

Toxic Weed Seed Contaminants in Soybean Processing

G. R. LIST and G. F. SPENCER, Northern Regional Research Center, Federal Research, Science and Education Administration, U.S. Department of Agriculture, Peoria, IL 61604 and W. HAWARD HUNT, Standardization Division, Federal Grain Inspection Service, U.S. Department of Agriculture, Beltsville, Maryland 20705

ABSTRACT

Soybeans are subject to potential contamination from toxic weed seeds during harvest, transportation and storage. The more common foreign seeds include those from jimsonweed, cocklebur, nightshade, cowcockle, corncockle, morning glory, castor, pokeweed, and crotalaria. This review addresses itself to the chemistry and toxic principles of these contaminants. Since jimsonweed seeds are probably the most toxic and most prevalent contaminant of soybeans, they are discussed in greatest detail. A gas liquid chromatographic method for the determination of tropane alkaloids in jimsonweed seeds is described together with the results from analyses of 11 samples found in grain shipments. Results of a nationwide survey of jimsonweed seeds in soybean samples are also included. The fate of jimsonweed seed alkaloids during solvent extraction of contaminated soybeans and alkali refining of crude oil was investigated. Extraction of a 50:50 mixture of soybeans and jimsonweed seeds with petroleum ether yielded meal and crude oil fractions; chemical analysis showed that virtually all the alkaloids remained in the meal. Alkali refining effectively removed atropine added to crude soybean oil.

INTRODUCTION

Many plants contain alkaloids, glucosides, irritant oils, organic acids, minerals, resins, and phytotoxins that are potentially toxic to man and domestic animals. Kingsbury (1) points out that while many poisonous principles are potentially disastrous to livestock, some have not been troublesome because of certain deep-seated protective feeding habits and, with few exceptions, animals will not become poisoned by plants unless forced to do so by some unusual or artificial conditions of husbandry. Also, it was emphasized that control of livestock poisoning should be sought in stock management.

On the other hand, herbicides, improved cultivation practices, and vigorous cleaning of food grains prior to processing render the possibility of man being poisoned from eating contaminated grains very unlikely. Even under ideal conditions, the poisoning of humans by toxic weed seeds and plants is extremely rare.

Nonetheless, a number of toxic weed seeds have been identified as contaminants in soybean samples taken by the Federal Grain Inspection Service. These include cocklebur, morning glory, castor, pokeweed, cowcockle, corncockle, nightshade, crotalaria, and jimsonweed. Weed seed contamination can result from the plants growing with the grain crops and subsequent pick-up during harvest. It has been suggested (2,3) that amounts of foreign material permitted by U.S. Federal Grain Standards (4) contribute to the problem. According to these standards, U.S. No. 1 grades of grain may contain up to 1% foreign material, i.e., dirt, weed seeds, and trash. Other grades may contain up to 5%. Hutchins (3) states that foreign material costs the soybean industry hundreds of thousands of dollars in capital investments for grain cleaning equipment and damage to processing equipment.

Aside from potential toxicity aspects, another obvious but virtually unexplored facet of the problem concerns the effects of foreign material on the quality of finished protein and oil products. Although it has been shown that the presence of foreign material during soybean processing contributes to lower flavor stability and darker colored oil products (5), little is known about the effects of foreign material on protein functionality.

Of the seeds cited in the introductory paragraph, jimsonweed is without a doubt the most common and the most toxic. Occasionally, herbicides are not effective in controlling this weed. For example, in 1973, sorghum exported to India was badly contaminated with jimsonweed seed because the seeds are about the same size and color as sorghum and cannot be separated during harvest or by screening and cleaning (2). That incident prompted investigations concerning the analysis, distribution, and fate of toxic alkaloids in soybean processing (6). This review will summarize these findings.

Much of the literature on toxic plants and weed seeds has been summarized by Kingsbury (7) and Muenscher (8). Several additional texts (9,10) are useful because they provide color photographs of many of the plants of interest. Radeleff (11) and Garner (12) have thoroughly discussed the poisoning of livestock and poultry by weed seeds in their textbooks on veterinary toxicology. Botanical information on toxic weed seeds can be found in Bailey and Bailey (13), and the Merck Index (14) is a valuable source of information of the chemistry of toxic principles found in weed seeds. The foregoing have been freely drawn on in preparing this review.

TOXIC WEED SEEDS - DISTRIBUTION TOXIC PRINCIPLES, AND POISONING

The toxic weed seeds identified as contaminants in soybeans, their geographical distribution, and toxic principles are given in Table I.

Cocklebur (*Xanthium strumarium*)

Cockleburs are widely distributed throughout North America. Authorities disagree as to the toxic principle. Radeleff (11) states that the germinating seeds and seedlings contain the highly toxic glucoside xanostumarium. However, Kingsburg (7) cites a critical pharmacological study (15) that proved beyond doubt that the poisonous principle in cockleburs is hydroquinone. This compound has been isolated from the plant, and the lesions of cocklebur poisoning can be reproduced in detail by administration of synthetic hydroquinone. Also, it has been impossible to demonstrate that hydroquinone is present in a glucosidic combination in the plant.

Cocklebur poisoning of all classes of domestic livestock has been reported from most areas of the United States (16). The poisonous principle in mature plants is contained in seeds, which are toxic at a level of ca. 0.3% of an animal's weight. However, since the seeds are encased within the burs, which are normally not eaten, poisoning rarely results. Plants in the cotyledonary state of growth are toxic at ca. 1.5% of an animal's body weight. However, as the plant germinates, hydroquinone is distributed into the

TABLE I
Toxic Weed Seeds Identified in Soybeans^a

Seed	Genus, species	Distribution, United States	Toxic principle(s)
Cocklebur	<i>Xanthium strumarium</i>	Widespread	Hydroquinone
Nightshade	<i>Atropa belladonna</i>	Cultivated	Tropane alkaloids
Cowcockle	<i>Saponaria vaccaria</i>	Northwestern states	Githagenin
Corncockle	<i>Agrostemma githago</i>	Widespread	Githagenin
Morning glory	<i>Ipomea</i> spp.	Widespread	Clavine, indole alkaloids
Castor	<i>Ricinus communis</i>	South central	Ricinine, ricin
Pokeweed	<i>Phytolacca americana</i>	Eastern	Saponins, glycoproteins
Crotalaria	<i>Crotalaria</i> spp.	Southwestern, southern	Pyrrrolizidine alkaloids
Jimsonweed	<i>Datura stramonium</i>	Widespread	Tropane alkaloids

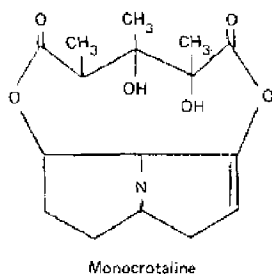
^aU.S. Federal Grain Inspection Service.

seedling. Since toxicity is not lost on drying (7), consumption of silage contaminated with cocklebur can poison livestock. Pigs are commonly poisoned (17), with symptoms of depression, nausea, vomiting, and muscular weakness. These symptoms are also common to other classes of livestock, except that vomiting may not be accomplished by ruminants. Fowl poisoned by cocklebur seeds exhibit no symptoms other than profound depression (7).

Crotalaria spp

Crotalaria species reported to be toxic include *C. sagittalis*, *C. spectabilis*, and *C. retusa*. The first is fairly widely distributed in areas bounded by Texas, Florida, Southern New England, and South Dakota, and forms extensive stands in bottomlands of the Missouri and other rivers. In the middle 1880s, *C. sagittalis* was reported to be toxic to horses feeding on the plant found in river bottoms. However, this condition known as "bottoms disease" became less important after 1900, and Kingsbury (7) states that *C. sagittalis* poisoning was exclusively a disease of the 19th Century.

C. spectabilis is distributed in fields and roadsides of the southern states north to Virginia and Missouri, while *C. retusa* is found in waste places and sandy soil regions in Peninsular Florida. Neal et al. (18) isolated an alkaloid from *C. spectabilis* seeds, leaves, and stems and named it monocrotaline. Subsequent studies by Adams and Rogers (19) showed monocrotaline to be a pyrrolizidine alkaloid, the structure of which is shown below.

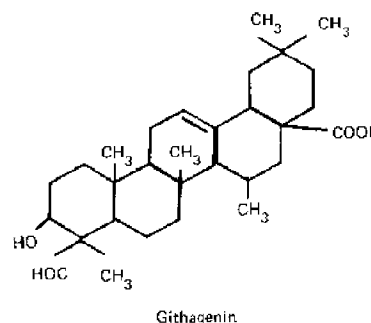


Monocrotaline has also been isolated from the seeds and other parts of *C. retusa* (19).

Becker et al. (20) have reviewed the toxicity of *C. spectabilis* and *C. retusa*. *C. spectabilis* has caused severe poisoning in fowl, cattle, and horses, but sheep, goats, and mules are less susceptible (7). Although *C. spectabilis* seeds have been reported as contaminants in shelled corn and possibly in soybeans (21), they reportedly can be easily removed by screening the grains (7). *C. retusa* is toxic primarily to fowl and is considered less acute than *C. spectabilis*. Since cattle find *C. retusa* distasteful, they will not eat it under ordinary circumstances (7).

Cowcockle (*Saponaria vaccaria*) and Corncockle (*Agrostemma githago*)

Cowcockle is distributed in the northwestern portions of the United States and Western Canada. The toxic principle is the saponin githagenin (7), (14).



Cowcockle seeds are only of academic interest because they are distasteful to animals, and no clear-cut cases of poisoning have been reported in North America.

Corncockle is widely distributed in North America and is commonly found where winter wheat is grown. The seeds contain 5 to 7% githagenin. Poisoning of poultry, hogs, horses, and cattle has been reported (7) and, prior to the advent of grain cleaning techniques, humans were poisoned from eating bread made from wheat contaminated with corncockle seed (22).

Morning Glory (*Ipomea* spp.)

Several morning glory subspecies have been reported as toxic. *I. fistulosa*, a South American vine, has become established in waste soils of Coastal Plains States from Texas to South Carolina and Peninsular Florida. Although *I. fistulosa* has not been reported to be toxic in the United States, sheep, goats, and cattle have been poisoned in Brazil (7). The widely distributed common morning glory *I. convolvulus*, however, contains purgative principles causing mild distress in hogs (7). Clavine and indole alkaloids have been identified in a number of morning glory varieties (23).

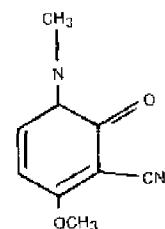
Castor Seed (*Ricinus communis*)

The castor plant is distributed in the South Central States. Most authorities (7) cite the toxic principle as the phytotoxin, ricin, a glycoprotein (mole wt 65,000). However, the Merck Index (14) also cites ricinine as a toxic principle of castor seeds and leaves.

Ricin is reportedly one of the most toxic plant substances known. In animals, the minimum lethal dose by injection is ca. 10^{-4} mg/kg, but it is several hundred times less toxic by oral ingestion (7).

The toxicity of castor seeds varies among livestock.

Horses are poisoned ca. 0.01% of their body weight. Cattle, sheep, and hogs require ca. 0.2%, while poultry require ca. 1.4% of their weight. Single oral lethal doses for various classes of livestock are given in Garner's Veterinary Toxicology (12).



Ricine

There are a few recorded cases of human deaths from eating castor bean seeds. According to Morton (10), two to three seeds are fatal to children. However, according to the reports to the national clearing house for poison control centers, other have become ill after eating up to 12 seeds, but did not die.

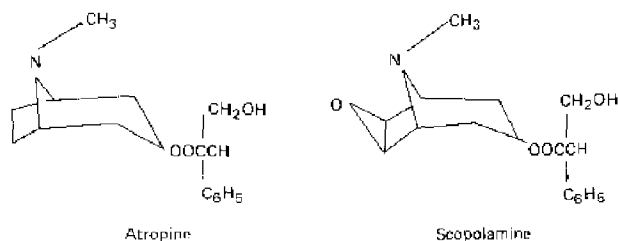
According to Kingsbury (7), toxic castor bean press cake can be made nontoxic simply by heating, since the phytotoxin ricin is heat labile. Ricin is not found in the oil. Morton (10) reports that castor seeds contain an allergen (presumably the aforementioned ricine) that causes bronchial asthma and dermatitis among castor oil factory workers.

Pokeweed (*Phytolacca americana*)

Pokeweed is distributed in the eastern United States. Although the toxic principle(s) has not been identified, extracts of the root show saponic characteristics (7). Dreisbach's Handbook of Poisoning lists glycoproteins as toxic principles (24). Animals are rarely poisoned, because the most toxic portions are underground and the plant is not very palatable. Pigs may be poisoned by eating the roots. The entire plant has produced toxic symptoms in cows when included in corn silage, but berries mixed in silage produced no toxic symptoms. Several human deaths have been reported, possibly as a result of eating the berries (7).

Nightshade (*Atropa belladonna*)

The presence of nightshade as a contaminant in soybeans is difficult to explain, because it is a cultivated plant and does not normally occur in the wild. The toxic principles are atropine and scopolamine, the structures of which are shown below.



Atropine

Scopolamine

Incidence of poisoning from nightshade is very rare. Kingsbury (7) states that in the United States, where the plant is uncommon, poisoning of livestock probably never occurs.

Jimsonweed (*Datura stramonium*)

D. stramonium is known by a number of common names including jimsonweed, thornapple, apple of Peru, tolgua, and Jamestown weed -- the most common. The latter stems from the mass poisoning of British troops sent to Jamestown to quell Bacon's rebellion in 1676 (7). The plant is widely distributed from Florida to Texas, north into Canada, in the far western states, and in waste areas. Jimsonweed is common to the rich soils of barnyards, pastures, and hog lots (10). Like the nightshade, the toxic principles are the tropane alkaloids, atropine and scopolamine.

In the United States, horses, cattle, sheep, hogs, and mules have perished from consuming whole jimsonweed plants present in hay or silage. Chickens have been poisoned by eating the seeds. Livestock find the whole plant distasteful and will not eat it under ordinary circumstances, and accounts of poisoning indicate that animals were forced to eat it through a lack of more desirable forages. Since the plant is commonly found in haylots and barnyards, it is frequently available under conditions where other feeds are not. However, because it is an annual, it can be readily controlled by cutting it before it goes to seed (7).

Compared to the previously discussed seeds, considerably more information is available on the human toxicity of *Datura*. This can be accounted for in part by the fact that the *Datura* alkaloids are useful in medicine and have been investigated both chemically and pharmacologically. The plant is unusual in that human poisonings are more commonly reported than are animal poisonings (7). According to Mitchell and Mitchell (25), 4 to 5 g of *Datura* seed or leaf constitutes a lethal dose to children, based on the toxicity of pure atropine. However, cases where the number of seeds consumed can be documented indicate that considerably less is required for development of toxic symptoms and/or death. McNally (26), in reviewing jimsonweed poisoning in man, states that a 2½ year-old died after consuming 16 *Datura* seeds, and a 7-year-old child died within 13 hr after eating 22 seeds.

Alkaloid Content of Jimsonweed (*Datura stramonium*) seeds

The close chemical relationship between the *Datura* alkaloids, their interconvertibility during extraction, and their presence within the plant in varying relative and absolute amounts, have hindered development of reliable analytical methods. A study of the distribution of tropane alkaloids in various species of *Datura* (27) demonstrated that every part of the plant contains alkaloids, with greater amounts found in younger plants. The alkaloid content in plant parts in decreasing order is: petiole, stem, vegetable tips, sprouts at the base of the leaves, fruits, leaves, and roots. According to Kingsbury (7), total alkaloid content of *D. stramonium* varies from 0.25 to 0.7%, whereas Radleff (11) gives a value of 0.25% or more. Neither author cites the source of his information.

Because of their importance in forensic medicine, considerable analytical methodology has been developed to rapidly and routinely identify plant alkaloids. A detailed discussion of these methods has been presented by Clarke (28). Prior to the advent of gas liquid chromatography, these methods consisted of paper and thin layer chromatography, ultraviolet spectrophotometry, and color tests.

A separation and analysis scheme for *D. stramonium* alkaloids has been described previously (6). Briefly, the method consists of pretreatment of the whole seed with base (NH_4OH) and extraction of both lipids and alkaloids with ethyl ether. The alkaloids are recovered by acid-base extractions and are converted to trimethylsilyl ethers (TMS) for GLC analysis.

Although atropine and scopolamine can be separated by GLC (29), preliminary studies showed that quantitation and separation can be improved by GLC as TMS ethers using ethyl arachidate as an internal standard as shown in

Figure 1. Ethyl arachidate was chosen as an internal standard because of its similarity in retention times to atropine and scopolamine and because its GLC response is very close to that of the alkaloids.

This separation/analysis scheme was applied to 11 samples of jimsonweed seeds (isolated from soybeans - see survey section) and gave the results shown in Table II. Atropine varied from 0.19 to 0.43%; scopolamine from 0.02 to 0.11%; while total alkaloids varied from 0.22 to 0.48%. Mean contents for atropine, scopolamine, and total alkaloids were 0.29, 0.05, and 0.34%, respectively. These values for total alkaloids are in the range quoted by Kingsbury (7) and Radeleff (11). Also, values for the weight in grams for 1000 seeds are given in Table II. The results show that 1000 jimsonweed seeds weigh an average of 7.6 g, 1 jimsonweed seed weighs ca. 8 mg, or in short, 125 seeds weigh 1 g. The latter value is in excellent agreement with that quoted by Nieser (30).

Jimsonweed Seed Survey in Soybeans. As cited in the Introduction, control of jimsonweed was difficult during 1973 and led to a nationwide survey conducted by the U.S. Federal Grain Inspection Service. The material passing through 8/64, 9/64, or 10/64 by 3/4 in. slotted sieves was examined for jimsonweed seed and identified by comparison to authentic jimsonweed seeds. The results of this survey are summarized in Table III.

Of the total of 274 samples broken down as truck and rail car, export, and other, 167 contained at least 1 jimsonweed seed, with an average of about 28 seeds per sample. It is interesting to note that exported soybeans (which elicited complaints from foreign importers) actually contained on an average fewer jimsonweed seeds than domestic shipments, yet the incidence of positive contamination was about the same (51 samples vs. 50 and 66). The seeds are very light materials. On an average, 28.4 jimsonweed seeds weigh ca. 215 mg and only amount to about 0.022% by weight (0.215/1000 x 100). Neiser (30), in discussing "Facts of Interest on Grain Impurities," states: "As the imports of U.S. soybeans grew in the years 1948 to 1957, analysis of a great many samples showed an average content of 0.02% and a maximum of 0.27% thornapple seeds." Thus, the results of the 1973 survey are in good agreement with these findings.

Fate of Jimsonweed Alkaloids in Processing of Soybeans. Jimsonweed seeds are small compared to soybeans and are easily separated by mechanical screening at the elevator. Furthermore, first-hand observations at domestic oil mills indicate that jimsonweed contamination is extremely unlikely because of the rigorous screening of dirt and small stones required to avoid damage to cracking and flaking rolls. Yet the question remained: "If jimsonweed should get into soybeans headed for the cracking rolls, what happens to the alkaloids - do they stay with the meal or are they carried along with the crude oil; if they end up in the oil, are they removed by caustic refining?" To answer these questions, we conducted some preliminary studies (6). Whole jimsonweed seed (10 g {0.31% atropine, 0.11% scopolamine}, sample 8, Table II) was ground finely in a Wiley mill and mixed with an equal portion of flaked soybeans. After extraction with petroleum ether in a Soxhlet, the crude oil and meal were separated into fractions weighing 4.68 and 14.43 g, respectively. Upon extraction, 10 g of jimsonweed seeds theoretically would yield 42 mg of total alkaloids (31 mg atropine; 11 mg scopolamine) for distribution within the crude oil and meal fractions. However, analysis of the crude meal showed that 37.4 mg of alkaloids (28.2 mg atropine, 9.2 mg scopolamine) was retained in the meal. It is estimated that the error associated with the alkaloid analysis amounts to ± 2 mg. Thus, the recovery of alkaloids amounted to ca. 90%. The crude oil contained only 0.04 mg of atropine and a trace of scopol-

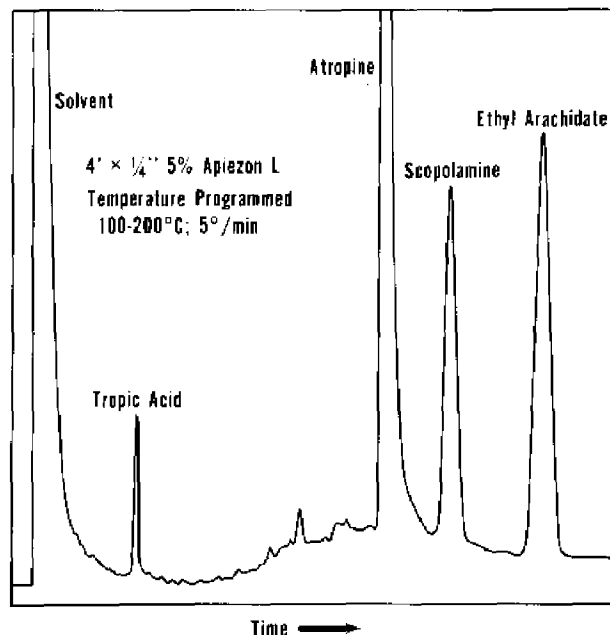


FIG. 1. GLC separation of atropine and scopolamine as trimethylsilyl (TMS) ethers.

amine.

To study the effect of further processing on removal of alkaloids from crude oil, a commercially extracted crude soybean oil was spiked with atropine (3 mg), refined with 14.4% Be' lye 0.5% excess, washed with water, and dried. Analysis showed that only 0.2 mg of atropine remained in the refined oil. Thus, over 93% of the added atropine was removed by caustic refining. Lack of a reliable sample of scopolamine prevented similar studies with this alkaloid, but there is no reason to believe it would not behave in a like fashion. The fact that atropine is readily removed by caustic refining is not surprising, because atropine and scopolamine are known to readily undergo hydrolysis in the presence of a base (31,32). Apparently, the hydrolysis products tropic acid and tropine (32) are more soluble in water than in oil and are thus removed during caustic refining and washing. No attempts were made to analyze for tropine. However, the lack of toxicity data on it would indicate that it may be relatively nontoxic. From preliminary studies, it may be concluded that (1) the jimsonweed alkaloids will be retained in the meal fraction during processing, (b) alkali refining will remove virtually all the alkaloids, and (c) any toxicity problems with jimsonweed-

TABLE II
Analytical Data of Jimsonweed Seeds

Sample	Wt/1000 seeds, g ^a	Percent alkaloids		
		Atropine	Scopolamine	Total
1	8.0	0.25	0.08	0.33
2	6.6	0.29	0.02	0.31
3	9.0	0.35	0.06	0.41
4	8.6	0.20	0.02	0.22
5	6.7	0.43	0.05	0.48
6	6.5	0.19	0.05	0.24
7	6.3	0.27	0.06	0.33
8	8.2	0.31	0.11	0.42
9	9.0	0.31	0.05	0.36
10	7.1	0.24	0.02	0.26
11	7.6	0.37	0.03	0.40
Mean	7.6	0.29	0.05	0.34

^aOne seed weighs ca. 8 mg, or 125 seeds weigh 1 g.

TABLE III

1973 Nationwide Survey of Jimsonweed Seed in Soybean Crop				
Method of movement	No. of samples ^a	Samples with jimsonweed seeds		Avg. no. jimsonweed seeds per sample ^b
Truck and rail car	100	50		30.7
Exports	77	51		22.6
Other	97	66		30.6
Total	274	Total	167	Avg. 28.4

^a1000-g samples.

^b28.4 Jimsonweed seeds weigh ca. 0.215 g.

contaminated soybeans will be encountered in the meal.

Obviously, much work remains to be done on the fate of jimsonweed alkaloids in soybean processing. For example, modern soybean processing entails toasting of the defatted flakes as a key step in producing high-quality protein products intended for animal and human food. Future studies should be directed toward ascertaining the effects of the toasting process on the toxicity of jimsonweed seed alkaloids. Other areas for study include: (a) determine fate and toxicity of hydrolysis products from atropine and scopolamine, and (b) investigate the fate of atropine and scopolamine N-oxides in soy processing. These compounds have recently been isolated from *Datura* seed (33).

REFERENCES

- Kingsbury, J.M., *J. Dairy Sci.* 41:875 (1958).
- Wennblom, R.D., *Farm J.* 97:26 (1973).
- Hutchins, R.P., *JAOCs* 45:624A (1968).
- Official Grain Standards of the United States Revised to 1970, USDA Consumer and Marketing Service, Grain Division, Washington, DC.
- Hutchins, R.P., *Oil Soap* 22:165 (1945).
- List, G.R., and G.F. Spencer, *JAOCs* 53:535 (1976).
- Kingsbury, J.M., "Poisonous Plants of the United States and Canada," Prentice Hall, New Jersey, 1964.
- Muenschler, W.C., "Poisonous Plants of the United States," The McMillan Co., New York, 1951.
- Evers, R.A., and R. Plink, "Poisonous Plants of the Midwest and Their Effects on Livestock," Special Publication 24, College of Agriculture, University of Illinois, Champaign, IL, 1972.
- Morton, J.F., "Plants Poisonous to People in Florida and Other Warm Areas," Hurricane House Publishers, Miami, FL, 1971.
- Radeleff, R.D., "Veterinary Toxicology," Lea and Febiger, Philadelphia, PA, 1964.
- Garner, R.J., "Veterinary Toxicology," Bailliere, Tindall and Cox, London, 1957.
- Bailey, L.H., and E.Z. Bailey, "Hortus Third, A Concise Dictionary of Plants Cultivated in the United States and Canada," MacMillan Co., New York, 1976.
- The Merck Index, 9th Edition, The Merck Co., Rahway, NJ (1976).
- Kuzel, N.R., and C.E. Miller, *J. Am. Pharm. Assoc.* 39:202 (1950).
- Forrest, G.P., *J. Am. Vet. Med. Assoc.* 93:42 (1938).
- Kingsley, A.T., *Vet. Med.* 17:282 (1922).
- Neal, W.M., L.L. Rusoff, and C.F. Ahman, *J. Am. Chem. Soc.* 57:2560 (1935).
- Adams, R., and E.F. Rogers, *ibid.* 61:2815 (1939).
- Becker, R.B., W.M. Neal, and P.T. Dix, *Florida Agric. Exp. Sta. Bull.* 361:33 (1941).
- Tumlin, J.T., *Southeast. Vet.* 10:60 (1959).
- Steyn, D.G., *Onderstepoort J. Vet. Sci. Anim. Ind.* 1:219 (1933).
- Taber, W.A., L.C. Vining, and R.A. Heacock, *Phytochemistry* 2:65 (1963).
- Dreisbach, R.H., "Handbook of Poisoning: Diagnosis and Treatments, Lange Medical Publications," Los Altos, CA, 1971, pp. 421-437.
- Mitchell, J.E., and F.N. Mitchell, *J. Pediatr.* 47:227 (1955).
- McNally, W.D., *J. Am. Med. Assoc.* 65:1640 (1915).
- Mateescu, E., and S. Bercovici, *Commun. Acad. Rep. Populace Ronume* 11:559 (1961) *CA* 56:5123f.
- Clarke, E.G.C., "The Alkaloids Chemistry and Physiology," Vol. XII, Academic Press, New York, 1970, pp. 513-589.
- Nieminen, E., *Farm. Aikak.* 80:263 (1971).
- Nieser, O., *Die Muehle* 101(5):30 (1964).
- Polesuk, J., and T.S. Ma, *Microchimica Acta (Wien)* 393 (1973).
- Holmes, H.L., "The Alkaloids Chemistry and Physiology," Vol. I, Academic Press, New York, 1950, pp. 271-374.
- Phillipson, J.D., and S.S. Handa, *Phytochemistry* 14:999 (1975).

[Received October 5, 1978]