Testimony from Nick Reinecker, Kansas Citizen

March 8, 2016 Senate Natural Resources Committee

HB2490- Regards to containing chemical toxins and other plant pests for public health.

Poisonous Food Plants;

Many plants commonly used as food possess toxic parts, are toxic unless processed, or are toxic at certain stages of their lives. Some only pose a serious threat to certain animals (such as cats, dogs, or livestock) or certain types of people (such as infants, the elderly, or individuals with pathological vulnerabilities). Most of these food plants are safe for the average adult to eat in modest quantities. Notable examples include:

Apple (Malus domestica). Seeds are mildly poisonous, containing a small amount of amygdalin, a cyanogenic glycoside. The quantity contained is usually not enough to be dangerous to humans, but it is possible to ingest enough seeds to provide a fatal dose.

Cassava (Manihot esculenta). Roots and leaves contain two cyanogenic glycosides, linamarin and lotaustralin. These are decomposed by linamarase, a naturally occurring enzyme in cassava, liberating hydrogen cyanide .[4] Cassava varieties are often categorized as either sweet or bitter, respectively signifying the absence or presence of toxic levels of cyanogenic glycosides. The 'sweet' cultivars can produce as little as 20 milligrams of cyanide per kilogram of fresh roots, whereas bitter ones may produce more than 50 times as much (1 g/kg). Cassavas grown during drought are especially high in these toxins.[5][6] A dose of 40 mg of pure cassava cyanogenic glycoside is sufficient to kill a cow. It can also cause severe calcific pancreatitis in humans, leading to chronic pancreatitis. Processing (soaking, cooking, fermentation, etc.) of cassava root is necessary to remove the toxins and avoid getting sick. In the tropics, where cassava farming is a major industry, "Chronic, low-level cyanide exposure is associated with the development of goiter and with tropical ataxic neuropathy, a nerve-damaging disorder that renders a person unsteady and uncoordinated. Severe cyanide poisoning, particularly during famines, is associated with outbreaks of a debilitating, irreversible paralytic disorder called konzo and, in some cases, death. The incidence of konzo and tropical ataxic neuropathy can be as high as 3 percent in some areas."[7] For some smaller-rooted sweet varieties, cooking is sufficient to eliminate all toxicity. The cyanide is carried away in the processing water and the amounts produced in domestic consumption are too small to have environmental impact.[4] The larger-rooted, bitter varieties used for production of flour or starch must be processed to remove the cyanogenic glycosides.[8] Industrial production of cassava flour, even at the cottage level, may generate enough cyanide and cyanogenic glycosides in the effluvia to have a severe environmental impact.[4]

Cherry (Prunus cerasus), as well as other Prunus species such as peach (Prunus persica), plum (Prunus domestica), almond (Prunus dulcis), and apricot (Prunus armeniaca). Leaves and seeds contain amygdalin, a cyanogenic glycoside.

Grape (Vitis spp.). Potentially toxic to dogs, although the precise mechanism is not fully understood. See grape and raisin toxicity in dogs.

Indian pea (Lathyrus sativus). A legume grown in Asia and East Africa as an insurance crop for use during famines. Like other grain legumes, L. sativus produces a high-protein seed. The seeds contain variable amounts of ?-N-Oxalyl-L-?,?-diaminopropionic acid or ODAP, a neurotoxic amino acid.[9] ODAP causes wasting and paralysis if eaten over a long period, and is considered the cause of the disease neurolathyrism, a neurodegenerative disease that causes paralysis of the lower body and emaciation of gluteal muscle (buttocks). The disease has been seen to occur after famines in Europe (France, Spain, Germany), North Africa and South Asia, and is still prevalent in Eritrea, Ethiopia and parts of Afghanistan when Lathyrus seed is the exclusive or main source of nutrients for extended periods.

Lemon, as well as lime, orange and other citrus fruits are known to contain aromatic oils and compounds of Psoralen which is toxic to dogs, cats, and some animals. The acid is found all over the entire plant. Symptoms include vomiting, diarrhea, depression and photosensitivity.

Mango tree. Mango peel and sap contain urushiol, the allergen in poison ivy and poison sumac that can cause urushiol-induced contact dermatitis in susceptible people. Cross-reactions between mango contact allergens and urushiol have been observed. Those with a history of poison ivy or poison oak contact dermatitis may be most at risk for such an allergic reaction. Urushiol is also present in mango leaves and stems. During mango's primary ripening season, it is the most common source of plant dermatitis in Hawaii.

Nutmeg (Myristica fragrans). Contains myristicin, a naturally occurring insecticide and acaricide with possible neurotoxic effects on neuroblastoma cells.[11] It has psychoactive properties at doses much higher than used in cooking. Raw nutmeg produces anticholinergic-like symptoms, attributed to myristicin and elemicin.[12] The intoxicating effects of myristicin can lead to a physical state somewhere between waking and dreaming; euphoria is reported and nausea is often experienced. Users also report bloodshot eyes and memory disturbances.[13] Myristicin is also known to induce hallucinogenic effects, such as visual distortions. Nutmeg intoxication has an extremely long delay before peak is reached, sometimes taking up to seven hours, and effects can be felt for 24 hours, with lingering effects lasting up to 72 hours.[14][15]

Onions and garlic. Many members of the Allium genus contain thiosulphate, which in high doses is toxic to dogs, cats and some types of livestock. Cats are more sensitive to Allium.

Potato (Solanum tuberosum). Potatoes contain toxic compounds known as glycoalkaloids, of which the most prevalent are solanine and chaconine. Solanine is also found in other members of the Solanaceae plant family, which includes Atropa belladonna ("deadly nightshade") and Hyoscyamus niger ("henbane") (see entries below). The concentration of glycoalkaloid in wild potatoes is sufficient to produce toxic effects in humans. The toxin affects the nervous system, causing headaches, diarrhea and intense digestive disturbances, cramps, weakness and confusion, and in severe cases coma and death. Poisoning from cultivated potatoes occurs very rarely, however, as toxic compounds in the potato plant are generally concentrated in the green portions of the plant and in the fruits, and cultivated varieties

contain smaller concentrations than wild plants.[16][17] Cooking at high temperatures (over 170 °C or 340 °F) also partly destroys the toxin. However, exposure to light, physical damage, and age can increase glycoalkaloid content within the tuber,[18] the highest concentrations occurring just underneath the skin. Tubers that are exposed to light turn green from chlorophyll synthesis, thus giving a visual clue as to areas of the tuber that may have become more toxic; however, this does not provide a definitive guide, as greening and glycoalkaloid accumulation can occur independently of each other. Some varieties of potato contain greater glycoalkaloid concentrations than others; breeders developing new varieties test for this, and sometimes have to discard an otherwise promising cultivar. Breeders try to keep solanine levels below 200 mg/kg (200 ppmw). However, when these commercial varieties turn green, even they can approach concentrations of solanine of 1000 mg/kg (1000 ppmw). The U.S. National Toxicology Program suggests that the average American consume no more than 12.5 mg/day of solanine from potatoes (the toxic dose is actually several times this, depending on body weight).

Rhubarb (Rheum rhaponticum). The leaf stalks (petioles) are edible, but the leaves themselves contain notable quantities of oxalic acid, which is a nephrotoxic and corrosive acid present in many plants. Symptoms of poisoning include kidney disorders, convulsions and coma, though it is rarely fatal. The LD50 (median lethal dose) for pure oxalic acid in rats is about 375 mg/kg body weight,[19] or about 25 grams for a 65 kg (~140 lb) human. Although the oxalic acid content of rhubarb leaves can vary, a typical value is about 0.5%,[20] so almost 5 kg of the extremely sour leaves would have to be consumed to reach the LD50. Cooking the leaves with soda can make them more poisonous by producing soluble oxalates.[21] However, the leaves are believed to also contain an additional, unidentified toxin,[22] which might be an anthraquinone glycoside (also known as senna glycosides).[23] In the edible leaf stalks, the concentration of oxalic acid is much lower, contributing only about 2–2.5% of the total acidity, which is dominated by malic acid.[24] This means that even the raw stalks may not be hazardous (though they are generally thought to be in the US). However, the tart taste of the raw stalks is so strong as to be unpalatable to most consumers.

Tomato (Solanum lycopersicum). Like many other members of the nightshade family (Solanaceae), tomato leaves and stems contain solanine that is toxic if ingested, causing digestive upset and nervous excitement. Use of tomato leaves as an herbal tea (infusion) has been responsible for at least one death.[25] Leaves, stems, and green unripe fruit of the tomato plant also contain small amounts of the poisonous alkaloid tomatine,[26] although levels are generally too small to be dangerous.[26][27] Ripe tomatoes do not contain any detectable tomatine.[26] Tomato plants can be toxic to dogs if they eat large amounts of the fruit, or chew plant material.[28]