Selenium toxicosis with focal symmetrical poliomyelomalacia in postweaning pigs in South Africa

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ABSTRACT

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An outbreak of paralysis in finisher pigs in South Africa after ingestion of feed containing 54,581 mg/kg of selenium is described. The main and entirely consistent lesion was bilaterally symmetrical focal poliomalacia of the ventral horns of the spinal cord, which was most severe and consistent in the lumbar intumescence. Acute and subacute lesions were characterized by malacia with large numbers of gitter cells. The main features of chronic lesions were loss of neurons and gliosis. Focal degeneration and necrosis of the myocardium and skeletal muscles were also consistent, but there were fewer specific changes. Endothelial swelling, mild fibrinoid degeneration and perivascular leukocytic infiltration were present in the acute stage. Dermatitis, coronitis and hoof sloughing, usually present in more chronic cases of intoxication, were not a feature of the present outbreak, although alopecia and crusting were evident on the backs of a few pigs several weeks after the episode of intoxication.

Serum- and tissue-selenium levels were elevated in the early stages after intoxication. Serum levels were nearly normal in chronic cases two months after the episode, while liver and kidney levels were still higher than normal. Higher levels were found in liver, kidney and serum than in muscle, with the highest levels in the kidney. Less than 20 % of affected pigs recovered sufficiently to be marketed.

Keywords: Focal symmetrical poliomyelomalacia, paralysis, pigs, selenium toxicosis

INTRODUCTION

Selenium is an essential micronutrient in the diet of several species, including pigs (Van Houweling 1979; Stowe, Eavey, Granger, Halstead & Yamini 1992). Addition of selenium to the diet to counteract deficiency has to be carefully controlled owing to its potential toxicity (Stowe *et al.* 1992). The Food and Drug Administration (FDA) of the United State of America approved maximum levels of 0,3 mg/kg (Van Houweling 1979; Stowe *et al.* 1992).

Field cases of acute selenium toxicosis in pigs usually manifest as a paralytic syndrome (Wilson & Drake 1982; Wilson, Scholz & Drake 1983; Harrison, Colvin, Stuart, Sangster, Gorgacz & Gosser 1983; Casteel, Osweiler, Cook, Daniels & Kadlec 1985; Hill, Allison & Halpin 1985; Van der Molen, Van Beek, Baars & Timmerman 1988; Sanford 1990; Stowe *et al.* 1992; Schoder, Weissenböck, Baumgartner & Truschner 1993). All the cases in which the spinal cord was examined were characterized by segmental focal symmetrical malacia in the ventral horns, and similar lesions have been reproduced experimentally (Herigstad, Whitehair & Olson 1973; Harrison *et al.* 1983; Wilson *et al.* 1983; Wilson, Hammerstedt, Palmer &

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DeLahunta 1988; Baker, James, Hartley, Panter, Maynard & Pfister 1989). Lesions associated with selenium toxicosis have been described in other organs by various authors, in both the presence and absence of poliomyelomalacia. These include hyaline degeneration and fibrinoid necrosis of arterioles in various organs, pulmonary oedema, myocardial degeneration and necrosis, hepatic degeneration with fatty change and necrosis, hyaline degeneration of muscle, extensive pancreatic necrosis with haemorrhage and oedema, necrosis of lymphocytes in spleen, thymus and lymph nodes, bone-marrow depletion, necrosis and haemorrhage of the adrenal cortex and hydropic degeneration of kidney tubular epithelium (Miller & Schoening 1938; Orstadius 1960; Herigstad *et al.* 1973; Hill *et al.* 1985; Stowe *et al.* 1992). Alopecia, coronitis and hoof cracking have been recorded in acute and chronic natural and experimental selenium intoxication in pigs (Miller & Schoening 1938; Herigstad *et al.* 1973; Mahan & Moxon 1984; Casteel *et al.* 1985).

In this paper the lesions in an outbreak of paralysis and mortality in finisher pigs, associated with feed accidentally contaminated with high levels of selenium, are described. Segmental focal bilaterally symmetrical malacia of the ventral horns of the spinal cord was present in 20 pigs examined over a period of 79 d. To our knowledge, this is the first incidence of selenium-induced poliomyelomalacia in pigs to be reported in South Africa (Robinson & Penrith 1994; Penrith & Robinson 1995).

HISTORY OF OUTBREAK

The outbreak occurred on a farm in Gauteng, where 450 pigs from 3 months of age are accommodated during finishing for market. The pigs are Large White/Landrace F2s with some Duroc terminal sires. They are fed a complete ration of reject dog food obtained from a milling company. The ration contains a large proportion of pelleted feed from broken packets, but may at times include feed rejected for unspecified reasons, presumably relating to poor quality. The feed consignment being fed at the time of the outbreak included factory sweepings composed of spilled feed and discarded additives.

Within a day of starting to consume a new consignment of feed, pigs in certain pens developed hindlimb paresis, and assumed a dog-sitting position (Fig. 1) with rapid progression to lateral recumbency and death. Necropsies performed on the farm did not reveal any specific lesions. Since the problem appeared to be feed-related, the feed was withdrawn, and no more pigs became paralysed. A week later, the same ration was fed in other pens. Within a day, pigs in two of the pens developed signs of anorexia and vomition, most became paralysed, and further deaths occurred.
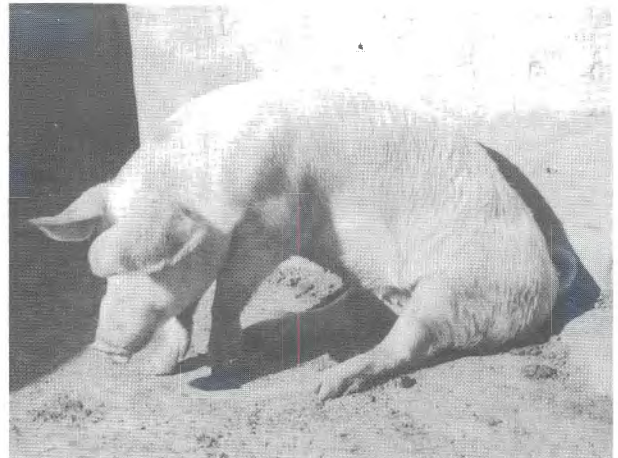

FIG. 1 Pig with selenium toxicosis in dog-sitting position, unable to rise

curred. Exact figures for each of the two episodes are not available, but a total of 42 out of 450 pigs developed clinical signs, six pigs died and eight were culled on the farm, prior to the commencement of this investigation.

Affected pigs examined on the farm the day after commencement of the second episode were in lateral recumbency. Hind- and forelimbs were areflexic, but most of the pigs were alert and could eat and drink. Ear movements appeared normal. Some of the pigs were depressed and a few were dyspnoeic, but all of them reacted to stimuli, vocalized and attempted to move away when lifted. Signs of congestive heart failure were present in one dead pig necropsied on the farm. Further examinations were carried out at the Onderstepoort Veterinary Institute (OVI).

A pig in extreme respiratory distress was euthanased and taken to the OVI for detailed post-mortem examination, followed by two pigs that died 2 d later. During the following two and a half months, another 17 affected pigs were euthanased at various times and examined at the OVI. Almost 3 months after the initial outbreak, only seven of the 42 pigs had recovered sufficiently to be marketed.

MATERIALS AND METHODS

Samples of heart, lung, liver, spleen, kidney and skeletal muscle (longissimus dorsi, diaphragm, quadriceps femoris) from a pig necropsied on the farm, were preserved in 10% buffered formalin for routine histopathological examination. Detailed necropsies were performed on 20 animals over a period of two and a half months. Brain, spinal cord, and samples of lung, heart, spleen, liver, kidney and skeletal muscle from all pigs, skin from pigs showing alopecia and crusting, and other tissues (gastrointestinal tract,

pancreas, lymph nodes, adrenal, thyroid, thymus, ovary, skin, ischiadic and brachial nerves) from selected pigs were preserved in 10% buffered formalin for histopathological examination. Sections were routinely processed and stained with haematoxylin and eosin. Selected sections of the spinal cord were also stained with luxol fast blue Holmes (LFB-H) and luxol fast blue/periodic acid Schiff/haematoxylin (LFB-PAS-H).

Samples of spinal cord and brain from two pigs, as well as tissues with macroscopic inflammatory lesions, e.g. pneumonic lungs, were taken in sterile containers for bacterial isolation. Blood from the pig euthanased on the farm, and stomach and caecal contents from the pig necropsied on the farm, were submitted for antibodies to botulinum toxin and botulinum-toxin determination by mouse bio-assay, respectively.

Samples (n = 12) of feed from pens with affected and unaffected pigs and from different types of dog food included in the consignment (e.g. pellets, meal), as well as blood (n = 26), liver (n = 18), kidney (n = 18), hoof (n = 1), skin (n = 2) and skeletal muscle (n = 1) from various pigs were submitted for determination of selenium levels. Selenium determinations were performed by means of an Inductively Coupled Plasma Mass Spectrometer.

RESULTS

The 21 pigs were divided into two groups, as follows:

Acute — Pigs (n = 4) examined 1–4 d after commencement of the outbreak, and pigs (n = 10) euthanased and examined 12 d after the outbreak.

Chronic — Pigs (n = 7) examined 27–79 d after the outbreak.

Only consistent macroscopical and microscopical pathological findings apparently associated with selenium intoxication are described. As is usual in finisher pigs, some pigs in all groups had pneumonia of varying degrees of chronicity and severity. These and other incidental macroscopical findings are mentioned for the sake of completeness only.

Macroscopical pathology

Acute group

Signs of congestive heart failure, including ascites and severe hydropericardium, were present in a dead pig examined on the farm. The myocardium had a pale, mottled appearance. Macroscopical lesions of heart failure were absent in the pigs examined subsequently. The spinal cord of this pig was not examined.
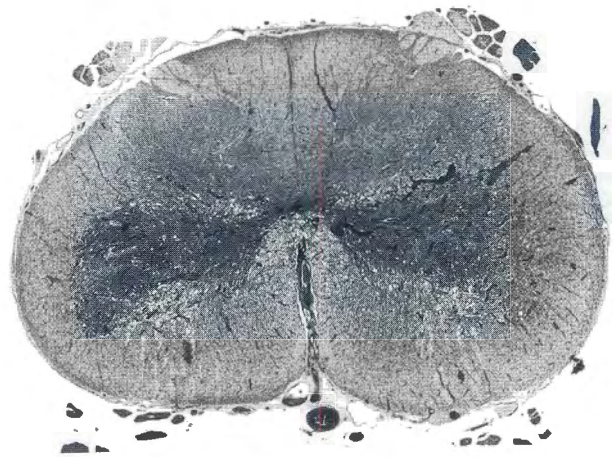

FIG. 2 Transverse section through lumbar intumescence of spinal cord of pig with selenium toxicosis to show bilaterally symmetrical malacia in ventral horns (x 6)

Striking bilaterally symmetrical segmental softening (malacia) of the spinal cord, which was most severe in the cervical and lumbar intumescences (Fig. 2), was present in all three pigs examined at the laboratory. The grey matter, particularly in the lumbar intumescence, appeared sunken on transverse section, and was a dull, dirty-brownish colour. The affected parts of the spinal cord had a soft, pasty consistency due to severe softening of the tissue. Petechiae were evident in the malacic areas as well as in other parts of the spinal cord.

The left caudal lung lobe of one of the pigs contained an abscess with extensive red hepatization of the adjacent tissue and adhesion of the apex to the parietal pleura. The right caudal lung lobe of one of the pigs contained areas of red hepatization and haemorrhage.

Chronic group

More extensive lesions were present in various organs in these pigs. Only one of the pigs was in fairly good condition—a gilt that was ambulatory, although her stance was abnormal, with the hind legs tucked well forward. She was euthanased after she had developed a rectal prolapse. The other pigs were emaciated, had remained recumbent and were euthanased because they showed no signs of recovery.

Bilaterally symmetrical sunken areas of malacia in the ventral horns of the spinal cord were consistently present in the lumbar intumescence, and variably present in the cervical intumescence of all the pigs. Petechiae and ecchymoses were present throughout the spinal cord.

Four of the pigs had alopecia, with crust formation, particularly along the dorsal midline.
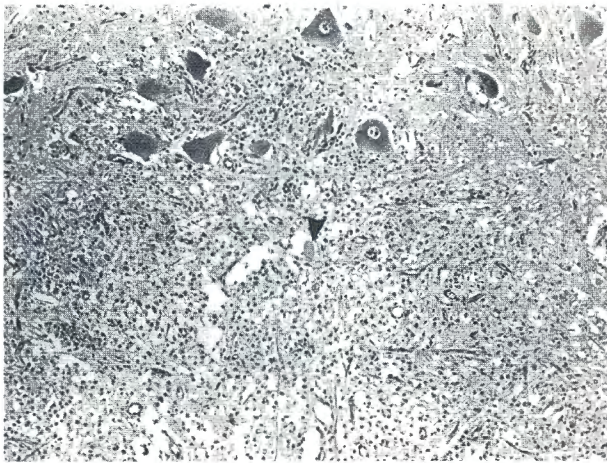
FIG. 3 Focal malacia in ventral horn of spinal cord, lumbar intumescence, in pig examined on day 2 after the episode of selenium intoxicaton. Note absence of neurons centrally and necrotic neuron (arrowhead) (HE; x 70)
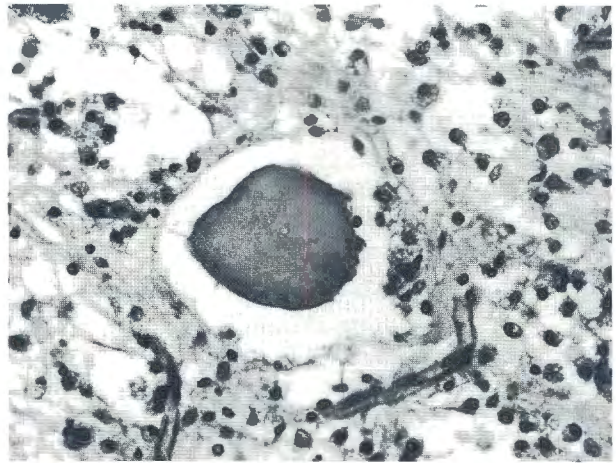

FIG. 5 Necrotic neuron and infiltrating gitter cells containing myelin remnants (LFB-PAS; x 270)
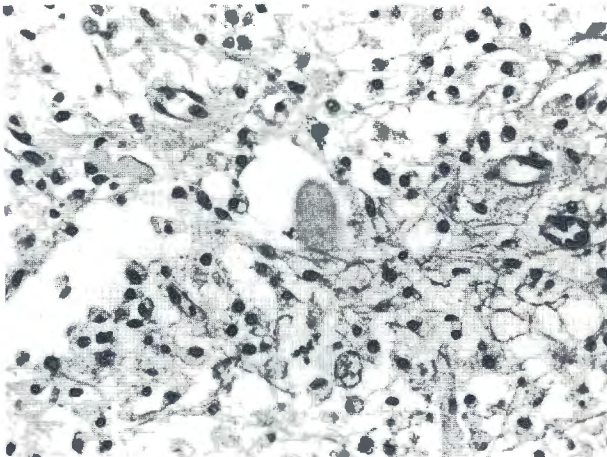

FIG. 4 Microcavitation of neuropil with infiltration of gitter cells and necrotic neuron in lesion shown in Fig. 3 (HE; x 270)
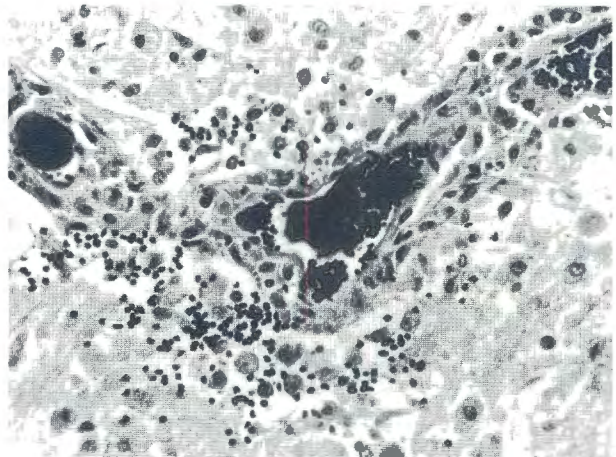

FIG. 6 Blood vessel in malacic area of ventral horn of spinal cord of pig examined on day 4 after selenium intoxication showing perivascular haemorrhage and mononuclear leukocyte infiltration (HE; x 270)

Five of the pigs had lobular bronchopneumonia and/or lung abscesses. One severely emaciated pig had an abscess in the dorsal lumbosacral musculature, with osteomyelitis of the adjacent dorsal vertebral spinous process, as well as pneumonia. The last three pigs examined (70 and 79 d after the initial outbreak), had lesions ranging from small erosions 1–2 mm in diameter, to more extensive superficial mucosal necrosis up to 100 mm in diameter near the cardia of the stomach. None of the pigs in any of the groups had coronitis or hoof separation.

Histopathology

Descriptions are restricted to changes observed in nervous tissue, myocardium, striated muscle and skin. Changes in other organs were either minimal and inconsistent, or were associated with macroscopical inflammatory lesions such as pneumonia.

Acute group

Well-circumscribed, round to oval, bilaterally symmetrical foci of malacia were present in the ventral horns of the spinal cord, consistently in the lumbar intumescence, usually in the cervical intumescence, and variably at other levels in the spinal cord (Table 1). Motor neurons in the middle of the affected foci were reduced in number, with those remaining undergoing central chromatolysis and necrosis, while neurons surrounding the lesion appeared normal (Fig. 3). Grey matter in the malacic foci was severely

FIG. 7 Myocardium of pig examined on day 4 after selenium intoxication, showing nuclear hypertrophy and dissolution of myofibres (HE; x 270)
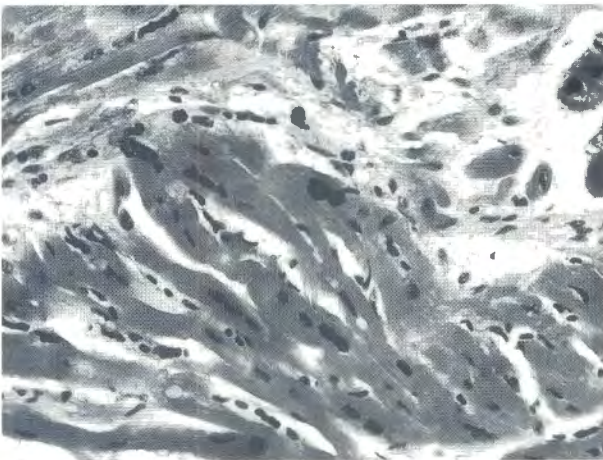

FIG. 8 Myocardium of pig examined on day 4 after selenium intoxication showing disorganization and dissolution of fibres, with a focal area of hyaline necrosis with nuclear pyknosis (HE; x 270)

TABLE 1 Distribution of poliomyelomalacia in the spinal cord in acute and chronic groups

Group	Cervical intumescence	Thoracic	Lumbar	Lumbo-sacral intumescence
Acute	+ − +++	0 − ++	0 − +++	+++
Chronic	0/+	0/+	0	+++

+++ = extensive: malacia affecting more than half of to entire ventral horn area

++ = moderately extensive, malacia affecting one-third to half of ventral horn area

+ = restricted: malacia affecting less than one-third of ventral horn area

0 = absent

oedematous, with microcavitation and necrosis of the neuropil, and very large numbers of gitter cells, containing LFB- and PAS-positive material (Fig. 4 and 5). Vascular changes included endothelial swelling, perivascular haemorrhage, mild fibrinoid degeneration in vessel walls and infiltration of the perivascular space with lymphocytes and monocytes (Fig. 6). Some of the blood vessels were surrounded by amorphous to fibrillar, deeply eosinophilic material. Changes in the adjacent white matter included oedema, swollen axons, and the presence of myelin fragments and infiltrating gitter cells.

Bilaterally symmetrical malacia, as described in the spinal cord, was present in the trigeminal nuclei of seven pigs, with multifocal haemorrhages and prominent angiogenesis. Lesions elsewhere in the brain were restricted to occasional perivascular and perineuronal oedema. Segmental Wallerian degeneration with mild, mainly lymphocytic infiltration was present in the ischiadic nerve of both pigs in which this specimen was taken, and Wallerian degeneration was evident in peripheral nerve branches visible in gluteal muscle sections of the other pigs.

Small to fairly extensive foci of hyaline degeneration and necrosis were present in the myocardium of all four pigs (Fig. 7 and 8), with occasional mild interstitial infiltration of mononuclear cells. Interstitial and occasional replacement fibrosis, with more prominent mononuclear cell infiltration, were evident in the apex of the heart of one pig.

Interstitial oedema and diffuse swelling of fibres were evident in striated muscle including diaphragm, longissimus dorsi, and inner thigh muscles. Lesions in individual fibres included segmental degeneration, hypercontraction and hyaline necrosis, with occasional hyperplasia of satellite nuclei, infiltration of a mixture of leukocytes, and early focal mineralization.

Chronic group

Bilaterally symmetrical lesions in the ventral horns of the spinal cord were pronounced only in the lumbar intumescence (Table 1). There was, however, considerable variation in the severity of the lesions. The main features were loss or absence of neurons and gliosis (Table 2). In most of the pigs, gitter cells were less prominent than in the acute group, but one of the pigs examined at 70 d had large numbers of gitter cells containing PAS-positive material (Fig. 5). Wallerian degeneration was present in the ischiadic nerve ($n = 4$).

Myocardial lesions were more advanced, and consisted of widespread hypertrophy, atrophy and disorganization of fibres, with focal to focally extensive interstitial and occasional replacement fibrosis, and marked medial hypertrophy of arterioles. Lesions as described in the acute stage were present in most

TABLE 2 Characteristics of poliomyelomalacia of the lumbosacral intumescence in pigs in the acute and chronic groups

Group	Number of pigs	Neuronal necrosis	Neuronal loss	Vascular changes	Swollen axons	Gliosis	Gitter cells	Microcav- itation
Acute	13	+++	+ − ++	+ − ++	+ − ++	+	+++	+++
Chronic	7	0/+	++	0/+	+/++	++/+++	+/++	0/+

+++ = severe
++ = moderate
+ = mild
0 = absent

of the striated muscles including the diaphragm, but diffuse atrophy of fibres and occasional extensive replacement fibrosis were present in the thigh muscles and longissimus dorsi.

Four pigs with alopecia and crusting had lesions of epidermal thickening due to acanthosis and hyperkeratosis, vacuolar degeneration of basal cells and acanthocytes, necrosis of individual keratinocytes (apoptosis) at all levels, and serocellular crusts. Most of the hair follicles were plugged with keratin. Dermal changes included increased fibrosis, mild to moderate perivascular and subepidermal infiltration of mainly mononuclear cells, and marked dilation of sweat glands.

Bacteriology

Pasteurella multocida was isolated from lung tissue of the pigs with bronchopneumonia, and the pigs with abscesses yielded *Actinomyces pyogenes*. No other pathogenic bacteria were isolated.

Botulinum-toxin determination

Samples tested for botulinum toxin were negative.

Selenium determinations

The high level of 54,581 mg/kg of selenium was determined in feed from a pen in which feed refusal was rapidly followed by paralysis. Samples of feed from the same consignment taken from the store and

TABLE 3 Selenium (mg/kg) in tissue samples

Group		Liver	Kidney
1	(n = 2)	7,218; 7,564	8,707; 9,622
2	(n = 10)	2,45–12,29 (mean = 6,12)	4,64–13,70 (mean = 10,27)
3	29 d (n = 2)	10,74; 14,00	13,86; 26,28
	43–79 d` (n = 5)	0,583 –2,657 (mean = 1,37)	3,655–7,381 (mean = 4,95)

Normal values: Liver less than 1 mg/kg, kidney less than 2 mg/kg (Casteel 1985; Sanford 1990), although 2,52 mg/kg was considered high normal for liver by Stowe *et al.* (1992)

other pens had levels of selenium ranging from too low to detect (i.e. less than 0,001 mg/kg) to 0,78 mg/kg (Robinson & Penrith 1994).

Liver and kidney selenium levels, determined at different stages of the disease, are given in Table 3. Levels in kidney were consistently higher than levels in liver, although there was considerable variation between pigs.

Selenium levels in blood measured 5,358 mg/kg in a pig in the acute stage, and 0,147–6,777 (mean = 2,512) in the subacute stage (n = 10). Ten blood samples taken on the farm from 12 affected pigs 6 weeks after the outbreak had selenium levels ranging from not detectable to 1,28 mg/kg, with an average of 0,36 mg/kg. Blood levels of 140–190 ng/mℓ (i.e. 0,14–0,19 mg/kg) in 71–180-day-old pigs (Stowe & Herdt 1992) are regarded as normal.

Owing to the expense of testing, only limited samples of other tissues were submitted for analysis. Selenium levels in muscle taken from two pigs 27 d after the outbreak were 5,84 and 9,64 mg/kg, considerably lower than the liver and kidney levels determined in the same two pigs. Muscle sampled from a pig at 43 d had a selenium level of 0,131 mg/kg. Selenium in the hoof of a pig in the subacute stage was only 6,92 mg/kg, but skin from two pigs at 29 d had very high levels of 22,69–22,08 mg/kg.

DISCUSSION

Selenium intoxication was diagnosed in pigs displaying the typical paralytic syndrome due to focal symmetrical poliomyelomalacia, after ingesting high levels of selenium in the feed. Elevated levels of selenium in the blood and tissues of the pigs, particularly liver and kidney tissues, supported the diagnosis.

This outbreak was of particular interest for a number of reasons. Firstly, it emphazises selenium intoxication as a differential diagnosis for a paralytic syndrome of pigs in South Africa. As far as we could ascertain, the present outbreak is the first reported incident of selenium intoxication in pigs in South Africa (Robinson & Penrith 1994; Penrith & Robinson 1995). Outbreaks have been reported in the United

States of America, Canada, Australia and Europe (Wilson & Drake 1982; Wilson *et al.* 1983; Harrison *et al.* 1983; Casteel *et al.* 1985; Hill *et al.* 1985; Van der Molen *et al.* 1988; Sanford 1990; Stowe *et al.* 1992; Schoder *et al.* 1993). A second point of interest in the South African outbreak was that it resulted from accidental contamination of feed with selenium, with intake of high levels in a short time. The selenium was believed to have originated from discarded premix that had been present in the factory sweepings included in the feed consignment. Thirdly, it was possible to examine a large number of affected pigs over a long period, so that both acute and chronic lesions of affected systems could be assessed, as well as the consistency of the lesions.

The present outbreak shared the essential features of the outbreaks reported elsewhere. Marked variation in the response of pigs to excess selenium is documented. Experimental studies have shown comparatively minor differences in response to selenium in different forms (Herigstad *et al.* 1973; Baker *et al.* 1989). Greater differences were noted between individuals, between pigs of different breeds and ages, and with different diets (Miller & Schoening 1938; Wahlstrom, Kamstra & Olson 1955; Wahlstrom & Olson 1959; Herigstad *et al.* 1973; Goehring, Palmer, Olson, Libal & Wahlstrom 1984a; b; Casteel *et al.* 1985; Mensink, Koeman, Veling & Gruys 1990). Even when intoxication was induced experimentally by feeding the incriminated feed to other pigs, there were differences between the findings in field and laboratory cases (Harrison *et al.* 1983). The severity of the manifestations probably depends on the level of selenium present, as well as the amount of selenium-containing feed that is consumed before the feed is rejected.

Feed refusal, sometimes with vomition, skin and hoof lesions and a paralytic syndrome, have been the most frequently described clinical findings in experimental studies and field outbreaks of selenium toxicosis in pigs. Severe loss of condition resulted when the period of feeding was prolonged. Since anorexia developed only after one or two feeds, Herigstad *et al.* (1973) concluded that a systemic toxic effect, rather than palatability, was responsible. Refusal of the feed containing selenium within a day of its being fed was a feature of the present outbreak, and several of the pigs vomited.

Intoxication with selenium in various forms, under experimental and natural conditions, resulted in a paralytic syndrome associated with bilaterally symmetrical segmental poliomyelomalacia affecting the ventral horns, particularly in the lumbar and, to a lesser extent, the cervicothoracic intumescences (Herigstad *et al.* 1973; Wilson & Drake 1982; Wilson *et al.* 1983; Harrison *et al.* 1983; Goehring *et al.* 1984b; Casteel *et al.* 1985; Van der Molen *et al.* 1988; Wilson *et al.* 1988; Baker *et al.* 1989; Sanford 1990; Stowe *et al.*

1992; Schoder *et al.* 1993). Similar lesions have been induced experimentally in pigs by the administration of 6-aminonicotinamide (O'Sullivan & Blakemore 1978; 1980), which causes acute nicotinamide deficiency, but this has apparently not been conclusively proven to occur naturally. Brain lesions were usually confined to, or were more pronounced, in the basal motor nuclei (Harrison *et al.* 1983; Wilson *et al.* 1983; Wilson *et al.* 1988; Baker *et al.* 1989; Schoder *et al.* 1993). In a number of field and experimental cases of selenium toxicosis, the paralytic syndrome either did not occur or was not recognized (Miller & Schoening 1938; Wahlstrom *et al.* 1955; Orstadius 1960; Wahlstrom & Olson 1959; Mahan & Moxon 1984; Hill *et al.* 1985). In the present outbreak, bilaterally symmetrical foci of poliomyelomalacia were consistently present in the lumbar intumescence of the spinal cord in all pigs examined at all stages, and variably present in the cervical intumescence and at other levels in the spinal cord. Similar lesions were present in nuclei of the brain stem in 50% of the pigs examined in the acute group. In pigs examined over a period of 79 d, after a single episode of intoxication, the progression of the spinal cord lesions from severe oedema and necrosis of grey matter, neuronal necrosis and large numbers of gitter cells to neuronal loss and gliosis, agreed with the findings of Van der Molen *et al.* (1988), although acute lesions were present in some pigs in the chronic group. The variation is probably not surprising, when one considers that the selenium was evidently not evenly distributed in the feed, and intake varied. Changes in the white matter adjacent to the areas of poliomalacia and in the ischiadic nerve were also similar to those described by Van der Molen *et al.* (1988).

Alopecia and scurfiness, usually most severe along the dorsal midline, and hoof cracking with detachment of the hoof at the coronary band, have frequently been reported (Miller & Schoening 1938; Wahlstrom *et al.* 1955; Herigstad *et al.* 1973; Wilson & Drake 1982; Harrison *et al.* 1983; Wilson *et al.* 1983; Goehring *et al.* 1984b; Mahan & Moxon 1984; Casteel *et al.* 1985; Van der Molen *et al.* 1988; Baker *et al.* 1989). Harrison *et al.* (1983) reported hoof lesions in pigs in which they reproduced intoxication by feeding them feed incriminated in a field outbreak, although no hoof lesions were observed in the field cases. No hoof lesions were present in any of the pigs examined in the present outbreak. Four of the seven pigs in the chronic group showed hair loss, particularly along the dorsal midline.

Effects of selenium intoxication in pigs on systems and organs other than the spinal cord, skin and hoof, are even more varied. Gastroenteritis and transient to severe diarrhoea were reported in a few studies (Miller & Schoening 1938; Orstadius 1960; Herigstad *et al.* 1973). Diarrhoea was not a feature of the present outbreak.

Most of the liver lesions described previously (Miller & Schoening 1938; Wahlstrom *et al.* 1955; Herigstad *et al.* 1973; Van Houweling 1979; Casteel *et al.* 1985; Hill *et al.* 1985; Baker *et al.* 1989) were observed in one or more of the pigs in the present outbreak, but were mild. They are interpreted as nonspecific changes, probably associated with anorexia, heart failure or parasitic infestation.

Focal degeneration and necrosis of individual skeletal muscle fibres, as reported by Herigstad *et al.* (1973) and Stowe *et al.* (1992), were present in skeletal muscle, including the diaphragm in most pigs, at all stages in the present outbreak. It is not known whether these changes are a direct effect of selenium on muscle or are secondary to nervous or vascular damage.

One pig each in the studies of Miller & Schoening (1938) and Wilson *et al.* (1983), and another pig that was examined, had striking macroscopical lesions of heart failure at necropsy. Myocardial necrosis was described by Baker *et al.* (1989) and Stowe *et al.* (1992), and was present in all the pigs examined. As in the case of skeletal muscle, it is uncertain whether the myocardium is a primary target for selenium intoxication.

Hill *et al.* (1985) reported severe oedema of all the tissues in the pig they examined, which had taken in very high levels of selenium. Acute pulmonary oedema was reported in acute or peracute selenite toxicosis (Herigstad *et al.* 1973; Nebbia, Fink-Gremmels & Gennaro Soffietti 1990; Nebbia, Gennaro Soffietti, Zittlau & Fink-Gremmels 1991), but otherwise lung lesions have not been specifically associated with selenium intoxication. The lung changes in the present outbreak were either incidental or induced by recumbency and resultant hypostasis with, particularly in the longer surviving cases, secondary bacterial pneumonia.

The present outbreak confirms the consistent association of focal symmetrical segmental poliomyelomalacia with paralysis caused by selenium toxicosis in pigs, and raises the question of whether focal degeneration and necrosis of skeletal muscle and myocardium might possibly be selenium-associated as well. Hair loss, although not consistently present, is considered to be a direct result of selenium intoxication. The remaining lesions that were found are probably either incidental or are secondary to other effects of intoxication such as anorexia, recumbency and stress.

The pathogenesis of selenium-induced neurotoxicity has not yet been elucidated (Jubb & Huxtable 1993). Vascular changes varying from capillary endothelial proliferation and leakage of eosinophilic ("fibrinoid") material to widespread fibrinoid degeneration have been described (Herigstad *et al.* 1973; Hill *et al.* 1985; Wilson *et al.* 1988). Marked endothelial proliferation in capillaries in the spinal-cord lesions, mild fibrinoid degeneration in blood-vessel walls, perivascular leukocytic infiltration, and deposits of eosinophilic ("fibrinoid") material around blood vessels in the spinal cords, were evident in early cases in the present outbreak. Oedema resulting from the vascular changes probably plays an important role in the development of malacia.

Selenium levels in serum and body tissues become elevated in toxicosis, and are useful in diagnosis and in determining when and whether recovered pigs can be safely marketed. Apart from hair, where very high levels may occur (Goehring *et al.* 1984b), the highest levels occur in the liver and kidneys (Miller & Schoening 1938; Herigstad *et al.* 1973; Wilson *et al.* 1983; Goering *et al.* 1984b; Mahan & Moxon 1984; Van der Molen *et al.* 1988; Baker et al. 1989), which are accumulator organs and will show elevated selenium levels in most cases of intoxication. Muscle accumulates selenium to a much lesser extent (Goehring *et al.* 1984b; Mahan & Moxon 1984; Hill *et al.* 1985; Van der Molen *et al.* 1988). In the present outbreak, muscle of a pig sampled at 43 d had a much lower selenium level (0,131 mg/kg) than was found in liver and kidney. At this stage, serum levels of selenium among recovered pigs ranged from too low for detection to 1,28 mg/kg. Since serum levels of 0,142–0,19 mg/kg are considered normal in finishing pigs (Stowe & Herdt 1992), serum monitoring seems to offer a conservative indication of whether the selenium levels in skeletal muscle could be considered acceptable, and it is also a practical way of monitoring live pigs.

Selenium in accumulator organs may remain elevated for some time, judging by the kidney levels obtained in the pigs examined 79 d after the initial outbreak (Table 3). This suggests that, if recovered pigs are marketed, condemnation of parenchymatous organs should be recommended, even if serum levels indicate that the meat would be fit for consumption. The wide range of variation in serum selenium levels observed in the present outbreak suggests that a large number of samples would be necessary for herd monitoring with a view to marketing, after an episode of toxicosis.

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