

TOP 10 POISONS AFFECTING DOGS & CATS

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INTRODUCTION

Each year, the [ASPCA Animal Poison Control Center](#) (APCC) manages hundreds of thousands of poisoning calls. At the ASPCA APCC, an estimated 50% of pet poisonings comprise human over-the-counter (OTC) and prescription medications. In this lecture, we will review the mechanism of toxicosis, clinical signs, and overall treatment of the top 10 most common poisons affecting dogs and cats. In the veterinary poisoned patient, the goal of decontamination is to “inhibit or minimize further toxicant absorption and to promote excretion or elimination of the toxicant from the body.”^{1,2} When treating the poisoned patient, the clinician should have an understanding of the toxic dose (if available), the pharmacokinetics (including absorption, distribution, metabolism, and excretion), the underlying mechanism of action, and the potential clinical signs that can be observed with the toxicant.² This will help determine appropriate decontamination and therapy for the patient. If this information is not readily available, the reader is advised to contact the [ASPCA Animal Poison Control Center](#) (888-426-4435) for life saving, 24/7 advice as needed. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

CALCIUM CHANNEL BLOCKERS, BETA-BLOCKERS, ACE-INHIBITORS, STATINS AND DIURETICS

Certain cardiac medications include broad categories such as calcium channel blockers (CCB), beta-blockers (BB), and angiotensin-converting enzyme (or “ACE”) inhibitors. These medications are commonly used in both human and veterinary medicine to treat underlying cardiac disease or hypertension. Each category of cardiac medication has different margins of safety. CCB and BB toxicosis should be treated aggressively, as these two categories of medications have a narrow margin of safety. Toxicosis of these agents can result in myocardial failure, severe bradycardia, and hypotension; untreated, cardiac output becomes reduced, and secondary severe hypoperfusion and acute kidney injury (AKI) can potentially develop.³⁻⁵ With ACE-inhibitors, severe overdoses can cause hypotension, dizziness, weakness, and hypotension. In general, there is a wider margin of safety with ACE-inhibitors, which are typically considered much safer. Pets ingesting small amounts of ACE-inhibitors can potentially be monitored at home, unless they have underlying disease (e.g., kidney failure, cardiac disease, etc.). With ACE-inhibitors, ingestions > 10-20X a therapeutic dose are generally considered toxic, and can result in severe clinical symptoms (e.g., hypotension).⁵ Treatment for any cardiac medication includes decontamination (e.g., emesis induction, gastric lavage, activated charcoal (AC) administration), blood pressure monitoring, aggressive IV fluid therapy if hypotension is detected, and blood work monitoring. With severe toxicosis, the use of high-dose insulin therapy or intravenous lipid emulsion may be warranted as a potential antidote for calcium channel blocker toxicosis.³

SELECTIVE SEROTONIN RE-UP TAKE INHIBITORS (SSRI)

Selective serotonin re-uptake inhibitors (SSRIs) are a class of medications that are commonly used in human medicine for depression. Common examples include the following drugs:

- Fluoxetine (Prozac[®] in human beings; Reconcile[™] in veterinary medicine)
- Citalopram (Celexa[®])
- Paroxetine (Paxil[®])
- Sertraline (Zoloft[®])

Other similar drugs include selective norepinephrine re-uptake inhibitors (SNRIs), which include common drugs like duloxetine (Cymbalta[®]), nefazodone (Serzone[®]), and venlafaxine (Effexor[®]). SNRI and SSRI drugs result in similar clinical signs of toxicosis, and therefore are treated the same. In veterinary medicine, SSRIs are used for a wide array of behavioral problems, including feline urine spraying, canine separation anxiety, lick granulomas, etc. These SSRI drugs work by blocking the reuptake of serotonin in the pre-synapse, thereby increasing the levels of serotonin in the pre-synaptic membrane. In small animal patients, common clinical signs from SSRIs include the following:

- Sedation or central nervous system (CNS) stimulation
- Anorexia
- Lethargy
- Serotonin syndrome

Clinical signs of serotonin syndrome include: gastrointestinal (GI) signs (e.g., hypersalivation, vomiting, diarrhea, abdominal pain) and CNS signs (e.g., stimulation, mydriasis, tremors, seizures, hyperthermia secondary to tremoring and seizuring). Treatment for antidepressant toxicosis includes decontamination (ideally done at a veterinarian, due to the rapid onset of clinical signs), sedation (e.g., with acepromazine or chlorpromazine), intravenous (IV) fluid therapy, blood pressure and electrocardiogram (ECG) monitoring, thermoregulation, muscle relaxants (for tremors; methocarbamol 22-55 mg/kg, IV, PRN), anticonvulsants (e.g., phenobarbital 4-16 mg/kg, IV, PRN; diazepam 0.25-0.5 mg/kg, IV, PRN), serotonin antagonists [e.g., cyproheptadine (1.1 mg/kg for dogs or 2-4 mg *total* per cat) PO or rectally q. 6-8], and supportive and symptomatic care. In general, the prognosis for antidepressant toxicosis is excellent.

AMPHETAMINES

Amphetamines are used for a variety of medical and illicit reasons. Legal forms include prescription medications for attention-deficit disorder/attention deficit-hyperactivity disorder (ADD/ADHD), weight loss, and narcolepsy. Examples of amphetamines include:

- Dextroamphetamine
- Amphetamine (Adderall[®])
- D-amphetamine (Dexedrine[®])
- Methamphetamine (Desoxyn[®])
- Lisdexamfetamine (Vyvanse[®])

Illegal forms of amphetamines include street drugs like methamphetamine, crystal meth, and ecstasy. This class of drugs acts as sympathomimetic agents, meaning they stimulate the sympathetic system. Amphetamines also cause stimulation of α and β -adrenergic receptors, and stimulate release of serotonin and norepinephrine; this results in increased catecholamine stimulation in the synapse. Amphetamines also increase release of serotonin from the presynaptic membrane, resulting in serotonin syndrome. With amphetamine toxicosis, secondary stimulation of certain body systems can result in significant clinical signs: GI (e.g., vomiting, diarrhea, hypersalivating), CNS (e.g., agitation, mydriasis, tremors, seizures), cardiovascular (e.g., tachycardia, hypertension), and respiratory (e.g., panting). Both clinical signs and treatment for amphetamine toxicosis are similar to SSRI toxicosis, and include IV fluids, cooling measures, sedation (e.g., with acepromazine or chlorpromazine), muscle relaxants, anticonvulsants, thermoregulation, blood pressure monitoring, and symptomatic/supportive care.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

NSAIDs are competitive inhibitors of prostaglandin synthesis (cyclooxygenase or “COX” inhibitors) and result in decreased prostaglandin, which is important for normal homeostatic function (including maintaining renal blood flow, maintaining mucous production in the stomach, etc.). Common OTC human NSAIDs include active ingredients such as ibuprofen and naproxen sodium. Examples of human NSAIDs include Advil[®], Aleve[®], certain types of Motrin[®], etc. Common prescription veterinary NSAIDs can also result in toxicosis, particularly when available in the chewable, palatable formulation. Examples of veterinary NSAIDs include carprofen, deracoxib, etogesic, previcoxib, etc. With NSAID toxicosis, the GI tract, kidneys, CNS, and platelets can be affected. Cats and certain breeds of dogs (e.g., German shepherds) seem to be more sensitive to NSAIDs, and should be treated aggressively. With cats, severe acute kidney injury (AKI) is often more clinically seen with NSAID toxicosis at lower doses (as compared to dogs). With dogs, signs secondary to GI ulceration (e.g., vomiting, diarrhea, melena, hematemesis, etc.) are more commonly seen initially, followed by secondary AKI. With NSAID toxicosis, it is important to keep in mind that each NSAID has a different toxic dose, margin of safety, half-life, and route of excretion, and an animal poison control should be contacted to identify what specific NSAID and toxic dose was ingested. For example, in dogs, ibuprofen results in GI signs at doses as low as 16-50 mg/kg, while severe GI signs may be seen at 50-100 mg/kg.⁶ Renal compromise may be seen at doses of 100-250 mg/kg (resulting in potential AKI), and fatalities have been reported at doses > 300 mg/kg.⁶ This differs tremendously from naproxen sodium (dogs), where severe clinical signs can be seen at doses as low as 5 mg/kg.⁶

Clinical signs of NSAID toxicosis include anorexia, vomiting, hematemesis, diarrhea, melena, abdominal pain, lethargy, malaise, uremic halitosis, dehydration, etc. Treatment includes decontamination, the use of activated charcoal (often multiple doses due to enterohepatic recirculation, if appropriate), GI protectants (e.g., H₂ blockers, sucralfate), aggressive IV fluid therapy (to help maintain renal blood flow), anti-emetic therapy, and symptomatic and supportive care. With high doses, anti-convulsants may also be necessary if CNS signs develop.

PYRETHRINS AND PYRETHROIDS

Pyrethrins and their synthetic derivative, pyrethroids, are commonly found in household insect sprays and insecticides (e.g., permethrin, cypermethrin, cyphenothrin, etc.). Due to a cat's altered

liver glucuronidation metabolism, cats are significantly more sensitive to pyrethrins than dogs. While a precise toxic dose for cats is not well established, products containing greater than a 5-10% concentration of pyrethrins may lead to systemic toxicosis. The diluted amount found in household insect sprays and topical flea sprays and shampoos is typically < 1%. Toxicosis from exposure to these products is highly unlikely. The application of canine spot-on pyrethrin/pyrethroid based insecticides (typically ~40-50% concentration) to cats is the primary cause of feline pyrethrin toxicosis. Cats that groom dogs following recent spot-on applications are also at high risk for toxicosis; ideally, pets should be separated until the spot-on product has completely dried on the dog to prevent cat exposure. Signs of systemic toxicosis in cats include GI signs (e.g., hypersalivation, vomiting, nausea), neurologic signs (e.g., disorientation, weakness, hyperexcitability, tremors, seizures) and respiratory signs (e.g., tachypnea, dyspnea). Tremors are extremely responsive to methocarbamol (22-220 mg/kg, IV PRN to effect), a centrally acting muscle relaxant, although oral absorption of methocarbamol is often slower in onset of action. In general, tremors are less responsive to benzodiazepines (e.g., diazepam). Seizures may be controlled with Phenobarbital (e.g., 4-16 mg/kg, IV PRN to effect) or general gas anesthesia. Dermal decontamination is crucial but should be performed after stabilization. This should be performed with a liquid dish detergent (e.g., Dawn, Palmolive). Supportive care including the monitoring and maintenance of hydration, body temperature and blood glucose levels are necessary. Signs may persist for 1-4 days, depending on the animal. The prognosis is excellent with aggressive dermal decontamination and treatment.

INSECT BAIT STATIONS

Household ant and roach bait stations are rarely toxic, as the active ingredient is often a low-concentration of abamectin (a macrocyclic lactone derivative in the same family as ivermectin). Certain breeds with the MDR-1 gene mutation (now known as the ABCB1-1Δ polymorphism), including collies, Border collies, old English sheepdogs, and collie-mixed breed dogs, may be more at risk when large amounts of bait stations are ingested. Typically, the plastic on the bait station is more of a problem, as it can result in GI signs or potentially foreign body obstruction (FBO), when ingested in large amounts.

HOUSEHOLD CLEANERS

Most surface cleaners are generally benign, and when ingested directly from the bottle, can result in minor GI signs. However, certain concentrated cleaners can be highly toxic or corrosive. Household bleach is a GI irritant, but “ultra” bleach can be corrosive, resulting in severe esophageal or upper GI damage. Concentrated lye products, toilet bowl cleaners, and oven cleaners are also corrosive, and immediate flushing out the mouth for 10-15 minutes should be performed prior to veterinary visit to minimize tissue injury. Appropriate pet-proofing (such as keeping toilet seats down or securing cleaners in a locked or elevated bathroom cabinet) are the easiest way to prevent this specific toxicosis.

GRAPES, RAISINS, AND CURRANTS

Grapes and raisins (*Vitis* spp) have been recently associated with development of acute renal failure (ARF) with ingestion. All types have been implemented with toxicosis, including organic grapes, commercial grapes, homegrown grapes, and seedless or seeded grapes. While the mechanism of toxicosis is unknown, there are several suspected hypotheses, including individual inability to metabolize certain components of the fruit (e.g., tannins, high monosaccharide

content),⁸ the presence of mycotoxins or pesticide residues on the fruit,⁸ or salicylate-like chemicals within the grape or raisin. Common kitchen items also contain grapes, raisins, or currants in their active ingredient, including raisin bread, trail mix, chocolate-covered raisins, cereal with raisins, etc. Currently, grapeseed extract has not been associated with nephrotoxicity.⁸ Treatment for grape and raisin ingestion includes aggressive decontamination as the first-line of therapy. Grapes and raisins seem to stay in the stomach for a prolonged period of time, and are not rapidly broken down or absorbed from the gastrointestinal (GI) tract; hence, delayed emesis induction even several hours post-ingestion can still be initiated to maximize decontamination methods. One dose of activated charcoal can also be administered to prevent absorption of the unknown nephrotoxin. In general, all ingestions should be treated as potentially idiosyncratic and be appropriately decontaminated and treated. Initially, vomiting may be observed within the first 24 hours of ingestion.⁸ Within the next 12-24 hours, clinical signs of lethargy, dehydration, vomiting, diarrhea, anorexia, abdominal pain, uremic breath, and diarrhea may be seen.⁸ Azotemia may develop within 24 hours, with hypercalcemia and hyperphosphatemia occurring first.⁸ Oliguria and anuria may develop 48-72 hours post-ingestion,⁸ at which point the prognosis is poorer. Treatment includes decontamination, aggressive intravenous (IV) fluid therapy, anti-emetics, blood pressure and urine output monitoring, and serial blood work monitoring (q. 12-24 hours for several days). In severe cases, hemodialysis or peritoneal dialysis may be necessary. Asymptomatic patients that have been adequately decontaminated and survive to discharge should have a renal panel and electrolytes monitored 48-72 hours post-ingestion. Overall, the prognosis varies from good to poor, depending on time to decontamination, response to therapy, and prevalence of oliguria or anuria. While 50% of dogs that ingest grapes and raisins never develop clinical signs or azotemia, aggressive treatment is still warranted.⁸

XYLITOL

Xylitol is a natural sweetener found in small quantities in certain fruit. Xylitol has gained recent popularity because it is sugar-free, and is often found in diabetic snacks, foods, baked foods, mouthwashes, toothpastes, chewing gum, mints, candies, and chewable multivitamins.⁹ Sugarless products, particularly those with xylitol listed within the first 3 to 5 active ingredients (AI), can result in severe toxicosis within 15-30 minutes of ingestion. Ingestion of xylitol results in an insulin spike in non-primate species, resulting in severe hypoglycemia. Many pieces of candy and gum (e.g., Orbit™, Trident™, Ice Breakers™) contain various amounts of xylitol ranging, on average, from 2 mgs to 1.0 grams/piece. Unfortunately, not all sources are disclosed by the company (e.g., how many grams of xylitol may be in each piece of gum) due to a proprietary nature. With xylitol toxicosis, it is imperative to calculate whether a toxic dose has been ingested. Doses > 0.1 g/kg are considered toxic and result in profound, sudden hypoglycemia from insulin stimulation.⁹ Higher doses (> 0.5 g/kg) of xylitol have been associated with acute hepatic necrosis. Clinical signs of xylitol toxicosis include lethargy, weakness, vomiting, collapse, anorexia, generalized malaise, tremors, and seizures (from hypoglycemia). When hepatotoxic doses are ingested, clinical signs and clinicopathologic findings may include melena, icterus, increased liver enzymes, diarrhea, hypoglycemia, hypocholesterolemia, decreased BUN, hypoalbuminemia, etc.

When presented a patient that has ingested a toxic amount of xylitol, a blood glucose should be checked immediately upon presentation; if hypoglycemic, a bolus of 1 ml/kg of 50% dextrose, diluted with an additional amount of 0.9% NaCl (in a 1:3 ratio) should be given IV over 1-2

minutes. Emesis induction should not be performed until the patient is euglycemic. Keep in mind that activated charcoal does *not* reliably bind to xylitol, and is not routinely recommended for xylitol toxicosis. Hypoglycemic patients should be hospitalized for IV fluid therapy [supplemented with dextrose (2.5 to 5% dextrose, CRI, IV)] for approximately 24 hours, and frequent blood glucose check should be performed every 1-4 hours. For patients ingesting a hepatotoxic amount of xylitol, the use of hepatoprotectants (e.g., SAME), anti-emetics, and supportive care (including frequent liver enzyme monitoring) are warranted.

CHOCOLATE (THEOBROMINE/CAFFINE)

Chocolate is one of the most well-known toxic foods that pet owners are aware of. Chocolate contains methylxanthines such as theobromine and caffeine. Methylxanthines antagonize adenosine receptors and inhibits cellular phosphodiesterases, causing an increase in cAMP. Methylxanthines also stimulate release of catecholamines (e.g., norepinephrine) and cause an increase of calcium entry into cardiac and skeletal muscle, resulting in central nervous system (CNS) stimulation, diuresis, and myocardial contraction. When ingested in toxic doses, clinical signs may include agitation, vomiting, diarrhea, panting, tachycardia, polyuria, hyperthermia, muscle tremors, and seizures. Clinical signs of theobromine toxicosis can be seen at within a few hours, up to 10-12 hours out (as the absorption time is slow). As theobromine has a very long half-life (e.g., 17 hours), treatment may be necessary for 72-96 hours.¹⁰ Toxic doses of theobromine can be seen at:

- > 20 mg/kg: mild signs of agitation and gastrointestinal distress (e.g., vomiting, diarrhea, abdominal pain)
- > 40 mg/kg: moderate signs of cardiotoxicosis can be seen in addition to aforementioned signs (e.g., tachycardia, hypertension)
- > 60 mg/kg: severe signs of neurotoxicosis can be seen in addition to aforementioned signs (e.g., tremors, seizures)
- 250-500 mg/kg: LD₅₀ (for dogs)¹⁰
- 200 mg/kg: LD₅₀ (for cats)¹⁰

Rarer secondary complications may also be seen from chocolate toxicosis, including pancreatitis and secondary aspiration pneumonia. In general, the darker and more bitter the chocolate, the higher the concentration of methylxanthines in the product. For example, a 20 kg dog would need to ingest approximately 14 oz. of milk chocolate, or 4.5 oz. of semi-sweet, or 2 oz. of unsweetened chocolate to cause moderate signs of toxicity (e.g., agitation, tachycardia). As chocolate tends to stay in the stomach for a prolonged period of time, delayed emesis induction (e.g., even several hours after ingestion) - provided the patient is asymptomatic – may help decontaminate the patient, as chocolate tends to remain in the stomach for quite some time. Further decontamination includes the administration of multiple doses of activated charcoal (1-2 g/kg every 4-6 hours X 4 doses), as methylxanthines undergo enterohepatic recirculation. Treatment includes gastrointestinal support (e.g., anti-emetics), supportive care, IV fluid therapy, frequent walks (to prevent reabsorption of methylxanthines from the urine across the bladder wall), sedatives for agitation (e.g., acepromazine, butorphanol), beta-blockers for persistent tachycardia or hypertension (e.g., propranolol), methocarbamol for tremors, and anticonvulsants for seizures, as needed.

CONCLUSION

Pet owners should be appropriately educated on how to pet-proof the house, and be trained on what common human medications can be toxic to pets. Pet owners should also be appropriately educated on crate training to help minimize toxin exposure. When in doubt, the [ASPCA Animal Poison Control Center](#) should be consulted for toxic ingestions that veterinarians are unaware of.



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REFERENCES

1. Peterson ME. Toxicological decontamination. In: Peterson ME, Talcott PA (eds.) *Small Animal Toxicology*, 3rd ed. Elsevier Saunders: Saint Louis, 2013: 73-85.
2. Lee JA. Decontamination and detoxification of the poisoned patient. In: Osweiler GD, Hovda LR, Brutlag AG, Lee JA (eds.) *Blackwell's Five-Minute Veterinary Consult Clinical Companion Small Animal Toxicology*, 1st ed. Wiley-Blackwell: Ames, 2011: 5-19.
3. Syring RS, Engebretsen KM. Calcium channel blockers. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2010, pp. 170-178.
4. Engebretsen KM, Syring RS. Beta-blockers. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2010, pp. 155-163.
5. Adams CM. Angiotensin-converting enzyme (ACE) inhibitors. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2010, pp. 131-135.
6. Syring RS. Human NSAIDs. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2010, pp.292-299.
7. Babski DM, Koenig A. Acetaminophen. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2010, pp. 687-695.
8. Craft EM, Lee JA. Grapes and raisins. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2011. pp. 429-435.
9. Liu TY D, Lee JA. Xylitol. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2011, pp. 470-475.
10. Craft EM, Powell LL. Chocolate and Caffeine. In: Osweiler G, Hovda L, Brutlag A, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*, 1st Ed. Iowa City: Wiley-Blackwell, 2011, pp. 421-428.

NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb's Veterinary Drug Handbook*.

Cannabis poisoning (from plants, leftover matter and edibles) is one of the most common plant poisonings vets see, especially in dogs. Delta nine tetrahydrocannabinol affects cat and dogs' neurological system. Incoordination, tremors, drooling, seizures, possible respiratory problems, depression, coma. Lilies. ABC Everyday helps you navigate life's challenges and choices so you can stay on top of the things that matter to you. More from ABC. We acknowledge Aboriginal and Torres Strait Islander peoples as the First Australians and Traditional Custodians of the lands where we live, learn and work. You are here: Home » Vet Care » Top Cat & Dog Poisons. Previous Next. Top Cat & Dog Poisons. View Larger Image. Common Cat & Dog Poisons. Cats who have been affected need to have the pyrethroid washed off them using soapy water. Some will require sedation and even anaesthesia to keep them from continuous tremors and convulsions. Treatment of pyrethroid poisoning in cats can be time-intensive and expensive. Pyrethroid Poisoning In Dogs. We provide a cat and dog poison hotline as part of our online vet services, just mention that your pet has been poisoned when you fill out your history form and our vets will immediately be able to assist. Depending on the toxicity, your pet may still need to visit a vet for decontamination. Facebook Comments. Top Products. Appliances. Babies & Kids. Most pet owners have heard they should keep dogs away from chocolate and cats away from lilies. But did you know that just a few sticks of sugar-free gum could cause liver failure in your dog? Or that half a tablet of acetaminophen could put your cat into a coma? The same goes for dogs. Each type of rat poison works differently in the body, so treatment after a poisoning will differ. Keep the box after placing rodenticides around the house, Wismer says, so you have the details on hand when you call your vet. Common Symptoms If Your Pet Ingests: Insecticides containing organophosphates, carbamates, pyrethroids, pyrethrins, and permethrins: Vomiting, diarrhea, tremors, convulsions, paralysis, respiratory failure. 15 Common Plants that Are Toxic + Poisonous to Dogs & Cats. Home. Knowledge Center. 15 Common Plants that Are Toxic + Poisonous to Dogs & Cats. Share. If you are aware that your dog or cat has ingested one of these plants, please call your vet immediately. And, the symptoms we list are only those that are likely, others are certainly possible. 1. Aloe. Signs of poisoning will include labored breathing, eye discharge and digestive discomfort. The plant is considered to be relatively mild in toxicity but still call your vet immediately if you suspect your pet has ingested it. Poinciana gilliesii is another type of flowering plant that's often referred to as a bird of paradise, however, it's even more toxic than the strelitzia reginae. 5. Chrysanthemum. Top 10 Pet Poisons. The ASPCA Animal Poison Control Center handles more than 100,000 cases of pet poisonings every year. Based on those cases, the top offenders are Bartonella is a type bacteria that can be transmitted to cats, dogs and humans from exposure to infected fleas and Why diagnosis can be difficult. The Best Fruits and Veggies for Dogs.