Chocolate toxicosis in pets

Chocolate ingestion is very common in pets, particularly dogs, but has also been reported in other animals including cats and rabbits. Most cases occur around Christmas and Easter, when large amounts of chocolate are often present in the home in the form of chocolate bars, snacks, confectionery, Easter eggs and chocolate figurines. Chocolate-covered raisins, nuts and coffee beans present an additional toxicological hazard in dogs. The type of chocolate is defined in law by the percentage of cocoa solids, with dark (plain) chocolate containing more than milk chocolate. The main toxic component of chocolate is theobromine, a methylxanthine, although caffeine, another methylxanthine, is also present. These cause central nervous system stimulation with cardiac and respiratory hyperactivity. Signs include gastrointestinal effects, excitability, hyperactivity, tachycardia, tremors, hypertension and hyperthermia. Signs of toxicosis generally occur within a few hours of ingestion. The mainstays of treatment are decontamination and supportive care with rehydration, control of central nervous system stimulation and arrhythmias as required. Most pets recover fully, and death is uncommon; however, prognosis is more guarded in pets with seizures or arrhythmias.

10.12968/coan.2023.0058

Nicola Bates BSc (Brunel), BSc (Open), MSc, MA Veterinary Poisons Information Service (VPIS). Corresponding author: nicola.bates@vpisglobal.com

Key words: poisoning | toxicity | chocolate | theobromine | methylxanthine

Submitted: 12 October 2023; accepted for publication following double-blind peer review: 29 October 2023

Chocolate ingestion is very common in pets, particularly in dogs. This article discusses the seasonality, clinical signs and management of chocolate toxicosis in pets.

Types of chocolate

Chocolate is made from the fermented, dried then roasted beans of *Theobroma cacao* (*Figure 1*). After roasting, the shell is removed, and the remaining nibs are ground to cocoa mass which is liquefied to produce chocolate liquor. This can then be processed into cocoa solids and cocoa butter. Cocoa nibs (cacao nibs) are roasted, cracked deshelled beans in broken pieces.

The type of chocolate is defined by the quantity (percentage) of cocoa solids the chocolate contains. Milk chocolate has some of the cocoa solids replaced by milk solids (from dried or condensed milk), giving it a sweeter taste and smoother texture than dark chocolate. In the UK and Ireland, the legal definition for 'milk chocolate' differs from that of the rest of Europe. In the UK and Ireland 'milk chocolate' must contain a minimum of 20% cocoa solids and 20% milk solids that include at least 5% milk fat. In the rest of Europe, this product is known as 'family milk chocolate', while 'milk chocolate' needs to have a minimum of 25% cocoa solids and 14% milk solids that include at least 3.5% milk fat.

Dark chocolate is chocolate made without milk. European law does not recognise the adjectives 'dark' or 'plain' and it is just known as 'chocolate'. Dark chocolate should contain a minimum of 35% cocoa solids, at least 18% of which should be cocoa butter, however, the cocoa content is often much higher, and can reach up to 90%. In the US this type of chocolate is called semi-sweet and bitter (or extra dark) chocolate. White chocolate is not real chocolate as it does not contain any cocoa solids, but it is made primarily of cocoa butter, sugar and milk solids. White chocolate must contain at least 14% milk solids, including 3.5% milk fat.

Cocoa powder is different. It is the non-fat part of the cacao bean (after the cocoa butter has been removed), which is ground into a powder. The quantity of theobromine in cocoa powder is very variable. 'Dutch-processed' cocoa powder is made from beans which have been treated with a potassium solution that neutralises their acidity and gives them a milder flavour.

Newer types of chocolates are also available. Ruby chocolate was introduced in 2017 and is notable for its natural pink colour. It is made from 'ruby' cocoa beans. The US Food and Drug Administration (FDA) defines ruby chocolate as containing a minimum of 1.5% non-fat cocoa solids and a minimum of 20% by weight of cocoa fat. Darkmilk chocolate (Cadbury's) contains 40% cocoa solids, so is a type of dark chocolate.

Sources of theobromine

Chocolate is available in many different forms and sources (*Figure 2*). In addition to bars, boxes and novelties (such as Easter eggs, chocolate bunnies and Santa figurines), chocolate is also found in numerous other sweet foods including biscuits, muffins and cakes. These are the most common sources in companion animals but in other animals, cocoa waste from processing or restaurant waste

Figure 1. Theobroma cocoa fruit (pods) (a), containing the beans (b) which are fermented, dried then roasted (c) to produce chocolate.

is an additional source. Chocolate-coated foods such as raisins, nuts and coffee beans may present an additional toxicological hazard.

Cocoa butter is found in some cosmetics and toiletries, often as a moisturiser, but it contains very little theobromine and is not likely to cause theobromine toxicity. Some laxative products also contain chocolate (and even come in a bar); these are another potential source of theobromine.

Cocoa mulch used in gardening is made of cocoa shells and is a potential source of theobromine found around the home. Cases of theobromine toxicosis in dogs after ingestion of cocoa mulch have been reported (Hovda and Kingston, 1994; Hansen et al, 2003) including death in one dog that ingested a large quantity (Drolet et al, 1984) but this is an uncommon source of chocolate toxicosis in pets.

Seasonality of cases

Chocolate exposure occurs throughout the year, with peaks around Easter and Christmas (Gwaltney-Brant, 2001; McFarland et al, 2017; Noble et al, 2017; Neagu et al, 2021; Weingart et al, 2021), sometimes with additional peaks around Valentine's Day and Halloween (Gwaltney-Brant, 2001; McFarland et al, 2017). Cases are most commonly seen around Christmas; almost one-fifth of all annual chocolate enquiries to the UK Veterinary Poisons Information Service occur in December (*Figure 3*).

Mechanism of toxicity

The main toxic component of chocolate is theobromine, a methylxanthine; it also contains a small amount of caffeine (another methylxanthine). Methylxanthine intoxication produces central nervous system stimulation with cardiac and respiratory hyperactivity.

Methylxanthines exert their effect by a variety of mechanisms. They competitively antagonise cellular adenosine receptors resulting in central nervous system stimulation, vasoconstriction and tachycardia. They also inhibit cellular phosphodiesterase causing an increase in cyclic adenosine monophosphate, increased release of catecholamines, increased entry of calcium and inhibition of calcium sequestration by the sarcoplasmic reticulum causing increased muscular contractility in both skeletal and cardiac muscle. In addition, methylxanthines increase the blood concentrations of adrenaline (epinephrine) and noradrenaline (norepinephrine).

Kinetics and metabolism

Cats and dogs

There is no information on the pharmacokinetics of theobromine in cats, but in dogs, theobromine is well absorbed from the gastrointestinal tract (Miller et al, 1984). At low doses, the peak theobromine concentration occurs within 3 hours in dogs; at higher doses absorption is slower (Gans et al, 1980).

The form of theobromine may also affect pharmacokinetics. In a study comparing the pharmacokinetics of pure theobromine (10 mg/kg) and the equivalent dose of chocolate in dogs, the peak blood concentration occurred at 4 hours in those given pure theobromine compared to 6 hours in those given chocolate. Theobromine could be detected in blood for 48 hours and in urine until at least 96 hours after dosing. The elimination half-life was 23 hours (Grip-Jonsson et al, 1994).

Theobromine (3,7-methylxanthine) is metabolised by demethylation to 7-methylxanthine and 3-methylxanthine in dogs (Miller et al, 1984). After dosing with radiolabelled theobromine, 60% was excreted in urine by 24 hours and 89% from 60–96 hours. Excretion in faeces was limited (Miller et al, 1984). In another study, the average total renal excretion over 96 hours was 13.7% of the dose (Grip-Jonsson et al, 1994). Theobromine and metabolites have been detected in the bile of dogs fed theobromine (Gans et al, 1980). Theobromine is itself a metabolite of caffeine (1,3,7-methylxanthine), which is also present in chocolate.

Rabbits

Theobromine is also well absorbed in rabbits; it is extensively metabolised with approximately 20% excreted unchanged and the remainder as metabolites. After oral dosing most is excreted in the urine with minimal elimination in the faeces (Miller et al, 1984). The elimination half-life of theobromine in rabbits is ap-

proximately 2–6 hours, but there is individual variation (Latini et al, 1984; Traina and Bonati, 1985).

Toxic dose

Dogs

Data from the American Society for the Prevention of Cruelty to Animals Animal Poisons Control Center suggest that toxic effects in dogs occur at theobromine doses of 20 mg/kg, with severe signs at 40–50 mg/kg and seizures at 60 mg/kg (Gwaltney-Brant, 2001). Fatal theobromine poisoning has been reported in dogs after ingestion of 80–300 mg/kg (European Food Safety Authority, 2008).

In an experimental study in dogs, a single oral dose of theobromine 500 or 1000 mg/kg caused panting, restlessness and muscle tremors 4–5 hours after ingestion and lasted 6–8 hours (Gans et al, 1980). No dogs given theobromine 200 mg/kg or less died, but one of 4 dogs given 300 mg/kg and one of two given 1000 mg/kg died within 5 hours. One of 8 dogs given 500 mg/kg died (Gans et al, 1980).

If the toxic dose of theobromine is 20 mg/kg, then it is important to understand the various types of chocolate. As stated earlier, the type of chocolate is defined by the quantity of cocoa solids present. The percentage of cocoa solids in a particular chocolate cannot be converted into the quantity of theobromine. It only describes the type of chocolate (dark, milk or white). Numerous sources list the range of theobromine content in various chocolate products. A review of original literature reporting the concentrations of theobromine in various chocolate products (Bates et al, 2015) provides the approximate dose of product equivalent to 20 mg of theobromine (*Table 1*). Dark chocolate can contain around four times the quantity of theobromine compared to milk chocolate, whereas the amount in white chocolate is minimal. It is important to note, however, that the amount of theobromine in products will vary due to natural differences in cocoa beans and the formulation of the product.

Cats

Cats are reportedly more sensitive to theobromine, but there is little information to substantiate this. There are no cases described in the literature and there is very limited information on the toxicity of chocolate in cats (European Food Safety Authority, 2008). A specific toxic dose for cats of theobromine has not been established.

Rabbits

Rabbits are not particularly sensitive to theobromine. There are no cases described in the literature. In experimental studies, which typically involved chronic dosing, death occurred due to cardiac failure (Soffietti et al, 1989).

A specific toxic dose for rabbits of theobromine has not been established, but rabbits tolerate higher doses than dogs. In a kinetic study, rabbits were given a single oral dose of theobromine up to 100 mg/kg. No clinical signs or deaths are mentioned in the report (Latini et al, 1984). In an experimental study, synthetic theobromine (300 mg/kg) was administered through oral gavage to 8 rabbits for 10 days. Half the rabbits died, but no information is provided on time of death or how many doses were given (Adeyina et al, 2008). In rabbits given theobromine 200 mg/kg/day for days 6–29 of gestation, 40% of the animals (4/10) died. The timing of death or the number of doses given was not reported (Tarka et al, 1986).

Figure 2. Chocolate is available in various forms, as (a) cocoa powder, (b) chocolate bars and (c) boxed chocolates.

Clinical signs

The amount of theobromine in products will vary due to natural differences in cocoa beans and the formulation of the product.

Dogs

Signs of chocolate toxicity generally occurs within 2–4 hours of ingestion (Dolder, 2013) but can sometimes occur after 6–12 hours (Gwaltney-Brant, 2001).

Initially, there may be vomiting, abdominal discomfort, diarrhoea, polydipsia, polyuria and restlessness, agitation, excitability and hyperactivity; then ataxia, tremors, tachycardia, panting, hypertension and hyperthermia. Dehydration, hypokalaemia, mild hyperglycaemia, hyperlactataemia, and mild elevation of alanine aminotransferase and aspartate aminotransferase may occur (Weingart et al, 2021).

Convulsions can occur after ingestion of chocolate (Weingart et al, 2021) but are rare (Noble et al, 2021). Other less com-

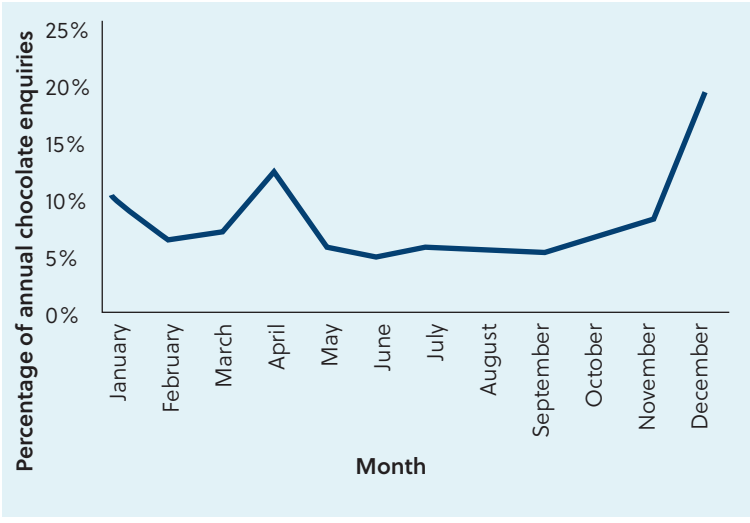

Figure 3. Enquiries to the Veterinary Poisons Information Service about chocolate by month as a percentage of annual chocolate enquiries, 2009–2022 (total enquiries: 12 050).

Table 1. Toxic doses of chocolate

Type of chocolate product	Approximate dose of product equivalent to 20 mg of theobromine
Milk chocolate	9.5–20 g (range), median 14.3 g
Dark (or plain) chocolate (semi-sweet, bitter or extra dark chocolate in the US)	2.3–4.5 g (range), median 3.8 g
White chocolate	Minimal risk of theobromine toxicity

(Bates et al, 2015)

mon effects include bradycardia, haematemesis, haematuria, bloat, tachypnoea, cyanosis and arrhythmias (classically premature ventricular contractions, but ventricular extrasystoles and ventricular tachycardia have been reports (Stosic et al, 2011)). Renal dysfunction may also occur but is uncommon. Pancreatitis may result 24–72 hours after ingestion because of the high fat content of some chocolate products (Gwaltney-Brant, 2001). Recovery from theobromine toxicosis can occur within 24 hours but may take 48–72 hours.

White chocolate, although low in theobromine, is high in fat and sugar and ingestion may cause gastrointestinal signs. Pancreatitis is also a potential risk with white chocolate.

The sweetener xylitol may be present in some chocolate or chocolate-containing products which may cause xylitol poisoning with risk of hypoglycaemia and liver failure. Chocolate-coated macadamia nuts, coffee beans or raisins also present additional risks of toxicosis.

Death is not common in dogs with chocolate toxicosis and is usually as a result of cardiac arrhythmias or less commonly, respiratory failure. Death has been reported in dogs within about 1 hour after ingestion (Stidworthy et al, 1997) or more than 12 hours later (Sutton, 1981; Strachan and Bennett 1994; Mays, 2022). Death can occur suddenly in the absence of any other signs (Sutton, 1981; Strachan and Bennett, 1994; Stidworthy et al, 1997). In these rare cases, dogs have been reported to collapse suddenly, sometimes have a brief seizure episode and then die.

Cats

Few cases of chocolate poisoning are reported in cats. Signs reported in cases encountered by the Veterinary Poisons Information Service include diarrhoea, vomiting, lethargy, polydipsia, inappetence, occasionally twitching, tachypnoea, tachycardia and hyperactivity. Convulsions are uncommon in chocolate poisoning. As in dogs, pancreatitis is a potential risk with some products due to their high fat content.

Rabbits

Few cases of chocolate poisoning are also reported in rabbits. Signs reported in cases encountered by the Veterinary Poisons Information Service and toxicity studies are soft stools or diarrhoea and lethargy. Less common signs are polydipsia, anorexia, twitching and tachycardia. Very high doses (used in experimental studies) can cause restlessness, pyrexia, tachypnoea, hyperaesthesia and seizures.

Prognosis

Most pets recover from chocolate toxicosis. Prognosis is more guarded in animals with seizures or arrhythmias (Dolder, 2013) and those with pre-existing cardiac disease may be more at risk of cardiac effects. Complications reported in dogs include arrhythmias (Stosic et al, 2011) and pulmonary oedema (Agudelo et al, 2013).

In cases encountered by the Veterinary Poisons Information Service with follow up data, approximately 1.5% of dogs died or were euthanised but in most years there are no fatal cases. There were no fatalities in 122 cases in dogs reported to two poison centres in Germany and Switzerland (Degrandi et al, 2016). In a retrospective evaluation of 156 cases from Germany, 44 dogs developed signs and one dog died (Weingart et al, 2021).

Treatment

Cats and dogs

If a potentially toxic dose has been ingested (eg equivalent to more than 20 mg/kg: see *Table 1*) gut decontamination should be attempted. An emetic can be given but emesis is best avoided in animals with hyperactivity or excitability as a result of theobromine toxicosis; however, activated charcoal can be given if practical (1 g/kg). In late-presenting animals, activated charcoal should be given. Repeated doses of adsorbents may be useful in enhancing elimination as theobromine appears to undergo enterohepatic re-circulation (Gans et al, 1980) and has a long half-life.

If possible, in symptomatic animals, the heart rate, body temperature and electrocardiogram should be monitored. There is no laboratory analysis that can aid diagnosis or predict toxicity in an emergency situation.

Treatment is supportive with anti-emetics if there is severe or persistent vomiting. Fluids are recommended to support the cardiovascular system and enhance excretion (as theobromine is excreted renally). Diazepam (0.5–1 mg/kg intravenously) to ef-

fect or methocarbamol (20–45 mg/kg orally every 8 hours, up to 330 mg/kg) can be used for the management of tremors with diazepam or barbiturates for hyperactivity or convulsions.

Where tachycardia is severe, a beta-blocker such as propranolol (0.1–0.2 mg/kg orally every 8 hours (dogs); 2.5–10 mg (total) orally every 8 hours (cats)), metoprolol or esmolol (0.05–0.5 mg/kg intravenous bolus over 5 minutes; 25–200 µg/kg/min constant rate infusion) can be given, although care should be taken when using these drugs as severe hypotension may result owing to unopposed alpha-adrenergic effects. Lidocaine (2 mg/kg slowly intravenously, bolus followed by 25–80 mcg/kg/min intravenous continuous rate infusion) is the drug of choice for ventricular arrhythmias unresponsive to a beta-blocker. In pets with bradycardia, atropine can be given (0.01–0.04 mg/kg intravenous or intramuscular).

Rabbits

Treatment of chocolate toxicosis in rabbits is supportive. Activated charcoal (1 g/kg) can be considered and the heart rate, body temperature and behaviour monitored.

Fluids are recommended to support the cardiovascular system and enhance excretion (theobromine is excreted renally). Rabbits should be monitored carefully for gastrointestinal signs and to ensure the animal is eating and drinking as normal. If required, suitable nutritional support should be (eg syringe feeding proprietary critical care products for rabbits, free access to good quality hay etc).

Diazepam (1–5 mg/kg intravenous) can be used for tremors and diazepam or barbiturates for hyperactivity or convulsions. Management of cardiovascular signs is the same as for cats and dogs.

Conclusions

Chocolate ingestion in pets, particularly dogs, is very common, but severe cases are rare. Gastrointestinal signs are common, but there may also be neurological and cardiovascular signs. The mainstays of treatment are gut decontamination and supportive care with rehydration, control of central nervous system stimulation and arrhythmias as required. CA

Conflicts of interest

The author declares that there are no conflicts of interest.

KEY POINTS

- Chocolate ingestion is very common in pets, particularly dogs, especially around Easter and Christmas.
- Gastrointestinal signs are common; neurological and cardiovascular signs may also occur.
- Management of chocolate toxicosis involves decontamination and supportive care with rehydration, control of central nervous system stimulation and arrhythmias.
- Death from chocolate toxicosis is uncommon, but prognosis is more guarded in animals with seizures or arrhythmias.

References

Adeyina AO, Oguntoye SO, Olatunde OA, Apata DF. Comparative effects of theobromine and cocoa bean shell (CBS) extract on the performance, serum constituent profile and physiological parameters in rabbits. Global J Pure Appl Sci. 2008;14(3):253–255. http://doi.org/10.4314/gjpas.v14i3.16604

Agudelo CF, Filipejova Z, Schanilec ZP. Chocolate ingestion-induced non-cardiogenic pulmonary oedema in a puppy: a case report. Veterinarni Medicina. 2013;58(2):109–112. https://doi.org/ 10.17221/6703-VETMED

Bates N, Rawson-Harris P, Edwards N. Common questions in veterinary toxicology. J Small Anim Pract. 2015;56(5):298–306. https://doi.org/10.1111/jsap.12343

Degrandi C, Hermanns-Clausen M, Hofer KE, Reicherta C. Chocolate poisonings in dogs: a consecutive case series [abstract]. Clin Toxicol. 2016;54(4):515. https://doi.org/ 10.3109/15563650.2016.1165952

Dolder LK. Methylxanthines: caffeine, theobromine and theophylline. In: Peterson ME, Talcott PA (eds). Small animal toxicology. 3rd edition. St Louis:Elsevier; 2013:647–652

Drolet R, Arendt TD, Stowe CM. Cacao bean shell poisoning in a dog. J Am Vet Med Assoc. 1984;185(8):902

European Food Safety Authority. Theobromine as undesirable substances in animal feed – scientific opinion of the panel on contaminants in the food chain.EFSA Journal. 2008;6(9):1–66. https://doi.org/10.2903/j.efsa.2008.725

Gans JH, Korson R, Cater MR, Ackerly CC. Effects of short-term and long-term theobromine administration to male dogs. Toxicol Appl Pharmacol. 1980;53(3):481–496. https://doi.org/10.1016/0041-008x(80)90360-9

Grip-Jonsson A, Nytrøen T, Bondesson U et al. (1994) Administration and detection of theobromine in the dog. Proceedings of the 10th International Conference of Racing Analysts and Veterinarians, Stockholm, Sweden: 354–6

Gwaltney-Brant S. Chocolate intoxication. Vet Med. 2001;96(2):108–111

Hansen S, Trammel H, Dunayer E, Dwaltney S, Farbman D, Khan S. Cocoa mulch as a cause of methylxanthine toxicosis in dogs. Clin Toxicol. 2003:41:741

Hovda LR, Kingston RL. Cocoa bean mulch poisoning in dogs. Vet Hum Toxicol. 1994;36(4):357

Latini R, Bonati M, Gaspari F et al. Kinetics and metabolism of theobromine in male and female non-pregnant and pregnant rabbits. Toxicology. 1984;30(4):343–354. https://doi.org/10.1016/0300-483x(84)90144-6

Mays T. Possible chocolate poisoning in a dog. 2022. https://tvmdl.tamu.edu/2022/02/24/possible-chocolate-poisoning-in-a-dog/ (accessed 7 October 2023)

McFarland SE, Mischke RH, Hopster-Iversen C et al. Systematic account of animal poisonings in Germany, 2012–2015. Vet Rec. 2017;180(13):327. https://doi.org/10.1136/vr.103973

Miller GE, Radulovic LL, DeWit RH, Brabec MJ, Tarka SM, Cornish HH. Comparative theobromine metabolism in five mammalian species. Drug Metab Dispos. 1984;12(2):154–160.

Neagu AM, Diac E, Codea AR, Nagy A, Oros A, Dreanca A. Incidence of pet intoxications in the emergency clinic, Faculty of Veterinary Medicine Cluj-Napoca. Bull Uni Agric Sci Vet Med Cluj-Napoca Vet Med. 2021;78(1):101–108. https://doi.org/10.15835/buasvmcn-vm:2020.0036

Noble PM, Newman J, Wyatt AM, Radford AD, Jones PH. Heightened risk of canine chocolate exposure at Christmas and Easter. Vet Rec. 2017;181(25):684. https://doi.org/10.1136/vr.104762

Soffietti MG, Nebbia C, Valenza F, Amedeo S, Re G. Toxic effects of theobromine on mature and immature male rabbits. J Comp Pathol. 1989;100(1):47–58. https://doi.org/10.1016/0021-9975(89)90089-3

Stidworthy MF, Bleakley JS, Cheeseman MT, Kelly DF. Chocolate poisoning in dogs. Vet Rec. 1997;141(1):28

Stosic A, Ondreka N, Henrich E, Hassdenteufel E, Schneider M. Chocolate intoxication in a dog. Tierarztl Prax Ausg K Kleintiere Heimtiere. 2011;39(2):111–115. https://doi.org/ 10.1055/s-0038-1623569

Strachan ER, Bennett A. Theobromine poisoning in dogs. Vet Rec. 1994;134(11):284. https://doi.org/10.1136/vr.134.11.284

Sutton RH. Cocoa poisoning in a dog. Vet Rec. 1981;109(25-26):563–564

Tarka SM Jr, Applebaum RS, Borzelleca JF. Evaluation of the teratogenic potential of cocoa powder and theobromine in New Zealand White rabbits. Food Chem Toxicol. 1986;24(5):363–374. https://doi.org/10.1016/0278-6915(86)90200-0

Traina GL, Bonati M. Pharmacokinetics of theobromine and its metabolites in rabbits. J Pharmacokinet Biopharm. 1985;13(1):41–53. https://doi.org/10.1007/BF01073655

Weingart C, Hartmann A, Kohn B. Chocolate ingestion in dogs: 156 events (2015–2019). J Small Anim Pract. 2021;62(11):979–983. https://doi.org/10.1111/jsap.13329