Poisonous plants of veterinary and human importance in southern Africa

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ABSTRACT

Southern Africa is inherently rich in flora, where the habitat and climatic conditions range from arid environments to lush, sub-tropical greenery. Needless to say, with such diversity in plant life there are numerous indigenous poisonous plants, and when naturalised exotic species and toxic garden varieties are added the list of potential poisonous plants increases. The economically important poisonous plants affecting livestock and other plant poisonings of veterinary significance are briefly reviewed. In addition, a synopsis of the more common plant poisonings in humans is presented. Many of the plants mentioned in this review are also used ethnobotanically for treatment of disease in humans and animals and it is essential to be mindful of their toxic potential.

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1. Introduction

Southern Africa has a rich and varied flora that includes a wide variety of plants with the potential to cause poisoning of animals and humans. Heavy stock losses caused by plant poisoning have occurred throughout the history of the region, and have given rise to considerable research. Although there is a large amount of information in the veterinary field, human poisoning appears to be less well documented.

Plant poisoning in animals is usually accidental, and most frequently occurs during unfavourable conditions when pastures are poor due to drought, veld fires and overstocking and trampling of the grazing. Consumption of hay contaminated with poisonous plants also occurs. In humans it may be accidental or intentional. Accidental poisoning in humans may be due to confusing poisonous with edible plants, contamination of food with poisonous plants, or by the use of plants as remedies.

Poisonous plants can affect the entire spectrum of organ systems, with some plants having several toxic principles that affect different systems. The dominant effect may depend on the condition, growth stage or part of the plant, the amount consumed, and the species and susceptibility of the victim.

While the active principles and mode of action are known for many plants, many others are known to induce poisoning, but the mechanism of intoxication has yet to be elucidated.

Diagnosis of plant poisoning of livestock depends on the history, clinical syndrome observed, post-mortem lesions, evidence that plants have been grazed, and remains of toxic plants in the gastro-intestinal tract. Where the toxic principle is known, confirmatory laboratory tests may be possible.

Only a proportion of plant poisonings results from the presence of toxic principles in the plant itself. Poisoning can result from contamination of non-toxic food plants with mycotoxin synthesizing fungi and from other interactions with organisms including insects, helminths and bacteria that result in the elaboration of toxins. It can also result from mineral imbalances that are linked to the consumption of certain plants under particular circumstances, such as enzootic icterus in sheep, which is a form of chronic copper poisoning. In this review, only poisoning due to toxins in the plant tissue itself will be discussed.

Many of the plants mentioned in this review are also used ethnobotanically for treatment of disease in humans and animals. The interrelationship of pharmacology and toxicology is important as therapeutic efficacy occurs at a lower dose, where overdosing can induce poisoning. However, poisonous plants may contain active compounds with useful biological activities (McGaw and Eloff, 2005). With the current emphasis on research and development of phytomedicines in southern Africa it is imperative to be aware of and have some information at hand regarding

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the more common plant poisonings occurring in man and live-stock.

When plants are to be utilized ethnobotanically it is essential to be mindful concerning their toxic potential. As it is not the intention of this review to include all aspects of ethnobotanical usage the interested reader is referred to the complete and comprehensive reference works compiled by Watt and Breijer-Brandwyk (1962), Hutchings et al. (1996), Neuwinger (1996) and Van Wyk and Gericke (2000).

Upon selecting plants for ethnopharmacological studies, researchers are encouraged to search available literature for known toxic properties of plants of interest, prior to conducting biological activity studies. Where toxic effects are unknown, parallel cytotox-icity studies, or inclusion of a panel of unrelated micro-organisms, are useful in detecting potential toxicity when screening plant extracts or isolated natural products for antibacterial, antifungal, antiviral and antiparasitic activity (Cos et al., 2006).

2. Major plant poisonings of livestock in southern Africa

The plant poisonings that cause major stock losses in southern Africa have been thoroughly reviewed by Kellerman et al. (1996) and Naudé et al. (1996).

2.1. Cardiac glycosides

Plants that contain cardiac glycosides are considered to be the most important cause of livestock poisoning (Kellerman et al., 1996). Cardiac glycosides affect a wide range of species, including humans, but cardiac glycoside poisoning in southern Africa is most frequently reported in cattle and sheep. Chemically, two forms of cardiac glycosides' are contained by plants, viz. car-denolides and bufadienolides. Plants that contain cardenolides include *Nerium oleander* (oleander), *Thevetia peruviana* (yellow oleander), *Strophanthus* spp. (poison rope), *Acokanthera* spp. (bush-man's poison bush), *Gomphocarpus* spp. (milkweeds), *Cryptostegia grandiflora* (rubber vine) and *Adenium multiflorum* (impala lily). As these are unpalatable plants they are rarely eaten by stock and therefore of little veterinary importance. Major stock losses are, however, recorded due to consumption of plants containing bufadienolides. Poisoning by bufadienolides falls into two major categories, acute poisoning by non-cumulative bufadienolides and chronic poisoning by cumulative bufadienolides (Kellerman et al., 2005).

Acute cardiac glycoside poisoning most frequently results from consumption of members of the genera *Moraea* (Iridaceae, tulp) and *Drimia* (Hyacinthaceae, slangkop). Other genera of Hyacinthaceae, *Thesium* spp. (Santalaceae) and *Melianthus* spp. (Melianthaceae) also contain bufadienolides and have rarely been suspected or incriminated. Tulp and slangkop appear before the rains and may be the only greenery available, particularly in the drier parts of their distribution areas. Contamination of hay with tulp has also caused poisoning, as desiccated plants retain their toxicity. Animals newly introduced to pastures where tulp grows are par-ticularly susceptible, as aversion to tulp has not yet developed (Snyman et al., 2003). Death is usually due to a cardiotoxic effect, although cardiac glycosides also affect three other systems, namely the gastro-intestinal, respiratory and nervous systems. Gastro-intestinal effects include colic, ruminal stasis, bloat and usually diarrhoea. Nervous signs are most frequently observed as posterior paresis. Respiratory effects are probably neuromuscular in origin and include dyspnoea, polypnoea and periods of apnoea (Kellerman et al., 2005). Acute cardiac glycoside poisoning may be treated by the administration of relatively large doses of activated char-coal.

Chronic bufadienolide poisoning is caused by a group of suc-culent plants of the family Crassulaceae (plakkies) of the genera *Tylecodon*, *Cotyledon* and *Kalanchoe*. Their bufadienolides have a cumulative, neurotoxic effect that produces a syndrome known as 'krimpsiekte' (=shrinking disease), a paretic syndrome of sheep and goats (Botha, 2003). Consumption of these plants can result in acute cardiac glycoside poisoning, as the manifestation depends on factors such as dose, duration of exposure, the bufadienolides involved and predisposing factors. Hungry animals that consume large amounts of the plants in a short period of time may die sud-denly or develop an acute syndrome that can include depression, bloat, regurgitation, paralysis of the tongue, salivation and recum-bency. This may progress to the typical chronic form of poisoning, which can also develop in animals that are exposed to the plants over a protracted period that allows low but continuous intake. Affected animals lag behind the flock, walk with the neck dangling and assume the characteristic 'shrinking' posture, with the back arched, the feet drawn together under the body and the head hang-ing down. Affected small stock tire quickly and lie down with the neck extended sometimes twisted to one side (torticollis). Mortal-ity can be as high as 90%. Poisoning has also been reported in horses and domestic chickens. Relay toxicosis or secondary poisoning has been described in dogs that have eaten the flesh of goats and horses that have died of krimpsiekte (Henning, 1926).

2.2. Gousiekte

Various plants of the family Rubiaceae (*Pachystigma* spp., *Fado-gia homblei* and *Pavetta* spp.) induce a syndrome known as 'gousiekte' (=quick sickness). Large numbers of domestic rumi-nants can die suddenly without any premonitory signs 6–8 weeks after ingestion of these rubiaceous plants. These plants occur in the north-eastern and central parts of southern Africa and may be grazed when they sprout before the new grass appears, or in the late summer when the grass becomes dry. Affected animals usually drop dead after exercise or other stimulation, although a few may mani-fest signs of congestive heart failure. Histopathological examination of the heart reveals typical lesions of degenerative cardiomyopathy and replacement fibrosis that affect mainly the endocardium of the heart. Both cattle and small stock are affected. A polyamine has been isolated and purified relatively recently and has been identified as pavetamine (Kellerman et al., 2005; Prozesky et al., 2005).

2.3. Gifblaar

Fluoracetate poisoning due to the consumption of *Dichapetalum cymosum* ('gifblaar', Dichapetalaceae) most frequently affects cat-tle, possibly because the distribution of the plant coincides with mainly cattle-raising areas. Gifblaar is deeply rooted and is gener-ally eaten in spring, because the underground water storage system allows it to sprout before the rains, when no other green forage is available. However, it is also reported to be particularly dangerous when the pasture is deteriorating at the end of the rainy season. Because the mechanism of action, which blocks the tricarboxylic acid cycle, drastically reduces cellular respiration, sudden death occurs as a result of acute heart failure induced by the affected animal drinking water or exercising, usually within 24 h of eating the plant (Kellerman et al., 1996).

2.4. Seneciosis

The consumption of species of *Senecio* (Asteraceae: *Senecio lati-folius*, *Senecio retrorsus*) that contain pyrrolizidine alkaloids results in hepatotoxicosis. About 10% of cattle deaths due to plant poi-soning and 5% of small stock deaths are attributed to seneciosis

(Kellerman et al., 1996). The plants tend to invade pastures that have been degraded by poor management or by droughts. Acute seneciosis occurs when animals consume large amounts of the plants over a relatively short period. Young animals are most often affected. They may die within a few days of consuming the plants. Clinical signs include loss of appetite, abdominal pain, diarrhoea, and icterus. The most significant pathological lesions are found in the liver, and consist of centrilobular necrosis, with severe congestion and haemorrhage, with degenerative changes in the hepatocytes surrounding the necrotic zones. Field cases of subacute to chronic seneciosis are more frequent. Adult cattle develop a syndrome known as Molteno straining disease that is characterised by tenesmus, blood-stained faeces and progressive loss of condition, as well as nervous signs that are ascribed to hepatic encephalopathy resulting from the severe liver damage. Sheep suffering from chronic seneciosis similarly lose condition and may develop ascites, but although lesions characteristic of hepatic encephalopathy may be present in the brain, nervous signs have not been described (Kellerman et al., 2005). Chronic seneciosis in horses manifests as a wasting disease, with progressively worsening incoordination, known as 'dunsiekte' (Botha and Naudé, 2002). Chronic seneciosis may be preceded by a latent period of up to 6 months before clinical signs appear. The liver in all species has characteristic lesions of fibrosis, often progressing to cirrhosis, with nodular regeneration. Megalocytosis is a typical feature owing to the inhibition of mitosis by pyrrolizidine alkaloids, and abnormal mitotic figures may be present. Bile duct proliferation is another typical feature of *Senecio*-induced hepatosis.

2.5. Lantana camara

Lantana camara (Verbenaceae) is an important cause of poisoning in cattle. Although this exotic weed is generally unpalatable to stock, its abundant availability at times when pasture is scarce results in ingestion by hungry cattle. Furthermore, there is some evidence that certain animals will develop a taste for *Lantana* and actually eat it in preference to other plants. Sheep and goats are susceptible but are rarely naturally poisoned, although one incident in relocated goats has been reported (Ide and Tutt, 1998). The toxic principles are pentacyclic triterpene acids. Affected animals develop photosensitivity and jaundice as a result of retention of phylloerythrin and bile stasis, which is ascribed to damage to the bile canaliculi caused by the action of the toxins. Apart from icterus and photodermatitis, clinical signs include loss of appetite, ruminal stasis, diarrhoea and severe depression. In addition to liver damage, severe nephrosis develops, and the animal may become uraemic (Fourie et al., 1987). Because the toxic effects depend upon the continuous passage and absorption of the toxins through the gastro-intestinal tract, the effects, if not too advanced, may be mitigated by the administration of activated charcoal. Microscopic lesions in the liver and kidneys confirm cholestasis, hepatic degeneration and necrosis, and severe damage to the renal tubular epithelium (Kellerman et al., 2005).

2.6. Tribulus terrestris

The most important plant poisoning causing photosensitivity in sheep, referred to as 'geeldikkop', is associated with the consumption of *Tribulus terrestris* (Zygophyllaceae). This plant is an important food plant for sheep in dry areas, but consumption of young wilted plants and, occasionally, plants that have not wilted, can result in hepatogenous photosensitivity. This is characterised pathologically by the presence of birefringent crystalline material in bile ducts that may result in their complete occlusion. Although the clinical signs of photosensitivity are similar to those of sporidesmin-induced facial eczema, the pathogenesis, involving the deposition of crystalline material in bile ducts that show evidence of epithelial damage, is different (Coetzer et al., 1983). In 'geeldikkop' it is probable that the effects are the result of the action of steroidal saponins that have been isolated from the plants. A similar syndrome occurs in sheep grazing *Panicum* grasses (Poaceae) that have wilted in hot dry conditions after rains (Kellerman et al., 2005).

2.7. Vermeersiekte

A syndrome known as 'vermeersiekte' in sheep, induced by the consumption of plants of the genus *Geigeria* (Asteraceae), is responsible for up to 13% of stock deaths due to plant poisoning and mycotoxicoses in South Africa (Kellerman et al., 1996). Affected sheep show one or more of the following signs: regurgitation of ruminal content, bloat, stiffness and/or paralysis. The name 'vermeersiekte' refers to the regurgitation of ruminal content. The toxic principles are believed to be sesquiterpene lactones, and their main target appears to be striated muscle, in which marked pathological changes occur (Van der Lugt and Van Heerden, 1993). Sheep that regurgitate usually show marked dilatation of the oesophagus, which may be more than four times the diameter of the normal oesophagus (Kellerman et al., 2005). Prompt removal of animals from pastures with abundant *Geigeria* can reduce mortality and promote recovery of most of the sheep, but production losses can be significant.

3. Other plant poisonings of livestock

Numerous plant poisonings have been documented as occasional events. These are nevertheless of economic importance to affected livestock producers, who may suffer the loss of most of their herd or flock as a result. 'New' poisonous plants have been discovered during the last two decades. Prolonged droughts and changes in animal management may result in the discovery of potentially poisonous plants that under different conditions would not be eaten in sufficient quantities to cause ill effects. Many poisonings affect more than one organ system, but for convenience the less frequent plant poisonings will be grouped under the main organ system affected.

3.1. Central nervous system

Plant poisonings affect the nervous system in various ways, which include stimulation, depression, tremors, convulsions, paresis, paralysis, and abnormal behaviour. A large number of plant species have been associated with the development of nervous signs in livestock (Table 1). These poisonings have been grouped by Kellerman et al. (2005) according to whether or not they are associated with specific pathological lesions in the central nervous system.

Poisoning of mainly cattle and rarely sheep and goats by consumption of the pods of *Albizia versicolor* and *Albizia tanganyicensis* occurs in late winter and spring when high winds cause large numbers of pods to fall to the ground and become accessible to livestock. Animals are usually found dead, with the ground around them disturbed as a result of violent convulsions before death. Animals observed in time can recover if treated with Vitamin B$_6$ (Gummow et al., 1992).

Toxic pregnane glycosides are responsible for the nervous syndromes resulting from consumption of the succulent creepers *Cynanchum africanum*, *Cynanchum ellipticum*, *Cynanchum obtusifolium* and *Sarcostemma viminale*. *Cynanchum* species, which are widely distributed along the coast of southern Africa, contain a milky, bitter latex, but nevertheless appear to be palatable to

Table 1
Plant poisonings that primarily affect the central nervous system

Plant	Species	Syndrome	Toxin	Mode of action
Albizia spp.	Ruminants	Hypersensitivity, convulsions	4′-Methoxypyridoxine	Vitamin B_6 antagonist
Cynanchum spp.	Ruminants, horses	Hypersensitivity, incoordination, convulsions, paralysis	Pregnane glycosides	Unknown
Sarcostemma viminale	Ruminants, horses	Hypersensitivity, incoordination, convulsions, paralysis	Pregnane glycosides	Unknown
Euphorbia mauritanica	Cattle, sheep	Tremors (+bloat and diarrhoea)	Unknown	Unknown
Dipcadi glaucum	Ruminants	Incoordination, diarrhoea	Unknown	Unknown
Lupinus spp.	Ruminants, pigs	Hyperactivity, paralysis	Alkaloids	Unknown
Melia azedarach	Animals, humans	Tremors, dyspnoea, vomition, diarrhoea	Tetranortriterpenes	Unknown
Datura spp.	Animals, humans	Tremors, hallucinations, convulsions, mydriasis, gastro-intestinal signs	Atropine, hyoscine, hyoscyamine	Parasympatholysis
Pteridium aquilinum	Horses, sheep	Tremors, incoordination	Thiaminase I	Thiamine depletion
Nicotiana glauca	Ruminants, pigs, humans	Incoordination, tremors, convulsions, dyspnoea	Anabasine (alkaloid like nicotine)	Ganglion blockade
Ficus ingens Ficus salicifolia	Cattle	Hyperaesthesia, ataxia, tremors, paddling	Unknown	Unknown
Nierembergia linariifolia	Cattle	Variety of nervous signs	Unknown	Unknown
Solanum tettense	Cattle	Loss of balance, epileptiform seizures	Unknown	Cerebellar atrophy
Ipomoea carnea	Goats	Ataxia, head tremors, nystagmus	Swainsonine, calystegine glycosidase inhibitors	Inhibition of mannosidase and glycosidases, resulting in lysosomal storage disease
Trachyandra spp.	Ruminants, horses, pigs	Progressive paresis and paralysis	Unknown	Unknown
Phalaris spp.	Ruminants	Staggers	Tryptamine alkaloids	Unknown
Helichrysum argyrosphaerum	Ruminants	Amaurosis, paresis, paralysis	Unknown	Unknown
Cotula nigellifolia	Cattle	Disorientation, head pressing	Unknown	Unknown

livestock. *Sarcostemma viminale* grows in drier areas and may be consumed when grazing is scarce. Poisoning caused by *Euphorbia mauritanica* is rare.

Dipcadi glaucum, unlike other poisonous members of its family (Hyacinthaceae), apparently does not contain cardiac glycosides, but is nevertheless toxic to ruminants. Consumption induces disorientation and eventually posterior paresis in cattle, commencing with knuckling over of the fetlocks, causing them to stumble; however, appetite is not affected. Sheep are more frequently poisoned than cattle, and in addition to disorientation and dyspnoea, develop severe diarrhoea and fever, and pregnant ewes may abort. Like many of the toxic lilies, *Dipcadi glaucum* sprouts rapidly after the first rains, and may be the only green feed available on dry pastures.

Poisoning by bitter lupins, mainly *Lupinus angustifolius* and *Lupinus digitatus*, is less common than lupinosis, a hepatotoxicosis that occurs when lupins are contaminated with the fungus *Phomopsis leptostromiformis*. Bitter lupine alkaloids have been associated with the development of nervous signs, rapidly culminating in asphyxia, in cattle, sheep and pigs in the Western Cape province of South Africa, as well as in Australia and America (Van Warmelo et al., 1970).

Datura spp. (*Datura ferox*, *Datura stramonium*), *Nicotiana glauca* (wild tobacco) and *Pteridium aquilinum* (bracken fern) are cosmopolitan poisonous plants that cause nervous disorders and are present in South Africa, although they are rarely responsible for livestock poisoning. *Datura* poisoning has been reported mainly in horses that have eaten feed heavily contaminated with the toxic seeds (Schulman and Bolton, 1998) or hay contaminated with plant parts (Naudé et al., 2005). Poisoning by *Nicotiana glauca* causes an acute nervous toxicity that culminates in death due to respiratory paralysis, as well as teratology in sheep, but rarely causes poisoning in southern Africa in spite of its abundance. Bracken fern has been associated with a variety of toxic effects worldwide. In South Africa, horses that have eaten fodder contaminated with bracken fern become drowsy and dyspnoeic, and develop an unsteady gait, tremors, staggers, arching of the back, and eventually recumbency with convulsions. Thiamine treatment in the early stages results in complete recovery.

Melia azedarach (Meliaceae) and *Nierembergia linariifolia* (Solanaceae) are introduced ornamental plants that have escaped from gardens and become established on natural pastures. Pigs are most susceptible to poisoning by *Melia*, in particular the ripe drupes, which have also been incriminated in poisoning in children, but cases of poisoning have also occurred in sheep and cattle (Kellerman et al., 2005). *Nierembergia* poisoning was recently described in calves in the Free State province of South Africa (Botha et al., 1999).

Another recently described neurotoxicosis involved the indigenous trees *Ficus ingens* and *Ficus salicifolia*, which may be consumed during times of drought. Cattle developed hyperaesthesia, ataxia and tremors, progressing to lateral recumbency with paddling movements and death (Myburgh et al., 1994).

Of the plant-induced neurotoxicoses with which specific pathological lesions are associated, the most recently described is a lysosomal storage disease in goats in southern Mozambique (De Balogh et al., 1999). *Ipomoea carnea* (Convolvulaceae) is a cosmopolitan shrub that is extensively used for hedging. Goats that consumed this plant developed ataxia, head tremors and nystagmus, associated with vacuolar lesions in neurons and other brain cells typical of a lysosomal storage disease. The plant material was demonstrated to contain toxic principles that inhibited mannosidase, β-glycosidase and α-galactosidases, resulting in lysomal storage disease.

The neurotoxicosis known as 'maldronksiekte' ('mad drunk disease') in cattle induced by *Solanum tettense* (Solanaceae) is also characterised by vacuolation of neurons, but has not been demonstrated to be a storage disease. The main lesion is cerebellar atrophy, which is macroscopically conspicuous in severely affected animals, and is microscopically characterised by loss of the Purkinje cells. As the name implies, the clinical signs include loss of balance and falling (Pienaar et al., 1976).

Progressive posterior paresis and paralysis induced in cattle, sheep, horses and pigs by consumption of *Trachyandra* spp. (*Trachyandra laxa*, *Trachyandra divaricata*) and *Phalaris* staggers in sheep and cattle are characterised by the accumulation of brownish pigment in neurons in the brain and spinal cord. The toxic principles have not as yet been identified.

Nervous signs and amaurotic blindness in sheep caused by *Helichrysum argyrosphaerum* are the result of severe brain swelling, necrosis of the retina, and bilateral status spongiosus especially of the mid-brain and thalamus (Van Der Lugt et al., 1996). In cattle, which are rarely affected by this plant, stiffness and posterior paresis associated with necrosis of skeletal muscles occurs, but not blindness.

The condition known as 'stootsiekte' (pressing disease) in cattle caused by *Cotula nigellifolia* (Asteraceae) is unusual among toxicoses in being accompanied by inflammatory lesions in the white matter of the cerebrum and mid-brain with occasional extension to the cerebellum (Newsholme et al., 1984). The lesions consist of perivascular gliosis with infiltration of inflammatory leucocytes, mainly lymphocytes and plasma cells, and are more suggestive of an infectious disease than a toxicosis.

3.2. Cardiovascular system

As described above, three of the most important plant poisonings of livestock (cardiac glycoside poisoning, gousiekte and gifblaar poisoning) primarily affect the cardiovascular system. The other cardiovascular toxicoses affect a wide range of species and involve more than one system, although death is most likely to be due to cardiac damage.

Gossypol is a reactive polyphenolic pigment that is present in cotton seed (*Gossypium* spp.). It is inactivated by binding to proteins, which occurs in the rumen, and adult ruminants can therefore tolerate higher amounts of gossypol in the diet than monogastric animals, including young ruminants. Pigs are severely affected. Clinical signs that may include dyspnoea, anorexia, unthriftiness and diarrhoea appear 1–3 months after gossypol has been included in the feed. On the other hand, finished pigs may die suddenly without warning during transport to the abattoir, without premonitory signs. Post-mortem examination reveals severe cardiomyopathy as well as severe hepatosis with centrilobular necrosis and haemorrhage that may affect the whole lobule. The hepatic lesions may be more pronounced than the lesions in the heart. Gossypol poisoning has also been associated with various manifestations of infertility in monogastric species including humans (Nicholson, 2007).

Argemone spp. (prickly poppies, or 'bloudissel') are unpalatable, spiny exotic weeds that are not intentionally eaten by livestock but cause poisoning when the plants are harvested together with lucerne or wheat. The toxic principles are isoquinoline alkaloids. Berberine and protopine are present in all parts of the plant, while the seeds also contain sanguinarine and dihydrosanguinarine. The most prominent sign in affected animals is dyspnoea. Necropsy reveals severe ascites and hydrothorax, with degenerative changes in the myocardium and the liver. The alkaloids present in the seeds have been demonstrated to cause vasodilation and increased vascular permeability (Kingsbury, 1964).

Avocado (*Persea americana*) has been associated with congestive heart failure related to severe cardiomyopathy in goats, sheep, horses and ostriches (Stadler et al., 1991; Burger et al., 1994). At lower doses aseptic mastitis has been reported in goats and horses. High intake of the fruits of certain cultivars has poisoned budgerigars and canaries, as well as possibly dogs (Kellerman et al., 2005).

3.3. Hepatotoxicoses

Three of the most important plant poisonings of livestock (geeldikkop, senecioses and Lantana poisoning) result in severe liver damage, and a number of less frequently encountered plant poisonings also cause hepatotoxicity.

Like *Senecio*, at least some members of the genus *Crotalaria* contain pyrrolizidine alkaloids. *Crotalaria spartioides* has occasion-

ally been associated with hepatotoxicity in cattle (Kellerman et al., 2005), and dosing cattle with *Crotalaria spartioides* and cattle and sheep with *Crotalaria dura* has resulted in cirrhosis of the liver. Various members of the genus are also associated with respiratory disease and laminitis (see below).

Several other plants induce hepatotoxicity without photosensitivity. Liver lesions are characterised by centrilobular to zonal necrosis and varying degrees of congestion and haemorrhage. *Cestrum* spp. contain kaurene glycosides that are very similar to carboxyatractyloside isolated from *Xanthium* spp. (Van Wyk et al., 2002). *Cestrum* poisoning has been reported in cattle and goats (Van Der Lugt et al., 1991), while pigs are most frequently the victims of cockle burr (*Xanthium*) poisoning. Sheep and sometimes goats in the western parts of southern Africa may be poisoned by eating *Hertia pallens* and *Pteronia pallens* (Asteraceae) and *Galenia africana* (Aizoaceae) (Kellerman et al., 2005). Animals poisoned by *Galenia* usually develop severe ascites, so that the condition has been named 'waterpens' (water belly). Myocardial involvement observed in field cases is believed to develop secondary to severe liver damage rather than as a primary effect of the toxin (Van der Lugt et al., 1992).

Furanosesquiterpenoids have been identified in four plants of the family Asteraceae that cause hepatotoxicity with photosensitivity. These are *Lasiospermum bipinnatum*, two *Athanasia* spp. (*Athanasia minuta*, *Athanasia trifurcata*) and *Nidorella foetida* (Kellerman et al., 2005). Poisoning of cattle and sheep by *Lasiospermum bipinnatum* is locally important in parts of the Eastern Cape Province and eastern Free State (Kellerman et al., 1996). The liver lesions that typically consist of midzonal and peripheral necrosis, with the centrilobular area relatively unaffected, are accompanied by lung lesions of emphysema, bronchiolar dilatation, and interstitial pneumonia characterised by proliferation of non-ciliated epithelial cells (Penrith and Van Vollenhoven, 1994).

3.4. Gastro-intestinal tract

A number of poisonous plants induce diarrhoea when consumed (Table 2), although the toxins and their modes of action, where known, differ. In addition to severe, foetid diarrhoea, cattle poisoned with *Ornithogalum* spp. (Hyacinthaceae, chinkerinchee) also become blind, and the blindness may be permanent or may resolve within a few weeks.

Poisoning of livestock by the toxalbumin-containing plants (Table 2) is unusual, as the plants are not palatable. Ricin poisoning has been associated with the accidental inclusion of castor oil seed cake in rations. A case of poisoning of cattle that grazed *Abrus precatorius* has been recorded (Kellerman et al., 2005).

Many species of the family Solanaceae, including food plants such as tomatoes and potatoes, contain glycoalkaloids that may reach toxic levels under particular conditions (e.g. unripe tomatoes, potato tubers that have turned green after exposure to light). Poisoning by Solanaceae is relatively rare, possibly due to a combination of unpalatability of the unripe fruits and the fact that solanine is rapidly hydrolysed to the less toxic aglycone in the gastro-intestinal tract. In addition, solanine is also poorly absorbed from the gastro-intestinal tract (Steyn, 1934).

Wild cucumbers (Cucurbitaceae: *Cucumis* spp.) contain cucurbitacins that are concentrated in the ripe fruits and the roots. These impart an extremely bitter taste to the plants, which are generally only eaten by cattle when nothing else is available. Clinical signs range from sudden death with lung oedema to severe, sometimes haemorrhagic diarrhoea (Rimington, 1935).

Ornithoglossum vulgare (Colchicaceae) is a lily-like plant that has occasionally been incriminated in stock losses. The toxic principle has not yet been identified but, although the clinical signs of exper-

Table 2
Plant poisonings that manifest as diarrhoea

Plant	Species	Syndrome	Toxin	Mode of action
Ornithogalum spp.	Ruminants, horses	Diarrhoea; blindness	Cholestane glycosides	Unknown
Ricinus communis	Multispecies	Diarrhoea	Toxalbumin (ricin)	Disrupts protein synthesis
Jatropha curcas	Multispecies	Diarrhoea	Toxalbumin (curcin)	As above
Abrus precatorius	Multispecies	Diarrhoea	Toxalbumin (abrin)	As above
Solanum nigrum, S. lichtensteinii	Multispecies	Diarrhoea	Glycoalkaloids	Acetylcholinesterase inhibition, saponin-like effects
Cucumis spp.	Multispecies	Diarrhoea	Cucurbitacins	Unknown
Ornithoglossum vulgare	Sheep and goats	Diarrhoea	Unknown	Unknown
Gnidia spp.	Ruminants	Diarrhoea, emphysema	Daphnetoxin (phorbol triterpene)	Unknown
Sesbania punicea	Multispecies	Diarrhoea	Sesbanamide	Unknown

imental poisoning in a sheep resembled those induced by cardiac glycosides, none have been isolated from the plant.

Plants of the genus *Gnidia* (*Gnidia polycephala*, *Gnidia burchellii)* can induce, upon ingestion, severe diarrhoea in stock, accompanied by emphysema that causes polypnoea. Lymphoid tissues may also be affected. The mechanism of action of the toxin is not completely understood, but dust from the dried plant has an irritant effect on the mucosa when inhaled (Terblanche et al., 1966).

Sesbania punicea (Fabaceae) is an introduced leguminous plant that has been incriminated in outbreaks of poisoning in domestic fowl and pigeons. Although it was possible to poison laboratory animals (guinea pigs, rats), there are no recorded cases of poisoning in mammals in southern Africa (Kellerman et al., 2005).

3.5. Respiratory system

All the plants associated with toxic effects on the respiratory system also affect other systems and have been described elsewhere. *Crotalaria* spp., mainly *Crotalaria dura* and *Crotalaria globifera*, have been associated with chronic pulmonary disease ('jaagsiekte') in horses and mules. Affected animals developed respiratory signs commencing with an increase in respiratory rate and progressing to severe dyspnoea particularly after exercise (Botha and Naudé, 2002). Necropsy revealed severe emphysema, thickening of interlobular septa, and microscopic lesions showing bronchiolar obstruction and desquamation of epithelial cells into fluid-filled alveoli. There was sometimes extension of the emphysema subcutaneously and into regional lymph nodes. The liver of affected horses usually showed changes typical of those induced by chronic seneciosis. Cattle poisoned experimentally with the same species of *Crotalaria* only developed hepatotoxicity. Two other plants that target the liver, *Lasiospermum bipinnatum* and *Hertia pallens*, also affect the respiratory system, as described above under hepatotoxicoses, while *Gnidia* spp. cause severe diarrhoea in addition to pulmonary emphysema (Kellerman et al., 2005).

3.6. Urogenital system

Primary nephropathy results from poisoning by plants that contain oxalic acid/soluble oxalates, tannins, and unknown toxic principles. Oxalate poisoning occurs when soluble oxalates bind with calcium in the blood, causing hypocalcaemia and the deposition in tissues, in particular the kidneys, of insoluble calcium oxalate, resulting in damage to renal tubular epithelium. High levels of oxalates can occur in a wide variety of plants, including many that are used as fodder (beetroot and related plants, prickly pears, agave) as well as plants that occur naturally on pastures and may be grazed (oxalis, sorrel, *Mesembryanthemum* spp.). Although soluble oxalates are bound in the rumen to form insoluble calcium oxalates that are excreted in the faeces and/or are detoxified in the rumen, ingestion of large amounts of oxalate-containing plants can over-

whelm this mechanism and result in oxalate poisoning (Naudé and Naidoo, 2007).

Poisoning by ingestion of oak (*Quercus robur*) has been reported in cattle, sheep and horses, although pigs can be fed acorns without any ill effects. The toxic effects are believed to be due to tannin derivatives, mainly digallic acid. The animals die as a result of kidney failure due to severe nephrosis, accompanied by necrotic lesions in the gastro-intestinal tract. One of the clinical signs may be severe, foetid diarrhoea (Neser et al., 1982).

Other plants that have been associated with primary nephrosis are *Anagallis arvensis* (Primulaceae), an introduced weed that is reported to have caused poisoning in sheep in the Western Cape province, and *Nolletia gariepina* (Asteraceae), which has recently been demonstrated to be nephrotoxic after an outbreak occurred in cattle in the Northern Cape province (Du Plessis et al., 2004).

Plant poisonings affecting reproduction include *Salsola tuberculatiformis* (Chenopodiaceae), a shrub that grows in dry areas, and the red and white subterranean clovers, *Trifolium* spp., which can affect fertility when they contain high levels of oestrogenic glycosides. *Salsola* spp. if consumed in sufficient quantities during pregnancy can result in retention of the foetus past term, with a large, post-mature lamb that, at up to three times the normal birth weight, is likely to cause dystocia (Basson et al., 1969).

3.7. Haemopoietic system

Plant poisonings that affect the haemopoietic system include toxic haemolysis, prussic acid poisoning, nitrite poisoning, plant-induced bone marrow suppression and haemorrhagic diathesis.

Plants of the onion family, including onions and garlic, and the cruciferous plants (Brassicaceae), can contain disulphides or their precursors that, if present in sufficient quantity, can cause haemolytic anaemia through interference with the hexose monophosphate pathway. Onions can affect both ruminants and monogastric animals, while conversion of S-methylcysteine sulphoxide in cruciferous plants to dimethyl disulphide in the rumen is the cause of Brassicaceae-induced poisoning (Taljaard, 1993).

A wide variety of plants can contain high concentrations of cyanogenic glycosides that can result in prussic acid poisoning in livestock. Ruminants are more susceptible owing to the higher pH in the rumen compared to the stomach of monogastric animals, allowing the release of prussic acid, but most species including humans can be affected. The presence of cyanogenic glycosides depends upon the growth stage of the plant as well as damage and wilting. Binding with iron in the cytochrome oxidase system causes animals to die from anoxia (Kellerman et al., 2005).

Plants are an uncommon source of nitrite poisoning compared to other sources such as underground water and fertilizers. Nevertheless, a number of plants contain levels of nitrates that may be converted in the rumen to nitrites sufficient to cause intoxication. These plants include pigweed *Amaranthus* spp. (Amaranthaceae)

and various members of the Brassicaceae, Chenopodiaceae, and some grasses (Poaceae). Nitrites induce poisoning due to fatal anoxia when methaemoglobin, with a much lower capacity to carry oxygen, is formed (Kellerman et al., 2005).

One of the toxicoses caused by bracken fern (*Pteridium aquilinum*) is a haemorrhagic syndrome caused by a radiomimetic toxin, ptaquiloside, that causes severe bone marrow suppression, thrombocytopenia, and widespread bleeding. Feeding spoilt sweet clover (*Melilotus alba*), in which coumarins have been converted to dicoumarol, to cattle can cause a haemorrhagic diathesis if sufficient quantities are fed over a long period (Kellerman et al., 2005).

3.8. Skin and adnexa

Mechanical damage to the skin and adnexa can result from contact with plants and is not considered here. Skin irritation due to contact with plants is also in general not considered to be a toxicosis, but the irritant effects of the latex of *Euphorbia ingens*, which can result in severe dermatitis as well as blindness, are sufficiently severe to be categorised as a poisoning.

Ingestion of certain plants can produce adverse effects mainly involving the skin or adnexa. Certain species of *Crotalaria*, principally *Crotalaria burkeana* and *Crotalaria barkae*, cause severe laminitis in cattle, while *Crotalaria juncea* has been associated with laminitis in cattle and a break in the wool in sheep (Steyn, 1934).

Chrysocoma ciliata (Asteraceae) is a shrub that is associated with alopecia in lambs and kids whose dams have ingested the plant during pregnancy (Steyn, 1934). Ingestion of large amounts of this plant also causes severe diarrhoea in adult sheep and goats. Additionally, the plant has, circumstantially, been linked to a nervous syndrome called falling disease ('valsiekte') in sheep. The spinal cord lesions in animals that die of 'valsiekte' resemble those caused by copper deficiency.

Hairy vetch (*Vicia* spp.) is associated with the development of pruritis and granular dermatitis in cattle; granulomas may also occur in internal organs, including the kidneys (Green and Kleynhans, 1989).

Primary photosensitivity in livestock caused by plants that contain hypericin has not been documented in southern Africa, although St John's wort, *Hypericum perforatum*, has been introduced, and in addition there are indigenous species of *Hypericum* (*Hypericum aethiopicum*, *Hypericum revolutum*) that have been demonstrated experimentally to be capable of causing photosensitivity (Kellerman et al., 2005).

4. Plant poisonings of humans

Plant poisonings of humans in eastern and southern Africa have been documented in a comprehensive treatise by Watt and Breijer-Brandwyk (1962). Plant poisoning in humans usually arises either from the unintentional use of toxic plants as food or from the use of poisonous plants for medicinal purposes. Accidental ingestion of plants resulting in acute poisoning is more common in pre-school children (Van Wyk et al., 2002). Human intoxication due to plant exposure is far less important than poisonings involving paraffin, pesticides, pharmaceuticals, household cleaning chemicals and cosmetics (Gaillard and Paquin, 1999; Van Wyk et al., 2002). It is, however, likely to be higher in societies where plant-based traditional medicines are commonly used. A survey conducted at a large hospital in such an area revealed that poisoning with traditional medicines is the second most common cause of acute poisoning representing 12.1%, mostly of plant origin (Joubert and Mathibe, 1989). Through the centuries traditional medicines based on plants known and selected for their therapeutic effects have been used to good purpose and have undoubtedly cured many more people

than they have killed. However, the levels of poisonous principles in plants are usually unpredictably variable, and occasional overdoses are likely. Furthermore, the cumulative effects of plants taken in over long periods may be subtle and not well understood in traditional medicine. The toxic effects of poisonous plants, particularly chronic effects, are not always easy to reverse, and it has been estimated that 15% of people in southern Africa poisoned by medicinal plants will die, as opposed to 2% suffering from acute, non-plant-induced poisoning (Gaillard and Paquin, 1999).

4.1. Nervous system

There are numerous plants that contain cyanogenic glycosides. Some of these, such as sorghum and cassava (*Manihot esculenta*), are cultivated as crops for human consumption. Cassava is a staple food in large parts of tropical Africa. Toxicity depends on the cultivar, and in general the sweet cassava varieties are less toxic than the bitter varieties. Various strategies have been developed to detoxify the tubers, such as peeling and soaking in water for a day or two before processing. Grating, chopping, and thorough cooking also reduce toxicity. However, acute prussic acid poisoning, often killing entire families, occurs sporadically in areas where cassava of low toxicity is usually used without particular precautions, and consequently tubers containing high concentrations of the cyanogenic glycoside are prepared in the same way. A chronic neurological condition is also associated with cassava consumption. Protein-deficient subjects with low sulphur amino acids are more susceptible and may develop konzo (cassava-associated spastic paraparesis) in Mozambique and East Africa and mantakassa (cassava-associated tropical ataxic myeloneuropathy) in Nigeria after consuming levels that do not affect healthy, well-nourished people (Ellenhorn, 1997; Tor-Agbidye et al., 1999). Long-term ingestion of *Lathyrus sativa* (chickling pea) may result in lathyrism, which is a peripheral neuropathy caused by excitatory amino acids contained by this plant. The disease manifests as a spastic muscle weakness and is clinically very similar to konzo (Ellenhorn, 1997).

Datura stramonium and *Datura ferox* (moon flower, jimson weed, stinkblaar, oliebome) are cosmopolitan weeds that contain parasympatholytic alkaloids such as atropine and hyoscine. Humans are extremely susceptible to their effects and hallucinations may occur, and the proverb "blind as a bat, red as a beet, dry as a bone and mad as a hatter" aptly describes atropine poisoning in humans. Consumption of these weeds is usually inadvertent. The plants may be mistaken for *Amaranthus* and prepared as "marog", resulting in severe poisoning, or the seeds may end up in harvested maize. On occasion, young children have been forced to swallow seeds ("malpitte") during initiation ceremonies at schools (Watt and Breijer-Brandwyk, 1962). Another plant of which the young seedlings may be mistakenly collected as 'marog' is *Nicotiana glauca* (wild tobacco), which contains a pyridine alkaloid, anabasine, which is very similar to nicotine. Ingestion may result in nausea, vomition, gait abnormalities, tremors, confusion and convulsive seizures (Steenkamp et al., 2002).

Boophane disticha (seeroogblom, bushman poison bulb) contains various alkaloids such as buphanidrine, buphanisine and buphanamine. Poisoning usually occurs in humans that utilize the bulb for medicinal purposes. Acute poisoning induces vomition, weakness, coma and mortality (Steenkamp, 2005).

4.2. Cardiovascular system

Cardiac glycoside-containing plants, which cause some of the most economically important livestock toxicoses, are also prominent in causing human plant poisoning (McVann et al., 1992). Cardenolide-containing plants are highly toxic and may be very

important in humans, as a number of garden plants contain cardenolides. *Nerium oleander* (oleander, selonsroos) is a popular ornamental plant widely used in gardens and contains oleandrin. It is extremely toxic, apparently soldiers of Alexander the Great were fatally poisoned when they roasted meat on skewers made from oleander branches, and there are anecdotes of people dying after chewing or sucking the leaves by accident. Yellow oleander (*Thevetia peruviana*) is also very toxic, highlighted by the fact that the fruit is referred to as "Be-still-nut". Certain cardiac glycoside-containing plants are used medicinally. *Digitalis purpurea* (foxglove) contains a cardenolide, digoxin, which is used for the treatment of congestive heart failure. Some of the bufadienolide-containing plants, in particular *Drimia sanguinea* (slangkop, sekanama) and *Bowiea volubilis* (climbing potato) are used by traditional healers in the treatment of various ailments and have been implicated in human poisoning (Marx et al., 2005; Steenkamp, 2005). The sap of *Acokanthera oppositifolia* (bushman poison bush) contains cardenolides and, as the name implies the sap has been used as arrow poisons for hunting by the San people. Humans may accidentally or intentionally become the target, resulting in fatal intoxication.

Although poisoning of humans by the consumption of animals that have died of 'krimpsiekte' induced by cumulative bufadienolides has not been documented, deaths of carnivores by relay poisoning is well known and the possibility that humans could be affected remains an aspect of concern (Henning, 1926). An unusual case of possible secondary poisoning by *Argemone* seeds has been reported in a family that consumed the meat of sheep that had died as a result of feeding on contaminated wheat (Brink et al., 1965). At the time a large number of sheep as well as people were poisoned by the contaminated wheat, but in one particular case consumption of the wheat could be definitively excluded and it seemed probable that the meat of the poisoned sheep had to be responsible.

4.3. Hepatotoxicosis

Hepatotoxic plants may be consumed either in traditional medicines or as contaminants of flour. *Callilepis laureola* (impila, ox-eye daisy, wildemagriet) is widely used as herbal medicine ("muti") and upon ingestion induces severe hepatotoxicity, nephrotoxicity and hypoglycaemia (Wainwright et al., 1977). An atractyloside has been isolated from the tuberous roots (Laurens et al., 2001; Steenkamp, 2005).

Seeds and other parts of the pyrrolizidine alkaloid-containing plants such as *Senecio* species and *Crotalaria* species may contaminate flour. In the 1930s bread poisoning occurred in poor people when they consumed wheat flour contaminated with *Senecio* plant material. They suffered from hepatotoxicity and veno-occlusive disease (Steyn, 1934). Exposure to pyrrolizidine alkaloids through the use of herbal remedies may also be a contributing factor to the high rates of liver cancer and cirrhosis seen in Africa (Steenkamp et al., 2000). Another pyrrolizidine alkaloid-containing plant that is often used as herbal medicine and has also been associated with poisoning is comfrey (*Symphyttum officinale*) (Betz and Page, 1998).

Poisoning by the fruits of cycads (broodbome) has been reported in humans and animals. They contain different glycosides that following ingestion are converted in the intestinal tract to methylazoxymethanol (MAM), which is hepatotoxic and carcinogenic (Spatz et al., 1967). Reitz described that during the Anglo-Boer War hungry soldiers ate the fruit of an *Encephalartos* species and were severely affected (Reitz, 1969).

4.4. Digestive system

Toxalbumins that occur principally in the seeds but sometimes also in other parts of plants are highly toxic plant lectins. Ricin,

derived from the castor oil plant *Ricinus communis*, is one of the most toxic substances known, and features very high on the list of substances likely to be used for bioterrorism. During the Cold War an assassination using ricin was documented where a Bulgarian dissident, Georgi Markov, was eliminated by the implantation in his body of a perforated metal sphere containing ricin. The assassin injected the sphere with a sharpened umbrella tip at a crowded bus stop, and had left by the time the victim collapsed and died (Farrell, 1992). Other members of the Euphorbiaceae family such as *Jatropha curcas* and *Jatropha multifida* also contain a toxalbumin, namely curcin, which can cause severe diarrhoea. The brightly coloured seeds of *Abrus precatorius* (Fabaceae) are used to produce bracelets and necklaces and contain a toxalbumin named abrin. Although abrin is highly toxic, poisoning in humans is unusual, probably because the seeds are extremely hard and indigestible and even if eaten are likely to pass through the digestive tract unbroken (Van Wyk et al., 2002).

Ingestion of the ripe berries of *Melia azedarach*, the exotic syringa tree introduced from India, has been associated with vomition, diarrhoea, dyspnoea, muscle tremors and convulsions in children (Van Wyk et al., 2002).

Vegetables such as green tomatoes, potatoes and brinjals may contain high concentrations of solanine, which can cause irritation of the digestive tract. Many of the Solanaceae produce attractive berries that might be picked and eaten by children, and which are poisonous when unripe, although some become harmless when ripe (Van Wyk et al., 2002).

An indigenous tree, *Spirostachys africana* (tamboti), which is sometimes used for medicinal purposes, is nevertheless highly toxic, and use of the wood in fires over which meat is grilled has resulted in severe diarrhoea after eating the meat. Even the smoke can cause headache and nausea (Palmer, 1981; Van Wyk et al., 2000).

Members of the family Araceae, such as *Alocasia macrorrhiza*, *Dieffenbachia*, *Philodendron* spp., *Monstera deliciosa* and *Zantedeschia aethiopica* (the only indigenous species) may cause severe stomatitis. These plants are grown for their beautiful foliage, sometimes as house plants, and contain insoluble calcium oxalate crystals (needle-sharp raphides), which are packed in specialised ampoule-shaped ejector cells, each with an operculum, called idioblasts. On pressure such as crushing of the stem when chewed the needle-like crystals are ejected and penetrate the surrounding tissue, resulting in intense irritation, discomfort and histamine release (Wiese et al., 1996). According to Gaillard and Paquin (1999), most of the victims in America are children, many under the age of 12 months.

4.5. Skin and adnexa

African poison ivy or pynboom (*Smodingium argutum*) causes an allergic dermatitis and pruritis in sensitive individuals. It even occurs when the individual just passes near the tree (Van Wyk et al., 2002).

Like animals, people who come into contact with the highly irritant milky latex of the candelabra tree or naboom, *Euphorbia ingens,* and the rubber hedge euphorbia, *Euphorbia tirucalli*, develop severe irritation and inflammation, especially when moist mucous membranes are affected (Van Wyk et al., 2002).

5. Discussion

Plant poisonings of livestock are responsible for considerable economic losses in southern Africa (Kellerman et al., 1996). Most of these losses are due to consumption of plants that induce seven well documented toxicoses, namely cardiac glycoside poisoning,

gousiekte, gifblaar, seneciosis, *Lantana camara* hepatotoxicosis, geeldikkop and vermeersiekte. Most if not all of these poisonings occur during periods when pastures are poor and the poisonous plants offer the most obvious source of green feed. In the case of geeldikkop poisoning is related to conditions that render a normally palatable and nutritious plant toxic. Plant poisonings are also more common in stock newly introduced to areas where toxic plants grow, as aversion can play an important role in protecting stock against eating poisonous plants.

Plant poisoning of humans is possibly overwhelmingly linked to the use of toxic plants as medicine, with many cases, including fatal cases, presumed to occur without diagnosis or documentation (Gaillard and Paquin, 1999). Cases of contamination of human foodstuffs, in particular flour, with toxic plants, as well as malicious administration of plant toxins, have also been documented.

The diagnosis of plant poisonings in livestock and humans is not always simple, and depends on a good case history and an evaluation of clinical signs and pathological lesions, as well as confirmatory laboratory tests where these exist. Treatment is not always possible or successful, and prevention remains the most important way to protect humans and animals. The study and documentation of traditional medicines will go a long way towards protecting people from being poisoned by substances that were meant to cure them. For livestock, optimal pasture management and supplementary feeding during times of adversity can prevent many of the outbreaks of poisoning, as well as well-informed farmers having a good knowledge of the local plants likely to cause poisoning and the circumstances under which this is likely to occur. Control of exotic weeds like *Lantana camara* and many other garden escapes with toxic potential is also of great importance in preventing poisoning.

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