Animal poisonings in Belgium: a review of the past decade

Intoxicaties bij dieren in België: een overzicht van de voorbije tien jaar

[1]V. Vandenbroucke, [2]H. Van Pelt, [1]P. De Backer, [1]S. Croubels

[1] Department of Pharmacology, Toxicology and Biochemistry, Faculty of Veterinary Medicine, Ghent University, Salisburylaan 133, B-9820 Merelbeke, Belgium
[2]Belgian National Poison Centre, Queen Astrid Military Hospital, Bruynstraat 1, B-1120 Brussels, Belgium

virginie.vandenbroucke@ugent.be

ABSTRACT

This review focuses on poisonings in companion animals, including horses, farm animals and wildlife, investigated and recorded during the past ten years at the Laboratory of Toxicology of the Faculty of Veterinary Medicine (Ghent University) and the National Poison Centre in Belgium. The causative agents of poisoning incidents vary among the different species. The Laboratory of Toxicology of the Faculty of Veterinary Medicine reports that the majority of poisoning incidents in companion animals, and especially in dogs and cats, are due to contact with insecticides and pesticides, whereas horses are more frequently poisoned by plant toxins. Farm animals, on the other hand, are mainly intoxicated by heavy metals, toxic plants and agrochemicals. The Belgian Poison Centre reports that intoxications in companion animals are mostly with agrochemicals, household products and drugs, whereas in farm animals intoxications with agrochemicals are a common problem. This review gives an overview of the most common causes of intoxication and their association with the different animal species. In addition, some rare or difficult to diagnose intoxications are described, which account for a small number of poisoning cases.

SAMENVATTING

In dit overzichtsartikel wordt de nadruk gelegd op vergiftigingen van huisdieren, paarden inbegrepen, landbouwdieren en wild, die gedurende de laatste tien jaar in het Laboratorium voor Toxicologie van de Faculteit Diergeneeskunde (Universiteit Gent) en het Antigifcentrum van België onderzocht en gerapporteerd werden. De oorzaak van vergiftiging varieert naargelang de diersoort.

Aan het Laboratorium voor Toxicologie van de Faculteit Diergeneeskunde stelt men vast dat de meerderheid van de intoxicaties bij huisdieren, vooral bij de hond en de kat, te wijten is aan contact met insecticiden en pesticiden, terwijl paarden eerder aangetast worden door plantentoxinen. Landbouwdieren worden vooral vergiftigd door zware metalen, toxische planten en landbouwchemicaliën. Het Antigifcentrum rapporteert bij kleine huisdieren vooral intoxicaties met landbouwchemicaliën, huishoudelijke producten en medicatie terwijl bij landbouwhuisdieren voornamelijk intoxicaties met landbouwchemicaliën een frequent voorkomend probleem zijn. In dit artikel wordt een overzicht gegeven van de meest voorkomende oorzaken van intoxicatie en hun associatie met de verschillende diersoorten. Bijkomend worden enkele zeldzame of moeilijk te diagnosticeren intoxicaties beschreven die een rol spelen bij een beperkt aantal vergiftigingsgevallen.

INTRODUCTION

The Laboratory of Toxicology of the Faculty of Veterinary Medicine at Ghent University (LTGU) is specialized in confirming cases of animal poisonings by chemical analysis. The Belgian Poison Centre (BPC), on the other hand, has a 24-hour telephone hotline service to respond to urgent questions and to provide expert medical and toxicological information for both human and animal poisoning cases. In this review, data obtained from the LTGU and the BPC is presented. The data from the LTGU represents mainly the results of the analysis of suspected poisoning cases and post-mortem examinations, whereas the data from the BPC gives an idea of the exposure risk of animals to various poisons.

One difficulty with investigating the causes of animal poisoning is that neither the veterinarian nor the animal owner is legally obliged either to report the case or to send samples for chemical analysis. Even when this does occur, the results are usually not published and there are only sporadic peer-reviewed papers dealing with regional incidences of animal poisoning. This limited reporting obscures the incidence of accidental or malicious poisoning, particularly when only a limited number of samples from the total number of animals involved, or only the baits used in

poisoning attempts, are sent for analysis. Furthermore, only known toxicants can be investigated in the laboratories, so that new chemicals are often not detected and, consequently, the real number of positive cases may be underestimated. The same problems also exist in other EU countries (Guitart *et al*., 2010a).

Figure 1 gives an overview of the species, including bait, most commonly involved in poisoning incidents observed at the LTGU and the BPC. Of all companion animals, the dog appears to be most commonly involved in poisoning incidents, followed closely by the cat and to a lesser extent the horse. Among farm animals, most of the poisonings occur in cattle, followed by sheep and to a lesser extent goats. Poultry and pigs are rarely involved in poisoning episodes, since these animals are kept in a confined environment, which precludes exposure to toxicants.

Remarkably, at the LTGU, the buzzard accounts for 20% of the animal species involved in poisoning, and it is involved with about 22% of the baits examined. The last few years, corpses of birds of prey or other predators have been brought mostly by the Belgian Wildlife Disease Society to the LTGU on a regular basis for routine diagnosis, as these animals can be intoxicated because of the direct ingestion of baits or, to a lesser extent, because of the ingestion of poisoned rodents.

The causative agents for intoxications vary among the different animal species and will be specified further on in this article.

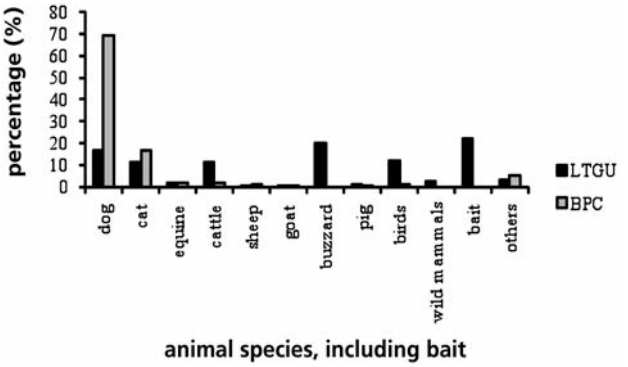

Figure 1. Species (including bait) most commonly involved in poisoning incidents recorded at the Laboratory of Toxicology of the Faculty of Veterinary Medicine (LTGU, Ghent University) and the Belgian Poison Centre (BPC).

COMPANION ANIMALS

Most common poisons involved

Data collected at the BPC over the period 1997-2006 showed that 84% of the 2300 telephone calls submitted annually dealt with oral intake of poisons, followed by cutaneous exposure (9.1%). For dogs, most of the calls concerned poisoning incidents with agrochemicals, followed by drugs and household products. In cats, exposure to agrochemicals and household products seemed to be the most frequent, followed by drugs. In horses, intoxications with agrochemicals and plants are most frequently reported.

Table 1 shows the most common poisons analytically confirmed in companion animals, including horses, over the past decade at the LTGU. Table 2 gives an overview of the most common types of poisoning incidents recorded in companion animals, including horses, at the BPC during the period 2000-2009.

Insecticides

Organophosphate (e.g. parathion) and carbamate (e.g. aldicarb and carbofuran) insecticides are marketed for control of insects on plants, animals, soils and buildings. They inactivate acetylcholinesterase, the enzyme responsible for the metabolization or breakdown of the neurotransmitter acetylcholine at cholinergic si-

Table 1. The most common poisons analytically confirmed in companion animals (including horses) over the past decade at the Laboratory of Toxicology of the Faculty of Veterinary Medicine, Ghent University.

	2000 (%)	2001 (%)	2002 (%)	2003 (%)	2004 (%)	2005 (%)	2006 (%)	2007 (%)	2008 (%)	2009 (%)
Insecticides										
Aldicarb	24.6	19.8	19.6	14.4	19.9	14.5	11.4	11.4	12.6	12.4
Carbofuran	2.4	4.8	3.1	4.3	8.1	8.4	14.4	14.8	11.9	21.5
Parathion	4.8	4.2	2.2	3.8	1.7	1.9	1.1	1.3	1.8	2.2
Others*	0.5	0.6	1.3	1.0	1.3	3.7	1.5	3.4	2.7	2.6
Pesticides										
Strychnine	5.8	1.2	5.3	4.2	3.4	1.9	3.7	0.8	0.7	0.4
Coumarins	1.5	1.8	3.1	1.8	3	0.5	2.6	5.5	5.1	10.6
Metaldehyde	0.48	0	0	0.5	1.3	0.9	0	0.4	1.4	0.4
Plants	0.5	0.6	0	0	0.4	0.5	0.7	1.3	0.7	0.4
Total (n)	207	167	225	236	236	203	271	237	277	274

* Others include e.g. mevinphos, malathion, diazinon, methiocarb, carbaryl, propoxur.

Table 2. The most common poisoning incidents recorded in companion animals (including horses) over the past ten years at the Belgian Poison Centre.

	2000 (%)	2001 (%)	2002 (%)	2003 (%)	2004 (%)	2005 (%)	2006 (%)	2007 (%)	2008 (%)	2009 (%)
Agrochemicals	40.4	40.7	41.2	43.4	42.4	40.7	39.6	41.5	38.0	35.6
Household products	25.7	24.4	23.9	22.2	22.7	23.3	21.0	19.6	19.2	19.5
Drugs	16.5	16.3	18.0	16.2	16.5	19.7	21.9	19.3	23.0	23.4
Plants	7.0	6.9	6.8	7.1	6.7	5.7	6.3	8.3	6.7	7.0
Veterinary drugs	1.7	1.5	1.8	1.9	2.8	2.6	2.6	2.5	3.0	4.1
Others (1)	8.8	10.2	8.4	9.2	8.9	8.1	8.5	8.8	10.1	10.3
Total (n)	2155	2277	2191	2154	2253	2417	2367	2660	2700	2478

(1) Others include eg. paint, industrial chemicals, cosmetics, mushrooms, gas, alcohol, tobacco, unknown substances.

tes by phosphorylation or carbamylation. As a consequence, acetylcholine continues to depolarize the postsynaptic membrane. The clinical signs that are seen in animals depend on the dose ingested, the age and the general health status, but in general they include diarrhea, vomiting, miosis, bronchoconstriction, bradycardia and salivation. Atropine should be given to reverse the cholinergic effects (Meerdink, 2004).

Temik® and Regent Plus® are pesticides whose active ingredient is aldicarb, which have been used as pesticides to protect a range of agricultural and horticultural crops worldwide. Aldicarb is a fast-acting cholinesterase inhibitor. Cats are extremely sensitive to the effects of aldicarb and are often the victim of deliberate poisoning with impregnated baits (De Bosschere *et al.*, 1999). In Belgium, the authorization for the application of aldicarb, for example in the cultivation of sugar beets to control infestations of lice, centipedes and beet flies, was withdrawn in late 2007, together with the authorization for carbofuran (e.g. Curater®) in late 2008. The data in Table 1 do indeed show a remarkable shift towards the misuse of carbofuran after the ban on aldicarb from 2006 onwards.

A retrospective study of the necropsy reports from dogs and cats for the years 1995-1998 at the LTGU revealed that intoxication with aldicarb and other carbamates (e.g. carbofuran) was the most frequent cause of death. Out of a total number of 1764 dogs submitted for post-mortem examination, poisoning was suspected in 75 cases (4.2%); in 50 of these cases, toxicological examination was performed, and 17 of these were found to be positive for aldicarb. Out of 916 necropsies performed on cats, 150 cases involved suspected poisoning (16%), 117 investigations were undertaken and 85 were found to be positive for aldicarb (De Bosschere *et al.*, 1999). Aldicarb poisoning in domestic and wild animals is also common in Wallonia (Delaunois *et al.*, 1997).

Pyrethroids, which are approved for use against ectoparasites on dogs that are more than 3 months old, can be found in many shampoos, sprays, dusts, dips, spot-on flea and tick products and household insecticides. Pyrethroids are not safe for use with cats due to the low tolerance that cats have towards them and to the fact that poisoning can occur when a cat is treated with a product labeled for dogs (Volmer, 2004). The problem also occurs when the cat ingests the product during grooming. The effects of pyrethroid poisoning in cats are manifested by excessive salivation, ear flicking, ataxia, depression, lethargy, muscle tremors, seizures, hyperthermia and death. The treatment depends on the severity of the signs and is aimed at controlling the seizures and tremors, together with supportive care and decontamination of the exposed skin (Bates, 2000).

Occasionally, thallium has been reported as a source of intoxication in dogs and cats after they have licked bait intended for ants and/or rodents (J. Declercq, personal communication, 2004).

Rodenticides

Anticoagulant rodenticides are substances that are widely used to control infestations with rats and mice (Berny *et al.*, 1995). 4-hydroxy-coumarins and indandione derivates, i.e. chlorophacinon and diphacinon, are the most common active constituents of these pesticides. Rodenticides continue to be a major cause of morbidity and mortality in companion animals, with dogs being more frequently involved in anticoagulant poisoning than cats. Anticoagulant rodenticides are vitamin K antagonists, with the main site of action being the liver. The poisoning is manifested by severe hemorrhage, with massive bleeding and poor coagulation. The primary clinical signs are caused by blood loss such as anemia, pale mucous membranes, weakness and tachycardia (DuVall *et al.*, 1989; Murphy and Gerkin, 1989; Berny, 2007). The antidote for treating dogs exposed to anticoagulant rodenticides is vitamin K_1 (phytomenadione). At the LTGU, a sensitive LC-ESI-MS/MS method was developed for the determination and confirmation of suspected anticoagulant rodenticide toxicosis in animals such as cats and dogs (Vandenbroucke *et al.*, 2008).

The BPC receives around 400 enquiries annually dealing with exposure to anticoagulant rodenticides in animals (79% dogs). The main products ingested in dogs were, in order of frequency, difenacoum (36%), brodifacoum (17%), difethialon (5%), coumatetralyl (3%), bromadiolone (4%), chlorophacinon (2%), flocouma-

fen (4%), warfarin (1%) and unknown products (30%). These percentages correspond well with the relative availability of the various products on the market. Strychnine and anticoagulant rodenticides were confirmed in max. 5.3% and 10.6%, respectively, of all poisoning cases examined at the LTGU over the last ten years, even though strychnine was banned in Belgium several years ago.

Molluscicides

At the BPC, exposure of dogs to molluscicides is reported frequently (approximately 100 cases annually). Metaldehyde is the most frequently encountered molluscicide, accounting for 57% of the cases, followed by methiocarb (19%), ferriphosphate (13%) and other unidentified products (11%). However, analytically confirmed cases of fatal metaldehyde poisoning in pets were rare (max. 1.4%) at the LTGU. Metaldehyde is the ingredient of most molluscicide preparations, which can be quite attractive to dogs. Severe intoxications frequently result in persistent convulsions and death (Puschner, 2001). To diminish the number of these intoxications, a number of initiatives were taken in December 2007 by the federal authorities. These initiatives included (1) better control for the presence of the potent, yet harmless bittering agent denatomium benzoate (Bitrex®), as previous tests showed little or no denatomium benzoate at all in some of the samples, (2) revised instructions for use on the label (e.g. number of granules/m² instead of grams/100m²), and (3) distribution of a leaflet explaining the correct use of these products. As can be seen in Table 3, no amelioration was observed. Compared with the two years before the start of the initiatives, there were more calls and more major symptoms (with or without convulsions) in the ensuing two years. Apparently, risk-reducing initiatives like the ones described above do not diminish the number of serious metaldehyde intoxications in dogs. Other measures are required to achieve a reduction of these kinds of intoxications.

Plants and fungi

In Belgium, the European yew (*Taxus baccata*) and tansy ragwort (*Senecio jacobaea*) are the most important poisonous plants for horses (Figures 2 and 3). The needles and seeds of the yew are highly poisonous, both in fresh and dry form. However, the red fleshy seed covering is not poisonous. The toxic principle is taxine and death will usually occur so rapidly that symptoms such as staggering gait, muscle tremors, convulsions, collapse, dyspnea and heart failure are often missed (Casteel, 2004). Poisoning of animals by pyrrolizidine alkaloids, as found in many plant species of the genus *Senecio*, is well described and is a main cause of chronic liver damage in the horse. Although herbivores seldom eat mature plants, poisoning can still occur when seedlings are grazed accidentally along with other forage or when there is a lack of other feed (Polhmann *et al.*, 2005). When present in hay, the dried plants pose a particular risk since they are more palatable and less avoided during eating. Because of the poor prognosis, prevention of the disease by means of pasture management and the exclusion of these plants from hay or silage is mandatory.

Table 3. Overview of molluscicide poisoning in dogs from 2006-2009 (data Belgian Poison Centre).

Year	Number of dogs intoxicated	Major symptoms (%)	+ Convulsions (%)
2006	27	10 (37)	7 (26)
2007	56	8 (14)	7 (13)
2008	53	16 (30)	12 (23)
2009	39	12 (30)	9 (24)

The black locust (*Robinia pseudoacacia*) has also been reported to cause fatal poisoning of horses in Belgium, usually after horses are tethered to and subsequently eat the bark of poles made of this wood (S. Croubels, personal communication). Also *Nerium oleander* is known to cause fatal intoxications in horses (Durie *et al.*, 2008).

Figure 2. *Taxus baccata*
(Source: http://caliban.mpiz-koeln.mpg.de/lindman/)

Figure 3. ***Senecio jacobaea*** **(Source: http://caliban.mpiz-koeln.mpg.de/lindman/)**

In 2004, an unusual case of wood poisoning in dogs that ate *Simarouba amara* shavings was described at the Department of Medicine and Clinical Biology of Small Animals at Ghent University. *Simarouba amara*, commonly referred to as marupa or caixeta, is a tropical hardwood tree in the *Simaroubaceae* family from South America. Two male Labradors developed bleeding erosions/ulcerations involving the oral mucosa, mucocutaneous junctions of the lips, node, prepuce and anus, ulcerated nodules on the chin and crusting lesions on the elbows, hocks and scrotum. The dogs had recently been exposed to bedding containing *Simarouba amara* shavings and, because of the striking similarities of the clinical signs to those described for horses, a probable diagnosis of wood poisoning was made. This assumption was supported by the clinical course, as healing of the skin lesions occurred when the dogs were no longer exposed to the bedding (Declercq, 2004).

The first confirmed case of ryegrass staggers in Belgium was described in horses at a riding school (Nollet *et al.*, 2007). The causative agents are tremorgenic compounds such as lolitrem B, produced by the endophytic fungus *Neotyphodium lolii* (Cheeke, 1994). About 30 horses were affected. Ryegrass staggers was diagnosed based on the clinical symptoms, the typical anamnesis and the demonstration of high lolitrem B concentrations in the hay. There was no mortality, despite a high morbidity, and all the animals recovered a few days after withdrawal of the hay.

Drugs

At the LTGU, most of the reports of drug toxicities have been limited to telephone calls or questions from owners concerning the accidental ingestion of barbiturates, paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs) by their pets (Berny *et al.*, 2010)

FARM ANIMALS

Poisoning is seldom recorded in food-producing animals in Belgium, probably as it is usually only suspected after the exclusion of common pathological disorders. Furthermore, poisoning in livestock is often only addressed when the economic loss is sufficient to justify immediate investigation. For instance, very few cases are reported in goats and sheep, which are of low economic value. Also, many species (swine, poultry) are kept in a confined environment, which precludes exposure to external toxicants and, consequently, poisoning is unlikely to occur, unless toxicants are present in (some of) the feed components or intoxication is caused by faulty feed formulation.

Species involved

Most of the reported poisonings occur in cattle (11.24%), followed by sheep and goats (0.38%). Poisoning episodes in swine and poultry are rarely described and are mainly caused by inadequate farm management.

Most common poisons involved

At the LTGU, toxic plants, heavy metals and agrochemicals are the most common toxicants found to be suspected of being involved in food animal poisonings. Intoxications by bacterial toxins such as botulism were not taken into account.

According to BPC records, poisoning incidents with cows most frequently involve agrochemicals, whereas intoxications in small ruminants most frequently involve toxic plants.

Pesticides

The possible effects of diverse agrochemicals on the health status and performance of animals, mainly cows and horses, are the frequent subject of calls addressed to the LTGU, particularly when herbicide spray has drifted onto neighboring pastures where the cattle have been grazing. However, toxicological analyses on pesticide poisoning are rarely undertaken, mainly due to the lack of scientific data about the toxic levels, the metabolism of the pesticides and the analytical methods required in the examination of livestock. Very often, products such as glyphosate (Round-up®,

Glialka Plus®), MCPA, fluroxypur and clopyralid (Bofix®) are involved. Although glyphosate has a high oral LD_{50} (rat: 4050 mg/kg), intoxication may occur due to the presence of surfactants such as polyoxyethylene amine in the commercial formulation (Martinez and Brown, 1991; Bradberry *et al.*, 2004). These surfactants may cause diarrhea, gastro-intestinal irritation, ptyalism, pulmonary edema, etc.

At the BPC, 51.1% of the calls concerning poisoning in cattle are about exposure to agrochemical substances (period 2000-2009).

Plants

Poisoning by plants often occurs when ornamental plants or trimmings are made available to the animals due to the negligence of the animal owners or neighbors. Farm animals seldom eat toxic plants, except when no other feed is available or if the plant is not recognizable, for example when it is mixed in the hay. Small ruminants, and especially goats, are inquisitive by nature and in their browsing habits. They commonly consume small quantities of poisonous plants without showing adverse effects, particularly when the rumen is filled with other feed stuff (Popay and Field, 1996; Matthews, 1999). In the period 2000-2009, 44.8% and 64.4%, respectively, of the cases in sheep and goats reported to the BPC had to do with exposure to toxic plants.

Livestock can be poisoned by many different plant species, but an analytically confirmed diagnosis is not always easy, and most cases are only suspected following necropsy and identification of leaves, stems or pods in the rumen.

Trimmings from the European yew tree (*Taxus baccata*) are undeniably known to be extremely toxic to animals, especially cows and horses, as already mentioned above. On a yearly basis, the LTGU diagnoses yew tree poisoning by macroscopic evaluation in 0.31% of all confirmed poisoning cases. The clinical signs in poisoned livestock reflect conduction disturbances in the heart, with sudden death as a result (Casteel, 2004).

Two cases of annual mercury (*Mercurialis annua*) poisoning in cattle were described at the Faculty of Veterinary Medicine (Ghent University) (Deprez *et al.*, 1996). On two cattle farms, several animals showed constipation or diarrhea, dullness, hemolytic anemia and red urine. The clinical symptoms, the results of the blood examinations (low hematocrit, high bilirubin levels, increased levels of urea and AST) and the presence of large amounts of the toxic plant on the field suggested a poisoning with *Mercurialis annua*.

More recently, two cases of intoxication in small ruminants with toxic garden plants were also reported (Baert *et al*., 2005): the first one involved the death of three goats after eating garden plants, including Japanese pieris (*Pieris japonica*). Japanese pieris belongs to the family of *Ericaceae,* which contains many ornamental plants such as azalea and rhododendron. The toxic substances are grayanotoxins, which exert their effect by binding to sodium channels in excitable cell membranes of nerve, heart and skeletal muscles. The toxins are found in nectar, flowers, stems and especially the leaves (Visser *et al.*, 1988). After exposure, a rapid onset of clinical signs is seen and the duration is usually about 1-2 days. Initially, depression develops with severe salivation and abdominal pain, vomiting, regurgitation and sometimes diarrhea. Later on, the animals may become recumbent and develop seizures, tachycardia, tachypnea and pyrexia. The poisoning is usually not fatal, but recovery may take several days. In the second case, a flock of sheep suddenly showed salivation followed by the death of five animals. Post-mortem examination revealed numerous leaves of the Japanese spindle (*Euonymus japonicus*) in the ruminal content, as well as edematous lungs and hemorrhagic fluid in the trachea and in the pleural and peritoneal cavities. The poisonous substances of *E. japonicus* include alkaloids and cardiac glycosides or cardenolides (Frohne and Pfänder, 1983; Van Genderen *et al.*, 1996).

Acorn poisoning (*Quercus robur*) is reported every year at the LTGU, especially in the autumn after a hot summer and after a storm (Deroo and de Kruif, 2003). The toxic syndrome is gastro-intestinal and renal dysfunction caused by gallotannins and polyhydroxyphenolic compounds or their metabolites. The signs include abdominal pain, poor appetite, diarrhea (often black or bloody in color) and occasional constipation. As the disease progresses, signs of kidney failure may appear. The affected cattle will exhibit dehydration, increased thirst, general weakness, weight loss, frequent urination and a rapid, weak pulse. Edema may be noted in the lower parts of the body, such as the chest, legs and ventral abdomen. Some body cavities may also become filled with fluid.

Recently, more attention has also been given to the dangers to cattle of *Senecio* spp. in their pastures and their hay (Vos *et al.*, 2002). The same symptoms as described above for horses can be seen.

Mycotoxins

Mycotoxins such as aflatoxins, deoxynivalenol, T2-toxin, zearalenone, fumonisins, ochratoxins and tremorgenic mycotoxins contaminate mainly cereal grains worldwide, and their presence in animal feed poses a potential health threat to farm animals (and human consumers) (Bimczok *et al*., 2007).

Currently, animal health services and universities are frequently asked to confirm a possible diagnosis of mycotoxin feed contamination. However, several problems typically arise in connection with this diagnosis. First, no background levels in organs such as the liver or in fluids such as the bile are available, but only guidance levels in feed (2006/576/EC). Second, there is insufficient knowledge about the elimination of mycotoxins in these animals, such as the mean residence time in the liver and bile and the half-life of elimination. Third, no typical postmortem histopathological lesions are known after exposure to most of the mycotoxins. Finally, the possibly contaminated feed is frequently consumed and therefore no longer available when clinical symptoms develop and analysis of the remaining feed is ham-

pered by the non-homogenous distribution of mycotoxins in feed and by 'masked' mycotoxins that are bound to feed components.

A severe and unique neurotoxicosis, comprising tremors, ataxia, paresis, recumbency and death, occurred simultaneously among several herds of beef cattle in Flanders. *Aspergillus clavatus* was found to be the dominant fungal species in a sample of compacted fodder containing malting residues that was consumed by all the affected herds. Measurable amounts of verrucologen and patulin could also be identified in the feed samples involved (Sabater-Vilar *et al.*, 2004).

Heavy metals and metalloids

In 2007, analyses of metal and metalloid (e.g. arsenic and selenium) levels in diverse matrices were requested in 80.4% of all analyses done in cattle, with selenium accounting for 56.1% of the metals and metalloids found, followed by copper (24.4%), lead (12.2%) and zinc (7.3%).

Historically, lead poisoning occurred frequently in large animals, particularly in cattle. The cause for this poisoning was the uptake of lead-containing paint used in stables and the licking of car batteries dumped in meadows (De Corte-Baeten and Debackere, 1973; Debackere and De Corte-Baeten, 1974). Nowadays, lead intoxication is diagnosed only occasionally.

Acute or chronic copper poisoning is reported in many countries (Guitart *et al.*, 2010a). Sheep are affected most often, though other animals such as cattle and to a lesser extent pigs are also susceptible. The addition of excessive amounts of copper to commercially prepared milk replacers caused chronic copper toxicity in veal calves on two different Belgian farms. A possible mixing error by the feed company resulted in copper levels ranging from 120 to 159 mg/kg in the milk powder, whereas the maximum level is set at 30 mg/kg. On one farm, four animals died, showing the typical clinical signs of chronic copper toxicity, including weakness, anorexia and severe icterus. In the liver, copper concentrations of 294 and 500 mg/kg fresh liver were found, which indicated chronic copper intoxication (Croubels *et al.*, 2001a). Hadrich (1996) suggests that normal copper concentrations in calf's liver range from 50 to 150 mg/kg fresh weight.

In the past, fluorosis in cattle has been ascribed to fluoride pollution caused by brickworks and enamel factories in the Flemish countryside (Debackere and Delbeke, 1978; 1981). In some regions, the intake of borehole water can lead to fluorosis, due to the high level of fluoride in deep-lying clay layers. This type of fluoride poisoning has been reported, for example, in turkeys, where it was mainly the fast growing male animals that were affected (P. De Backer, personal communication). In 2000, a special case of fluoride poisoning was reported at the LTGU. Superphosphate is a fertilizer produced by the action of concentrated sulphuric acid on powdered phosphate rock. $CaSO_4$ or plaster is formed as a residual product from this reaction and can be used as a binding agent in the making of beet pulp. However, it has been reported that such use of $CaSO_4$ can lead to high levels of fluoride in the beet pulp (normal level of fluoride in beet pulp: 15 mg/kg dry matter), which in turn can lead to chronic fluoride intoxication in cattle. In this case, levels of up to 452 mg/kg dry matter were found in the pulp (S. Croubels, personal communication). Animals that have a high calcium metabolism, such as growing animals, pregnant animals and dairy cows, are more sensitive to fluoride intoxication than others (Osweiler, 2004).

At the LTGU, a selenium deficiency is diagnosed more often than selenium intoxication. In cattle, 52.2% of all selenium analyses revealed a deficiency, 26.1% showed normal selenium levels and 21.7% showed excessive selenium levels. Adequate levels in the serum were set at 0.08 to 0.30 mg/L, and levels below 0.025 mg/L were considered to be deficient (Puls, 1994).

In 2005, a case of arsenic poisoning in cattle was confirmed at the LTGU. Three animals died after consumption of the ashes of old packages of arsenic-containing herbicides that were burned on a meadow. The symptoms were hemorrhagic enteritis, erosions and bleeding of the gastro-intestinal tract, and nervous symptoms such as ataxia and salivation (J. Wullepit, personal communication).

Industrial chemicals

In 1999, the contamination of animal feeds with polychlorinated biphenyls (PCBs), dioxins and dibenzofurans led to an unprecedented food crisis in Belgium. Several poultry farms reported a sudden drop in egg production as the initial symptom, followed by a marked reduction in egg hatchability, reduced weight gain and increased mortality of the chicks. The degenerative changes of the skeletal and cardiac muscles seen on histological examination resembled the classical manifestations of 'chicken edema disease', as described in the 1950s to the 1970s in outbreaks of poultry poisoning by polyhalogenated hydrocarbons (Gilbertson *et al.*, 1991; Bernard *et al.*, 1999; Vanhoutte and Paque, 2000; Bernard *et al.*, 2001; Van Larebeke *et al.*, 2001; Guitart *et al*., 2010a).

Other toxicants

Nitrite intoxication is known in several species as a cause of methemoglobinemia. Three cases of acute mortality in pigs raised in different commercial farms in Belgium have been described in which still standing water within the water distribution system was identified as the source of nitrite. The nitrite formation was likely due to a bacterial reduction of nitrate within the pipes (Vyt *et al.*, 2005).

Gas intoxication by hydrogen sulphide (H_2S) and ammonia (NH_3) is well known in the pig industry and can result in sudden death, especially when the animals are kept in stables with open grids or when the ventilation systems are working inefficiently. One case of NH_3 intoxication was reported in bovines after the consump-

tion of large amounts of soybean meal with symptoms such as head pressing, agitation and aggression. Soybean meal overconsumption by ruminants can cause acute carbohydrate fermentation and excessive ammonia release leading to ammonia toxicosis and lactic acidosis (Osweiler *et al.*, 1996).

A case of acute doxycycline intoxication in veal calves was described during treatment of enzootic pneumonia. The animals had accidentally received 5-6 times more than the recommended dose of doxycycline (Chiers *et al.*, 2004). In 2001, an acute tiamulin-salinomycin intoxication in pigs was described following the administration of salinomycin in the feed and the presence of traces of tiamulin in the drinking water, resulting from the administration of tiamulin to fattening pigs housed in the stable four months previously (Croubels *et al.*, 2001 b).

WILDLIFE

In the last few years, corpses of birds of prey and other predators have frequently been suspected of malicious poisoning. In Flanders, the common buzzard (*Buteo buteo*) is frequently the victim of deliberate poisoning. Of 162 buzzards presented for diagnosis during the period 2003-2006, 91 appeared to have been maliciously killed either by poisoning (n=79) or shooting (n=12). Other birds involved in poisoning incidents were the kestrel (*Falco tinnunculus*), sparrow hawk (*Accipiter nisus*), red kite (*Milvus milvus*), magpie (*Pica pica*), carrion crown (*Corvus corone*) and pheasant (*Phasianus colchicus*). Sixty suspicious baits were examined in the LTGU during the period 2003-2006, and 77% of these were found to contain pesticides (Guitart *et al*., 2010b). Carbofuran was the most common poison detected, followed by aldicarb, carbaryl, strychnine and chlorophacinon (P. Tavernier, personal communication, 2006).

Lead poisoning was diagnosed in 16% live and 4.04% dead anseriformes presented at the Bird Clinic of the Veterinary Faculty of Ghent University in the period 2002-2004 (Tavernier *et al.*, 2005a). Acute poisoning caused by lead shot ingestion was also diagnosed in two young racing pigeons (Tavernier *et al.*, 2005b).

CONCLUSION

It appears that poisoning is an uncommon cause of illness in animals compared to other clinical manifestations such as infectious diseases and inflammation. This review reveals that most published cases of poisoning are rather special and/or unusual cases, since the common causes of poisoning are rarely published, and reliable and complete toxicoepidemiological data are not easy to obtain.

In Belgium, the National Poison Centre plays an important role as a 24-hour telephonic medical permanence service and as an information center for toxicological questions.

The Laboratory of Toxicology of the Faculty of Veterinary Medicine (Ghent University) performs chemical analyses of suspected poisoning cases and post-mortem examinations and plays a crucial role in the confirmation of both accidental and deliberate animal poisoning. That deliberate poisoning plays an undeniable role in poisoning incidents involving both wildlife and companion animals (especially dogs and cats) is proven by the number of baits (22% of all analyses) that are examined yearly at the Laboratory of Toxicology of the Faculty of Veterinary Medicine.

REFERENCES

Baert K., Croubels S., Steurbaut N., De Boever S., Vercauteren G., Ducatelle R., Verbeken A., De Backer P. (2005). Two unusual cases of plant intoxication in small ruminants. *Vlaams Diergeneeskundig Tijdschrift 74*, 149-153.

Bates N. (2000). Pyrethrins and pyrethroids. In : Campbell A., Chapman M. (editors). *Handbook of Poisoning in Dogs and Cats*. Blackwell Science, Oxford, United Kingdom, p. 42-47.

Bernard A., Broekaert F., De Poorter G., De Cock A., Hermans C., Houins G., Saegerman B. Hallet L. (2001). De PCB/dioxinevergiftiging in België (1999). Analyse van de voedselketencontaminatie en evaluatie van de gezondheidscrisis. *Vlaams Diergeneeskundig Tijdschrift 70*, 81-87.

Bernard A., Hermans C., Broekaert F., De Poorter G., De Cock A., Houins G. (1999). Food contamination by PCBs and dioxins. *Nature 401*, 231-232.

Berny P.J. (2007). Pesticides and the intoxication of wild animals. *Journal of Veterinary Pharmacology and Therapeutics 30*, 93-100.

Berny P.J., Buronfosse T., Lorgue G. (1995). Anticoagulant poisoning in animals: a simple new high-performance thin-layer chromatography (HPTLC) method for the simultaneous determination of eight anticoagulant rodenticides in liver samples. *Journal of Analytical Toxicology 19*, 576-580.

Berny P., Caloni F., Croubels S., Sachana M., Vandenbroucke V., Davanzo F., Guitart R. (2010). Animal poisoning in Europe. Part 2: Companion animals. *The Veterinary Journal 183*, 255-259.

Bimczok D., Döll S., Rau H., Goyarts T., Wundrack N., Naumann M., Dänicke S., Rothkötter H.-J. (2007). The Fusarium toxin deoxynivalenol disrupts phenotype and function of monocyte-derived dendritic cells in vivo and in vitro. *Immunobiology 212*, 655-666.

Bradberry S., Proudfoot A., Vale J. (2004). Glyphosate Poisoning. *Toxicological Reviews 23*, 159-167.

Casteel S.W. (2004). Taxine alkaloids. In: Plumlee K.H. (editor). *Clinical Veterinary Toxicology*. Mosby, Missouri, USA, pp. 379-381.

Cheeke P.R. (1994). Endogenous toxins and mycotoxins in forage grasses and their effects on livestock. *Journal of Animal Science 73*, 909-918.

Chiers K., Weyens P. Deprez P., van Heerden M., Meulemans G., Baert K., Croubels S., De Backer, P., Ducatelle R. (2004). Lingual and pharyngeal paralysis due to acute doxycycline intoxication in veal calves. *The Veterinary Record 155,* 25-26.

Croubels S., Baert K., Torck T., Deprez P., De Backer P. (2001a). Chronic copper intoxication in veal calves. *Vlaams Diergeneeskundig Tijdschrift 70*, 142-146.

Croubels S., Vrielinck K., Baert K., Vermaut I., Castryck F.,

De Backer P. (2001b). A special case of an acute tiamulin-salinomycin intoxication in pigs due to residual tiamulin four months after medication. *Vlaams Diergeneeskundig Tijdschrift 70*, 54-58.

De Bosschere H., Baert K., Ducatelle R., De Backer P. (1999). Aldicarb intoxications in dogs and cats: a retrospective study. *Vlaams Diergeneeskundig Tijdschrift 68*, 148-152.

De Corte-Baeten K., Debackere M. (1973). Onderzoek naar loodgehalten bij gezonde en geïntoxiceerde runderen en gezonde varkens. *Vlaams Diergeneeskundig Tijdschrift 42*, 233-248.

Debackere M., De Corte-Baeten K. (1974). Onderzoek naar loodgehalten bij gezonde en geïntoxiceerde honden en paarden. *Vlaams Diergeneeskundig Tijdschrift 43*, 405-415.

Debackere M., Delbeke F.T. (1978). Fluoride pollution caused by a brickworks in the Flemish countryside of Belgium. *International Journal of Environmental Studies 11*, 245-252.

Debackere M., Delbeke F.T. (1981). Fluoride pollution caused by an enamel factory in the Flemish countryside of Belgium. *International Journal of Environmental Studies 17*, 201-207.

Declercq J. (2004). Suspected wood poisoning caused by *Simarouba amara* (marupa/caixeta) shavings in two dogs with erosive stomatitis and dermatitis. *Veterinary Dermatology 15*, 188-193.

Delaunois A., Lessire F., Fanal H., Ansay M., Bloden S., Gustin P. (1997). Temik® poisonings in domestic and wild animals: an alarming problem in Wallony. *Annales de Médecine Vétérinaire 141*, 353-358.

Deprez P., Sustronck B., Mijten P., Vande Vijvere B., Muylle E. (1996). Two cases of *Mercurialis annua* poisoning in cattle. *Vlaams Diergeneeskundig Tijdschrift 65*, 92-96.

Deroo M., de Kruif A. (2003). Eikelvergiftiging bij een zoogkalf. *Vlaams Diergeneeskundig Tijdschrift 72*, 302-305.

Durie, I., van Loon G., De Clercq D., Deprez P. (2008). Nerium oleander intoxication in horses, 3 cases. In: *Proceedings of the 47th BEVA Congress*, p. 133.

DuVall M.D., Murphy M.J., Ray A.C., Reagor J.C. (1989). Case studies on second generation anticoagulant rodenticide toxicities in nontarget species. *Journal of Veterinary Diagnostic Investigation 1*, 66-68.

Frohne D., Pfänder H.J. (1983). Celastraceae. In: Frohne D., Pfänder H.J. (editors). *A Colour Atlas of Poisonous Plants*. Wolfe Publishing Ltd, London, p. 87-88.

Gilbertsen M., Kubiak T.J., Ludwig J.P., Fox G. (1991). Great lakes embryo mortality, edema, and deformities syndrome (GLEMEDS) in colonial fish-eating birds: similarity to chick edema disease. *Journal of Toxicology and Environmental Health 33*, 455-520.

Guitart R., Croubels S., Caloni F., Sachana M., Davanzo F., Vandenbroucke V., Berny, P. (2010a). Animal poisoning in Europe. Part 1: farm livestock and poultry. *The Veterinary Journal 183*, 249-254.

Guitart R., Sachana M., Caloni F., Croubels S., Vandenbroucke V., Berny P. (2010b). Animal poisoning in Europe. Part 3: Wildlife. *The Veterinary Journal 183*, 260-265.

Hadrich J. (1996). High amounts of copper in calf's livers. Recent data and estimation of potential health hazards. *Deutsche Lebensmittel-Rundschau 92*, 103-113.

Martinez T., Brown K. (1991). Oral and pulmonary toxicology of the surfactant used in roundup herbicide. In: *Proceedings of the Western Pharmacology Society 34*, 43-46.

Matthews J. (1999). Plant poisoning. In: Matthews J. (editor). *Diseases of the Goat*. Blackwell Science, Oxford, pp. 285-294.

Meerdink G. (2004). Anticholinesterase insecticides. In: Plumlee K. (editor). *Clinical Veterinary Toxicology*, Mosby, Inc., Missouri, p. 178-180.

Murphy M.J., Gerkin D. (1989). The anticoagulant rodenticides. In: Kirk R.W. (editor). *Current Veterinary Therapy X*. WB Saunders, Philadelphia., p. 143-146.

Nollet H., Vanschandevijl K., Lefère L., Deprez P. (2007). First confirmed case of ryegrass staggers in horses in Belgium. *Vlaams Diergeneeskundig Tijdschrift 76*, 355-358.

Osweiler G. (1996). Feed-related toxicoses. In: Osweiler G. (editor). *Toxicology*. Williams and Wilkins, Philadelphia, USA, p. 344-345.

Osweiler G. (2004). Fluoride. In: Plumlee K. (editor). *Clinical Veterinary Toxicology*. Mosby, Inc., Missouri, p. 197-200.

Pohlmann J., van Loon G., Lefère L., Vanschandevijl K., Nollet H., De Clercq D., Delesalle C., Deprez P. (2005). Hepatoencephalopathy caused by *Senecio jacobaea* intoxication in five horses. *Vlaams Diergeneeskundig Tijdschrift 74*, 440-445.

Popay I., Field R. (1996). Grazing animals as weed control agents. *Weed Technology 10*, 217-231.

Puls R. (1994). *Mineral Levels in Animal Health: Diagnostic Data*. 2nd Ed. BC, Canada: Sherpa International.

Puschner B. (2001). Metaldehyde. In: Peterson M. and Talcott P. (editors). *Small Animal Toxicology*. WB Saunders Company, Philadelphia, p. 553-563.

Sabater-Vilar M., Maas R.F.M., De Bosschere H., Ducatelle R., Fink-Gremmels J. (2004). Patulin produced by an *Aspergillus clavatus* isolated from feed containing malting residues associated with a lethal neurotoxicosis in cattle. *Mycopathologia 158*, 419-426.

Stegelmeier B. (2004). Pyrrolizidine alkaloids. In: Plumlee K. (editor). *Clinical Veterinary Toxicology*. Mosby, Inc., Missouri, p. 370-377.

Tavernier P., Cauwerts K., Van Caelenberg A., Pasmans F., Martel A., Hermans K. (2005a). Lead poisoning in anseriformes diagnosed in Flanders. *Vlaams Diergeneeskundig Tijdschrift 73*, 303-306.

Tavernier P., Roels S., Baert K., Hermans K., Pasmans F., Chiers K. (2005b). Lead intoxication by ingestion of lead shot in racing pigeons. *Vlaams Diergeneeskundig Tijdschrift 73*, 307 - 309.

Van Genderen H., Schoonhoven L.M., Fusch A. (1996). Kardinaalsmutsfamilie - *Celastraceae*. In: *Chemisch-Ecologische Flora van Nederland en België.* Stichting Uitgeverij van de Koninklijke Nederlandse Natuurhistorische Vereniging, Utrecht, p. 157-159.

Van Larebeke N., Hens L., Schepens P., Covaci A., Baeyens J., Everaert K., Bernheim J.L., Vlietinck R., De Poorter G. (2001). The Belgian PCB and dioxin incident of January-June 1999: exposure data and potential impact on health. *Environmental Health Perspectives 109*, 265-273.

Vandenbroucke V., Desmet N., De Backer P., Croubels S. (2008). Multi-residue analysis of eight anticoagulant rodenticides in animal plasma and liver using liquid chromatography combined with heated electrospray ionization tandem mass spectrometry. *Journal of Chromatography B. Analytical Technologies in the Biochemical and Life Sciences 869*, 101-110.

Vanhoutte P., Paque L. (2000). *Report of the Belgian Parliament on the Organization and Production of Meat, Dairy and Eggs in Belgium and on the Political*

Responsibilities in the Dioxin Crisis. Parliament of Belgium. Document 50 0018/007.
Visser I.J.R., van den Hoven R., Vos J.H., van den Ingh T.S.G.A.M. (1988). *Pieris japonica* (pieris)-intoxicatie bij twee geiten. *Tijdschrift voor Diergeneeskunde 133*,185-189.
Volmer P.A. (2004). Pyrethrins and pyrethroids. In: Plumlee K. (editor). *Clinical Veterinary Toxicology*. Mosby, Inc. Missouri, p. 188-190.
Vos J.H., Geerts A.A.J., Borgers J.W., Mars M.H., Muskens J.A.M., van Wuijckhuise-Sjouke L.A. (2002). Jacobskruiskruid: bedrieglijke schoonheid. Vergiftiging met *Senecio jacobea. Tijdschrift voor Diergeneeskunde 127*, 753-756.
Vyt P., Maes D., Vrielinck J., Castryck F. (2005). Nitrite intoxication in pigs. *Vlaams Diergeneeskundig Tijdschrift 74*, 359-363.

Uit het verleden

VOORBEELDIG MOEDERINSTINCT BIJ EEN KOE

Dr. J. Baeke, dierenarts, Bassevelde

(tekst verschenen in het VDT jg 23, 1954, p. 225-226)

Wanneer we de literatuur nagaan, krijgen we vaak de indruk dat sommige auteurs onze hedendaagse koeien als abnormale dieren beschouwen – als wandelende melkmachines.

Sommigen gaan nog verder en vinden van de natuurlijke wildheid bij onze hedendaagse koeien niets meer terug. Dat onze koeien hun levensdoel ver voorbijgestreefd zijn, geven we grif toe. De enorme plas melk gedurende tien en meer maanden kunnen we beschouwen als een van de edelste veroveringen van de wetenschap, omdat het de mens ten goede komt. Maar nodig voor de instandhouding van de soort is dat zeker niet. Nochtans, met degenen die beweren bij de hedendaagse koe niets natuurlijk meer terug te vinden, kunnen we niet akkoord gaan. Het relaas dat volgt, toont aan dat het moederlijk instinct bij de koe, niettegenstaande de scheiding over tientallen generaties heen van moeder en kalf, nog maar weinig is veranderd.

De koe “Mele” was een zeer oude (20 jaar) zwart-bonte koe met een erg laaghangende uier, een echt melktype. De fokker E.D.M. had ze 18 jaar lang als zijn beste koe uitgebaat en anderhalf jaar terug bij de uitscheiding van het bedrijf verkocht. Ze was toen weer drachtig en had nog een benijdenswaardige melkgift. Op enkele maanden tijd veranderde Mele viermaal van eigenaar. De lelijke magere koe die elk bedrijf ontsierde, moest er telkens weer uit als een eigen koe gekalfd had en er weer voldoende melk was. Het laatst werd Mele aangekocht in oktober 1953 door een voeder handelaar V.Z. te B. die tevens een varkenshouderij uitbaatte. Mele kalfde zonder droogstand begin november en moest voor rekening van haar eigenaar in de strenge wintermaanden twee kalveren groot brengen – haar eigen kalf plus een tweede vreemd kalf.

Alles verliep zeer gunstig – ze verbleef de hele dag buiten en zocht gedurende de nacht enkel beschutting in een primitief strohok waar ook een varken verbleef dat moeilijk kon lopen wegens erge rachitis.

Op zekere morgen bemerkte de verzorger dat dit varken (+/- 70 kg) aan het zuigen was terwijl Mele rustig te herkauwen lag. Dat varken deed zulks geregeld, herstelde zeer vlug en bleef de koe achterna lopen om te zuigen. Na ongeveer 10 weken werden de kalveren afgetrokken – het varken bleef zijn gewoonte getrouw.

Mele gaf echter te veel melk voor het éne varken en door melkretentie werd de uier hard. Toevallig weigerde één van de twee kalveren alle voedsel op stal zodat besloten werd het groot kalf terug bij de koe te laten plus nog een nuchter kalf. Alles verliep weer goed en Mele kweekte *tegelijkertijd één groot kalf, één vers geboren kalf en één varken*.

Nadat dit zowat 4 weken geduurd had, werd het varken afgetrokken en ten titel van proef werden 5 lopervarkens van 40 kg bij Mele gebracht. Ofschoon de lopers ook over samengesteld meel beschikten naar beliefte, duurde het geen week of ze hadden allemaal de uier van Mele ontdekt en ze gingen er geregeld aan zuigen. Mele voedde dan *tegelijkertijd één klein kalf, één jong rund en 5 lopers van 40 à 45 kg.*

Drie lopers zijn bij Mele gebleven tot ze ongeveer 80 kg wogen en werden dan afgetrokken. De kalveren zijn bij de koe gebleven en zogen tot de dag dat ze van uitputting gestorven is in de zomer van 1954. Het ene kalf woog toen 258 kg en het andere 350 kg. Mele liet zich steeds stilstaande, terwijl ze herkauwde, leeg zuigen. De varkens en runderen leefden in de beste verstandhouding, want nu eens zogen 4 biggen tegelijk, dan eens twee biggen en twee kalveren, dan weer 3 biggen en het groot en het klein kalf.

Wekenlang hebben veel kijklustigen kunnen genieten van dit voorbeeld van moederzorg dat men vanaf de openbare weg kon gadeslaan.

Luc Devriese