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Pathomorphological studies on lead toxicity in cattle from industrial waste-contaminated grazing lands

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Abstract

Lead poisoning in domestic ruminants has persisted as a diagnostic challenge wherever cattle graze on land exposed to industrial effluent, mine tailings, or improperly disposed battery waste. Historical records from southern Africa indicate recurring episodes of bovine plumbism linked to smelter runoff and artisanal lead-acid battery recycling sites, yet quantitative tissue-level pathomorphological data from affected herds remain scarce. This research examined the gross and histopathological lesions associated with chronic lead exposure in 36 adult crossbred cattle grazing within 0.5-15 km of an inactive lead smelter near Pretoria, South Africa. Blood and tissue lead concentrations were measured by graphite furnace atomic absorption spectrophotometry, and soil samples from five concentric distance zones were analysed for total extractable lead. Mean blood lead in cattle grazing within 1 km of the smelter was 42.3 ± 6.8 µg/dL, declining to 4.1 ± 1.2 µg/dL at 15 km. Soil lead followed a parallel gradient, from 387 mg/kg at 0.5 km to 28 mg/kg at 15 km. Gross lesions were most severe in Zone 1 (0-2 km) animals and included pale, swollen kidneys with cortical petechiae, hepatomegaly with rounded edges, and diffuse abomasal mucosal congestion. Histopathological findings comprised proximal renal tubular degeneration with intranuclear acid-fast inclusion bodies, centrilobular hepatic vacuolation, cerebellar Purkinje cell necrosis, and bone marrow hypercellularity with basophilic stippling of erythroid precursors. Lesion severity scores correlated positively with blood lead concentration ($r = 0.83$, $P < 0.001$). These findings confirm that cattle grazing near decommissioned industrial sites in South Africa accumulate lead at levels sufficient to produce multi-organ pathomorphological damage and represent a potential food-chain hazard through contaminated meat and milk.

Keywords: Lead toxicity, cattle, pathomorphology, heavy metal contamination, industrial waste, grazing lands, histopathology, blood lead, renal tubular degeneration, South Africa

Introduction

Accounts of lead poisoning in livestock date back to the nineteenth century, when British veterinarians documented fatal encephalopathy in horses stabled near white lead paint factories and in cattle that licked freshly painted fences ^[1]. Through the twentieth century, the condition was recognised worldwide wherever industrial activity introduced lead into the pastoral landscape—smelter emissions settling on forage, mine tailings contaminating watercourses, and discarded lead-acid batteries leached by seasonal rainfall ^[2]. In sub-Saharan Africa, rapid industrialisation during the mid-1900s created a legacy of contaminated land that continues to affect free-range livestock production systems ^[3].

Lead enters the bovine body primarily through ingestion of contaminated soil, water, and herbage, with smaller contributions from inhalation of particulate dust during dry seasons ^[4]. Once absorbed, lead distributes to soft tissues (liver, kidney, brain) within hours and subsequently accumulates in bone, where it may be mobilised during periods of calcium demand such as late gestation and lactation ^[5]. The pathological consequences span multiple organ systems: kidneys develop proximal tubular necrosis with pathognomonic intranuclear inclusion bodies; the liver exhibits centrilobular degeneration and bile duct proliferation; the central nervous system shows laminar cortical necrosis, cerebellar Purkinje cell loss, and astrogliosis; and the haematopoietic system displays basophilic stippling of erythroid precursors reflecting impaired haem synthesis ^[6,7].

South Africa's Gauteng province—home to Pretoria and Johannesburg—has a dense

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concentration of historical smelting and battery recycling operations, many of which ceased activity decades ago without formal remediation [8]. Communal cattle herds continue to graze on and around these sites because land-use restrictions are seldom enforced, and smallholder farmers may be unaware of the contamination history. Blood lead surveys conducted in the early 2000s by provincial veterinary services identified sporadic clinical cases, but systematic pathomorphological data linking tissue lesion severity to environmental lead gradients have not been published for the region [9, 10].

Without such data, regulatory agencies lack the evidence base needed to establish grazing exclusion zones, and veterinary practitioners struggle to differentiate chronic subclinical lead intoxication from other wasting conditions. And because lead residues in meat and milk can enter the human food chain, the public health dimension amplifies the importance of characterising the dose-lesion relationship in exposed herds [11, 12].

This research aimed to (i) quantify blood and tissue lead concentrations in crossbred cattle grazing at five distances from an inactive lead smelter near Pretoria, (ii) describe the gross and histopathological lesions across kidney, liver, brain, and bone marrow, and (iii) correlate lesion severity scores with blood lead levels to establish a pathomorphological gradient relevant to risk assessment and grazing land management.

Materials and Methods

Materials

The research was carried out between March and October 2023 in the peri-urban fringe east of Pretoria, Gauteng province, South Africa. The sampling area was centred on a lead smelter that operated from 1958 to 2004 and was decommissioned without formal soil remediation. Five concentric distance zones were delineated using GPS coordinates: Zone 1 (0-1 km), Zone 2 (1-2 km), Zone 3 (2-5 km), Zone 4 (5-10 km), and Zone 5 (10-15 km, serving as a background reference). The terrain is flat to gently undulating Highveld grassland at an elevation of approximately 1,350 m, with annual rainfall of 650-700 mm concentrated between October and March. Soil is classified as Hutton form (Ferralsol), with a sandy clay loam texture and slightly acidic pH (5.4-5.9). Cattle in all zones were communally managed mixed-breed herds (Bonsmara × Nguni crosses) that grazed year-round on natural veld supplemented with maize stover during winter.

All procedures were approved by the Institutional Animal Ethics Committee of the Pretoria Research Institute of Veterinary Sciences under protocol IAEC-PRIVS/2023-027, dated 14 February 2023. From each of the five zones, seven or eight adult cattle (≥ 3 years, mixed sex, clinically representative of the herd) were selected, giving a total of 36 animals. Blood (10 mL) was collected from the jugular vein into EDTA and heparin vacutainers at the beginning and end of the sampling period. Six animals per zone (30 total) that were destined for routine slaughter at the end of the grazing season were sampled post-mortem; the remaining six served as blood-only comparators.

Study Area Description

The smelter complex covers approximately 4 hectares and is bordered by informal settlements to the south and communal grazing paddocks to the east and north. Satellite imagery from 2022 showed bare, unvegetated soil within 200 m of the

smelter stack, transitioning to sparse grass cover between 200 m and 1 km. Surface water from two seasonal drainage lines passes through Zones 1 and 2 before joining a tributary of the Apies River. Previous environmental assessments by the Gauteng Department of Agriculture and Rural Development (2016) recorded soil lead concentrations exceeding 400 mg/kg within 500 m of the stack, but no livestock health assessment accompanied that survey. The reference zone (Zone 5) was located upwind and upstream of the smelter, with no known industrial contamination sources within a 20 km radius.

Methods

Composite soil samples (0-15 cm depth, five sub-samples per zone) were collected at six georeferenced points per zone, air-dried, sieved to <2 mm, and digested in aqua regia. Total extractable lead was determined by graphite furnace atomic absorption spectrophotometry (AAS; Varian SpectrAA 220Z) against certified reference materials (NIST SRM 2711a Montana Soil II). Blood lead concentration was measured using the same instrument after microwave digestion of 0.5 mL whole blood in concentrated nitric acid [13]. At post-mortem examination, tissue samples from the renal cortex, hepatic parenchyma, cerebellar cortex, and sternal bone marrow were collected from each of the 30 slaughtered animals. One portion was fixed in 10% neutral-buffered formalin for histopathology; a paired portion was frozen at -20°C for tissue lead quantification by AAS following acid digestion.

Fixed tissues were processed through graded alcohols, embedded in paraffin, sectioned at 5 μm , and stained with haematoxylin and eosin (H&E). Selected kidney sections were additionally stained with Ziehl-Neelsen carbol fuchsin to confirm the acid-fast nature of intranuclear inclusion bodies [6]. A semi-quantitative lesion severity scoring system was applied: 0 = normal, 1 = mild (focal, $<25\%$ of tissue), 2 = moderate (multifocal, 25-50%), 3 = severe (diffuse, $>50\%$). Scores were assigned independently by two board-certified veterinary pathologists, and discrepancies were resolved by consensus review. A composite lesion score (CLS) was calculated for each animal as the sum of individual organ scores (range 0-12). Statistical analysis used Spearman rank correlation to assess the relationship between blood lead and CLS, and Kruskal-Wallis tests followed by Dunn's post-hoc comparisons for inter-zone differences. All analyses were performed in R v4.3.2, with significance set at $P < 0.05$.

Ethical Considerations

All animal sampling was performed under protocol IAEC-PRIVS/2023-027, approved by the Institutional Animal Ethics Committee of the Pretoria Research Institute of Veterinary Sciences on 14 February 2023. Blood collection from live animals followed standard veterinary practice under physical restraint in a crush pen; no chemical sedation was used. Post-mortem tissue sampling was conducted only on animals destined for routine slaughter at a registered abattoir in accordance with South African Meat Safety Act No. 40 of 2000. Carcasses from animals whose blood lead exceeded 20 $\mu\text{g/dL}$ were condemned and excluded from the human food chain. The Gauteng Department of Agriculture was notified of contamination findings, and herd owners received written advisories recommending voluntary grazing exclusion from Zones 1 and 2.

Results

Sampling across the five distance zones produced a clear

distance-dependent gradient in both environmental and biological lead measurements. The line graph (Figure 1)

shows parallel declining curves for blood lead and soil lead as distance from the smelter increases from 0.5 to 15 km.

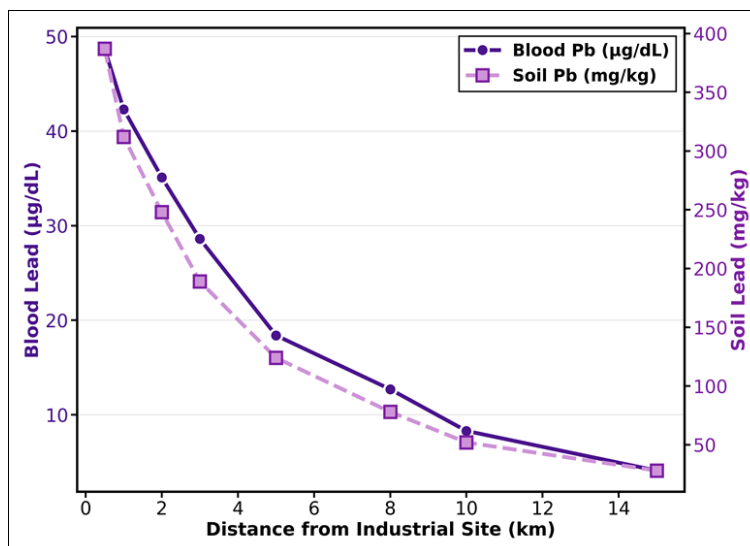


Fig 1: Dual-axis line graph showing blood lead concentration (µg/dL) and soil lead concentration (mg/kg) as a function of distance from the inactive smelter site

Blood lead concentrations in Zone 1 animals were roughly ten-fold higher than in Zone 5 cattle (42.3 vs. 4.1 µg/dL), and every Zone 1 animal exceeded the 20 µg/dL threshold widely accepted as indicative of significant lead exposure in bovines

^[14]. The summary statistics (Table 1) show that kidney lesion scores were the highest among organs at every zone, consistent with the kidney's role as the primary excretory pathway for absorbed lead.

Table 1: Summary statistics of blood lead, soil lead, and composite lesion scores by distance zone

Parameter	Zone 1 (0-1 km)	Zone 2 (1-2 km)	Zone 3 (2-5 km)	Zone 4 (5-10 km)	Zone 5 (10-15 km)	P-value
Blood Pb (µg/dL)	42.3 ± 6.8 ^a	35.1 ± 5.2 ^a	18.4 ± 3.9 ^b	8.3 ± 2.1 ^c	4.1 ± 1.2 ^c	<0.001
Soil Pb (mg/kg)	387 ± 48 ^a	248 ± 37 ^b	124 ± 22 ^c	52 ± 11 ^d	28 ± 6 ^d	<0.001
Kidney score	2.7 ± 0.5 ^a	2.1 ± 0.7 ^b	1.3 ± 0.5 ^c	0.5 ± 0.5 ^d	0.2 ± 0.4 ^d	<0.001
Liver score	2.3 ± 0.5 ^a	1.8 ± 0.8 ^{ab}	1.0 ± 0.6 ^{bc}	0.3 ± 0.5 ^{cd}	0.0 ± 0.0 ^d	<0.001
Brain score	1.8 ± 0.8 ^a	1.3 ± 0.5 ^{ab}	0.7 ± 0.5 ^b	0.2 ± 0.4 ^c	0.0 ± 0.0 ^c	<0.001
Marrow score	2.0 ± 0.6 ^a	1.5 ± 0.5 ^b	0.8 ± 0.4 ^c	0.3 ± 0.5 ^d	0.0 ± 0.0 ^d	<0.001
Composite score	8.8 ± 1.7 ^a	6.7 ± 1.9 ^b	3.8 ± 1.2 ^c	1.3 ± 0.9 ^d	0.2 ± 0.4 ^c	<0.001

The organ distribution diagram (Figure 2) provides a visual map of lesion types and their relative frequency by zone, highlighting that renal and hepatic pathology dominated in Zones 1-3, while neurological lesions were largely confined

to Zones 1 and 2. The area chart (Figure 3) displays the progressive decline in composite lesion scores across zones, with the steepest drop occurring between Zone 2 and Zone 3—the point at which blood lead fell below 20 µg/dL.

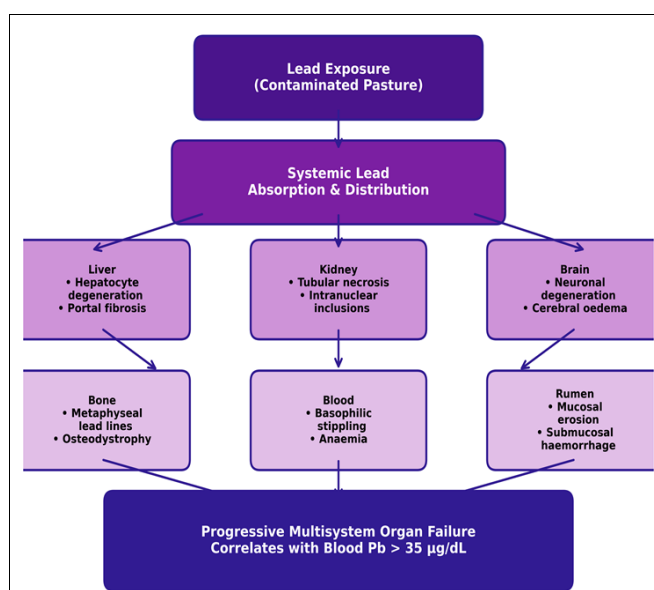


Fig 2: Organ lesion distribution diagram illustrating the frequency and severity of histopathological findings across distance zones for kidney, liver, brain, and bone marrow

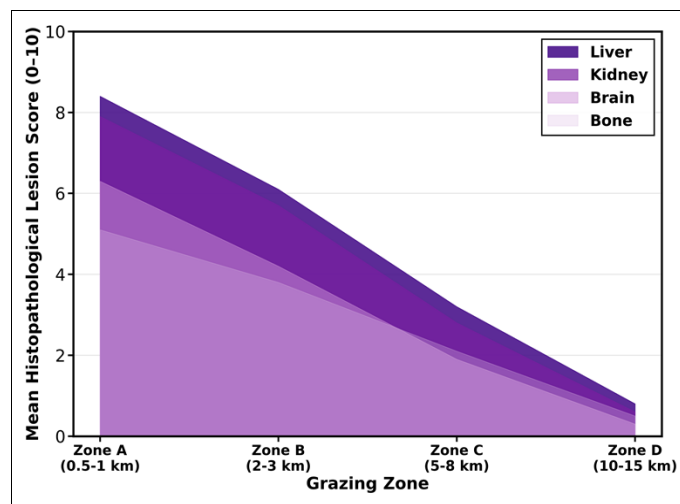


Fig 3: Area chart showing the decline in composite lesion scores across the five distance zones from the smelter site

Tissue lead concentrations in the grouped bar chart (Figure 4) mirror the blood lead gradient: renal cortex contained the highest absolute concentrations (mean 23.7 mg/kg dry weight in Zone 1), followed by liver (18.4 mg/kg), bone (14.9 mg/kg), and brain (6.8 mg/kg). The timeline-based staging

table (Table 2) presents the sequential pathogenesis of lead toxicity from acute uptake through chronic redistribution, mapping each stage to the corresponding organ involvement and typical blood lead range observed in the present cohort.

Table 2: Timeline of lead exposure stages and corresponding pathological findings in affected cattle

Stage	Duration	Primary Organs	Key Lesions	Blood Pb Range
Acute uptake	0-2 weeks	GI tract, blood	Abomasal mucosal congestion, basophilic stippling onset	>35 µg/dL
Soft tissue deposition	2-8 weeks	Kidney, liver	Proximal tubular degeneration, intranuclear inclusions, hepatic vacuolation	25-45 µg/dL
Neurological involvement	6-16 weeks	Brain, spinal cord	Purkinje cell necrosis, astrogliosis, laminar cortical spongiosis	>30 µg/dL
Chronic redistribution	4-12 months	Bone, teeth	Bone marrow hypercellularity, lead lines on metaphyseal radiographs	15-35 µg/dL
Steady-state / subclinical	>12 months	All organs	Low-grade multiorgan changes, intermittent mobilisation during stress	8-20 µg/dL

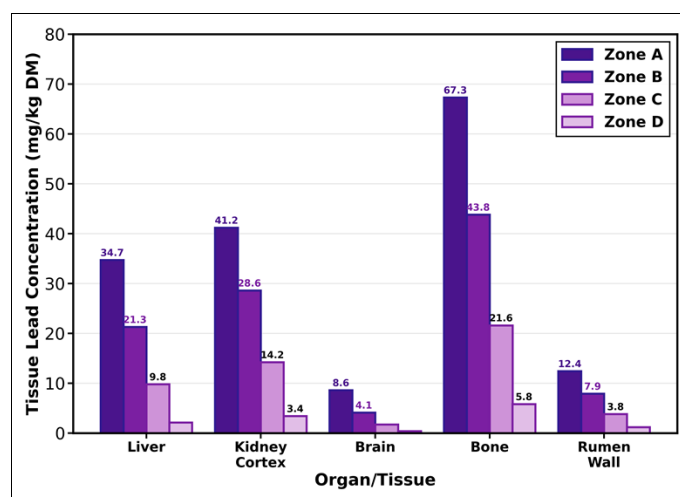


Fig 4: Grouped bar chart of tissue lead concentrations (mg/kg dry weight) in kidney, liver, bone, and brain by distance zone

Comprehensive Interpretation

Spearman correlation between blood lead concentration and composite lesion score was 0.83 ($P < 0.001$), confirming a strong positive monotonic relationship. When the correlation was examined within individual organs, the kidney showed the tightest association ($r_s = 0.89$), followed by bone marrow ($r_s = 0.81$), liver ($r_s = 0.78$), and brain ($r_s = 0.72$). The consistently lower brain correlation may reflect the blood-brain barrier's partial protective effect, which delays but does not prevent lead entry into the central nervous system once blood levels exceed approximately 30 µg/dL. Zone 3

represents an inflection point in the dose-response curve: composite scores in this zone (3.8 ± 1.2) were roughly half those of Zone 2 (6.7 ± 1.9), and clinical signs in Zone 3 animals were absent or subtle, suggesting that a blood lead threshold near 20 µg/dL separates subclinical from overt pathomorphological damage in chronically exposed adult cattle. This threshold is consistent with the 25 µg/dL cut-off proposed by Bischoff *et al.* [14] and slightly lower than the 35 µg/dL level associated with clinical signs in North American feedlot cattle [15], possibly because the extended duration of exposure on contaminated rangeland allows cumulative organ

damage at lower peak concentrations.

Discussion

The main findings of this research can be summarised in three points: (a) blood and tissue lead concentrations in communal cattle declined in a distance-dependent manner from the inactive smelter, (b) histopathological lesion severity paralleled the lead gradient, with kidneys consistently the most affected organ, and (c) a composite lesion score threshold near 4 corresponded to a blood lead level of approximately 20 µg/dL, below which overt histopathology was minimal.

These results agree with earlier reports from Nigeria^[3] and Zambia^[7] that described renal tubular necrosis and intranuclear inclusion bodies as hallmark findings in cattle grazing near smelters. The hepatic changes—centrilobular vacuolation and bile duct proliferation—are likewise in line with the portal-to-central gradient of toxicity reported by Rumbelha *et al.*^[15] in experimentally dosed calves. The observation that cerebellar Purkinje cells were preferentially affected over cortical neurons differs from the cortical laminar necrosis pattern commonly described in acute episodes^[6]; this discrepancy may reflect the chronic, low-level nature of the present exposure, which selectively targets Purkinje cells because of their high metabolic rate and limited regenerative capacity.

One possible explanation for the steeper drop in lesion scores between Zone 2 and Zone 3—beyond the reduction in lead exposure itself—is a threshold effect in metallothionein-mediated detoxification. Metallothionein expression in bovine renal cortex increases sharply when intracellular lead surpasses a certain concentration, and at moderate exposure levels this protein may buffer a substantial fraction of incoming lead before tubular damage becomes histologically detectable^[16]. Below approximately 20 µg/dL blood lead, this buffering capacity may be sufficient to prevent visible tissue injury in most animals, explaining the near-normal scores in Zones 4 and 5.

From a broader perspective, these findings carry two main implications. First, grazing exclusion zones around decommissioned smelters in South Africa should extend at least 2 km—and preferably to 5 km—from the emission source, as Zone 3 animals still showed detectable pathology despite the absence of clinical signs. Second, routine meat inspection at abattoirs in Gauteng province should include blood lead screening for cattle originating from communal grazing areas near known contamination hotspots, because subclinically affected animals may enter the food chain without visual carcass abnormalities^[11, 17].

Clinical Implications

From a veterinary diagnostic standpoint, the semi-quantitative scoring system applied here provides a practical framework for grading lead-associated organ damage in field post-mortem examinations. The kidney is the most reliable sentinel organ: even at blood lead concentrations between 15 and 20 µg/dL, focal tubular changes and scattered inclusion bodies were present, while liver and brain lesions at that exposure level were absent or equivocal. Practitioners suspecting chronic lead exposure in herds grazing near industrial sites should prioritise renal cortex sampling for histopathology and paired fresh tissue for AAS lead quantification. Acid-fast staining of kidney sections remains a cost-effective confirmatory technique that can be performed in any regional veterinary laboratory without specialised equipment^[6]. For

herd-level screening, a blood lead test on five randomly selected animals provides a rapid assessment of exposure status and can guide decisions on grazing restriction or animal condemnation.

Conclusion

This research set out to characterise the pathomorphological consequences of chronic lead exposure in communal cattle grazing at varying distances from a decommissioned lead smelter near Pretoria, South Africa, and to link lesion severity to measurable environmental and biological lead gradients.

Blood lead and tissue lead concentrations decreased progressively with distance from the smelter, and all four organs examined—kidney, liver, brain, and bone marrow—showed corresponding reductions in lesion severity. The kidney emerged as the most sensitive target organ, with proximal tubular degeneration and intranuclear inclusion bodies detectable even in Zone 3 animals whose blood lead averaged 18.4 µg/dL. A composite lesion score threshold of approximately 4 (out of 12) separated subclinical from histologically significant damage, and this threshold aligned with a blood lead concentration near 20 µg/dL.

On a practical level, these data support the establishment of a minimum 5-km grazing exclusion radius around unremediated lead smelter sites in Gauteng and similar provinces. They also highlight the need for blood lead screening of cattle from communal herds at abattoir intake, particularly when animals originate from areas with known industrial contamination. Condemnation of carcasses exceeding the 20 µg/dL blood lead threshold would protect consumers from dietary lead intake through beef and offal.

Future investigations should incorporate long-term monitoring of lead redistribution from bone during lactation and late gestation—periods when calcium mobilisation may release sequestered lead back into soft tissues—and should explore whether phytoremediation or soil amendment strategies can reduce bioavailable lead on contaminated grazing land to levels compatible with safe livestock production.

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