) and protoporphyrin IX. Levels of lead in blood are not measured by these techniques. Instead, the disruption of biochemical pathways highly sensitive to the action of lead is measured. A marked and prolonged suppression of delta-ALAD and a rise in values of protoporphyrin IX occurs in the presence of lead. Recovery of protoporphyrin IX to normal is somewhat more rapid than for delta-ALAD after cessation of exposure to lead. Because these test systems are highly sensitive to lead, they are useful for detecting sublethal levels of exposure.

The preceding measurements provide the same basic information—evidence of exposure to lead on an individual bird basis. None of these techniques alone provides direct unequivocal evidence of site-specific exposure of lead because the time of lead exposure relative to the time of sample collection is unknown. However, sequential sampling of populations over time with any of these techniques can provide strong evidence of sources and sites of lead exposure when considered along with bird movement patterns, hunting activities, and the pathogenesis of lead poisoning.

The usefulness of any of these techniques is a function of two variables: (1) cost factors associated with sample collection and analysis, and (2) acceptability of data generated. Area differences in the acceptability of data generated appear to be caused by too little attention given to developing a sound understanding of the strengths and weaknesses of the various techniques.

Environmental Indices

Lead shot density in soil and bottom sediments and area hunting pressure are two criteria sometimes used for evaluation of lead poisoning problem areas. Both criteria provide a measurement of potential exposure to lead shot by foraging waterfowl. Field sampling provides direct evidence for the presence of lead shot, as well as data regarding the relative abundance of shot in specific substrates sampled. Hunting pressure, whether measured in birds bagged per unit area, or hunter-use days, provides an indirect measure of annual lead shot deposition. Variables influencing the ingestion of lead shot and its toxicity preclude direct extrapolation of the presence or absence of lead poisoning. Instead, the usefulness of these data lies in their value as environmental indices of the potential for exposure to lead shot.

Characteristics of Lead Poisoning as a Disease

Avian botulism, avian cholera, and lead poisoning are three of the most common waterfowl diseases. Two major differences between lead poisoning, botulism, and avian cholera, other than the specific cause for each are that lead poisoning usually requires a prolonged period between exposure and death, and that exposure tends to be on an individual rather than a group basis. A common misconception by biologists is that losses from all three diseases are equally visible; that is not true because of differences in the disease processes.

The significance of differences between disease processes causing lead poisoning, avian cholera, and avian botulism lies in the different approaches needed for problem detection. The acute nature of avian cholera and avian botulism, and the absence of good "markers" for detection of exposure in clinically normal birds make mortality the best criterion for problem identification. Avian cholera is an infectious disease with the capability for rapid transmission through susceptible bird populations. Because of the disease's acute course sick birds are seldom observed and the time between exposure and death is often as brief as 6 to 12 hours. Carcasses accumulate rapidly when gregarious species such as waterfowl are infected. As a result, problem visibility is enhanced and exposure most likely occurred in the vicinity where most birds have died.

Avian botulism is a toxic process like lead poisoning. However, the source of intoxication is maggots and other invertebrates. The toxin involved is one of the most powerful known; a single dead bird can be the source of thousands of toxic maggots and death in other birds can occur following ingestion of only two or three of these maggots. As a result, the disease pattern is similar to an infectious disease process. The time between exposure and death is usually several days; therefore, sick birds are commonly observed in various stages of intoxication. However, since loss of flight is an early consequence of botulism

intoxication, the problem is often detected in the immediate vicinity of the disease's source.

In contrast, lead poisoning of waterfowl is a debilitating disease requiring an average of 2 to 3 weeks between exposure and death. During this time, affected birds lose mobility, experience marked behavioral changes involving increased seclusion, and become increasingly susceptible to predation and other causes of mortality. Large-scale concentration of lead-poisoned carcasses does not usually occur, nor does this perspective reflect the disease's true nature.

The prolonged course of lead intoxication results in clinically ill birds. However, because exposure is on an individual bird basis, only small numbers of sick birds are usually present. Ingestion of lead shot and its retention in the gizzard for a sufficient time so that lead absorption occurs are essential for intoxication and visible illness. Unlike avian cholera and avian botulism, a number of sensitive and specific "markers" are available for detection of lead exposure before the onset of clinical signs. In addition, other indices are available to identify areas of high potential for exposure to lead shot.

Summary

Determination of lead poisoning problem areas is complicated by the nature of the disease process. Rigorous documentation of lead poisoning as a cause of mortality in birds requires the integration and evaluation of pathological and toxicological data by an experienced diagnostician. No single technique provides unequivocal proof that lead exposure occurred at the site of death. However, evaluation processes that integrate knowledge regarding the course of lead poisoning in birds, bird movement patterns in specific geographic areas, and findings from studies involving criteria commonly used to measure exposure to lead shot provide a sound basis for determination of specific problem areas. Sequential sampling during the period of bird use is an important requirement for establishing strong cause and effect relations. Knowledge of lead poisoning characteristics as a disease process are also useful in identifying lead poisoning problem areas.

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A list of current *Wildlife Leaflets* follows.

512. Construction and Operation of Cable-chain Drag for Nest Searches, by Kenneth F. Higgins, Leo M. Kirsch, Harold F. Duebbert, A. T. Klett, John T. Lokemoen, H. W. Miller, and Arnold D. Kruse. 1977. 14 pp.
513. Guide for Collecting and Seeding Native Forbs for Wildlife in Douglas-fir Clearcuts, by Dan L. Campbell and Larry E. Johnson. 1981. 13 pp.
514. Distribution of Animal Damage in Southwestern Oregon Forests, by James Evans, Dan L. Campbell, Gerald D. Lindsey, Victor G. Barnes, Jr., and R. Michael Anthony. 1981. 12 pp.
515. Senegal's Trade in Cage Birds, 1979–81, by Philippe Ruelle and Richard L. Bruggers. 1983. 11 pp.

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