



Summer hazards to avoid in our pets and patients

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Summer is finally here, and with that comes a myriad of fun outdoor activities along with home and garden projects. While summer is meant for relaxing at the lake with friends picnicking, watching fireworks, and cleaning up and readying yards and gardens for the upcoming growing season, it's potentially fraught with toxic exposure to your pets! Summertime brings new opportunities for potential pet exposures to harmful and dangerous substances. During the summer months, Pet Poison Helpline (PPH) is inundated with calls involving yard and garden products (including bone meal, fertilizers, and insecticides), mulch and compost pile ingestions, and exposures to outdoor plants and mushrooms.¹ As with all poisonings, early recognition and decontamination (including emesis induction and activated charcoal administration) are key to a successful outcome. Here is some basic information for you to know about when dealing with these exposures.

Compost bins or piles: While we applaud you for composting, make sure to do so appropriately – your compost shouldn't contain any dairy or meat products, and should always be fenced off for the sake of your pets and wildlife. These piles of decomposing and decaying organic matter and molding food products have the potential to contain tremorgenic mycotoxins, which are toxic to both pets and wildlife. Even small amounts ingested can result in clinical signs within 30 minutes to several hours. Clinical signs include agitation, hyperthermia, hyper-responsiveness, panting, drooling, and vomiting, and can progress to serious CNS signs (including incoordination, tremors, and seizures!). Ruleouts for this include toxins that cause “shake and bake,” such as metaldehydes (snail bait), strychnine, organophosphates, and methylxanthines. Prompt decontamination is the key if the patient isn't demonstrating clinical signs yet – this includes inducing vomiting and giving activated charcoal. Once the patient is symptomatic, aggressive supportive care includes the use of IV fluids, temperature regulation, cooling methods (cooling down to a temperature of 103.5° F/39.7° C), IV muscle relaxants (methocarbamol), and anticonvulsants (i.e., diazepam, phenobarbital).



Slug and Snail Baits: Slug and snail baits are commonly used on the West coast and in warm-weather conditions, and are available in a variety of forms (pellets, granular, powder, and liquid). The active ingredient is typically metaldehyde, which is toxic to all species (particularly dogs).² When ingested, metaldehyde results in clinical signs that resulted in the nickname “shake and bake.” Within 1 to 2 hours of ingestion, clinical signs of salivation, restlessness, vomiting, and incoordination are seen, which then progress to tremors, seizures, and secondary severe hyperthermia. Treatment

consists of early decontamination, supportive care, temperature regulation (cooling down to a temperature of 103.5° F/39.7° C), anticonvulsants, and muscle relaxants. Generally, the prognosis is favorable if treatment is quickly and aggressively implemented.



Mole and Gopher Bait: Surprisingly, most veterinary professionals aren't very familiar with mole and gopher baits, which typically contain zinc phosphide. Other types may contain bromethalin. Neither of these active ingredients have an antidote and both can result in rapidly developing, life-threatening symptoms. Zinc phosphide is often manufactured in a poisoned "peanut" form but can also be found in a pelleted or powdered form. When zinc phosphide combines with gastric acid, it results in rapid phosphine gas formation within the stomach. This toxin is made worse by the presence of food in the stomach, so make sure acutely poisoned pets aren't fed anything when this toxicity occurs! This gas causes severe gastrointestinal inflammation, abdominal distension, and cardiovascular insufficiency (similar to symptoms of a GDV or bloat). Pulmonary congestion and edema may also occur.¹ Clinical signs develop rapidly within 15 minutes to several hours and include vomiting, salivation, abdominal discomfort, bloating, depression, labored breathing, tremors, and weakness.¹ Once clinical signs have developed, the prognosis is guarded.² A word of caution to veterinary staff: second hand phosphine gas exposure can result in significant health risks to healthcare providers working in unventilated areas. By the time the phosphine gas odor has been recognized (which smells like rotten fish and garlic), there has already been significant exposure to staff.² So, whenever inducing emesis in a patient with this toxicity, do so in a well ventilated, outdoor area, and contact Pet Poison Helpline for more information on treatment.

The other toxin is bromethalin, a neurotoxin, which is found in a pelleted grain or as a gummy worm-shaped strip. These lanced gummy worms are placed underground as mole bait. Dogs can readily dig this product up and ingest it. Because cats aren't typically digging outside, there are fewer exposures to cats – that said, cats are very sensitive to bromethalin also. Bromethalin results in signs of cerebral edema (mentally obtunded, seizures, abnormal pupils, etc.), incoordination, and paralysis. As no antidote is available, treatment is centered around aggressive decontamination to limit absorption, supportive care, and drugs to decrease cerebral edema (i.e., Mannitol). The prognosis is based on the amount ingested and the severity of clinical signs. The more severe the symptoms, the more guarded the prognosis becomes.

Plants: Most garden and food producing plants are non-toxic to pets, and only result in mild gastrointestinal upset when ingested. That said, here are a few common summer plants that can cause concerns when eaten by pets:

Tomato plants are in the Nightshade family and contain tomatine. Tomatine is found in concentrations of up to 5% in the leafy greens, the fruit blossoms, and in small green tomatoes; this concentration rapidly decreases as the tomato ripens. When stems, vines and green fruit are ingested, clinical signs can include gastrointestinal irritation, ataxia, and weakness. Treatment is purely supportive with an overall good prognosis.

Rhubarb leaves contain oxalic acid, calcium oxalate and potassium oxalate and can result in oral and gastrointestinal irritation causing vomiting and diarrhea.⁴ Treatment includes symptomatic and supportive care.

Onions and garlic, when ingested in large amounts, can result in Heinz body formation and anemia. Cats are more sensitive than dogs to Allium toxicosis.⁵ Clinical signs are generally secondary to the anemia, with resultant weakness, lethargy and pale mucous membranes.

Grapes (or raisins) grown in home gardens can present significant concerns when dogs ingest them. Although the mechanism of action is not clearly understood at this time, grapes can result in anorexia, vomiting, diarrhea, and potentially severe acute renal failure. The toxicity is not necessarily dose-dependent, and symptoms can occur with even

small ingestions. Decontamination, aggressive supportive care, IV fluid therapy, and BUN/creatinine monitoring is recommended.



Mushrooms: There are various types of mushrooms located throughout the United States that may be non-toxic; however, other types of mushrooms may be gastric irritants, hallucinogenic, or hepatotoxic (from cyclopeptides, hydrazine toxins, isoxazoles, or psilocybin compounds).⁵ The frequency of mushroom toxicity is low, but the lack of readily available identification of mushrooms lands all ingestions in the category of toxic until proven otherwise. With ingestion of any mushroom, immediate emesis is recommended, provided the animal is alert, asymptomatic, and able to adequately protect his or her upper airway. Gastric lavage may be necessary for animals already exhibiting clinical signs. Clinical symptoms are dependent on the species of mushroom ingested, the specific toxin within that mushroom, and the individual's own susceptibility.⁵ Early clinical signs include vomiting, diarrhea, abdominal pain, ataxia, CNS depression, tremors, and seizures, with liver and renal damage occurring later. One can collect all the pieces of the mushroom in a paper towel, place them in a labeled (DO NOT EAT! POISONOUS) paper bag, and refrigerate the sample for future possible identification.



Mulch Products: Cocoa bean mulch, a byproduct of chocolate production, is the discarded hulls or shells of the cocoa bean. This mulch is frequently used for home landscaping and is often very fragrant, especially when first placed in the yard and warmed by the sun. This tempting smell of warm chocolate often attracts and encourages dogs (Labradors!) to ingest the mulch. Through the processing procedure of creating cocoa bean mulch, much of the methylxanthine poison is removed, but still potentially contains 0.19% to 2.98% theobromine and 0.5% to 0.85% caffeine.² All animals can be affected by methylxanthylates, but dogs tend to have more frequent exposure opportunities to the chocolates, coffee beans and cocoa mulch that contain them. Clinical signs include vomiting, diarrhea, hyperactivity, tremors, tachycardia, tachypnea, and potential seizures.^{1,2} Symptoms are dose-dependent and an accurate dose is very difficult to determine because of the variation of the concentration of methylxanthylates from one mulch product to next.

There have been anecdotal reports of a dog dying after ingesting cocoa bean shell mulch, and this has been rapidly circulating on the Internet. That said, how toxic is this stuff, *really*? The first report of this poisoning was actually reported by Pet Poison Helpline's Drs. Lynn Hovda and R. Kingston at the 1993 International Congress of Clinical Toxicology. Dr. Steve Hansen from ASPCA published this again 10 years later (Clin Tox 2003;41:5). Recently, Dr. Hansen stated that the cause of the one fatality (a young Labrador) was "highly suspect." While theobromine and caffeine (methylxanthines) can be toxic, clinical signs are usually more PROGRESSIVE - such as vomiting, diarrhea, *more* vomiting, trembling, a racing heart rate, and then seizures in very high doses. Cocoa bean mulch is very unlikely to result in sudden death without showing other signs. Nevertheless, play it safe and don't allow pets to ingest this product! Typically, after a first rain, the smell dissipates, making the mulch less attractive to pets.



Salt water toxicity: While it's probably not at the top of your toxin lists, salt water is a dangerous poison, particularly if you take your dog to the beach! If your dog loves to play on the ocean beach, heed caution. Dogs don't realize that salt water is dangerous, and excessive intake can result in severe hypernatremia, or salt poisoning. While initial signs of hypernatremia include vomiting and diarrhea, salt poisoning can progress quickly to neurologic signs like walking drunk, seizures, progressive depression, and ultimately, severe brain swelling. Hypernatremia needs to be treated very carefully with IV fluids by your veterinarian. Help avoid the problem by carrying a fresh bottle of tap water and offering it to your dog frequently while he's frolicking on the beach.

Fertilizers: Fertilizers come in a variety of forms - granular to water soluble – and are soil amendment products routinely used in lawn, garden and farming. There are virtually hundreds of products and product formulations or mixes out there, and most contain nitrogen, phosphorus and potassium in various concentrations. The three numbers that you see listed behind a fertilizer name (i.e., 10:20:10) represent the concentration of these three elements. Typically, limited ingestions of these ingredients generally do not result in significant concerns and are a relatively low level toxicity risk. With most case of fertilizer ingestion, clinical signs are limited to gastrointestinal irritation and foreign body obstruction risk (particularly if organic compounds such as bone meal are mixed in, adding in a risk for pancreatitis!). Keep in mind that there are some fertilizers that contain iron, along with other herbicide and pesticide additives, and these pose additional concerns and can result in significant health concerns.

Herbicides: Herbicides rarely result in concerns when used and applied according to the label directions, provided pets have been kept off the treated surfaces until the applied product has dried completely. However, when applied inappropriately, or when pets chew containers of concentrated product, there is a significant increase in the likelihood of potential toxicity. Clinical signs are dose and product dependent. Glyphosate and 2,4-D are two of the most commonly used herbicides.³ Ingestions of glyphosate concentrates can result in drooling, vomiting, diarrhea, anorexia and lethargy.^{1,2} With ingestion of concentrated 2,4-D, clinical signs include vomiting and weakness.³ While there are no antidotes for these products, prompt, supportive care (including IV or SC fluids and anti-emetics) generally provide a good prognosis.

Pesticides: Today's pesticides are much safer than products used 30 years ago. Examples of newer pesticides include fipronil, imidicloprid, sulfuramide and hydramethylnon. Many of these products have very low percentages of the active ingredient and are poorly absorbed systemically in mammalian species. Pesticides typically have wide margins of safety and are relatively low risk to pets. Like herbicides, when these products are applied appropriately and according to the label directions, these are minimal concern to pets. That said, these products often are mixed with bone meal, which makes it attractive and palatable to dogs. While the bone meal does not pose a significant *toxicity* concern, it can result in gastrointestinal irritation, severe pancreatitis and a possible foreign body obstruction (as it creates a big "ball of bone" in the stomach). More importantly, this increase in palatability can greatly increase the amount of the pesticide ingested.

Organophosphates and carbamates are dangerous pesticide exposures because they are competitive inhibitors of acetylcholinesterase. These pesticides are easily and rapidly absorbed from a variety of routes.⁴ The anticholinesterase properties result in clinical signs with an easy-to-remember acronym SLUDGE: salivation, lacrimation, urination, defecation, and gastroenteritis. Other clinical signs include weakness, bradycardia, mydriasis or miosis, ataxia,

paralysis, and respiratory depression. Death typically occurs from severe bronchial secretions, resulting in the patient drowning in their lungs and secondary, severe hypoxemia. The two antidotes are pralidoxime chloride (2PAM) and high-dose atropine.² These antidotes must be given rapidly for the best prognosis. Rapid decontamination in asymptomatic animals includes inducing emesis, gastric lavage, and activated charcoal. Once patients are clinically symptomatic, aggressive supportive care, oxygen saturation monitoring, anti-convulsant therapy, diphenhydramine for tremors, IV fluid therapy, and intensive monitoring is necessary.

Fireworks: These can result in burns; injury to the mouth, eyes, or paws; and possible heavy metal toxicity if ingestion. Fireworks can contain iron, copper, barium, mercury, phosphorus and magnesium in the coloring agents.¹ The amount of heavy metal varies widely, depending on the type of fireworks, quantity ingested, and coloring agent used.¹ Clinical symptoms seen with fireworks ingestion include vomiting, diarrhea, jaundice, tremors and seizures. Induction of emesis and activated charcoal are not recommended and animals should be immediately evaluated at a veterinary clinic for injury.

Blue-green algae or *Cyanobacteria*: Growth of toxic algae can be found in both fresh and salt water throughout the warm regions of the world. Blue-green algae becomes concerning when algae accumulates on the surface of the water during hot, dry weather with wind that can shift concentrated algae mats along the shorelines.¹ Affected water may have the appearance of pea soup with thick layers of algae on the surface. Blooms of blue-green algae can contain hepatoxins and/or neurotoxins, depending on the species. Exposures occur when dogs ingest or swim water that contains the cyanobacteria. Clinical signs with the hepatotoxin variety are vomiting, diarrhea, lethargy, weakness, shock, icterus, and potentially death within 24 hours to several days. Clinical signs seen with ingestion of neurotoxin species occur acutely with onset of tremors, lethargy, seizures and respiratory distress and death within a hour.³

Spring and summer preventative wellness visits to the clinic are the perfect time for client education – these visits also serve as an opportunity to remind pet owners about potential hazards that may have been out of sight during the colder months of the year. Education of staff and pet owners has proven to be the best method of preventing exposures to potentially harmful substances in animals. This coupled with information on when to seek prompt veterinary intervention and care will help keep your patients happy and healthy through the busy summer months ahead!

Resource: Pet Poison Helpline (PPH) is an Animal Poison Control that provides treatment advice and recommendations relating to exposures to potential dangerous plants, products, medications, and substances, to veterinarians, veterinary staff and pet owners 24 hours a day, 7 days a week. Please be aware there is a **\$35.00**/per case consultation fee. Pet Poison Helpline is located in Bloomington, Minnesota. The Helpline number is **1-800-213-6680**. For further information regarding services, visit the PPH website at www.petpoisonhelpline.com.

Saving pets' lives...24 hours a day, 7 days a week



References:

1. Pet Poison Helpline Case Database: Unpublished data, Bloomington, MN, 2004 – 2009.
2. Plumlee KH: *Clinical Veterinary Toxicology*, St Louis, MO, Mosby, 2004. pp322-325.
3. Peterson ME, Talcott PA (eds): *Small Animal Toxicology*, ed. 2, St Louis, MO, Elsevier Saunders, 2001.
4. Gfeller RW, Messonnier SP: *Handbook of Small Animal Toxicology and Poisoning*, St Louis, MO, Mosby, 1998.
5. Burrows GE, Tyril RJ: *Toxic Plants of North America*, Ames. Iowa, Iowa State University Press, 2001.