Clinical and pathological studies on intoxication in horses from freshly cut Jimson weed (*Datura stramonium*)-contaminated maize intended for ensiling

R Binev[a*], I Valchev[a] and J Nikolov[a]

ABSTRACT

Spontaneous intoxication in 34 horses after ingesting freshly harvested maize that was to be used for ensiling and heavily contaminated with young *Datura stramonium* plants, is described. The clinical status of all horses was monitored for 7 days, and included body (rectal) temperature, respiratory and heart rates, colour and moistness of visible mucosae, changes in pupil size, appetite, thirst, general behaviour, locomotion, sensory perceptions, urination and defaecation. The intoxication was accompanied by altered clinical status, namely mild hyperthermia, tachycardia, polypnoea, dyspnoea and shallow breathing, mydriasis, dry oral, rectal, vaginal and nasal mucosae, acute gastric dilatation and severe intestinal gas accumulation, anorexia to complete refusal of feed, decreased or absent thirst, absence of defaecation and urination. As a result of the treatment, the clinical parameters normalised between days 2 and 5. Necropsies and pathological studies were performed on two horses that died, revealing toxic liver dystrophy, cardiac lesions and substantial dystrophic and necrotic processes in the kidneys. The observed clinical signs, the pathomorphological changes and the applied therapy could be used in the diagnosis, differential diagnosis, prognosis and treatment of Jimson weed intoxication.

Key words: atropine, horses, intoxication, Jimson weed (*Datura stramonium*), scopolamine, tropane alkaloids.

Binev R, Valchev I, Nikolov J **Clinical and pathological studies on intoxication in horses from freshly cut Jimson weed (*Datura stramonium*)-contaminated maize intended for ensiling.** *Journal of the South African Veterinary Association* (2006) 77(4): 215–219 (En.). Department of Internal Medicine, Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria.

INTRODUCTION

Jimson weed (*Datura stramonium*) is an annual plant in the family Solanaceae. The toxic principles are tropane belladonna alkaloids which possess strong anticholinergic properties[5,31,32,37]. They include hyoscyamine (stems, leaves, roots, seeds)[27], hyoscine (roots), atropine (*d,l*-hyoscyamine) and scopolamine (*l*-hyoscine)[11,16,20,23,27,31,32,41]. All parts of the plant are toxic, but the greatest amount of alkaloids are contained in ripe seeds[7,13,16,37], young, dried leaves[28] and stems and leaves of young plants[27]. They act as competitive antagonists to acetylcholine at peripheral and central muscarinic receptors at a common binding site[7,20,37]. Poisoning results in widespread inhibition of parasympathetic innervated organs[7,15,20,35,40].

The wide distribution, high toxicity and the potential for occurrence in foodstuffs are responsible for the numerous incidents in humans[1–3,6,7,9–12,14–16,18,19,21,25,30,32–35,38,41,42].

Cases of Jimson weed intoxication are considerably less frequent in animals such as cattle[29], swine[23,24,26,45], dogs[22,43], sheep and goats[17] and poultry[13,46]. In horses, poisoning has occurred after ingestion of Jimson weed seeds[4,39,44] and dried tef hay contaminated with young *Datura* plants (*Datura stramonium* and *D. ferox*)[28].

There are no published reports of cases of intoxication in horses from freshly cut Jimson weed (*Datura stramonium*)-contaminated maize intended for ensiling.

The objective of this study was to evaluate the usefulness of clinical and morphological parameters of horses after Jimson weed intoxication for rapid and correct diagnosis, differential diagnosis, prognosis and effective treatment of this intoxication.

MATERIALS AND METHODS

In October 1999, 3 stallions were referred to the Clinic of Internal Diseases and Clinical Toxicology at the Faculty of Veterinary Medicine, Stara Zagora Bulgaria. The history revealed that 18 hours previously, 34 horses, owned by the Horse Station of the Faculty of Agriculture of the Trakia University, were fed *ad libitum* with freshly harvested and chopped maize that was to be used for ensiling and heavily contaminated with young *D. stramonium* plants. All animals that ingested the forage manifested signs of intoxication to varying degrees. The animals with the most evident clinical signs were referred to the clinic.

The examination *in situ* showed that the horses were 3–14 years old, with a live body weight of 400–600 kg, of various breeds (Trakehner, Hanoverian, Danube, East-Bulgarian, Arabian etc.) and type. The animals were of both sexes: 18 mares, 12 stallions and 4 geldings.

Depending on the severity of clinical signs, the animals were divided into 3 groups:

Group 1 (n = 18) – horses with typical clinical signs: 9 stallions, 1 gelding, 8 mares, 3 of them pregnant.

Group 2 (n = 16) – horses with less obvious signs of intoxication: 3 stallions, 3 geldings, 2 pregnant mares, 8 lactating dams (the suckling foals were not included in the group).

Group 3 (n = 18) – horses owned by the Mounted Police, housed on the same premises under similar conditions, but fed another forage, served as controls.

The complete clinical status of all groups of horses was assessed on post-intoxication days 1, 2, 3, 4, 5, 6 and 7, and comprised body (rectal) temperature *via* an electronic thermometer (GT 2038 Geratherm Medical, Germany), respiratory and heart rates, colour and moistness of visible mucosae, changes in pupil size, appetite, thirst, general behaviour, locomotion, sensory perceptions, urination, and defaecation using routine clinical diagnostic methods.

Two horses that died were necropsied and specimens for histological examination were fixed in 10 % neutral formalin and processed using routine histological techniques. The specimens were embedded in paraffin blocks, sectioned on a microtome (cross-section thickness of 5 μm) and stained with haematoxylin and eosin.

Data were statistically processed using ANOVA (Statistica, Statsoft Inc., http://

[a]Department of Internal Medicine, Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria.

*Author for correspondence. E-mail: binew@abv.bg

Received: November 2005. Accepted: November 2006.

Table 1: **Change in clinical parameters in horses after intoxication with Jimson weed** (*Datura stramonium*) **(groups 1 and 2) and in the control group (group 3).**

Parameter	Group	Day after the intoxication						
		1	2	3	4	5	6	7
Body temperature (°C)	1	38.8 ± 0.3[c]	38.6 ± 0.2[c]	37.9 ± 0.1	38.0 ± 0.2	37.6 ± 0.3	37.9 ± 0.3	37.5 ± 0.2
	2	37.8 ± 0.2[b]	37.4 ± 0.1	37.4 ± 0.1	37.6 ± 0.2	37.8 ± 0.2	37.2 ± 0.2	37.4 ± 0.3
	3	37.5 ± 0.2	37.2 ± 0.1	37.9 ± 0.2	37.7 ± 0.3	37.3 ± 0.2	37.5 ± 0.2	37.4 ± 0.2
Heart rate (per min)	1	98.8 ± 8.3[c]	72.4 ± 5.4[c]	54.5 ± 6.3	41.3 ± 5.8	50.2 ± 6.3	43.0 ± 5.0	38.7 ± 4.2
	2	65.4 ± 7.1[b]	58.3 ± 6.7[a]	48.3 ± 6.2	38.8 ± 6.3	48.4 ± 4.2	46.6 ± 5.8	42.3 ± 3.3
	3	37.8 ± 5.8	39.1 ± 4.8	40.2 ± 7.1	38.8 ± 5.5	42.4 ± 4.8	37.8 ± 5.2	35.0 ± 5.0
Respiratory rate (per min)	1	64.5 ± 7.3[c]	34.2 ± 5.8[b]	15.8 ± 2.6	14.5 ± 2.6	12.7 ± 1.8	14.4 ± 2.9	13.2 ± 2.6
	2	30.2 ± 5.6	16.2 ± 2.8	18.2 ± 2.1	13.7 ± 1.4	14.4 ± 2.3	12.7 ± 1.8	13.4 ± 1.1
	3	16.2 ± 2.3	14.3 ± 2.4	16.8 ± 1.4	15.6 ± 2.3	14.8 ± 1.8	13.8 ± 2.1	15.2 ± 2.7

[a] $P < 0.05$; [b] $P < 0.01$; [c] $P < 0.001$.

www.statsoft.com). Differences between groups 1–2 and the control group, for each time interval, were considered statistically significant when $P < 0.05$.

RESULTS

Clinical studies

The changes in the clinical parameters body temperature, heart and respiratory rates are presented in Table 1. In horses from the group 1, statistically significant changes ccurred by post-intoxication days 1 and 2. The rectal temperature was higher on days 1 and 2 (38.8 ± 0.2 °C, $P < 0.01$, and 38.6 ± 0.2 °C, $P < 0.01$, respectively) than in the control group (37.5 ± 0.2 °C and 37.2 ± 0.1 °C, respectively.

Heart rates were also higher on days 1 and 2 in group 1 (98.8 ± 8.3/min, $P < 0.01$, and 72.4 ± 5.4/min, $P < 0.01$, respectively) than in the control group (37.8 ± 5.8/min and 39.1 ± 4.8/min, respectively). A weak, soft and rhythmic pulse and visible cardiac shocks in the cardiac region were registered.

The respiratory movements were faster on days 1 and 2 (64.5 ± 7.3/min, $P < 0.01$, and 34.2 ± 5.8/min, $P < 0.01$, respectively that in the control group (16.2 ± 2.3/min and 14.3 ± 2.4/min, respectively). The movements were shallow and the nostrils were flared. By post-intoxication day 3, the parameters returned to normal.

The clinical signs in this group were conspicuous, comprising mydriasis, complete refusal of feed, lack of thirst, defaecation and urination, dry mucosae (oral, nasal, vaginal and rectal), diffusely reddened conjunctivae, disturbances in locomotion, hyperaesthesia, muscle cramps and hyperreflexia.

Colic symptoms in horses from this group were moderately severe and continuous. The animals struck the ground with their forelimbs, turned their heads towards the abdomen (mostly to the left side) and stood with legs widely apart (urinating posture).

Gross examination revealed bilateral enlargement of the abdomen (in most cases more evident on the left side). Rectal examination of all animals revealed strong intestinal gas accumulation, especially in *colon primum ventrale et dorsale sinistrum.* In three-quarters of the animals this was accompanied by an increase in caecal and gastric volume.

Within 3–5 days most of the clinical signs had normalised, the last ones to do so being moistness of the mucosae, appetite and normal colour of conjunctivae (by post-intoxication days 5–6).

By day 3, polydipsia, frequent urination and defaecation were observed in 12 horses (66.6 %). The faeces were soft to aqueous, with a very unpleasant odour and greenish in colour. These changes receded after the 5th to 6th day.

In spite of therapy (see below), 2 horses died on the 2nd day.

In group 2, statistically significant changes in body temperature were not detected. The heart rate was higher by days 1–2 (65.4 ± 7.1/min, $P < 0.01$, and 58.3 ± 6.7/min, $P < 0.05$, respectively) than in the control group (37.8 ± 5.8/min and 39.1 ± 4.8/min, respectively). The pulse was weak and soft. The respiratory rate was higher than in the control group only on day 1 (30.2 ± 5.6/min, $P < 0.05$, *vs* 16.2 ± 2.3/min).

In group 2 the colic symptoms alternated with short, painless episodes. The other clinical singns were less obvious and transient. Only on post-intoxication day 1 were the following clinical signs observed: mydriasis in 9 horses (56.2 %), complete refusal of feed ($n = 4$) (25 %), anorexia ($n = 12$) (75 %), lack of thirst ($n = 4$) (25 %), lack of defaecation ($n = 8$) (50 %), dry mucosae ($n = 5$) (31.2 %), diffusely reddened conjunctivae ($n = 2$) (12.5 %) and nervous signs ($n = 3$) (18.8 %). The frequency of urination remained unchanged.

Gross examination revealed no enlargement of the abdomen and the rectal examination showed all horses to have a weak intestinal gas accumulation in *colon primum ventrale et dorsale sinistrum* and the caecum. The stomach was not dilated.

The suckling foals ($n = 8$) of dams from this group did not show signs of intoxication. Only 2 of them (25 %) had diarrhoeic faeces, and more frequent defaecation was evident on days 2 and 3.

Morphological studies

Several hours (3–5) before two of the horses in group 1 died, they were recumbent with stretched forelegs, often followed by lateral recumbency accompanied by kicking in the direction of the abdomen and looking in the same direction. Sweating, grinding of teeth, flared nostrils, clonic seizures, hypothermia (35–35.5 °C), accelerated, weak, soft and hardly perceptible heart rate (100–120/min), elevated respiratory rate (50–60/min) and shallow respiration were also observed.

The pathoanatomical study evidenced a strong intestinal and gastric gas accumulation. In 1 horse the stomach was ruptured and in the other one, the diaphragm. The wound edges were irregular and impregnated with clotted blood, thus showing that the rupture took place before death. The mesenterial blood vessels were highly hyperaemic, and on the mesenterium and intestinal serosa, petechial haemorrhages were present. The gastric, intestinal and bowel mucosae were diffusely reddened. In the stomach and intestinal content, seeds and seed pods of Jimson weed were found. In the animal with the ruptured stomach, similar contents were found in the abdominal cavity as well. Multiple small erosions and superficial necroses on the bowel mucosa were observed. Mesenterial lymph nodes were oedematous, with a moist rose-reddish cross-section and single haemorrhages. The liver had a clay-yellow colour and in some areas, the enlarged and blood-filled central veins of lobules were

clearly distinguishable. The mucosae of the spleen, kidneys, epicardium and the endocardium of both atria exhibited petechial and more extensive haemorrhages. The lungs were strongly reduced in size, hyperaemic with an increased density. The meninges were strongly hyperaemic.

The mucosae of intestines and the colon manifested various degrees of dystrophic-necrotic processes, often resulting in complete desquamation of the lining and glandular epithelium.

Massive hyperaemia of vessels and mucosal oedema, more apparent in the large intestines, was present. The sinuses of mesenterial lymph nodes were filled with erythrocytes and in the parenchyma, single haemorrhages were present. In the lungs, strong congestive hyperaemia and infiltration of the interstitium with erythrocytes was noticed. The cardiac musculature revealed granular dystrophy, cloudy swelling, hyalinisation of the myocardium, hyperaemia and haemorrhages with interstitial oedema (Fig. 1).

In the liver, varying degrees of granular and fatty dystrophy, karyolysis and pyknosis of hepatocytic nuclei was observed (Fig. 2). The central veins and their capillaries were highly distended and overfilled with blood. In the renal parenchyma, extensive, strong and general dystrophic and necrotic changes and disintegration were present, with epithelial desquamation of the renal tubules.

The lethal histopathological changes were similar in both animals.

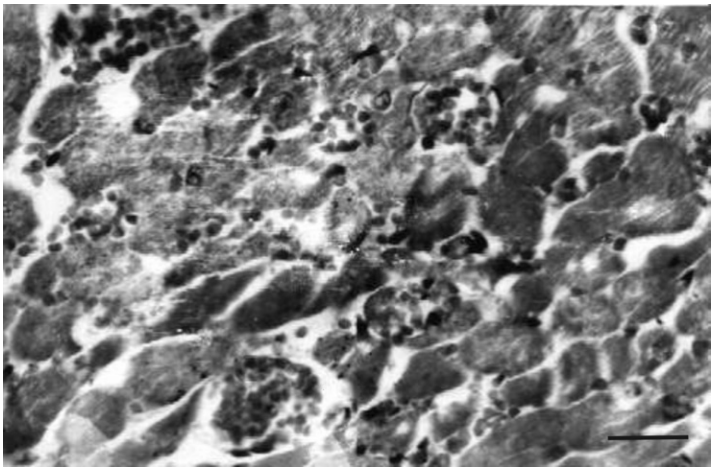

Fig. 1: **Morphological changes in horses after intoxication with Jimson weed** (*Datura stramonium*)**. Granular dystrophy and hyalinisation of the cytoplasm of myocardiac fibres. Strong blood vessel hyperaemia.** Scale bar = 3 µm.

Treatment

In horses in group 1, a single gastric lavage with suspension of activated charcoal was performed *via* naso-oesophageal intubation. Half an hour later, a solution of 300 g $MgSO_4$ (Biovet, Pestera, Bulgaria) and 500 g activated charcoal in 5 ℓ water was administered *via* naso-oesophageal intubation.

The inflated intestines were perforated *via* the rectum by means of a long needle covered by the hand. The procedure was performed after identifying the site with the strongest intestinal gas accumulation. For controlling the exiting gas, the needle was connected to a rubber tube, the distal end of which was inserted into a container with water.

A single bloodletting was done in all horses. The amount of blood was calculated according to body weight, on average 10 mℓ/kg. For 4 consecutive days intravenous administration of 3 ℓ Ringer solution (Balkanpharma, Troian, Bulgaria), 10 g ascorbic acid (Vitamin C, Biovet, Pestera, Bulgaria), 1 g thiamine (Vitamin B_1, Biovet, Pestera, Bulgaria), 100 mℓ sodium N-acethyl methionine (5 % Metionin, Sinaps, Veliko Tarnovo, Bulgaria) and 20 mℓ 20 % caffeine sodium benzoate (Biovet, Pestera, Bulgaria) was continued. Medication therapy was extended to intramuscular administration of benzylpenicillin procaine and dihydrostreptomycin (Intramicin, Ceva Sante Animale, France) at a dose of 5 mℓ/100 kg body weight, every 24 hours. As an antidote, pilocarpine hydrochloride (5 % Pilocarpin, Biovet, Pestera, Bulgaria) was administered subcutaneously: on post-intoxication day 1, 100 mg 6 times at 2-hour intervals; on post-intoxication day 2, 100 mg 4 times at 4-hour intervals, and on post-intoxication days 3 and 4, 100 mg 3 times at 6-hour intervals.

During the application of pilocarpine hydrochloride, the clinical status of animals was monitored with regard to the restoration of the moisture levels of visible mucosae, the pupils, heart and respiratory rates, defaecation and urination. After 3–5 administrations of pilocarpine hydrochloride, it was noticed that mucosal moistness was restored, although for a very short time in the beginning (from 30–60 minutes). After 2–3 days, this interval increased to several hours (3–5), but a complete restoration of moistness was only observed by days 5–6. The other clinical parameters normalised between the 3rd and 5th days. The dilated pupils were not influenced during the first 3 days of pilocarpine hydrochloride application, but returned to normal between days 4 and 5.

In horses from group 2, venesection was done on post-intoxication day 1 and 100 mg pilocarpine hydrochloride was administered thrice at 4 hourly intervals.

DISCUSSION

The toxic effect of Jimson weed results from the antimuscarinic action of the alkaloids it contains[3–5,7,16,20,23,35,37]. The increased heart rate observed by us and by other authors[2,4,10,11,14,15,17,30,44] is due to removal of the parasympathetic component of vagal block and correlated with

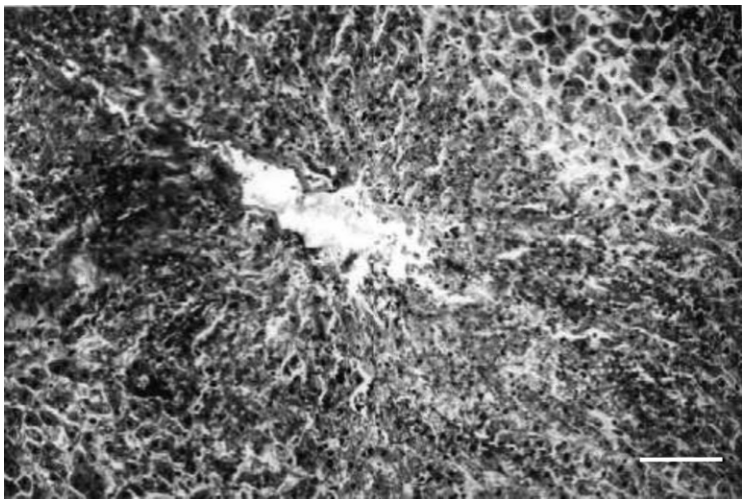

Fig. 2: **Morphological changes in horses after intoxication with Jimson weed** (*Datura stramonium*)**. Toxic liver dystrophy in the region of the central vein.** Scale bar = 2.5 µm.

observed histopathological alterations (granular dystrophy, cloudy swelling, hyalinisation of the myocardium, hyperaemia, haemorrhages with interstitial oedema). The observed polypnoea with dyspnoea is in accordance with data from literature[4,10,11,14,15,17,30,44], and are probably a consequence of the colic seizure and corresponds to morphological changes in lung parenchyma – the strong congestive hyperaemia and infiltration of the interstitium with erythrocytes. The dilatation of the pupils is a clinical sign of this intoxication[1,6,14,22,28,29,32,37,39] and was a further manifestation of parasympathetic block[6,10,11,14,15,30,34]. The observed hyperthermia is noteworthy, taking into consideration that peripheal vasoconstriction in humans could not be a cause for elevation in body temperature[8]. On the other hand, owing to the rapid development of the intoxication, we did not see histopathological signs of inflammatory processes in horses at the time of death. It is assumed that hyperthermia was resulting from impaired thermoregulation, correlating with strongly hyperaemic meninges. Alkaloids inhibit the secretion of cholinergic innervated glands and this is an explanation for the dry mucosae (oral, nasal, rectal, vaginal)[2,6,10–12,14–17,19,30,37,39]. Subsequent to the inhibition of parasympathetically innervated organs[16,20,35] a paralytic ileus occurs[39], followed by secondary intestinal gas accumulation, clinically manifested by acute gastric dilatation, increased bowel volume (especially the colon and caecum) and lack of defaecation. An anticholinergic sign is the constriction of urinary bladder sphincter, resulting in the accumulation of urine, correlating with observed anuria[2,11,14,16,29]. Our observations on the lack of thirst, urination and defaecation are in accordance with the literature[4,11,13–15,17,28,35,45] but differ from cited cases of intoxication[44], accompanied with polyuria, polydipsia, and defaecation. In our opinion, these are characteristic features that counteract the intoxication. The nervous signs (disturbances in locomotion, hyperaesthesia, hyperreflexia and tremor, correlating with meningeal hyperaemia) observed by us as well as other investigators[11,21,29,33,42] is in accordance with the well-known CNS signs due to these alkaloids.

Most investigators[2,12,14,19,38] consider physostigmine therapy as the most effective in anticholinergic toxicity. In critical cases, however, the preparation has no effect[38] and it can produce complications such as seizures and cardiac arrhythmia[32,36]. In Bulgaria, the use of physostigmine has been discontinued and instead its synthetic analogue rivastigmine is employed, but it is for internal use cannot be used in horses with intoxication. We therefore we used pilocarpine hydrochloride as an antidote – a cholinomimetic with a direct effect (antagonist of cholinoreceptors). There are no data on the relative efficacy of these preparations and whether physostigmine or pilocarpine hydrochloride is more or less effective in Jimson weed (*Datura stramonium*) intoxication remains to be determined.

CONCLUSIONS

The clinical signs manifested after intoxication with Jimson weed were hyperthermia, tachycardia, polypnoea with dyspnoea, acute gastric dilatation, secondary intestinal gas accumulation, mydriasis, dry mucosae, complete refusal of feed, lack of thirst, defaecation and urination. In lactating mares, the course of Jimson weed intoxication was more rapid, without the typical signs. The suckling foals did not manifest signs of intoxication. The prognosis is more favourable if polydipsia, polyuria and frequent defaecation are observed during the recovery from intoxication. The pathomorphological studies showed a toxic liver dystrophy and extensive dystrophic and necrotic changes in the kidneys and myocardium. The applied schedule of treatment is effective in the initial stage of the intoxication.

The clinical signs, morphological alterations and the effect of the treatment in Jimson weed intoxication are directly related to the stage of intoxication.

REFERENCES

1. Al-Shaikh A M, Sablay Z M 2005 Hallucinogenic plant poisoning in children. *Saudi Medical Journal* 26: 118–121
2. Amlo H, Haugeng K L, Wickstrøm E, Koss A, Husebye T, Jacobsen D 1997. Poisoning with Jimson weed. Five cases treated with physostigmine. *Tidsskrift for den Norske Laegeforening* 117: 2610–2612
3. Arouko H, Matray M D, Braganca C, Mpaka J P, Chinello L, Castaing F, Bartou C, Poisot D 2003 Voluntary poisoning by ingestion of *Datura stramonium.* Another cause of hospitalization in youth seeking strong sensations. *Annales de Medecine Interne* 154: 46–50
4. Barney G H, Wilson B J 1963 A rare toxicity syndrome in ponies. *Veterinary Medicine* 58: 419–421
5. Berger E, Ashkenazi I 2003 Jimson weed poisoning. *Harefuah* 142: 364–367+397
6. Birmes P, Chounet V, Mazerolles M, Cathala B, Schmitt L, Lauque D 2002 Self-poisoning with *Datura stramonium*: 3 case reports. *Presse Medicale* 31: 69–72
7. Boumba V A, Mitselou A, Vougiouklakis T 2004 Fatal poisoning from ingestion of *Datura stramonium* seeds. *Veterinary and Human Toxicology* 46: 81–82
8. Brown J H, Taylor P 2001 Muscarinic receptor agonists and antagonists. In Hardman J G, Limbird L E, Gilman A G (eds) *Goodman & Gilman's the pharmacological basis of therapeutics* (10th edn). McGraw-Hill, New York: 141–160
9. Calbo Mayo J M, Barba Romero M A, Broseta Viana L, Medrano Gonzalez F 2004 Accidental familiar poisoning by *Datura stramonium. Anales de Medicina Interna* 21: 415
10. Castanón López L, Martinez Badás J P, Lapena López de Armentia S, Gomez Mora J, Garcia Arias M L 2000 *Datura stramonium* poisoning. *Anales Espanoles de Pediatria* 53: 53–55
11. Chang S S, Wu M L, Deng J F, Lee C C, Chin T F, Liao S J 1999 Poisoning by *Datura* leaves used as edible wild vegetables. *Veterinary and Human Toxicology* 41: 242–245
12. Crouch B I, Caravati E M, Summers E H 2001 Jimsonweed toxicity and treatment. *Journal of Pharmaceutical Care in Pain and Symptom Control* 9: 25–30
13. Day E J, Dilworth B C 1984 Toxicity of jimson weed seed and cocoa shell meal to broilers. *Poultry Science* 63: 466–468
14. Dewitt M S, Swain R, Gibson L B J 1997 The dangers of jimson weed and its abuse by teenagers in the Kanawha Valley of West Virginia. *West Virginia Medical Journal* 93: 182–185
15. Djibo A, Brah Bouzou S 2000 Acute intoxication to the 'sobi-lobi' (*Datura*). Apropos of 4 cases in Niger. *Bulletin de la Société de Pathologie Exotique* 93: 294–297
16. Dugan G M, Gumbmann M R, Friedman M 1989 Toxicological evaluation of jimson weed (*Datura stramonium*) seed. *Food and Chemical Toxicology* 27: 501–510
17. El Dirdiri N I, Wasfi I A, Adam S E, Edds G T 1981 Toxicity of *Datura stramonium* to sheep and goats. *Veterinary and Human Toxicology* 23: 241–246
18. Ertekin V, Selimoglu M A, Altinkaynak S 2005 A combination of unusual presentations of *Datura stramonium* intoxication in a child: rhabdomyolysis and fulminant hepatitis. *Journal of Emergency Medicine* 28: 227–228
19. Forno F J, Terry R A 1998 Accidental ingestion of jimsonweed by an adolescent. *Journal of the American Osteopathological Association* 98: 502–504
20. Friedman M, Levin C E 1989 Composition of jimson weed (*Datura stramonium*) seeds. *Journal of Agricultural Food Chemistry* 37: 998–1005
21. Haas L F 1995 *Datura stramonium* (Jimsonweed). *Journal of Neurology Neurosurgery and Psychiatry* 58: 654
22. Hanse P, Clerc B 2002 Anisocoria in the dog provoked by a toxic contact with an ornamental plant: *Datura stramonium. Veterinary Ophthalmology* 5: 277–279
23. Janssens G, DeWilde R 1989 Toxicity of thornapple (*Datura stramonium* and/or *D. ferox*) seed present in pig feed. *Vlaams Diergeneeskundig Tijdschrift* 58: 84–86
24. Keeler R F 1981 Absence of arthrogryposis in newborn Hampshire pigs from sows ingesting toxic levels of jimsonweed during gestation. *Veterinary and Human Toxicology* 23: 413–415
25. Kurzbaum A, Simsolo C, Kvasha L, Blum A 2001 Toxic delirium due to *Datura stramonium. Israel Medical Association Journal* 3: 538–539
26. Leipold H W, Oehme F W, Cook J E 1973 Congenital arthrogryposis associated with ingestion of jimsonweed by pregnant sows. *Journal of the American Veterinary Medicine*

Association 162: 1059–1060
27. Miraldi E, Masti A, Ferri S, Barni Comparini I 2001 Distribution of hyoscyamine and scopolamine in *Datura stramonium. Fitoterapia* 72: 644–648
28. Naude T W, Gerber R, Smith R J, Botha C J 2005 *Datura* contamination of hay as the suspected cause of an extensive outbreak of impaction colic in horses. *Journal of the South African Veterinary Association* 76: 107–112
29. Nelson P D, Mercer H D, Essig H W, Minyard J P 1982 Jimson weed seed toxicity in cattle. *Veterinary and Human Toxicology* 24: 321–325
30. Nogue S, Pujol L, Sanz P, De la Torre R 1995 *Datura stramonium* poisoning. Identification of tropane alkaloids in urine by gas chromatography – mass spectrometry. *Journal of International Medical Research* 23: 132–137
31. Nuhu H, Ghani A 2002 Alkaloid content of the leaves of three Nigerian *Datura* species. *Nigerian Journal of Natural Products and Medicine* 6: 15–18
32. Oberndorfer S, Grisold W, Hinterholzer G, Rosner M 2002 Coma with focal neurological signs caused by *Datura stramonium* intoxication in a young man. *Journal of Neurology Neurosurgery and Psychiatry* 73: 458–459
33. Parissis D, Mellidis C, Boutis A, Apostolidis K, Ignatiadis M, Kiosses V, Milonas I 2003 Neurological findings in a case of coma secondary to *Datura stramonium* poisoning. *European Journal of Neurology* 10: 745–746
34. Pinilla Llorente B, Portillo A, Muino Miguez A, Garcia Castano J 1992 *Datura stramonium* poisoning. *Annales de Medecine Interne* 9: 208
35. Roblot F, Montaz L, Delcoustal M, Gaboriau E, Chavagnat J J, Morichaud G, Pourrat O, Scepi M, Patte D 1995 Datura stramonium poisoning: The diagnosis is clinical, treatment is symptomatic. *Revue de Medecine Interne* 16: 187–190
36. Rocco V G 2000 Poisoning. In Young G B, Ropper A H, Bolton C F (eds) *Coma and impaired consciousness.* McGraw-Hill, New York: 460–462
37. Ruhwald M 2005 Jimsonweed poisoning and the anticholinergic syndrome. *Ugeskrift for Laeger* 167: 2041–2043
38. Salen P, Shih R, Sierzenski P, Reed J 2003 Effect of physostigmine and gastric lavage in a *Datura stramonium*-induced anticholinergic poisoning epidemic. *American Journal of Emergency Medicine* 21: 316–317
39. Schulman M L, Bolton L A 1998 *Datura* seed intoxication in two horses. *Journal of the South African Veterinary Association* 69: 27–29
40. Sopchak C A, Stork C M, Cantor R M, Ohara P E 1998 Central anticholinergic syndrome due to Jimson weed: physostigmine therapy revisited. *Journal of Toxicology and Clinical Toxicology* 36: 43–45
41. Steenkamp P A, Harding N M, Van Heerden F R, Van Wyk B E 2004 Fatal *Datura* poisoning: identification of atropine and scopolamine by high performance liquid chromatography/photodiode array/mass spectrometry. *Forensic Science International* 145: 31–39
42. Thabet H, Brahml N, Amamou M, BenSalah N, Hedhill A, Yacoub M 1999 *Datura stramonium* poisonings in humans. *Veterinary and Human Toxicology* 41: 320–321
43. Tostes R A 2002 Accidental *Datura stramonium* poisoning in a dog. *Veterinary and Human Toxicology* 44: 33–34
44. Williams S, Scott P 1984 The toxicity of *Datura stramonium* (thorn apple) to horses. *New Zealand Veterinary Journal* 32: 47
45. Worthington T R, Nelson E P, Bryant M J 1981 Toxicity of thornapple (*Datura stramonium* L) seeds to the pig. *Veterinary Record* 108: 208–211
46. Yaman I, Eroksuz H, Ozer H, Ceribasi A O 2005 Toxicity of dietary *Datura stramonium* seed to broilers. *Indian Veterinary Journal* 82: 379–383