VET 406 Feed Toxicants

Feed	Toxin	MOA or ADME	Symptoms	Treatment
Malamine/Cyanuric Acid	Combination of Melamine and Cyanuric acid *individually they are fairly safe		Crystals in urine (feline) Development of acute renal failure in cats when given melamine and cyanuric acid combined	No specific antidote Supportive therapy Fluid therapy
Ionophores (Feed additives)	Monensin Lasalocid Narasin Salinomycin Laidlomycain	Rapid absorption from the gut Significant first pass effect meaning minimal amounts reach systemic circulation Rapid metabolism in the liver by P450 enzymes Minimal urinary excretion Minimal accumulation in tissues Influx of Na^+, Efflux of K^+ Increased Net Influx of Ca^{2+} Excess uptake of Ca^{2+} by mitochondria Mitochondrial damage leading to a lack of energy elevated cytoplasmic Ca levels and muscle necrosis	Horses/cattle/camelids Cardiac effects Sheep/swine/dogs Skeletal muscle effects (weakness) Poultry: Cardiac and muscle effects Cats: Polyneuropathy Long-term effects: Possible cardiac fibrosis Sudden death	

Ionophore Specific ex. (Monensin)	Monesin	Excess Ca^{2+} uptake leading to mitochondrial damage, elevated cytoplasmic Ca^{2+} levels and muscle necrosis	Horse: Anorexia, uneasiness Sweating profuse to intermittent Abdominal pain Stiffness, progressive ataxia, posterior paresis, recumbency, tachycardia, hypotension Death 24-72 hours Cattle: Anorexia 6-12 hrs followed by 12-24 hrs with diarrhea, weakness, dyspnea Death typically follows 5-9 days after exposure	Prevent further exposure Activated charcoal +/- sorbitol cathartic Fluids for shock and acidosis Correct cardiac arrhythmias Vit. E/Selenium Long-term effects may persist for months and sporadic death may occur due to cardiac lesions
Cotton	Gossypol (free portion is toxic)	Metabolites exert oxidative stress Enzyme inhibition leading to potassium and sodium dysregulation Blockage of gap junction intercellular communication	Cardiac failure Reproductive effects Malnutrition	Symptomatic and supportive care Alleviate edema Provide nutrients (Vit. A)

Sodium Ion Poisoning and/or Water Deprivation	Too much sodium and not enough water	Initial brain dehydration Fluid balance will lead to sodium shift across blood-brain-barrier -Na^+ rapidly absorbed, diffuses passively into CSF High Na^+ depresses glycolysis leading to decreased ATP and CNS signs Na^+ trapped in CNS attracts water (osmotic) Cerebral edema formation as water re-enters the CSF	NaCl ingestion: -GI signs -Polydipsia, polyuria, ataxia, disorientation, tremors, recumbency Water deprivation: -Thirst, depression, anorexia -Neurologic signs *Dog sitting, knuckling of forelimbs, head tilt and bobbing swine *Eosinophilic meningoencephalitis and perivascular cuffing swine	Rehydrate SLOWLY Diuretics
Urea	Urea *ruminants	Rapidly hydrolyzed by urease in rumen to form ammonia	Elevated blood ammonia accounts for signs Frothy salivation, teeth grinding, abdominal pain, bloat, regurgitation Polyuria Muscle tremors, incoordination Weakness, rapid breathing Violent struggling and terminal tetanic spasms	May not be effective because of rapid onset Acute death within 24 hours Vinegar orally to decrease rumen pH Cold water to decrease rumen urease activity

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