# Chapter 115

# JEQUIRITY BEAN and ABRIN (*Abrus precatorius* L.)

## HISTORY

In India, there is a long history of the use of abrin from the jequirity bean plant to poison animals and humans.[1] Administration of small doses of seeds (beans) from *Abrus precatorius* to protect animals from abrin poisoning is one of the earliest examples of active immunization.[2] The seeds from *Abrus precatorius* are called gumchi in Hindustani and gunja in Sanskrit. The physical properties (e.g., uniformity, durability) of these seeds were used in Southeast Asia for weighing jewels and gold. An old system of weights called the Ganda system was based on multiples of the weight of four *A. precatorius* seeds.[2] In addition, jequirity beans were used by some African and Madagascar tribes as an ordeal poison, but the delayed toxicity and the development of immunity limited the effectiveness of the these seeds as an ordeal poison. In Arab countries, jequirity beans (coq's eye) were an aphrodisiac. Extracts of these seeds were used to treat chronic eye diseases during the 19th century, particularly trachoma.[3] In India and Sri Lanka during the early 20th century, abrin was used as a homicidal agent by stabbing the victim with a hardened, needle-shaped abrin paste.[21]

## BOTANICAL DESCRIPTION

**Common Name:** Jequirity Bean, Rosary Pea, Buddhist Rosary Bead, Indian Bead, Corral Peas, Paternoster Beans, Lucky Beans, Minnie-Minnies, Seminole Bead, Prayer Bead, Crab's Eye, Weather Plant, Ojo de Pajaro, Pois Rouge, Tento Muido

**Scientific Name:** *Abrus precatorius* L.

**Botanical Family:** Fabaceae (pea)

**Physical Description:** Small (1 cm/~0.4 in.) alternate compound leaflets develop on a twisting vine that grows up to 20 ft (6 m) in length. The bright scarlet seeds are 3–8 mm (0.1–0.3 in.) long and ovoid with a jet black end. The three color variations of these seeds include a red seed with a black eye (Indian native), a black seed with a white eye, and a white seed with a black eye.

**Distribution and Ecology:** *Abrus precatorius* is a vine used as an ornamental plant in tropical regions, such as southern Florida and the Florida Keys. *Abrus precatorius* also inhabits tropical and subtropical climates in Southeast Asia, India, Hawaii, Virgin Islands, and Puerto Rico.

## EXPOSURE

Traditional herbal uses of *Abrus precatorius* include an anticonvulsant, insecticide, and the treatment of corneal opacities and trachoma by oculists.[4,5] In the Andaman Islands of India, aborigines eat boiled jequirity beans during extreme famines.[6] The jequirity bean is also a decorative bead in jewelry (e.g., necklace), shoes, and rosaries. Potential uses of abrin as a chemical weapon include aerosolization as a dry powder or liquid droplets and/or the contamination of food and water sources.[9] However, to date there is no documentation of the use of abrin as an aerosolized weapon.

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## PRINCIPAL TOXINS

### Physiochemical Properties

Constituents of the jequirity bean include *N*-methyl tryptophan, abric acid, glycyrrhizin (the active principle of licorice), a lipolytic enzyme, a heterodimeric glycoprotein (abrin), and a heterotetrameric glycoprotein (*Abrus* agglutinin-I). The latter two structurally similar substances are class II ribosome-inactivating proteins (ribotoxin). However, *Abrus* agglutinin-I is significantly less toxic than abrin, probably as a result of a difference in the secondary structural elements in chain A.[7] Like ricin, abrin has two dissimilar, disulfide-linked polypeptide chains composed of a lectin with two D-galactose moiety-binding sites (B chain) and an RNA-specific *N*-glycosidase that inhibits protein synthesis (A chain).[8] The A chain contains 251 residues divided into three folding domains with a molecular weight of approximately 30 kDa.[9] The B chains share the same 256 residues in addition to 12 other amino acids. There are at least three variants of abrin within *Abrus precatorius* species that differ in toxicity, binding ability, and lag period.[10] The toxic constituents of the jequirity bean are heat-labile and water soluble.[11] A thorough boiling removes these toxic ingredients.[6] The abrin compounds consist of four isolectins (A to D), which are monovalent compounds with molecular weights ranging from 63,000 to 67,000 Da. However, ricin and abrin are not identical disulfide-linked polypeptide chains. Based on minimal lethal intravenous doses to mice, abrin (0.7 µg/kg) is approximately four times more potent than ricin (2.7 µg/kg).[12]

### Poisonous Parts

The seeds of the jequirity bean plant (*Abrus precatorius*) contain four toxic lectins (i.e., abrin compounds) that bind to carbohydrates containing terminal nonreducing galactose residues.[13]

## DOSE RESPONSE

There is a marked variation between animal species and abrin toxicity with an intravenous minimal lethal dose (MLD) in mice and rabbits of 0.7 µg/kg and 0.06 µg/kg, respectively.[14] Predicting the fatal oral dose of jequirity beans is difficult because of the lack of data on the bioavailability of abrin in jequirity beans. A 20-year-old male died about 4 days after the ingestion of part of a mixture containing 20 pulverized jequirity beans.[15] The patient did not seek medical treatment until one day before his death, and the case report did not include a detailed case history or results of any laboratory analyses (e.g., drug screens) except the hematocrit.

### Mechanism of Toxicity

Most cases of human poisoning after the ingestion of jequirity beans involve gastrointestinal toxicity. These clinical features are consistent with abrin-induced damage to vascular endothelial cells, interstitial edema, and extravasation of fluids and proteins similar to the vascular leak syndrome associated with ricin toxicity.[16] The general structure of abrin compounds is similar to ricin with two polypeptide chains (A chain and B chain). At the cellular level, abrin inhibits protein synthesis and causes cell death. Like ricin, the A chain inactivates the 60 S ribosomal subunits enzymatically after the B chain attaches the A chain to cell surface receptors. *In vitro* studies indicate that the B chain of each abrin molecule rapidly binds to nonreducing β-galactosyl residues of cell surface glycoproteins, particularly to mannose receptors on cells of the reticuloendothelial system.[17] Following the entry of the A chain into the cell, this *N*-glycosidase cleaves adenine from positions 4 and 324 from a loop on the 28S rRNA.[18] The result is the inhibition of protein synthesis after a 30-minute delay and subsequent cell death.[19] The roots of *Abrus precatorius* also contain glycyrrhizin, the active ingredient in licorice; therefore, the clinical syndrome of hyperaldosteronism characterized by sodium retention and hypertension potentially may develop following large doses of roots. However, there are no data to confirm this adverse affect in humans.

Like ricin, the pathology associated with inhalation of abrin is limited to the lungs with little evidence of significant systemic toxicity. The target cell for inhaled abrin and ricin is the type I pneumocyte. Animal studies indicate that abrin binds to cell surface receptors on type I pneumocytes and initiates acute alveolitis and necrosis of the lower respiratory tract epithelium.[8] A rapidly progressive pulmonary edema develops that produces severe hypoxia and death in exposed animals. Proliferation of type II pneumocytes occurs in response to the injury during the resolution phase of the pulmonary damage.

## TOXICOKINETICS

There are few data on the toxicokinetics of abrin. The hard coat surrounding the jequirity bean limits the gastrointestinal absorption of abrin. Release of abrin from jequirity beans requires chewing or grinding of the bean prior to ingestion, and nonmasticated seeds pass through the gastrointestinal tract without causing toxicity. The high molecular weight (i.e., about 65 kDa) of abrin also limits the gastrointestinal absorption of this toxin. Based on animal studies, elimination of abrin probably occurs by the renal excretion of metabolites.[20]

## CLINICAL RESPONSE

The ingestion of jequirity beans frequently involves children, who are attracted to the bright colors of the seeds. These ingestions are usually asymptomatic because the whole bean passes through the gastrointestinal tract without absorption of abrin from the bean.[21] Serious abrin ingestion produces a severe gastroenteritis several hours after consumption, followed by the development of bloody diarrhea. Delayed symptoms do not usually occur during abrin intoxication; however, rare case reports associated the development of cerebral edema, altered sensorium, and seizures 4–6 days after ingesting 7–10 crushed jequirity beans.[22] One of these patients died, but the cause of death was unclear because of the lack of imaging studies or an autopsy. The latter case is typical of fatalities associated with exposure to jequirity beans. These case reports lack sufficient clinical details to determine the cause and mechanism of death. Jequirity beans are highly antigenic and exposure to material in these beans may produce allergic or anaphylactic responses, particularly in atopic patients.

## DIAGNOSTIC TESTING

Older analytical techniques include the detection of aqueous extracts of *A. precatorius* by characteristic ultraviolet absorption on spectrophotometry.[23] Radioimmunoassays have limits of detection near 50–100 pg abrin/mL, and there is little cross-reactivity of abrin with ricin on radioimmunoassay.[24] In animal experiments, sublethal parenteral doses of abrin cause a leukocytosis and mild to moderate elevation of serum hepatic aminotransferases.[25] There are few data on the laboratory abnormalities in humans following the ingestion of jequirity beans.

## TREATMENT

The treatment of jequirity bean poisoning is supportive.[26] Decontamination measures are usually unnecessary after the ingestion of whole seeds. The administration of activated charcoal is a therapeutic option for patients, who present to a health care facility within one hour of the ingestion of well-masticated seed, but there are no clinical data to determine the efficacy of decontamination measures including activated charcoal in this clinical setting. There are no commercial antidotes or efficacious methods to enhance the elimination of abrin. Merck and Company (Whitehouse Station, NJ) developed an antiserum in large animals (Antiabrin® or Jequiritol®) to control abrin-induced ocular inflammations (i.e., secondary to trachoma treatment), but no clinical trials have ever been conducted in humans. Consequently, there is no commercial source of the antiserum. Careful fluid and electrolyte replacement is the most important aspect of management. In general, supportive care is similar to treatment for ricin (castor bean) poisoning. Asymptomatic children may be observed at home; symptomatic patients should be hospitalized. Gram-negative sepsis from the passage of intestinal bacteria across damaged intestinal mucosa should be considered when a patient with serious abrin ingestion develops fever and hypotension.

## References


1. Stirling RF. Poisoning by gumchi or suis (needles). Vet J 1924;80:473–484.
2. Olsnes S. The history of ricin, abrin and related toxins. Toxicon 2004;44:361–370.
3. Martin S. The proteins of the seeds of *Abrus precatorius*. Proc Roy Soc 1887;42:3331–3333.
4. Moshi MJ, Kagashe GA, Mbwambo ZH. Plants used to treat epilepsy by Tanzanian traditional healers. J Ethnopharmacol 2005;97:327–336.
5. Molgaard P, Nielsen SB, Rasmussen DE, Drummond RB, Makaza N, Andreassen J. Anthelmintic screening of Zimbabwean plants traditionally used against schistosomiasis. J Ethnopharmacol 2001;74:257–264.
6. Rajaram N, Janardhanan K. The chemical composition and nutritional potential of the tribal pulse, *Abrus precatorius* L. Plant Foods Human Nutr 1992;42:285–290.
7. Bagaria A, Surendranath K, Ramagopal UA, Ramakumar S, Karande AA. Structure-function analysis and insights into the reduced toxicity of *Abrus precatorius* agglutinin I in relation to abrin. J Biol Chem 2006;281:34465–34474.
8. Griffiths GD, Rice P, Allenby AC, Bailey SC, Upshall DG. Inhalation toxicology and histopathology of ricin and abrin toxins. Inhal Toxicol 1995;7:269–288.
9. Dickers KJ, Bradberry SM, Rice P, Griffiths GD, Vale JA. Abrin poisoning. Toxicol Rev 2003;22:137–142.
10. Hedge R, Podder SK. Studies on the variants of the protein toxins ricin and abrin. Eur J Biochem 1992;204:155–164.
11. Niyogi SK, Rieders F. Toxicity studies with fractions from *Abrus precatorius* seed kernels. Toxicon 1969;7:211–216.
12. Gill DM. Bacterial toxins: a table of lethal amounts. Microbiol Rev 1982;46:86–94.
13. Wu AM, Wu JH, Herp A, Chow L-P, Lin J-Y. Carbohydrate specificity of a toxic lectin, abrin A, from the seeds of *Abrus precatorius* (jequirity bean). Life Sci 2001;69: 2027–2038.
14. Fodstad O, Johannessen JV, Schjerven L, Pihl A. Toxicity of abrin and ricin in mice and dogs. J Toxicol Environ Health 1979;5:1073–1084.
15. Davis JH. *Abrus precatorius* (rosary pea) the most common lethal plant poison. J Florida Med Assoc 1978;65: 188–191.

16. Baluna R, Rizo J, Gordon BE, Ghetie V, Vitetta ES. Evidence for a structural motif in toxins and interleukin-2 that may be responsible for binding to endothelial cells and initiating vascular leak syndrome. Proc Natl Acad Sci U S A 1999;96:3957–3962.
17. Tsuzuki J, Wu HC. Temporal behavior of abrin in the intoxication of Chinese hamster cells (line CHO). J Cell Physiol 1982;113:94–98.
18. Liu C-L, Tsai C-C, Lin S-C, Wang L-I, Hsu C-I, Hwang M-J, Lin J-Y. Primary structure and function analysis of the *Abrus precatorius* agglutinin A chain by site-directed mutagenesis. J Biol Chem 2000;275:1897–1901.
19. Benson S, Olsnes S, Pihl A, Skorve J, Abraham AK. On the mechanism of protein synthesis inhibition of abrin and ricin. Inhibition of the GTP hydrolysis site on the 60 S ribosomal subunit. Eur J Biochem 1975;59:573–580.
20. Fodstad O, Olsnes S, Pihl A. Toxicity, distribution and elimination of the cancerostatic lectins abrin and ricin after parenteral injection into mice. Br J Cancer 1976;34: 418–425.
21. Hart M. Hazards to health jequirity-bean poisoning. N Engl J Med 1963;268:885–886.
22. Subrahmanyan D, Mathew J, Raj M. An unusual manifestation of *Abrus precatorius* poisoning: a report of two cases. Clin Toxicol (Phila) 2008;46:173–175.
23. Niyogi SK. The toxicology of the *Abrus precatorius* Linnaeus. J Forensic Sci 1970;15:529–536.
24. Godal A, Olsnes S, Pihi A. Radioimmunoassays of abrin and ricin in blood. J Toxicol Environ Health 1981;8: 409–417.
25. Niyogi SK. Elevation of enzyme levels in serum due to *Abrus precatorius* (jequirity bean) poisoning. Toxicon 1977;15:577–580.
26. Kinamore PA, Jaeger RW, deCastro FJ. Abrus and ricinus ingestion: management of three cases. Clin Toxicol 1980; 17:401–405.