Potential plant poisonings in dogs and cats in southern Africa

C J Botha[a*] and M-L Penrith[b,c]

ABSTRACT

Plant poisoning occurs less commonly in dogs and cats than in herbivorous livestock, but numerous cases have been documented worldwide, most of them caused by common and internationally widely cultivated ornamental garden and house plants. Few cases of poisoning of cats and dogs have been reported in southern Africa, but many of the plants that have caused poisoning in these species elsewhere are widely available in the subregion and are briefly reviewed in terms of toxic principles, toxicity, species affected, clinical signs, and prognosis. The list includes *Melia azedarach* (syringa), *Brunfelsia* spp. (yesterday, today and tomorrow), *Datura stramonium* (jimsonweed, *stinkblaar*), a wide variety of lilies and lily-like plants, cycads, plants that contain soluble oxalates, plants containing cardiac glycosides and other cardiotoxins and euphorbias (*Euphorbia pulcherrima, E. tirucalli*). Poisoning by plant products such as macadamia nuts, onions and garlic, grapes and raisins, cannabis (marijuana, dagga) or hashish and castor oil seed or seedcake is also discussed. Many of the poisonings are not usually fatal, but others frequently result in death unless rapid action is taken by the owner and the veterinarian, underlining the importance of awareness of the poisonous potential of a number of familiar plants.

Keywords: cats, dogs, intoxications, poisoning, poisonous plants.

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INTRODUCTION

Plant toxicoses occur worldwide, but southern Africa's rich and varied flora places the region high on the list of areas where stock losses due to plant poisoning are likely. Plant poisoning in farm animals causes serious financial losses in South Africa[33]. In contrast, there are fewer documented cases of plant poisoning in dogs and cats in this region than in the Americas, Europe and Australia. The greatest majority of plant poisonings in dogs and cats are the result of exposure to house or garden plants, or to substances derived from plants that are used for food or other purposes in the home. Most of the plants involved are not indigenous to southern Africa, but are widely grown in homes and gardens throughout the region. Dogs and cats are not normally plant eaters, but might chew plants for various reasons, in particular boredom when confined in the owner's absence and curiosity or teething in puppies. Some causes of plant poisoning in pets, like onions, grapes and raisins, may be fed to them by owners who have no idea that they could be harmful. Dogs and cats sometimes ingest plants apparently to induce emesis and these reactions should not be confused with poisoning[57], but the possibility that the animal could select a poisonous plant for this purpose should be borne in mind.

This paper does not present a complete list of the potentially poisonous plants to which dogs and cats might be exposed, but a review of the more common or interesting plant intoxications occurring in dogs and cats. Intoxications are grouped according to the target organs or systems affected. Cyanobacterial intoxications, mycotoxicoses and mushroom poisonings are not included, and neither is mechanical damage and irritation caused by sharp seeds and awns. Therapeutic modalities are not discussed, because in the majority of cases there are no specific remedies and the treatment is supportive and symptomatic. Managing cases of poisoning usually involves removal or binding of the toxin, for example by the use of an emetic if indicated, or by oral administration of activated charcoal as a general adsorbent and supportive treatment to address the clinical signs. Since poisoning often results in an emergency, rapid intervention may be needed without confirming the diagnosis first. However, submission of plant samples for botanical confirmation is useful in order to determine ongoing treatment protocols and prognosis and to help owners to make the home a safer place for their dogs and cats.

[a]Department of Paraclinical Sciences, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

[b]TAD Scientific, 40 Thomson Street, Colbyn, 0083 South Africa.

[c]Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

*Author for correspondence:
E-mail: christo.botha@up.ac.za

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COMMON PLANT POISONINGS IN DOGS AND CATS

Plant poisoning in dogs and cats has been surveyed in veterinary practices, veterinary toxicological assistance services, poison information centres and animal welfare organisations in North America, Europe and Australia. Because the majority of ornamental garden and house plants have a worldwide distribution regardless of their area of origin, the more frequent causes of intoxication of dogs and cats occur in all the areas surveyed, and a similar situation might be expected in southern Africa.

Nervous system

- *Melia azedarach*

  Syringa berry tree, Chinaberry tree, Persian lilac, *seringboom*

Native to Asia, the Syringa is a medium to large, deciduous tree with dark green foliage and sprays of sweet-scented lilac flowers. The fruits are yellow drupes (Fig. 1) with a single ribbed stone. Syringas are widespread in all but the most arid parts of the country and occur both as invaders and as cultivated ornamental trees in gardens[79]. The principal toxins are tetranortriterpene meliatoxins A_1, A_2, B_1, B_2[28,32] that are present throughout the plant, but the highest concentrations occur in the ripe berries. Toxicity is dose-related and is also apparently influenced by environmental factors, with some trees not being toxic at all[28]. Poisoning usually follows ingestion of the fruits and has been documented in a wide variety of species (pigs, sheep, cattle, goats, rabbits, rats, guinea pigs, poultry and humans)[28]. Poisoning in monogastric animals has a high case fatality rate, but affected humans usually recover. Clinical signs generally develop within hours of ingestion of the fruits, and may be predominantly neurological or gastrointestinal. Neurological signs that have been described include

excitement or depression, ataxia, paresis and convulsions and the patient may lapse into a coma, while the principal gastrointestinal signs are anorexia, vomiting, diarrhoea, which may be bloody, and colic, although constipation has also been described[28]. Signs indicative of respiratory and cardiac distress have also been reported[28] and respiratory arrest is likely to be the cause of death[57]. There is little information about the pathological lesions of *Melia* poisoning in dogs, but experimentally poisoned pigs showed congestion and necrosis of the gastric and intestinal mucosa macro- and microscopically, and other histopathological changes described were lymphoid necrosis, necrosis of skeletal muscle and moderate degenerative changes in the liver and kidneys[46].

Fig. 1: **The yellow drupes of *Melia azedarach.***

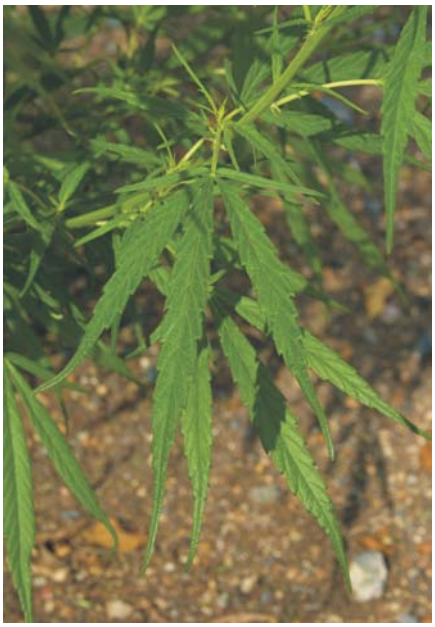

Fig. 2: ***Cannabis sativa* leaves with serrated edges.**

• *Cannabis sativa*
Cannabis, Indian hemp, marijuana, hashish, *dagga*

The plant is an erect annual forb that can grow to a height of several metres, with dark green palmately branched narrow leaves with serrated edges and hairs (Fig. 2). The leaves and flowers are dried to produce marijuana or dagga, which is smoked, or sometimes eaten in baked products, for its euphoric effect. Hashish refers to the dried resin from the flower tops[10]. The active substance is THC (delta-9-tetrahydrocannabinol), for which there is an unique receptor in the brain (CB1), and its effects are due to modification of the function of various neurotransmitters including dopamine, serotonin, acetylcholine, histamine, 5-hydroxytryptamine, opioid peptides, prostaglandins and noradrenalin[13,30,31]. Toxicity is variable because the amount of THC in plants varies. Clinical signs in dogs induced by inhalation or ingestion of a small amount of the drug have been documented, but it has been demonstrated that the lethal dose in dogs is more than 3 g and therefore cannabis poisoning in dogs is rarely fatal[30,54]. Few instances of intoxication in animals other than dogs have been reported; in a veterinary practice survey that spanned 5 years in Queensland, 62 cases of cannabis poisoning in dogs and only 1 in a cat were recorded[44]. Dogs become intoxicated owing to wilful exposure to smoke or drug-laced cookies by their owners, by ingestion of marijuana cigarette butts or, in 1 documented case, cut green marijuana[19]. Clinical signs develop shortly after intake, usually within 1–3 hours. The predominant clinical signs may be neurological or gastrointestinal or a combination of both. In a survey of 213 dogs poisoned by cannabis, 99 % exhibited neurological signs, while only 30 % developed gastrointestinal signs[31]. In this survey only 10 dogs had ingested cookies containing cannabis, the other 203 having eaten either leaves or cigarettes. The most frequent clinical signs were depression (60 %), ataxia/incoordination (59 %), vomiting (25 %), tremor (18 %) and mydriasis (10 %), all other signs described by various authors such as hypersalivation, urinary incontinence, weakness, head pressing, disorientation, hyper-excitability, coma, an irregular pulse and hyperthermia occurred in less than 10 % of the affected dogs[31]. Snapping at the air has been reported and is suggestive of hallucination[30]. Increased heart and respiratory rates have been reported[19], as well as on rare occasions convulsions[10]. The signs may be mild and non-specific, making diagnosis difficult if a history of ingestion of cannabis is not provided. A 4-month-old Pekinese puppy that consumed half a marijuana cigarette became ataxic 45 minutes later; the ataxia persisted for 6 hours, followed by sleepiness for 18 hours, after which recovery was uneventful[11]. A case of allergic inhalant dermatitis (canine atopy) in a dog living in a house where the previous owners had cultivated *Cannabis sativa* in- and out of doors has been reported[17]. Most animals recover within a period of 48–72 hours and no specific serum chemistry changes or pathological lesions have been described. In the absence of a history of exposure to cannabis, which may be concealed by the owner, THC can be detected in plasma or urine in laboratories that are equipped to perform the test[30].

• *Macadamia integrifolia*, *M. tetraphylla*
Macadamia nuts

Macadamia nuts (Fig. 3) are produced by trees of the genus *Macadamia,* which were introduced into South Africa from Australia and are cultivated in the warmer northeastern parts of the country. The toxic principle is unknown. While some

varieties of macadamia nuts contain toxic levels of cyanogenic glycosides, these have a bitter flavour and are not used as food. Dogs are the only species in which toxicity has been reported, and the reaction appears to be consistent in dogs that have eaten macadamia kernels, suggesting that it is not due to allergy[45]. The dose required to induce toxicity has not been determined accurately, but 5–40 kernels were capable of inducing clinical signs in a range of dogs whose cases were reported[45]. Clinical signs develop within 12–24 hours after ingestion of the nuts and are characterised by posterior weakness, stiffness or paresis and muscular tremors[26,45,57]. Swelling and pain in the hind limbs, including the joints, have been reported[45], with elevated GGT, ALT and AST in animals where these parameters were measured. These effects may be secondary to the locomotor difficulties experienced and were not reported in an experimental study in which macadamia poisoning was induced in 4 dogs[27]. Some dogs are hyperthermic[26,27,45]. Vomiting occurs in some dogs[26,27], as well as abdominal pain and mucosal pallor[57]. Clinical pathology was within normal limits in the experimental study except for a transient increase in serum triglycerides[27]. Dogs usually recover uneventfully within 48 hours or less[57]. No pathological lesions are described. While macadamia poisoning is generally unusual, 83 cases were reported in Queensland, Australia, over a period of 5 years[44]; Queensland is a major area for macadamia cultivation and farm dogs may have access to stores. Macadamia nut poisoning has also been reported in the United States of America[57].

Fig. 3: **Macadamia nuts**.

❖ Solanaceae

- *Solanum* spp. *Brunfelsia* spp.
  Poison apples, nightshade, yesterday, today and tomorrow

Many species of *Solanum* contain solanine, a glycoalkaloid that induces both neurological and gastrointestinal disturbances. Poisoning of dogs and cats due to ingestion of annual or perennial flowering *Solanum* species, which bear round berry-like fruits and are most poisonous when green, is reported to be fairly common in the USA[51]. Typical gastrointestinal signs are vomiting and diarrhoea, but in 1 case traced back to consumption of black nightshade berries (*S. nigrum*), the clinical signs noted were a foul, garlic-like breath and incontinence when asleep[12].

Poisoning due to ingestion of *Brunfelsia,* a genus native to South America and the Caribbean, but widely used as an ornamental shrub in gardens, has been reported in Australia and North America, mainly in dogs but also in cats[35,43,44,65]. It is an attractive erect shrub with bright green shiny foliage and clusters of flowers that change from purple through mauve to white, giving rise to the common name (Fig. 4). The fruits are considered to be the most poisonous part of the plant, but all parts have been able to induce intoxication[65]. Brunfelsamidine, a convulsive substance, has been isolated from *B. grandiflora*[78]. Onset of clinical signs is rapid and diagnosis can usually be confirmed by the presence of the plant in the gastrointestinal tract and/or vomitus and faeces[65]. Clinical signs described include salivation, oro-nasal irritation, choking and gagging, vomiting, foetid to haemorrhagic diarrhoea, frequent urination, weakness, anxiety, depression, ataxia and seizures, sometimes prolonged and only able to be controlled with general anaesthesia[4,35,43,53,65]. Most dogs recover if treated early. The plant species may influence the severity of effects. The first known intoxication with *B. calycina* var. *floribunda* (= *B. pauciflora*) involved the death of an 11-week-old Schipperke puppy within 2 hours of developing acute onset anxiety, persistent sneezing, vomiting, generalised muscle tremors and pyrexia of 40.7 °C that progressed to severe disorientation, staggering, ataxia, proprioceptive deficits, loss of the righting reflex and seizures, culminating in death. The seeds were found in loose stools passed during this period and toxicity was confirmed in rats and mice[69]. Clinical pathological changes were associated with dehydration, and no specific pathological lesions were observed in laboratory animals in which the intoxication was reproduced[65,69] or in a puppy that died[35]. Moderate oedema and hyperaemia of the terminal ileum was observed in 2 dogs that were poisoned experimentally and developed severe clinical signs and mild necrosis and desquamation of the villous epithelium was observed microscopically in the dog that received the higher dose[43].

- *Datura stramonium*
  Jimsonweed, thorn apple, moonflower, *stinkblaar*, *olieboom*

Datura stramonium, an annual weed with large, dentate, foul-smelling leaves, tubular white or purple flowers and spiny

Fig. 4: **A *Brunfelsia* species.**

fruits that contain small pitted seeds, is sometimes grown as an ornamental plant. The toxic principles are parasympatholytic alkaloids, atropine, hyoscine and hyoscyamine[32]. Toxicity in animals is rare, but several cases in horses have been described and were due to contaminated feed[52]. A fatal case occurred in a 1-year-old miniature poodle after clinical signs that lasted for 2–3 hours. These were described as agitation, aggression, ambulatory delirium, tachycardia, tachypnoea, mydriasis, seizures and convulsions, progressing to coma and death. At necropsy multiple organs were severely congested, there was acute lung oedema, and acute cardiac dilatation with multiple pericardial petechiae; histopathology revealed multifocal centrilobular hepatic necrosis, renal tubular degeneration, and widespread congestion and haemorrhages, including the central nervous system[74]. The pathological changes were considered to suggest an evolution towards hypotension, heart failure, disseminated intravascular coagulation and multiple organ failure[74]. A case of repeated anisocoria in a dog that was caused by contact with damaged plant material has been described[25].

Gastrointestinal tract

❖ Bulbs and bulb-like plants

• *Ornithogalum* spp.
Chinkerinchee, star of Bethlehem, *tjienkerintjee*

Chinkerinchees are bulbous plants of the family Hyacinthaceae that produce heads consisting of star-shaped white flowers and are frequently used as ornamental plants in gardens or in flower arrangements or bouquets. There are a number of species that have been identified as toxic, with *O. thyrsoides* (Fig. 5) and *O. saundersiae* particularly incriminated in cases of livestock poisoning in South Africa[32]. Reports of natural cases of poisoning due to ingestion of *Ornithogalum* by dogs are lacking, but 0.5 g of dried, pulverised bulbs of *O. saundersiae* proved fatal to dogs within 4 days[32] and there is also an account of experimental poisoning of a dog by *O. amoenum* in North Africa that resulted in haemorrhagic enteritis and death within 5–6 days[18]. The toxic principle appears to be cholestane glycosides, isolated from South African species of the genus[32]. The clinical signs produced in dogs have been severe haemorrhagic enteritis[18].

Phenanthridine alkaloids are present in many popular garden lilies (*Narcissus narcissus,* jonquil, daffodil; *Tulipa* – tulip; *Amaryllis* – amaryllis, crinum; *Hyacinthus* – hyacinth) that can cause mild to severe gastrointestinal signs depending on the amount ingested[57]. Besides the alkaloids

Fig. 5: ***Ornithogalum thyrsoides.***

the plants also contain glycosides[10]: the alkaloids include narcissine, narciclasine, galanthamine and lycorine, of which the last is present at the highest concentration; glycosides include scillitoxin. Calcium oxalate crystals are also present (daffodil). Poisoning is usually due to ingestion of bulbs but can occur after eating flowers or drinking the water in which the cut flowers have stood. Clinical signs include vomiting, diarrhoea, abdominal tenderness, anorexia, salivation, pyrexia, lethargy and mucosal pallor[10]; in severe cases ataxia, collapse, hypothermia, hypotension, bradycardia, severe abdominal pain, hyperglycaemia and dehydration may be present, and the affected animal may not recover. Contact with sap can cause pruritis and erythema. *Post mortem* changes are non-specific, consisting of evidence of severe gastroenteritis and plant material in the intestine. A popular South African lily, *Clivia miniata* (clivia) (Fig. 6), contains phenanthridine alkaloids (lycorine, clivonine, clivatine, miniatine, hippeastrine) in the leaves, stems and bulbs and is widely used as a garden or house plant, but rarely causes poisoning[36].

Irises (*Iris* spp.) contain irritant resins (irisin, iridin and irigenin) in the rhizomes that can cause vomiting, diarrhoea and colic, with congestion of the gastrointestinal tract, and sometimes also damage to the liver and pancreas[37].

❖ Plant lectins

• *Ricinus communis*
Castor oil plant, *kasterolieboom*

• *Abrus precatorius*
Lucky bean tree, *minnie-minnie*

The castor oil plant is a cosmopolitan

Fig. 6: **Flowers of *Clivia miniata.***

shrub or small tree with many grey-green to reddish branches bearing large palmate leaves. The spiny 3-lobed fruits contain 3 hard shiny seeds that resemble engorged ticks (Fig. 7). The toxic principle, ricin, is a toxalbumin (lectin, glycoprotein) that is present in all parts of the plant, but the seeds contain the highest concentration and are usually the source of natural cases of poisoning. Pure ricin is one of the most lethal toxins known, but the oral LD_{100} in mice is 25 000 times lower than when the toxin is administered parenterally[32]. The lethal dose for dogs is 1–2 g castor seed/kg body mass, which is equivalent to 0.03–0.04 mg ricin/kg[39].

Absorption of ricin from the gut is relatively poor, although in spite of its protein nature it can survive the action of proteolytic enzymes in sufficient quantities to cause toxicity. Once absorbed into cells, its mode of action is to interrupt protein synthesis[32]. All species investigated are highly susceptible to ricin poisoning. Although the plant is a widespread invader, is also cultivated for oil and has even been described as an ornamental plant[67], it is not generally available to companion animals. Toxicosis in dogs is rare and inevitably associated with ingestion of the seeds, which must be broken by mastication or other means for the toxin to become available. Most of the reported cases resulted from ingestion of the beans that were available in homes for various reasons, including in decorative handicraft[2]. The castor seed cake (containing 3–5 % ricin) obtained in the process of extracting castor oil is toxic unless heat treated and poisoning of dogs due to consumption of the cake when used as fertiliser on garden or house plants has been described[39,67]. A survey in the USA indicated that 76 % of dogs exposed to castor beans developed clinical signs[2], suggesting that in a percentage of dogs the beans pass through intact and toxin is not absorbed. When toxin is absorbed from the gut, clinical signs usually develop within hours; the survey cited above found an average time of 6 hours after ingestion, with a range of 0.5–42 hours[2]. The most prominent clinical sign is vomiting, followed by depression and watery diarrhoea, which frequently contains blood. Abdominal pain, anorexia and haematemesis are reported in fewer animals, and 5–8 % of dogs surveyed showed weakness, ataxia, hypersalivation, recumbency or tachycardia. Fewer than 10 % of the dogs died or were euthanased due to poor prognosis. However, this depends on the amount of toxin absorbed and the length of time between ingestion and treatment. All of the dogs poisoned by consumption of castor seed cake ferti-

Fig. 7: **The ripe fruits and seeds of *Ricinus communis.***

liser died as a result of circulatory collapse or hypovolaemic shock[39,67]. The mode of action of the toxin implies that many cell types can be affected. Two fatal cases of ricin poisoning in dogs indicated that hepatic necrosis (both cases), cardiac haemorrhage and necrosis (1 case) and elevated blood urea (1 case) occurred in addition to gastrointestinal lesions, as well as macro- and microscopic lesions in lymphoid tissues[50,67]. Elevated hepatocellular enzymes were the most consistent blood chemistry abnormality in dogs investigated[2]. Confirmation of the diagnosis is usually based on a history of consumption of castor beans and their identification in gastrointestinal content or faeces, but detection of ricinine in gastric content by liquid chromatography/mass spectrometry has been described[50]. Ricin is antigenic and immunity can be induced by the repeated administration of small quantities[32].

The seeds of *Abrus precatorius* (love bean, lucky bean, *minnie-minnie*) (Fig. 8) also contain a toxalbumin, abrin, that has been suspected in an outbreak of poisoning in cattle in Limpopo Province, South Africa[32], but poisoning in dogs or cats has not been documented. The seeds are much smaller than castor beans, with a very hard shell, and therefore unlikely to be chewed.

❖ *Araceae*

- *Alocasia* and *Colocasia* spp.
  Elephant's ear
- Dieffenbachia spp.
  Dumb cane
- *Philodendron* spp.
- *Monstera deliciosa*
  Delicious monster
- *Zantedeschia aethiopica*
  Arum lily, *varkoor* (Fig. 9)

The plants belonging to this family are ornamental garden or house plants with attractive foliage and flowers. The stems and the large, green or varicoloured smooth leaves contain insoluble calcium oxalate monohydrate crystals or raphides that have a highly irritant effect on mucous membranes[32]. The crystals are contained in specialised cells called idioblasts that, when ruptured, release the sharp spicules formed by the crystals, which become embedded in the mucous membranes of the mouth, tongue and throat. Severe irritation results from physical damage by

Fig. 8: **Pods and seeds of *Abrus precatorius.***

the sharp crystals and the release of plant proteolytic enzymes that provoke the release of inflammatory mediators including histamines[24,37,41,51]. Probably all species are susceptible, but as these plants are most often grown indoors, poisoning has mainly been reported in humans, dogs and sometimes cats[37]. Clinical signs appear immediately to up to 2 hours after exposure. Pain is indicated by head shaking, pawing and rubbing and sometimes vocalisation; increased salivation, anorexia and depression may occur and, if large amounts have been ingested, vomiting and diarrhoea, usually mild but sometimes haemorrhagic, and abdominal pain may be present[37,51,57]. Gastrointestinal signs were succeeded by debilitation, twitching and trembling, and rarely opisthotonus was reported in animals after ingestion of *Philodendron,* and there were indications that the toxin accumulated[24]. Swelling and oedema of the oral mucous membranes are often evident and in a severe fatal case extensive mucosal necrosis with acute respiratory distress was reported[41]. Most animals recover uneventfully with or without supportive treatment. For this reason, descriptions of pathology are scant and superficial, referring only to mucosal inflammation and necrosis.

• *Hedera* spp.
English ivy

Ivies are perennial creeping plants used as climbers, ground cover and house plants. The berries in particular contain pentacyclic terpenoids that cause hypersalivation, vomiting, abdominal pain and diarrhoea. In most cases the clinical signs are mild, but severe manifestations may occur[57].

• *Lonicera* spp.
Honeysuckle

Honeysuckle is a popular climbing plant that bears sweet-scented cream and pink flowers. Ingestion of fruit can cause mild gastrointestinal effects within 2 hours due to valerianic acid and xylostein, which have an irritant effect. Vomiting, diarrhoea, which may be bloody, depression, lethargy, and in 1 case convulsions, have been reported[10].

• *Laburnum* spp
Laburnum, golden rain

Laburnums are ornamental shrubs that grow to a height of several metres, with small green leaves and pendulous racemes of bright yellow flowers. Pods, leaves and flowers have caused poisoning in dogs that ingested them[10]. The toxic principle is a quinolizidine alkaloid, cytosine, that has a similar mode of action to nicotine on autonomic ganglia receptors. The principal clinical signs are salivation, vomiting and diarrhoea that commence soon after ingestion and vomiting may persist for 1–2 days. In severe cases, which are very rare, neurological signs including delirium and convulsions may occur, followed by respiratory paralysis and death. There are no specific pathological lesions apart from acute gastroenteritis[10].

Fig. 9: ***Zantedeschia aethiopica* flower.**

• *Erythrophleum lasianthum*
Swazi ordeal tree

The Swazi ordeal tree (Fig. 10) is a medium to large tree with pinnate leaves, dense spikes of cream or yellowish flowers, and flat brown seed pods; it is described as poisonous[55]. It is found on sandy soil in a small area in northeastern South Africa, Swaziland and southern Mozambique. An unpublished account of suspected poisoning of a puppy by ingestion of pods of the Swazi ordeal tree exists in correspondence with the Department of Paraclinical Sciences, Faculty of Veterinary Science, University of Pretoria. A 2-month-old Yorkshire terrier puppy suddenly started vomiting and vomited continuously for an hour and a half. Anorexia and lethargy followed, as well as diarrhoea, although the owner believed that this may have been induced by de-worming as part of the symptomatic treatment given by a veterinarian. Within 24 hours the dog was very weak, retching and having occasional convulsions, as well as diarrhoea. Recovery was gradual, but appeared to be complete. About 3–4 weeks later another episode of vomiting occurred. Pods of the Swazi ordeal tree were observed in the vomitus and the dog recovered within a few hours, possibly because most of the toxic material was eliminated rapidly.

Liver

❖ Cycads

• *Encephalartos* spp., *Cycas revoluta*, *Macrozamia* spp., *Zamia* spp.
Cycad palms, *broodboom*

Cycads are palm-like plants with dark

Fig. 10: **Pod of *Erythrophleum lasianthum* (Swazi ordeal tree).** (Courtesy of Geoff Nichols.)

green fronds that bear male and female fruits, the latter somewhat resembling pine cones. A strictly protected genus indigenous to southern Africa is *Encephalartos*[55]. Twelve of 28 species have been shown to be toxic and all should be regarded as potentially dangerous[75]. Exotic cycads (Fig. 11) are also widely grown in gardens or, in the young stages, as house plants. Most of 60 cases of cycad poisoning in dogs in USA[3] and the only case reported in South Africa[7] involved *Cycas revoluta*. *Macrozamia* sp. has caused poisoning of dogs in Australia[48], where a 5-year survey indicated that cycad poisoning is relatively common in dogs and was also reported in 1 cat[44]. Fatal poisoning of 2 dogs by *Zamia floridana* has been described[64]. Any part of the plant can be toxic but the seeds appear to be the most toxic part; however, the case described in South Africa involved ingestion of the stem[7]. One of the toxins, a glycoside, cycasin, is converted by β-glucosidase to methylazoxymethanol, which is teratogenic, mutagenic and carcinogenic, and causes hepatic damage with gastrointestinal signs. Two other toxins have been reported, an amino-acid β-methylamino-L-alanine that experimentally causes ataxia in rats and an unidentified compound with a high molecular weight that may cause axonal degeneration and hind limb paralysis in cattle[3,57]. A variety of species may be affected, including humans. In a survey in dogs in the United States of America , the onset of clinical signs occurred from 15 minutes to 3 days after ingestion and lasted for 24 hours to 9 days. Dogs usually develop gastrointestinal signs, with vomiting, sometimes with haematemesis, being the most consistent. Other signs include depression, diarrhoea that may be haemorrhagic and anorexia; neurological signs that include weakness, ataxia, proprioceptive deficits, coma or seizures have been reported[57], and occurred in more than half of the 60 dogs surveyed in the USA[3]. Elevated blood levels of alanine aminotransferase, bilirubin and alkaline phosphatase appear to be consistent[3,7,51,57] and indicate damage to the liver. Alterations in white blood cell counts and thrombocytopaenia are probably indicative of inflammation and compromised liver function. Elevated levels of ammonia and blood urea nitrogen (BUN) have also been reported[3,37]. Pathological lesions described in sheep were mostly in the liver and consist of megalocytosis, nuclear hyperchromasia, bile stasis, fatty change and fibrosis, while renal tubular necrosis appeared also to be a fairly consistent change[34]. In the American survey, 67.7 % of dogs that were treated survived the poisoning.

Fig 11: **An exotic *Cycas* species.**

Kidney

❖ Soluble oxalate-containing plants

Numerous plants contain soluble oxalates, including common garden plants and vegetables eg. Mesembryanthemaceae (mesems, *vygies*), *Beta vulgaris* (beetroot), *Spinacia oleracea* (spinach), *Oxalis* spp., *Parthenocissus quinquefolia* (Virginia creeper) (Fig. 12), *Rheum* spp. (rhubarb) and *Opuntia* spp. (prickly pear, *turksvy*). Soluble oxalate poisoning has been described in several species of livestock[32] but is relatively rare in dogs and cats[37]. Poisoning occurs when the absorbed oxalates bind serum calcium, resulting in systemic hypocalcaemia. Clinical manifestations include muscle fasciculations, tetany, convulsions, cardiac arrhythmia and nephrosis, which is indicated by polyuria that may progress to anuria. Animals may be weak and lethargic, and show generalised incoordination and signs of abdominal distress[37]. Blood chemistry reveals low calcium and elevated ammonia and BUN. The most prominent histopathological lesions are severe nephrosis with deposition of oxalate crystals in the kidney tubules, dilatation and necrosis of tubules with hyaline and cellular casts, and sometimes tubular regeneration[32].

❖ Liliaceae

- *Lilium*, *Hemerocallis* spp.
  Day lilies, tiger lilies, Easter lilies

The genera *Lilium* and *Hemerocallis* (Fig. 13) contain various species that are widely used as house and garden ornamental plants. They have large, attractive open tubular flowers with prominent stamens. A study has shown that toxicity is caused by soluble compounds in the aqueous fraction of leaves and flowers, but these have not yet been identified; the concentration appears to be higher in the flowers than in the leaves[63]. Cats are the only animals to which these plants are known to be nephrotoxic; attempts to induce the syndrome in rabbits and rats failed, and dogs are reported to develop only gastrointestinal signs[23]. Although it is relatively unusual for cats to eat plants,

Fig. 12: **Berries of *Parthenocissus quinquefolia* (Virginia creeper).**

there have been a large number of reports of nephrotoxicity in cats due to ingestion of Liliaceae[20,22,23,40,47,57,68,70,73]. Consumption of less than 1 leaf is capable of causing the syndrome[57,80]. In a survey of 52 cats that had eaten day lilies, 77 % showed gastrointestinal signs of vomiting and hypersalivation, 36 % developed neurological signs (ataxia, depression, tremors and seizures), and 32 % progressed to acute renal failure, which commenced 24–48 hours after ingestion and was always fatal[22]. Vomiting, anorexia and depression may be observed within 2 hours after ingestion of the plant, but signs of renal failure appear from 24 to 72 hours. Vomiting, anorexia and lethargy persist, polyuria develops and abdominal pain is usually present due to swelling and tenderness of the kidneys[20]. After about 12 hours, polyuria and uraemia occur, with marked elevation of BUN and serum creatinine, and the fact that creatinine is often extremely high in comparison to BUN has been reported to be a unique feature of lily nephrotoxicosis[73]. Later, polyuria is succeeded by oliguria progressing to anuria. Mortality is high, even when cats are treated; recovery may be uneventful or may be incomplete and the cat may eventually succumb to chronic kidney failure[20]. Pathological changes at necropsy are most prominent in the kidney and consist of severe necrosis of the proximal convoluted tubules with the basement membranes remaining intact, and interstitial oedema[63,68,73]. The occasional presence of birefringent crystals is probably the result of decreased excretion of oxalates[73]. Cytoplasmic vacuolation of exocrine pancreatic acinar cells and hepatic lipidosis were also observed in experimentally poisoned cats[63]. Electron microscopic examination indicated that the toxin targets mitochondria[63].

❖ Grapes and raisins

• *Vitis* sp.

Grapes and raisins have frequently been described as a cause of toxicity in dogs[9,16,21,42,49,56,76]. The toxic principle is as yet unknown. Investigation of available cases appears to rule out contamination of the grapes with pesticides, heavy metals or mycotoxins like Ochratoxin A[49]; factors like the excessively high sugar content of grapes and raisins leading to hypercalcaemia and the possible involvement of vaso-active substances, as well as possible anaphylaxis, have been discussed[49,56]. Grape and raisin toxicity has only been described in dogs. Toxicity appears to be highly variable. The amount consumed is often not known, particularly if grapes are eaten off the vine, but dogs have been seriously affected by amounts varying from 2.8–36.4 g/kg body mass of raisins and a few grapes to 19.6–148.4 g/kg body mass[16]. Furthermore, not all dogs appear to be equally susceptible, although no breed or age predisposition has been identified. Many dogs are reported to have eaten grapes or raisins with no ill effects[16], and in 1 reported case 1 of 3 dogs was so severely ill that the owner opted for euthanasia, while 2 other, older dogs that also ate grapes and grape residues were unaffected[76]. Clinical signs have been reported to develop 12–72 hours after ingestion[16]. Vomiting was reported in all cases[9,16,21,42,49,56,76]. Lethargy and anorexia have been reported in more than 70 % of cases, diarrhoea and decreased urine output in around 50 %, and abdominal pain, ataxia and weakness in a lower percentage[16]. Animals with oliguria or anuria are considered to have a poor prognosis. Hypercalcaemia, hyperphosphataemia and elevated serum creatinine and BUN have been reported in most cases for which data are available and are typical of azotaemia[16]. The prognosis is guarded, particularly if the dog is already in acute renal failure, but it can be improved by aggressive therapy[16,56]. A bull terrier that had consumed an entire punnet of grapes and was immediately submitted to the veterinarian for therapy did not develop clinical signs[15,] although with the variability in development of the syndrome it is impossible to know whether toxicity would have occurred had the dog not been treated. Pathological lesions have been described consistently as acute renal tubular degeneration and necrosis, with cast formation; basal membranes remain intact and there is evidence of regeneration in cases of longer duration[16,42,49]. A golden intracytoplasmic pigment was present in a number of cases submitted to detailed histopathological examination, but its nature could not be determined histologically[49].

Fig. 13: **A *Hemerocallis* species.**

Skin

❖ *Euphorbiaceae*

• *Euphorbia pulcherrima*
Poinsettia

• *E. tirucalli*
Rubber euphorbia, hedge euphorbia, *kraalnaboom*

Poinsettias are ornamental plants with cream, pink to bright red bracts (Fig. 14) of which there are many varieties that are widely used as garden and house plants, and whose stems contain milky latex that is severely irritating to the skin, mucous membranes and gastrointestinal tract. The toxic principles in the latex of euphorbias are diterpenoid esters that cause increased salivation, vomiting, and rarely diarrhoea[10,57]. They are sometimes regarded as irritant rather than toxic[51]. Nevertheless, poisoning by poinsettias was the most frequently encountered phytotoxicosis at an Italian toxicology assistance centre, and most often involved cats[1]. *Euphorbia tirucalli* is a shrub or small to medium-sized tree without spines and with small cylindrical leaves that rapidly fall off the slender, cylindrical branches that occurs in the eastern parts of southern Africa[79]. It is used as a hedge and has caused severe skin irritation and damage to cattle[32] and could therefore potentially harm dogs forcing their way through hedges as well.

Cardiovascular system

❖ Cardiac glycosides

Cardiac glycoside poisoning is very important in farm animals in South Africa[32], but is rarely encountered in companion animals. It is likely that more companion animals experience the beneficial effects of the digitalis glycosides that are used to

manage congestive heart failure than are poisoned by ingesting plants that contain them. However, a few ornamental garden plants contain cardenolide cardiac glycosides and can cause acute and rapidly fatal cardiac glycoside poisoning[57]. The most commonly encountered are the ornamental shrubs *Nerium oleander* (oleander, *selonsroos*) and *Thevetia peruviana* (yellow oleander) (Apocynaceae). Both are tall woody shrubs with narrow dark green leaves and fragrant flowers, those of *Nerium* being white, pink or red, while *Thevetia* has yellow flowers with the tips of the petals twisted to the right. *Digitalis purpurea* and *D. lanata* (foxglove) are woody flowering perennials that bear clusters of purple or white flowers that are sometimes cultivated in gardens. *Convallaria majalis* (lily of the valley) is also sometimes grown, although more commonly in the northern hemisphere. *Kalanchoe, Cotyledon* and *Tylecodon* spp. (*plakkies*) contain bufadienolides. These are succulent plants with attractive flowers and are popular as house and garden plants. Cardiac glycosides inhibit the sodium-potassium adenosine triphosphatase (Na-K pump), which results in disturbances of transmembrane iso-electrical gradients. Cardiovascular, gastrointestinal, neurological and respiratory effects may be induced. In acute cardiac glycoside poisoning death is usually due to cardiac and/or respiratory failure[32]. The physiopathology of chronic cardiac glycoside poisoning (*krimpsiekte*) induced by *plakkies* is less well understood, and may involve binding of the cumulative bufadienolides to nicotinic acetylcholine receptors at the neuromuscular junction[6]. Toxicity is generally high, with small amounts of plants ingested being able to induce severe illness. Toxicity is almost invariably caused by ingestion of plant parts, but secondary chronic cardiac glycoside poisoning has occurred in dogs (and humans) after eating the meat of sheep and goats that have died of *krimpsiekte*[32]. Here, it is interesting to note that there are anecdotal reports of secondary monofluoroacetate poisoning in dogs after ingesting part of the intestinal tract and its content of ruminants that have succumbed as result of *gifblaar* poisoning (*Dichapetalum cymosum).*

Clinical signs ascribed to cardiac glycosides develop soon after ingestion of the toxic material, and death may be almost immediate, or occur up to 2 days after the onset of clinical signs[57]. Clinical signs include cardiac arrhythmias, abdominal pain, anorexia, vomiting and diarrhoea and terminal seizures[51,57]. It has been reported that male dogs are more likely to develop clinical signs[47]. Dogs suffering from *krimpsiekte* exhibit a stiff or high-stepping gait, with the head bent downward so that the muzzle points at the chest, slackness of the jaw with salivation, and, like other affected animals, tire easily and become recumbent[32]. Serum chemistry changes include hypoxaemia, hypercarbia, hyperkalaemia and acidosis[32]. Death as a result of acute cardiac glycoside poisoning is often too rapid for significant pathological lesions to develop, but animals that survive for a day or 2 may show scattered foci of myocardial necrosis[61].

Fig. 14: **The bright red bracts of a poinsettia.**

• *Rhododendron* spp.
Rhododendrons, azaleas

Several genera of shrubs belonging to the family Ericaceae are used as ornamental plants or hedges. They are native to the northern hemisphere, but azaleas in particular are widely used as ornamental plants in southern Africa. They are flowering shrubs (Fig. 15) that grow well in acidic soils. Various plants of the family including azaleas contain diterpene resins known as grayanotoxins, which bind to receptors of cell membrane sodium channels, blocking them and causing cell membranes to remain in a state of depolarisation[10,37,47]. As may occur with cardiac glycosides, the poisoned animal will usually exhibit gastrointestinal, nervous, cardiac and respiratory signs. Death is usually due to the cardiovascular effects[47]. Vomiting is the most consistent clinical sign[57]. Death of a cat within hours of presumed ingestion of azalea plant material has been described[62]. Clinical pathology and *post mortem* findings are non-specific[10,57].

• *Persea americana*
Avocado

Poisoning by the Hass and Fuerte varieties of avocado has been documented in a wide variety of animals, but only a single suspected case has been reported in dogs[32]. The toxic principle appears to be persin, which has experimentally induced cardiomyopathy in goats at higher doses and mastitis at lower doses[32]. Two dogs with a reported fondness for Fuerte avocadoes, to which they had had free access for less than a year, were presented to the University Veterinary Teaching Hospital in Nairobi, Kenya, with a history of poor appetite, exercise intolerance, constipation, abdominal enlargement and dyspnoea[8]. One of the dogs was dead when presented, and the other was emaciated, with signs of congestive heart failure that included orthopnoea, ascites, pitting oedema of the hind limbs, a jugular pulse, muffled heart sounds and rales and gross cardiomegaly revealed by thoracic radiography. Serum levels of alanine aminotransferase, lactate dehydrogenase and alkaline phosphatase were moderately elevated. Necropsy revealed anasarca of the hind limbs, ascites, lung oedema and fluid in the pericardial and pleural cavities. Microscopic changes consisted of multifocal degeneration of myocardial fibres with infiltration of mononuclear leucocytes.

• *Taxus* spp.
Yew

Yew is an evergreen shrub or tree commonly planted in gardens and churchyards and often used to form hedges in the northern hemisphere, but is not commonly encountered in southern Africa. It contains cardiotoxic alkaloids, taxines, that depress conduction through the heart. Animals frequently die without developing clinical signs, but these may be delayed for up to 2 days and consist of trembling, incoordination, diarrhoea and collapse, with death due to acute cardiac arrest[10,51].

Fig. 15: **An azalea (*Rhododendron* species).**

Haematopoietic system

• *Allium cepa*, *A. sativum*
Onions, garlic

Onions and garlic are common vegetables widely used in human food and therefore available to household pets that are fed table scraps or, more rarely, are fed meals prepared for them at home instead of commercial pet food. Garlic is even sometimes intentionally given to dogs as it is perceived to be a health supplement. There are many reported cases of haemolytic anaemia in dogs after eating either raw or cooked onions, and the toxicosis has been reproduced experimentally in both dogs and cats[38,60,72]. Sodium n-propylthiosulphate isolated from boiled onions has been demonstrated to cause haemolysis, which is apparently aggravated by the presence of 2 other compounds, sodium trans-1-propenylthiosulphate and sodium cis-1-propenylthiosulphate[81]. Further studies have indicated that sodium *n*-propylthiosulphate increases methaemoglobin concentration and Heinz body count and decreases the concentration of reduced glutathione in erythrocytes, while another compound present in onions, *n*-propyl disulphide, causes a marked decrease in glucose-6-phosphate dehydrogenase (G6PD) activity[72]. Most of the cases reported involved ingestion of large amounts of onions either as a single dose or over time. Toxicosis was experimentally induced in small dogs at 30 g/kg body mass[72]. Haemolytic anaemia due to consumption of onions or garlic has been reported in horses, cattle, sheep, dogs and cats[38,60]. Haemoglobinuria developed within 12 hours of ingestion in dogs experimentally fed pure onions[72]; other signs observed in experimental and field cases were anorexia, lethargy, depression, weakness, pale mucous membranes, abdominal pain, vomiting, diarrhoea or soft faeces, tachycardia, tachypnoea, seizures, and splenic enlargement[14,29,66,72,77]. Changes in blood parameters indicative of anaemia were evident within 24 hours and included a decrease in haematocrit, increased reticulocyte count, increase in Heinz bodies (with a peak at day 3), anisocytosis, leptocytosis, poikilocytosis, displacement of methaemoglobin to one side in erythrocytes and intravascular haemolysis[14,29,38,60,66,72]. Serum chemistry was generally normal apart from high levels of serum bilirubin (both direct and total)[72]. Neutrophilia with left shift was also reported[29]. Most of the changes peaked at around Day 3 and then returned to normal over a varying period of time. Recovery is usual and pathology has not been described.

• *Cotoneaster* spp., *Pyracantha* spp.
Cotoneaster, firethorn

Cotoneaster and firethorn are shrubs that are used as ornamental bushes or hedges. They bear small dark green leaves and bright orange or red berries. They are mildly poisonous, containing the cyanogenic glycosides prunasin (which occurs in the bark, leaves, flowers and fruits) and amygdalin (in the fruits). Cyanide prevents cellular respiration by binding to cytochrome oxidase; oxygen is not released from haemoglobin and a state of acute cellular hypoxia results[32]. Additional clinical signs such as salivation, vomiting, and diarrhoea, sometimes bloody and, in 1 case, hypothermia, have been reported after ingestion of cotoneaster and firethorn[10], but the quantity was apparently insufficient to cause mortality. The kernels of various stone fruits, mainly peach and apricot, also contain cyanogenic glycosides in sufficient quantities to cause fatal poisoning[57], but poisoning is unusual, as the stones usually pass through the gastrointestinal tract unbroken so that the seed is inaccessible.

DISCUSSION

Surveys from various countries show that, in comparison with other poisons, plant poisoning of dogs and cats is uncommon. In 2 surveys in North America, involving 2199 calls to poison control centres in 1978 and 41 854 calls in 1990, plant poisoning in dogs and cats accounted for 11.6 % and 12.1 % of the calls respectively[51]. In a survey in Western Australia, 7.6 % of poisonings in 158 dogs and 2.8 % of 36 cats presented to an academic veterinary hospital over a period of 12 years were due to plants[59]. During a prospective survey of 138 veterinary practices in Western Australia over an 18-month period, 231 cases of poisoning of dogs and 32 of cats were reported, but the incidence of plant poisonings was extremely low; none of the cats was poisoned by plants, and only 1.3 % of dogs among rural practices and 0.9 % at urban practices[58]. Among 965 cases of poisoning in dogs and cats reported in Melbourne, in eastern Australia, over a 12-year period (1971–1983), none were attributed to plants[71]. Finally, a survey by a poison reporting centre at the University of Turin, Italy, which receives calls from all over Italy, reported that of 4297 calls relating to poisoning in dogs and cats in the first 7 years of operation, 5 % involved plant poisoning in dogs and 11 % in cats, plant poisonings in the 2 species being the subject of 6.1 % of the calls; a further 0.6 % of calls related to hashish and cannabis poisoning in dogs[1]. In all the surveys chemical poisoning with pesticides and drugs accounted for by far the greatest number of cases, so that these substances pose a far higher risk than plants, especially to dogs, which are more likely to be poisoned owing to their more omnivorous habits. In all the surveys where a distinction was made between the 2 species, the majority of cases were reported in dogs. The overwhelming majority of plant poisonings in dogs and cats are caused by ingestion of plants[24], with only occasional reports of contact poisoning.

Plants containing calcium oxalate raphides emerge as the most frequent cause of poisoning of dogs and cats in the American surveys that give an indication of frequency[24,51], followed by plants with cardiac effects and nightshades (Solanaceae)[51]. In contrast, Araceae accounted for only a small percentage of plant poisonings in Western Australia[44], where poisoning by macadamia nuts headed the list, followed by cannabis, *Brunfelsia*, onion, and cycad seeds. In the Italian survey house plants were the main causes

of phytotoxicity, with *Euphorbia pulcherrima* and *Ficus* spp. predominating. In an overview of serious plant toxicoses reported to a control centre in the USA, the most frequently reported involved Liliaceae, azaleas, plants containing cardiac glycosides (*Nerium oleander* and *Kalanchoe*), cycads, castor beans and autumn crocus (*Colchicum autumnale*)[47]. The autumn crocus is cardiotoxic, but is not commonly encountered in southern Africa and is therefore not included in the review.

The majority of plant poisonings in dogs and cats do not have a high mortality rate, particularly if treatment is initiated early. Exceptions are nephrotoxicity in cats caused by members of the Liliaceae, toxicity caused by *Melia azedarach,* phytotoxicoses that affect cardiac function, and toxicity caused by high intake of ricin (for example as seed cake), cycad seeds and *Brunfelsia.* Poisoning with onions and grapes and raisins is also potentially lethal[44]. Some of the rarer toxicoses, like ingestion of *Datura,* also appear to be highly fatal[74]. On the other hand, poisoning with macadamia nuts, cannabis and insoluble calcium oxalates usually results in spontaneous recovery and these toxicoses are listed as non-life-threatening by McKenzie[44]. The prognosis always depends upon early detection of illness by the owners, including a history of ingestion of potentially poisonous plants or their derivatives, and aggressive treatment. Owner awareness is important, and it is useful for clinicians to make the necessary information available to their clients.

Not all of the plants are invariably poisonous. Toxicity is often dependent on the variety, part, growth stage or condition of the plant; for example, *Melia azedarach* may not be toxic when growing under marginal conditions[28]. This seems likely, as a dog belonging to 1 of the authors (MLP) frequently ate syringa berries (as well as avocadoes and grapes) with impunity in a garden in Windhoek, Namibia, which must be close to the limit of the tree's tolerance for aridity. There is also apparent variability in individual susceptibility. This has been noted in the case of grapes and raisins, as well as onions[16,29]. Both species are susceptible to poisoning by most of the plants listed, although lilies apparently cause nephrotoxicity only in cats. A number of the poisonings have only been reported in dogs, probably owing to the level of exposure and the more fastidious eating habits of cats. Poisoning by syringa berries, macadamia nuts, grapes and raisins, ricin and cycad seeds has apparently not been documented in cats.

It has been suggested that plant poisoning can often be prevented, for example by removal of poisonous parts such as cycad seeds and *Brunfelsia* fruits, and that creating awareness by providing information in veterinary practices and nurseries would help owners to protect their pets from plant poisoning[44].

One plant, catnip (*Nepeta cataria*), is eagerly eaten by cats and appears to have a beneficial effect, since genetically predisposed cats are reported to become 'ecstatic' after intake, without any ill effects following the 'trip'[5]. Since 70 % of plant poisonings in cats in Italy were caused by house plants, it might be advisable to provide catnip as an acceptable alternative if cats are to be left alone in an environment that includes potentially toxic plants.

CONCLUSIONS

Although plant poisoning of cats and dogs is not as common as intoxication of herbivorous livestock, many cases of poisoning, some with a fatal outcome, have been reported, and it is important for both veterinarians and pet owners to be aware of potentially dangerous plants and their derivatives. Lives are saved by the prompt action of owners and veterinarians when a cat or dog has ingested a poisonous plant. Clinical signs are often non-specific and the exposure may not have been observed, but chewed plants and/or remnants of plants in vomitus or stools can be an indicator that a plant poisoning is involved.

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