

## COPPER TOXICITY IN RUMINANTS: AIR POLLUTION AS A POSSIBLE CAUSE

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### ABSTRACT

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Pathological findings and liver and kidney analyses confirmed that cattle had died of chronic copper poisoning on a farm in the north-eastern Transvaal. This is the first known published record of chronic copper intoxication of cattle in southern Africa. An epidemiological study revealed that a source of copper was air pollution which could have arisen from a nearby copper smelting unit. Buffalo and impala in an adjacent area of the Kruger National Park were found to have significantly higher liver copper levels than animals elsewhere in the Park. Prophylactic licks, containing zinc sulphate and sulphur, seemed to be successful in protecting cattle against the effects of the copper in the contaminated grazing.

### PART I: DIAGNOSTIC INVESTIGATION

#### INTRODUCTION

##### *Pathogenesis of copper poisoning*

Copper toxicity is rare in cattle, probably because cattle are more tolerant to copper intake than other animals, such as sheep. An oral dose of 200 mg/kg copper is likely to rapidly kill an adult cow (Booth & McDonald, 1982). Intoxication may, however, also result when low copper levels (20–125 mg/kg) are ingested over long periods (Booth & McDonald, 1982). This is because most of the copper absorbed from the intestine in ruminants accumulates in the liver which can only store a limited amount. High liver copper concentrations eventually inhibit essential enzymes and result in liver malfunction and ultimately necrosis. The liver then releases large amounts of copper and other oxidative substances into the blood stream where they cause vascular damage, loss of fluid and an increased blood colloid osmotic pressure and packed cell volume. Stress is often the trigger for this release. A sudden massive lysis of erythrocytes then occurs, with resultant renal failure, caused by clogging of the renal tubules with haemoglobin and the necrotizing effect of the high copper levels on the tubular cells. A high mortality rate and sudden death occurs once signs of the haemolytic crisis are seen. This sequence of events is termed chronic copper poisoning, but the term is misleading, as the ingestion of copper is chronic, while the onset of toxicosis is acute owing to the sudden release of copper from the liver.

##### *History of outbreak*

In May 1989, reports were received that 39 cattle in the Phalaborwa area had died after showing signs characteristic of a haemolytic crisis, i.e., depression, anaemia, haemoglobinuria, jaundice and constipation, followed by sudden death. A presumptive diagnosis of copper poisoning was made by Dr J. G. Pienaar of the State Veterinary Laboratory at Potgietersrus.

##### *Objectives of the investigation*

The objectives of the investigation were to confirm the diagnosis of copper poisoning, establish the source/s of the copper and prevent further mortalities.

### MATERIALS AND METHODS

#### *Macropathology*

Necropsies were performed on 3 cattle that had died on the farm.

#### *Histopathology*

Various tissues from the 3 cattle were collected in 10 % buffered formalin, sectioned, and stained routinely with haematoxylin and eosin (HE) for examination under the light microscope. The kidney, liver and brain were examined from all 3 cattle, the spleen from 2 of them, and the lung, rumen and omasum from one of the cattle.

#### *Liver copper analysis*

Portions of livers were collected in 10 % formalin from 15 animals that had died on the farm between the beginning of May and the end of July. Eight of these animals had accompanying kidney samples. These organs were analysed for copper concentrations, using an atomic absorption spectrophotometry method (Boyazoglu, Barrett, Young & Ebedes, 1972).

### RESULTS

#### *Macropathology*

All carcasses showed a moderate to severe icterus, excess fluid in the body cavities and pericardial sac, enlarged friable livers, severe congestion of the spleen and kidneys and a marked haemoglobinuria, all of which are consistent with copper poisoning (Booth & McDonald, 1982).

#### *Histopathology*

(a) Kidney: Moderate to severe hyperaemia was present throughout all regions of the kidney. The renal lesions were characterized by a haemoglobinuric nephrosis which differed in extent and severity from case to case. Scattered throughout the cortex were large focal groups of tubules exhibiting intense eosinophilia which was ascribed to a severe hyaline droplet degeneration of the tubular cells and the presence of haemoglobin casts within the tubular lumens and dilated Bowman's spaces of the glomeruli. The remaining cortical tubules revealed hydropic degeneration or lysis. Small isolated groups of regenerating tubules were also present. There was a mild mesangial cell proliferation of the glomeruli together with mild hydropic or hyaline degeneration of some glomerular cells. Areas of acute infarction characterized by coagulative necrosis were seen in 2 cases. One case also revealed severe venous throm-

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TABLE 1 Cattle liver and kidney copper concentrations (ppm WM)

Animal No.	Liver conc.	Kidney conc.	Animal No.	Liver conc.	Kidney conc.	Animal No.	Liver conc.	Kidney conc.
1	465*	—	6	277	—	11	161	7,5
2	600*	45	7	296	—	12	278	8,1
3	350*	21	8	563	—	13	434	81
4	258	—	9	233	37	14	444	83
5	343	—	10	462	6	15	228	—

\* = animals where histopathology was done

bosis and vasculitis. The arteries of all 3 cases showed a mild vacuolar degeneration of the cells of the tunica media.

The medulla revealed moderate diffuse hydropic degeneration with some protein and cytoplasmic debris in the lumens of the collecting tubules. A few focal isolated areas of hyaline droplet degeneration with associated haemoglobin casts were present.

(b) Liver: The livers showed fatty degeneration, moderate to severe intracanalicular and intraductule bile stasis with intracellular accumulation of bile pigments, moderate bile ductule proliferation, and mild portal fibrosis and round cell infiltration.

Fatty degeneration was diffusely present throughout the hepatic lobules but was more pronounced centrilobularly. Single or focal groups of hepatocytes, particularly in the centrilobular regions, exhibited lytic or coagulative necrosis. Some of the latter foci were infiltrated by neutrophils.

(c) Spleen: A moderate to severe atrophy of the white pulp and severe congestion of the red pulp were evident.

(d) Brain: The brain showed moderate congestion and mild oedema.

(e) Rumen and omasum: No significant lesions were evident in these organs.

#### Liver copper analysis

Liver copper levels ranged from 161 ppm to 600 ppm on a wet mass basis (WM) (Table 1), with a mean value of  $359,4 \pm 129,3$  ppm [i.e.  $c 1078,2 \pm 387,9$  ppm dry mass basis (DM)]. Kidney copper levels ranged from 6 to 83 ppm WM (Table 1), with a mean value of  $36,1 \pm 31,7$  ppm (i.e.  $c 108 \pm 95,1$  ppm DM).

## DISCUSSION

### Pathology

The principal pathological lesions could all be attributed to the effects of a haemolytic crisis. Based on the findings of high copper levels and the absence of *Babesia* or *Anaplasma* parasites in the erythrocytes, the conclusion was reached that the haemolysis was the result of an excessive copper intake.

The haemoglobinuric nephrosis was directly attributable to the haemolysis. Degeneration and necrosis of the renal tubular cells were probably due in part to the effects of haemolysis as well as the direct toxic effect of the high copper levels (Booth & McDonald, 1982).

Fatty degeneration of the livers was ascribed to the anaemia induced by the haemolysis. Accumulation of bile in the bile channels and bile pigment in the hepatocytes were seen as evidence of a breakdown of erythrocytes. Lytic and coagulative necrosis of hepatocytes, which accompanied the other liver

lesions, may have been due either to anaemia or the direct toxic effects of the copper.

A mild cerebral oedema was probably the result of the anaemia.

### Organ analysis

Examination of the literature revealed that the liver copper concentrations (Table 1) were consistent with known cases of copper poisoning in cattle. Mylrea & Byrne (1974) reported that calves, in which they had induced copper toxicosis by subcutaneous injection, had liver copper levels of 290 ppm DM ( $c 97$  ppm WM) or greater [mean = 558 ppm DM ( $c 186$  ppm WM)] and kidney copper levels of 15 to 97 ppm DM ( $c 5-32$  ppm WM) (mean = 39 ppm DM). Shand & Lewis (1957), on the other hand, found the liver copper levels of calves, poisoned by a milk substitute powder containing 150 ppm copper, to be 1400–2500 ppm DM. Field cases of copper toxicity in cattle that could be traced back to copper smelting units were reported by Wiemann (1939) and Parada (1987). The mean liver copper levels of these field cases were 196 ppm (basis unknown) in Wiemann's (1939) case and 925,7 ppm DM in Parada's. It would therefore seem that a wide range of liver copper levels can be expected in copper poisoning of cattle but that these levels are consistently higher than the average liver copper levels for normal cattle, reported as 50–85 ppm WM (Spector, 1956; Heine, Dekker & Hudson, 1989). The liver copper levels obtained in our investigation were therefore consistent with those associated with copper poisoning elsewhere, and it was concluded that these animals had died of copper poisoning. This conclusion was further supported by the kidney copper levels which in all cases exceeded the minimum 5 ppm WM concentration suggested by Mylrea & Byrne (1974) for calves and in 62,5 % of the cases exceeded the 15 ppm WM concentration laid down by Osweiler, Carson, Buck & Van Gelder (1985) for sheep. For the purpose of this paper liver copper levels in excess of 150 ppm WM (450 ppm DM) were considered as diagnostic for copper poisoning in cattle.

## PART II: EPIDEMIOLOGICAL INVESTIGATION

### INTRODUCTION

Having proved that cattle on the farm were dying of copper poisoning an epidemiological investigation was undertaken to locate the source of the copper.

An area survey established that a large opencast copper mine, with a smelting unit, was situated in the vicinity of the farm (Fig. 1). Weather data from the local airport revealed that the prevailing wind direction was from the south-east, which meant that any emissions from the mine would be blown predominantly in a north-westerly direction towards the



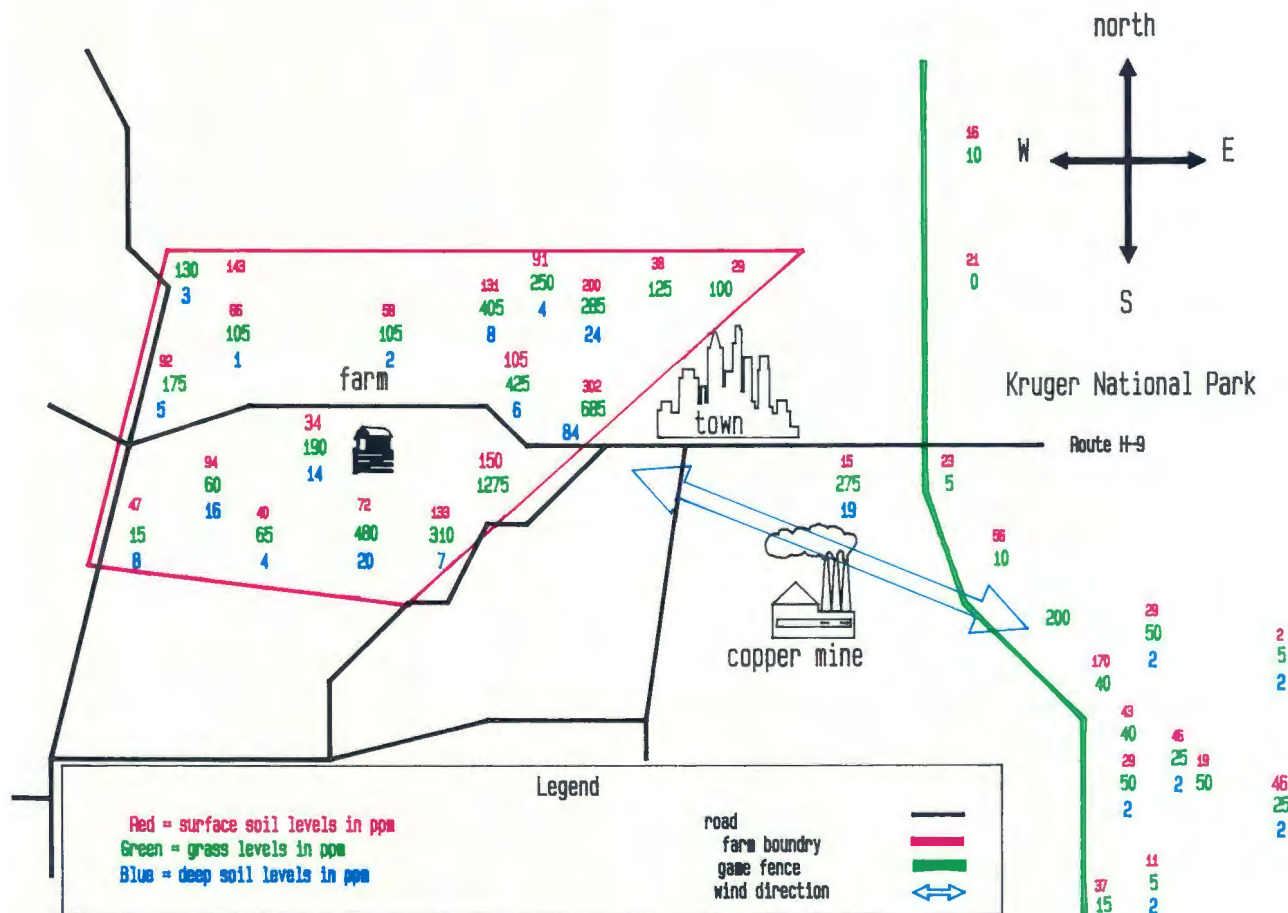


FIG. 1 The distribution of soil and grass copper levels given on a dry mass basis in ppm

farm (Fig. 1). Less frequently the wind blew the opposite way, which was in the direction of the Kruger National Park (Fig. 1).

From these observations it was postulated that the source of the copper that caused the toxicities was either air pollution or a copper-rich soil.

MATERIALS AND METHODS

Sampling procedures

To investigate the above postulation, 86 soil and grass samples were collected on the farm and game reserve at the points indicated on the map (Fig. 1). Soil samples were taken from the surface layer (top 2-3 cm) and again at a depth of approximately 30 cm. A bulk grass sample was taken at each sampling point (Fig. 1) and consisted of the major grass types growing in the immediate radius of the sampling point. In addition, 5 crude, filter paper traps were set up on the farm no less than 2 m off the ground to catch particulate wind-borne matter. Three traps were placed in the game reserve but were destroyed by baboons before any data could be accumulated from them. The traps consisted of circular (diameter = 22 cm) filter paper discs (grade 1, Whatman) fitted into the base of a bucket that had a mouth diameter of 30 cm. The surface of the filter paper was moistened with liquid paraffin after being inserted into the bucket to ensure that dust particles remained adhered to it. The copper content of the filter paper was determined each month.

Method of analysis

The soil, grass and filter paper samples were pro-

cessed and analysed for copper content according to the analytical methods for atomic absorption spectrophotometry published by The Perkin-Elmer Corporation, Norwalk, Connecticut, USA.

RESULTS

The results of the soil and grass analysis done in May are reflected in Fig. 1. The mean level of copper in the surface soil on the farm was  $103,1 \pm 73,9$  ppm and that of the deep soil  $16,6 \pm 21,4$  ppm. In the game reserve the surface soil levels in the areas east and south-east of the mine averaged at  $67,5 \pm 63$  ppm and the deep soil at  $2 \pm 0$  ppm. Surface soil copper levels in the game reserve north and far east of the mine averaged at  $14,6 \text{ ppm} \pm 8,4$  ppm. Analysis of filter papers for the month of June showed copper levels ranging from 20-141 ppm DM at various points on the farm. The average for June was  $103,75 \pm 45$  ppm. The areas of highest filter paper catchment corresponded to the areas with highest copper levels in grass and soil. These levels dropped for the month of July to an average of 43,85 ppm, which coincided to the time when the mine commissioned its new electrostatic precipitator. Four of the grass samples were analysed before and after rinsing (Table 2).

TABLE 2 Copper levels in rinsed and unrinsed grass (ppm DM)

Unrinsed grass	Rinsed grass	Unrinsed grass	Rinsed grass
1275	945	425	350
250	205	190	100



## DISCUSSION

The levels of copper in the grass and surface soil downwind of the smelting unit were above the accepted maximum of 10 ppm in grass (Osweiler *et al.*, 1985). Samples taken outside the direction of the prevailing wind from the smelting unit and those further away from the unit had lower levels of copper. This trend was later confirmed by the filter paper determinations. The distribution of copper therefore appeared to be related to the direction of the prevailing winds and the distance from the smelting unit. The copper levels of the deep soil samples were in almost all cases considerably lower than those of the surface soil and in most cases were within acceptable levels. This significant difference between surface copper levels and deeper levels suggested that the main source of the copper was air pollution. In support of this, the lower levels of copper in washed grass suggested that much of the copper was contained in dust on the plants.

## PART III: TREATMENT AND ITS EVALUATION

## INTRODUCTION

Owing to the lack of available information on the treatment of copper toxicosis in cattle, data for sheep were extrapolated to cattle. The 2 drugs commonly used for prophylaxis in sheep are molybdate (usually ammonium molybdate) and sulphate (usually sodium sulphate) (Booth & McDonald, 1982), with some evidence that sulphur and zinc may also be beneficial (G. Bath, Faculty of Veterinary Science, University of Pretoria, personal communication, 1989; Cousins, 1985). Successful licks used by sheep farmers in the copper-rich Karoo areas of the country also contained zinc sulphate and sulphur (Bath, G., personal communication, 1989). With this background knowledge, a treatment lick was formulated for the farmer. At first a lick containing ammonium molybdate was envisaged, but a lack of availability and the high cost of the compound precluded its use. By trial and error it was found that the lick best accepted by cattle contained: 200 kg of salt, 150 kg of yellow maize, 100 kg of high protein concentrate, 80 kg of molasses meal, 50 kg of dicalcium phosphate, 12 kg of sulphur and 400 g of zinc sulphate. Such a lick served 20–30 animals and, if each animal consumed 300–800 g per day, this gave a rough dose of 6–16 g of sulphur and 200–500 mg of zinc sulphate per animal per day. It was recommended that the lick be used for an indefinite period. Before the lick was manufactured an interim treatment of 500 mg of ammonium molybdate and 2 g of zinc sulphate was dosed orally per day per animal for the first 3 days of the trial, after which the lick was put into use. Some of the more severely affected animals continued to receive oral treatment for another 2–3 days in addition to the lick.

## MATERIALS AND METHODS

Ninety-six cattle out of a herd of 180 on the farm were bled by means of venipuncture on Day 1 of treatment. The resultant serum samples and all subsequent serum samples were analysed for the enzyme gamma glutamyltransferase (GGT), using Boehringer Mannheim, France SA, CBR kits (Cat No. 543101), according to the method described by the manufacturers (Persijn & Van der Slik, 1976). From these results 15 cattle were selected for further analysis. Thirteen of these had high GGT values (>45 U/l) and 2 had low GGT values (<10 U/l).

These 15 cattle were bled again 21 days and 64 days after initial exposure to the lick. Seven of this group were bled a 4th time 147 days after treatment had begun. Forty-nine animals were selected from the herd 147 days after commencement of treatment to assess the herd's GGT status at that time. Serum GGT levels >25 U/l were taken as indicative of liver damage. This figure was based on in-house normal values obtained over many years of analysis.

## RESULTS

The mean GGT levels for Days 1, 21, 64 and 147 of the respective groups are shown in Table 3.

TABLE 3 Mean GGT levels for sample groups at various time intervals

Days since treatment commenced	Mean GGT For n=15	Mean GGT For n=7	Herd GGT	% of herd with liver damage
1	75,6	71,1	32,7	42,71 %
21	59,7	57,3	—	—
64	41,0	34,7	—	—
147	—	14,4	22,7	22,45 %

## DISCUSSION

The serological results confirmed that a large percentage (43 %) of the herd had liver damage. Prophylactic treatment with zinc sulphate and sulphur appeared to have an advantageous effect as only 22 % of the herd showed liver damage after 147 days of treatment compared with an initial 43 %. This trend also occurred in the selected groups of 15 and 7 animals which showed a steady decline in GGT activity over the period of investigation to a point where eventually all in the group of 7 animals had normal GGT activity. A protracted period of prophylactic treatment however, does seem to be necessary to bring the liver enzyme activity back to normal.

## PART IV: IMPACT ON WILDLIFE

## INTRODUCTION

Examination of the soil and grass copper levels from the game reserve (Fig. 1) indicated that the wildlife was possibly at risk. This led to an expansion of the investigation to try to establish the extent to which the wildlife in that area of the game reserve was being affected by high copper levels. To prevent unnecessary killing of wildlife, only those animals which were being routinely culled for conservation purposes or were found dead were used in the investigation. The 2 species thought to be the best indicators of possible problems were buffalo and impala. Both species tend to remain area bound for long periods of time, if grazing is sufficient.

Furthermore, as they are to a large extent grass eaters they were considered to be the species most at risk.

## MATERIALS AND METHODS

*Buffalo*

In August 1989 57 buffalo were shot in the Kruger National Park (KNP) during routine culling operations. Thirty-seven of these shot in an area designated X, east of the KNP boundary fence, south of route H-9, west of the Tshutshi River and north of the Olifants River (Fig. 2). This area was situated within a 10 km radius east and south-east of the mine





FIG. 2 The liver and kidney copper levels for individual impala shot during October and November given on a wet mass basis in ppm

and town. The remaining 20 buffalo came from the areas Dan Punda, Shitshova, Nkovakulu and Lala-Palms, all situated within the Kruger National Park (KNP). These areas were designated A, B, C & D respectively. Five buffalo were shot at each of the latter locations. None of these locations were closer than 100 km to the mine. The buffalo shot at A, B, C & D were used for control purposes. The liver of each buffalo was analysed for its copper concentration according to the method described above.

*Impala*

A total of 21 impala were shot and 2 were found dead during the month of October 1989. Of those shot, 5 were shot as controls at Skukuza (designated area E), which is situated more than 100 km from the mine. A further 6 were shot at Phalaborwa, either near the boundary fence but north of route H-9 and east of route H14, or far to the east just beyond the Mulalani River. These areas, designated Y, were considered to be outside any pollution zone but still within the greater Phalaborwa area (Fig. 2). The 6 impala from area Y would determine if natural background levels of copper were having any influence on the game. The remaining 10 culled and the 2 dead impala came from the area X described above, which was the area suspected of being polluted (Fig. 2). Liver and kidney copper levels were determined for each of the animals concerned.

A second study was done on impala in the month of November 1989 when 5 impala were shot east and south-east of the mine in what was essentially area X but differing in that 3 of the impala were shot west instead of east of the boundary fence (Fig. 2). Eleven impala were shot in area Y or on the south

side of the Olifants River (Fig. 2). These 11 impala were used to further establish the possible effect of background levels of copper in the Phalaborwa area on organ levels.

*Statistical analysis*

The mean liver and kidney copper levels for both species in the various areas investigated were compared, using simple one-way analysis of variance together with Scheffé's method of multiple comparisons (Browne, 1985).

RESULTS

*Buffalo*

The mean liver copper concentration of buffalo shot in area X (Table 4) was  $80,4 \pm 35,7$  ppm WM. Buffalo shot at A had a mean liver copper concentration of  $28,8 \pm 12$  ppm WM; those at B,  $33,6 \pm 15$  ppm WM; those at C,  $18 \pm 4,2$  ppm WM; and those at D,  $18,6 \pm 12,4$  ppm WM (Table 4). The overall mean liver copper concentration for buffalo shot away from Phalaborwa (A, B, C & D) was  $24,75 \pm 12,7$  ppm WM. The difference between the means of buffalo shot in other regions of the KNP (A, B, C & D) and those shot in the Phalaborwa area (X) was significant ( $\alpha = 0,05$ ). No significant difference existed between the means of the buffalo shot at A, B, C & D.

*Impala*

The organ copper levels for individual animals for October and November are shown in Fig. 2. The levels, in ppm WM, are placed at the appropriate point where each impala was shot.

The mean liver and kidney concentrations for

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TABLE 4 Liver copper levels of buffalo shot in the Kruger National Park

X - Buffalo shot in the Phalaborwa region

Animal	Sex	Age	ppm Cu	Animal	Sex	Age	ppm Cu	Animal	Sex	Age	ppm Cu
1	M	5 m	54	14	M	30 m	57	26	F	42 m	45
2	M	6 m	99	15	M	30 m	111	27	F	42 m	39
3	M	6 m	78	16	M	30 m	15	28	F	42 m	39
4	M	12 m	144	17	F	30 m	120	29	F	42 m	132
5	F	12 m	81	18	F	30 m	42	30	F	42 m	78
6	M	18 m	42	19	F	30 m	84	31	F	54 m	69
7	M	18 m	81	20	F	30 m	81	32	M	54 m	126
8	F	18 m	81	21	F	30 m	126	33	F	60 m	90
9	M	24 m	87	22	F	30 m	132	34	M	60 m	72
10	M	24 m	75	23	F	30 m	27	35	F	>60	69
11	M	24 m	165	24	F	42 m	48	36	F	>60	75
12	M	30 m	57	25	F	42 m	129	37	F	>60	87
13	M	30 m	39								

Animal	Sex	Age	ppm Cu	Animal	Sex	Age	ppm Cu
A- Buffalo shot at Dan Punda				C- Buffalo shot at Nkovakulu			
1	F	8 m	24	1	F	6 m	24
2	M	12 m	45	2	M	18 m	15
3	F	old	33	3	F	30 m	15
4	F	8 yr	12	4	F	4 yr	15
5	F	10 yr	30	5	F	6 yr	21
B- Buffalo shot at Shitshova				D- Buffalo shot at Lala-Palms			
1	F	10 m	51	1	F	22 m	18
2	F	16 m	42	2	F	12 m	6
3	F	3 yr	39	3	M	3,5 yr	12
4	M	4 yr	21	4	F	4 yr	18
5	F	8 yr	15	5	M	6 yr	39

F = Female; M = Male; m = months; yr = years  
ppm Cu = ppm copper on a wet mass basis

impala shot in October in various parts of the Park were as follows:

- (a) at E (Skukuza): liver =  $25,8 \pm 15,96$  ppm; kidney =  $3,6 \pm 1,34$  ppm;
- (b) at X (south of route H-9, east of the boundary fence, west of the Tshutshi River and north of the Olfiants River) (Fig. 2): liver =  $226,67 \pm 96,68$  ppm; kidney =  $11,67 \pm 1,80$  ppm;
- (c) at Y (north of route H-9 and east of the Tshutshi River) (Fig. 2): liver =  $88,28 \pm 46,05$  ppm; kidney =  $7,28 \pm 1,60$  ppm.

It was shown that the mean liver copper levels of the impala shot in area X [i.e. east and south-east of the copper smelting unit (Fig. 2)] were significantly ( $\alpha = 0,05$ ) higher than the liver copper levels of impala at E (Skukuza) and of those of impala shot at Y (north-east or far east of the smelting unit). No significant difference between the mean liver copper levels of impala shot at E (Skukuza) and impala shot at Y (north east of the mine) could be demonstrated. The liver copper levels for the impala shot at E were of the same magnitude as those reported by Boyazoglu *et al.* (1972) (i.e.  $26,9 \pm 11,9$  ppm) and were considered normal for impala.

The mean liver and kidney concentrations for impala shot in November 1989 were as follows:

- In area Y: liver =  $64,6 \pm 26$ ; kidney =  $5,7 \pm 2,8$ .
- In area X: liver =  $161,2 \pm 79,5$ ; kidney =  $7,8 \pm 3,4$ .

A significant difference ( $\alpha = 0,05$ ) in mean liver copper levels still existed between impala shot at X and those shot at Y for the month of November but

TABLE 5 Liver and kidney copper levels for impala shot in the KNP during October and November 1989

October					
Area	Liver Cu ppm WM	Kidney Cu ppm WM	Area	Liver Cu ppm WM	Kidney Cu ppm WM
X	138	9	Y	90	9
X	261	12	Y	96	6
X	207	12	Y	42	6
X	444	9	Y	51	6
X	279	12	Y	57	6
X	129	12	Y	105	9
X	222	12			
X	153	15	E	18	3
X	177	9	E	3	3
X	207	15	E	39	3
X	153*	141*	E	27	6
X	132*	90*	E	42	3

\* = impala found dead

November					
Area	Liver Cu ppm WM	Kidney Cu ppm WM	Area	Liver Cu ppm WM	Kidney Cu ppm WM
X	222	6	Y	63	3
X	264	9	Y	45	6
X	144	12	Y	48	6
X	89	9	Y	33	6
X	87	3	Y	69	6
			Y	48	3
			Y	48	3
Y	66	3	Y	111	12
Y	66	6	Y	114	9



again no difference in liver copper levels could be established between unexposed impala shot at E in October and impala shot at Y in November.

#### DISCUSSION

The mean liver copper level of 24,75 ppm for the buffalo shot in the 4 areas away from Phalaborwa compared well with Boyazoglu *et al.*'s (1972) findings of 23,7 ppm WM for normal buffalo, yet the liver copper levels of buffalo in area X (east and south-east of the mine) were significantly greater than either of these norms. This showed that buffalo in the area of suspected aerial pollution had accumulated more copper than buffalo elsewhere in the Park and were therefore potentially at risk of intoxication.

The impala results for October and November followed the same pattern as those of the buffalo and it was apparent that impala south-east of the copper smelting unit (area X) had accumulated high levels of copper. Two of the impala shown in Fig. 2 were found dead and differed from those shot by having very high kidney copper levels (141 and 90 ppm WM). It is thus possible that these 2 impala had in fact died of copper poisoning.

Since impala apparently remain fairly localized in their movements these results confirm what the soil and grass samples demonstrated, that the area of the KNP contaminated by copper was fairly well defined (Fig. 1) and supports the postulation that aerial pollution occurred. In addition, the results for both months investigated showed that impala outside the suspected pollution zone had liver and kidney levels that could not be distinguished from the norm. This strongly suggests that natural background copper levels around Phalaborwa had no significant effect on liver copper levels and supports the soil and grass results which found no evidence of a naturally occurring high copper environment.

#### CONCLUSION

The pathological, chemical and epidemiological findings, showed that cattle on the farms under investigation had died of copper poisoning, which probably arose from air pollution. It was further established that copper had some effect on certain game species in the Kruger National Park but that this effect was limited to a fairly well-defined area. Prophylactic treatment of cattle with zinc sulphate

and sulphur seemed to be successful in this case of poisoning.

#### ADDENDUM

Discussions with the mine officials revealed that during the period of investigation an electrostatic precipitator, serving the reverberating furnace, was out of operation while it was being replaced by a new, more efficient model. This new precipitator improves metallurgical performance as well as pollution control.

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