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PLANT POISONING IN FREE-LIVING WILD ANIMALS: A REVIEW

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INTRODUCTION

Plant poisoning is of major concern to the livestock industry throughout the world, especially in those areas where rangeland and pasture grazing are practiced. In most non-intensive agricultural locations, livestock cohabit with wild herbivores. Little consideration has been given to the prevalence of plant poisoning in wild species. Do wild animals eat poisonous plants? Are they affected by poisonous plants? If not, how do they cope with poisonous plants?

Several facets of the interaction of wild animals and poisonous plants are of interest. Poisonous plants may be a factor in the coevolution of plants and animals. Wildlife biologists, researchers and diagnosticians should be aware that plant poisoning can and does occur in free-living herbivores. Plant poisoning of wild animals is often difficult to diagnose, and may be overlooked or neglected.

Much of the literature on poisonous plants deals with problems of acute or chronic toxicity that are readily apparent; i.e., the animal dies or exhibits known signs of poisoning. Reports of less obvious effects indicate that the ingestion of certain plants may inhibit growth, interfere with reproduction, cause weight and fur loss, shorten life span or cause neurological impairment, resulting in adverse behavioral changes or physical deficits (Freeland and Janzen, 1974).

BASIC CONSIDERATIONS

Plants and animals share similar metabolic and biochemical characteristics. Both grow, mature and reproduce as a result of complex biochemical processes. Since not all metabolic reactions are chemically balanced, waste products must be eliminated, detoxified or sequestered. In animals, this is accomplished through the action of specialized organs and by voiding urine and feces. In plants which lack such mechanisms, metabolic byproducts accumulate. As a result, two differing sets of plant chemicals have been identified. Primary plant compounds are those chemicals that are known to function within metabolic pathways necessary for the life of the plant. The second group, classified as secondary plant compounds (SPC), have no known function within the plant (Brower, 1969).

Many theories have been postulated to account for the presence of SPC. The major concern for the wildlife biologist is not why they were formed, but the fact that they are present and may produce pronounced toxic effects on individual and, ultimately, populations of animals. Also of interest is the fact that many wild animals are able to deal with SPC.

Chemical defenses of plants against consumption by herbivores are usually determined by non-nutrient compounds such as fiber, lignin, cellulose, toxicants, or essential oils and other volatile substances (Radwan, 1974). Some volatile substances are overtly toxic; others are merely unpleasant. Essential oils, which are mixtures of terpene and nonterpene compounds are highly unpalatable and interfere with digestion in deer. Mule deer (*Odocoileus hemionus*) limit intake of sagebrush, *Artemisia tridentata*, and juniper, *Juniperus* sp., because they contain high levels of essential oils (Smith, 1950; Dietz et al., 1962; Nagey et al., 1964; Radwan, 1974).

The concept that plants contain compounds that serve as defenses against herbivores is not new. Stahl suggested this theory in 1888. Numerous authors, especially during the past two decades, have defended the theory.

Arguments for coevolutionary development of plant and specialist feeding insects have been well substantiated in the literature (Brower, 1969; McBee, 1971; Freeland and Janzen, 1974; Feeny, 1975; Levin, 1976). Most of the literature on this topic deals with insect and plant interaction and has resulted in a subdiscipline of biology called chemical or phytochemical ecology (Laycock, 1978). A classical example of phytochemical ecology is illustrated by the relationship between milkweed, *Asclepias* sp., and the monarch butterfly, *Danaus plexippus* (Brower, 1969).

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TABLE 1. Comparisons of insects and large mammals.

Insects	Mammals
May eat only one species or even a single leaf.	Move from plant to plant. Take time to eat.
Short lifespan.	Long lifespan.
High fecundity.	Low fecundity.
Develop specific detoxification mechanisms.	Detoxification mechanisms more generalized.
Can adapt rapidly to new SPC in plant.	Adaptation is slow. Must depend on taste, smell and learning to avoid.

Specialized insect feeders that spend their entire life cycle on one plant are ideal subjects for studies of plant-animal coevolution (Freeland and Janzen, 1974). It is more difficult to demonstrate such coevolution in large generalist herbivores. Whether large herbivores coevolved with secondary plant compounds or have developed resistance mechanisms by accidental exposure to already existing plant substances has not yet been elucidated. A comparison of these two groups can be seen in Table 1.

The herbivorous generalist faces the daily task of securing sufficient nutrients to satisfy maintenance and other population requirements, such as reproduction. The animal is presented with food choices. That wild herbivores have definite forage preferences is unquestioned, but the basis for such selection is difficult to determine. Species survival dictates that populations of wild herbivores must select nutritious forage. It is interesting to note, however, that some plants avoided by a given species may be equally as nutritious as those preferred (Longhurst et al., 1968).

Individual wild animals acquire feeding habits by genetically controlled food identification mechanisms, parental education (imprinting), interaction with con-specifics (in social species), and experience (random plant selection). Critical field observations of black-tailed deer, *Odocoileus hemionus columbianus*, have shown that a deer first sniffs a new plant. If the odor is acceptable it then tastes and, if still acceptable, eats it. Once familiar with the plant, deer eat it directly (Longhurst et al., 1968). Deer preferentially select the more nutritious forages, choosing plants from plots which have been fertilized with nitrogen over plants from unfertilized plots. The protein content of the preferred plants is higher than that of the rejected (Longhurst et al., 1968). It is likely that volatile indicator compounds found in less desirable forage plants stimulate rejection.

COPING WITH INJURIOUS PLANTS

Animal anatomic and physiologic adaptations

Unique anatomical structures enable certain animals to counter plant defenses. The incisors of the Great Basin kangaroo rat, *Dipodomys microps*, are highly adapted for removing the hypersaline outer layers of the stems of salt brush, *Atriplex confertifolia*, allowing the rat to harvest the starchy inner layers low in electrolytes (Kenagy, 1972). Other species of *Dipodomys* are granivorous, thus *D. microps* can coexist with granivorous *Dipodomys* without competition and without suffering from sodium toxicity. The giraffe, *Giraffa camelopardalis*, uses its long tongue to reach through the formidable thorn screen of acacia and grasp the highly nutritious leaves.

SPC in a plant may serve as a defense against herbivory if the substances are extremely toxic, unpalatable, or cause adverse conditioning. The effects of all three of these factors can be observed in livestock and wild animals. All wild herbivore vertebrates feed in an environment replete with plants containing potentially poisonous substances. That wild animals have adapted to these substances is beyond question. Understanding how animals adapt enables more effective management of wildlife resources.

Animals cope with SPC in plants by utilizing one or more of the following strategies: avoidance, dilution, degradation or detoxification. Should the strategies fail, the animal will be affected in some detrimental manner, the ultimate of which is death.

Avoidance

In a feeding experiment, mule deer were transported from one habitat to a new area containing 200 unfamiliar plant species. Thirty-five of the plants were known to be toxic. The deer began to eat small amounts of many plants and appeared to reject the more toxic plants. Some plants (i.e., locoweed, *Astragalus* sp., bracken fern, *Pteridium* sp., and Elk's clover, *Psoralea* sp.) known to be toxic to livestock were palatable to and eaten by the deer, without apparent harm (Nichol, 1938). On the other hand, when

a yearling eland, *Taurotragus oryx*, was given an opportunity to browse gifblaar, *Dichapetalum venenatum*, it investigated the plant but refused to eat a single leaf (Norval and Basson, 1974).

Plant populations vary in content of SPC. Bracken fern is polymorphic as to the presence of cyanogenic glycosides. Red deer, *Cervus elaphus*, and sheep have avoided fronds and rhizomes which contained the glycoside and readily consumed acyanogenic plants (Cooper-Driver and Swain, 1976).

The wild Norway rat, *Rattus norvegicus*, is a highly adaptive herbivore. Its ability to avoid poisoned baits is legendary, but also has been documented scientifically (Chitty, 1954; Barnet, 1963; Rozin, 1967) and is attributed to a learning process. Wild rats exhibit neophobia or suspicion and/or avoidance of strange food items. A new food item is lightly sampled until it is determined to be safe. Rats become more suspicious with repeated attempts to poison them, refusing strange or adulterated food items up to the point of starvation (Rozin, 1967).

Behavioral adaptation is a significant factor in the ability of a wild animal to avoid poisonous plants. Avoidance is a crucial skill that must be learned early in life. It is important to note that environmental pollutants such as chlorinated hydrocarbons, organic phosphates, mercury and lead may cause behavioral modifications that change avoidance patterns. Infectious and parasitic diseases affecting the central nervous system also alter behavior. Eleophorosis of Rocky Mountain elk, *Cervus elaphus nelsoni*, may be an example. The parasite *Eleophora schneideri* causes blockage of cerebral arteries, thus decreasing cerebral functions.

Consumption of locoweeds, *Astragalus* sp. and *Oxytropis* sp., causes neuronal degeneration. Elk eventually will eat these plants in preference to others and devlop locoism (Adcock and Keiss, 1969). In addition, altered behavior may allow consumption of other toxic plants.

Many wild species will avoid dangerous plant species in their native habitat that are high in SPC unless driven by extreme hunger, a factor that can override basic avoidance patterns. Animals are more likely to consume poisonous plants when they are denied adequate forage (Longhurst et al., 1968).

Wildlife biologists who plan to translocate animals from one location to another should take note of plant life. Plant communities may vary markedly even though short distances apart. Variation in elevation, soil types and moisture all may contribute to the development of an entirely different flora. A transplanted animal may lack the avoidance behavior repertoire necessary to distinguish between safe and dangerous unfamiliar plants.

Dilution

Wild herbivores usually eat a large variety of plants. Such a diet tends to dilute the toxic agent from any one plant. Perhaps dilution would satisfactorily explain the ability of wild animals to deal with SPC if SPC were not so ubiquitous, but it is estimated that over 40% of the plants in the environment of herbivores contain SPC (Levin, 1976). A large mammal could not obtain sufficient food by simply eating small amounts of all the plants. The animal must have intrinsic mechanisms for dealing with SPC.

Degradation and detoxification

Two major methods of destroying dangerous SPC are degradation of the toxicant within the gastrointestinal tract and both general and specific detoxification mechanisms that become active following absorption from the gut. Some of these metabolic pathways have been studied in livestock, but little has been done with wild species (Freeland and Janzen, 1974).

Numerous reports in the literature indicate that wild animals safely consume many plants that are lethal to livestock. In most instances little factual data are presented to establish whether or not the animal has specific detoxification mechanisms.

Kudu, *Tragelaphus strepsiceros*, are reported to safely eat the toxic euphorbid, *Synadenium cupulare*, ouabin, *Strophanthus* sp., and candelabrum, *Euphorbia candelabrum* (Pirie, 1968). However, if the kudu inadvertently rubs the plant juices of candelabrum into the eye, blindness ensues.

The colobus monkey, *Colobus guereza caudatus*, is a leaf-eating primate. A favorite food item is *Rauwolfia* sp., a known poisonous plant. It is not known precisely how this monkey copes with the alkaloidal tranquilizing compounds, but the unusual, complex, fermenting-type stomach undoubtedly plays a part (Kuhn, 1964).

The mountain viscacha, *Lagidium peru-*

anum, utilizes *Senecio* sp. as a major food item (Pearson, 1948). *Senecio* contains pyrrolizidine alkaloids. The white-footed deer mouse, *Peromyscus leucopus*, consumes the seeds of *Prunus* sp. at up to 5% of the diet without ill effects (Whitaker, 1963).

In a limited feeding trial, white-tailed deer, *Odocoileus virginianus*, were able to consume the leaves of mountain laurel, *Kalmia latifolia*, and rhododendron, *Rhododendron maximum*, without being poisoned. Deer preferentially would exclude these shrubs from the diet if given a choice, but apparently were able to tolerate a restricted intake. Force-feeding fresh laurel leaves at 1.75% of a deer's body weight resulted in death with signs and lesions of andromedotoxin poisoning (Forbes and Bechdel, 1931).

Dromedary camels, *Camelus dromedarius*, were introduced to Australia as early as 1840 (McKnight, 1969). Feral populations built up to over 30,000 in the 1940's. Camels are successful as feral animals because they are able to eat many different plants and will sample almost anything (Williams, 1963). They are even known to consume plants that are extremely bitter, such as white wood, *Atalaya* sp., which even goats refuse to eat (McKnight, 1969). There are, however, many reports of plant poisoning of domestic camels in Australia and other countries and it is presumed that feral animals are likewise poisoned (Leese, 1927, 1942; Peck, 1942; McKnight, 1969).

Schmidt-Nielsen et al. (1955) have shown that camels are able to conserve nitrogen, especially when on a low protein diet. Thus, camels would theoretically be better able than other mammals to cope with poisonous plants because rumen microbial action would be more efficient on a marginal diet than would rumen action of cattle and sheep.

It is interesting to note that deer were introduced into Australia at about the same time as the camels (Bentley, 1978). No mention is made of mortality caused by toxic plants in a recent review of the status of Australian deer (English, 1979). Perhaps different feeding habits and habitats account for the variance. Deer were introduced into the southeastern areas where desirable forage is generally more available, whereas camels populated more arid sections of the country.

The American pronghorn, *Antilocapra americana*, is a grazer-browser of the western ranges of North America. Studies indicate that this ruminant utilizes a considerable amount of four plant species known to be toxic for livestock: locoweed, *Astragalus* sp.; peavine, *Astragalus emoryanus*; woolly senecio, *Senecio longilobus*; and paper flower, *Psilostrophe* sp. Pronghorn feed on locoweed in the spring and summer and they sometimes then show neurological signs indicative of poisoning. No detrimental effects on the pronghorn have been ascribed to the other species of plants, even though they may provide a major portion of the forage during critical seasons (Beuchner, 1950).

Gastrointestinal modifications are the most common anatomical adaptations that mitigate the effects of plant toxins. Digestive physiologic adaptations are also important. The microflora and protozoa of the gastrointestinal tract are of extreme importance to all species. Herbivores, in particular, require assistance from microorganisms to utilize nutrients encased in lignin and cellulose. Wild herbivores have numerous gastrointestinal configurations but all are of two basic types: hindgut fermenters and stomach fermenters. Further background on this highly complex topic can be found in the paper by Moir (1968) and the monograph by Hoffman (1973).

Ruminants are considered to have the most highly evolved complex stomach arrangement of any mammal (Moir, 1968). Although all ruminants share a basic similarity, there is a gradation of complexity within the suborder Ruminata, from the chevrotain, *Hyemoschus* sp., to members of the Bovidae.

Ruminants are not unique in the development of complex stomachs or other gastrointestinal fermentation organs. Certain marsupials, particularly the macropods, have evolved complex stomachs in parallel with ruminants (Moir, 1968). Other examples exist among rodents, edentates, lagomorphs, perissodactylids, sirenians and simple-stomached artiodactylids. Examples among primates are langurs, *Presbytis* sp. and colobus monkey, *Colobus* sp.

Lagomorphs and some other species practice coprophagy. The animal consumes a fecal pellet enveloped by a mucoid sheath composed primarily of bacterial cells arising from the cecum (McBee, 1971). The bacteria are recycled, digested and utilized by the animal.

Some animals are also able, in concert with their resident bacteria, to degrade numerous secondary plant compounds that could otherwise be lethal. Species vary in the ability to respond to different plant environments with a shift in bacterial populations (Freeland and Janzen, 1974).

Numerous plant substances have an inhibitory effect on rumen microorganisms. The essential oils from the needles of Douglas fir, *Pseudotsuga menziesii*, contain 40 different chemical compounds. Some of these compounds have pronounced antimicrobial action on rumen microorganisms. In one study, the antimicrobial action was most pronounced in domestic sheep, followed by black-tailed deer which had never been exposed to fir needles before the study. Deer which had been reared in a fir habitat suffered the least from the essential oils (Oh et al., 1967). In another study, black-tailed deer browsed heavily on seedlings and new buds of Douglas fir, yet avoided mature fir needles containing substances that have inhibitory effects on the rumen (Longhurst et al., 1968).

Black-tailed deer may browse California bay, *Umbellaria californica*, and sagebrush, *Artemisia tridentata*, both of which contain bacteriostatic compounds which inhibit rumen function. If deer are forced to subsist on diets which are high in these plants, mortalities occur (Longhurst et al., 1968).

Artificially reared fawns showed similar forage preferences to their free-ranging counterparts when first introduced to native forages in a feeding experiment. This indicates a genetic basis for forage selection (Longhurst et al., 1968).

New Zealand's flora evolved without interaction with populations of large herbivores. When European livestock and wild species were introduced, the animal populations increased exponentially. Plants were not equally vulnerable to animal exploitation, but more species had lower resistance to animals than might be expected had plant and herbivore populations coevolved (Howard, 1967; Longhurst et al., 1968).

Well-nourished animals are more likely than poorly nourished animals to support a gastrointestinal microflora that is capable of detoxifying SPC. A shortage of either carbohydrate or nitrogen in the diet reduces the richness of the flora (Longhurst et al., 1968). Cattle which are fed a ration including concentrates can cope with nitrate levels in the diet that would be lethal to animals fed a maintenance ration. It is obvious that wild animals are frequently subjected to starvation or bare maintenance situations that may alter the gut flora and allow poisoning, which would not occur when better forage is available.

Once a toxic agent is absorbed from the gastrointestinal tract, the body must either excrete the substance unchanged, sequester it into a nonactive storage site, detoxify it by molecular rearrangement or suffer the ill effects of the toxicant. All vertebrates have general detoxification pathways that can deal with many different toxicants, such as alkaloids, glycosides, saponins, or tannins. Detoxification is accomplished chemically by oxidation, reduction, hydrolysis, esterification, N-dealkylation and conjugation. No vertebrate could exist in the general environment without the operation of these mechanisms. Besides the general mechanisms, species may develop a specific detoxification system to deal with unique toxins (Buck et al., 1976).

Much detoxification is carried out by hepatic microsomal enzyme activity. More limited microsomal activity takes place in the kidney, intestinal mucosa, lungs, and skin (Freeland and Janzen, 1974).

There is good evidence that some detoxification mechanisms are inherent. Variation within an animal population may be continuous or discontinuous (Freeland and Janzen, 1974). All wild and domestic rabbits, *Oryctolagus cuniculus*, have some resistance to ingestion of plants containing atropine, but some individuals are able to eat the plant in bulk because they have inherited the ability to produce large quantities of the enzyme atropinase (Swain and Glick, 1943; Schmidt-Nielsen et al., 1955).

Factors other than genetics operate in detoxification systems. Only minimal quantities of a particular enzyme will be present unless the enzyme system has been stimulated by prior exposure to a toxicant. An animal may survive consumption of an otherwise lethal dose of a poisonous plant if it has experienced microsomal stimulation, which is induced by eating non-lethal amounts of the plant. Free-ranging wild herbivores eating a variety of foods have an opportunity for repeated non-lethal exposure to many SPC, and are thus more capable

of coping with toxicants than are livestock or captive-reared wild animals (Freeland and Janzen, 1974).

The efficiency of microsomal enzyme systems may also depend on age, size, sex and the reproductive status of an animal. Young animals have less fully developed microsomal enzyme systems than older animals. Older animals may also have more materials to draw from for conjugation. Sex hormones may either enhance or diminish the effects of various toxins. Cortisol increases the capacity for detoxification by microsomal enzymes (Freeland and Janzen, 1974).

The overall picture of detoxification is compounded by the potential interaction of time as a result of the ingestion of multiple SPC at the same time. One compound may detoxify another or there may be a synergistic effect that produces a more severe poisoning. Also, metabolites from SPC may alter an animal's overall metabolism and affect the action of other compounds (Freeland and Janzen, 1974).

Prior ingestion of some toxic substances is not always beneficial. If the toxicant is cumulative, tolerance resulting from small doses cannot develop. Examples include bracken fern, *Pteridium aquilinium*, rubberweed, *Hymenoxys* sp. and orange sneezeweed, *Helenium hoopesii* (Laycock, 1978).

EXAMPLES OF SPECIFIC TOXINS AND METHODS OF COPING

Oxalates

Soursob, *Oxalis cernua*, grows in south Australia and may contain up to 14.5% oxalates (Everist, 1974). In certain areas this plant provides almost the total diet of cattle and sheep. Cattle are never poisoned by consumption of soursob, whereas sheep may be. This is especially true early in the grazing season, when ruminal bacteria lack the capacity to degrade the oxalates. If consumption is low and prolonged, the sheep gradually become tolerant to the oxalic acid and can ultimately eat the plant with impunity (Dodson, 1959). That this tolerance is based on rumen degradation is indicated by the fact that tolerant sheep can be poisoned by sodium oxalate dripped into the abomasum or bloodstream (Dodson, 1959). Most microorganisms do not utilize oxalates, but a few bacteria and molds do. If sheep, cattle and, presumably, other ruminants are given sufficient time, the oxalate utilizing microorganism populations build up and offer protection to the host. This has been demonstrated in wild rodents also (Shirley and Schmidt-Nielsen, 1967).

Experiments with packrats, *Neotoma albigula*, sand rats, *Psammomys obesus* and hamsters, *Mesocricetus auratus*, demonstrate that these desert-dwelling species can utilize plants containing soluble oxalates which are toxic for non-adapted species. The oxalate moiety is degraded by intestinal microorganisms, freeing calcium ions which can then be absorbed and utilized by the rodent (Shirley and Schmidt-Nielsen, 1967).

Pyrrolizidine alkaloids

Pyrrolizidine alkaloid poisoning is a serious problem for livestock managers in many arid to semiarid grazing areas of the world. Cattle and horses are highly susceptible, while sheep are much less vulnerable.

Pyrrolizidine alkaloids are not toxic themselves but are converted by liver microsomal enzymes to pyrroles which are hepatotoxic. The conversion is modified by genetic ability for microsomal enzyme action, a diet which may modify production of enzymes, and exposure to agents causing depletion of enzymes (Shull et al., 1976; Johnson, 1978).

Domestic mammal and bird species differ substantially in the rate of hepatic pyrrole production from pyrrolizidine alkaloids (Shull et al., 1976). Rapid pyrrole production is generally correlated with increased susceptibility to alkaloid poisoning. The status of wild animal resistance or susceptibility is unknown.

In one black-tailed deer feeding trial with senecio as a diet component, no lesions or clinical signs were noted after 42 days on the feed and a total senecio consumption of 24% of the deer's body weight. Cattle and horses would have been adversely affected with this level of intake (Dean and Winward, 1974). However, white-tailed deer have been known to become sick and die from the effects of pyrrolizidine alkaloid poisoning. Drought conditions in a coastal marsh of Louisiana forced white-tailed deer to graze on *Crotalaria* sp. and *Heliotropium* (Seger et al., 1969). Other species may also be affected. Hepatosis typical of pyrrolizidine alkaloid poisoning has been seen when red kangaroos (*Megaleia rufa*) have been grazing

on plants containing alkaloids (Hartly, pers. comm.).

Phytoestrogens

An interesting aspect of the toxic effects of SPC on wild mammals involves possible direct action on the reproductive system. It is known that many plants contain chemicals that are structurally and functionally similar to gonadal steroids. Other plant compounds, though structurally unlike estrogen of animal origin, may produce estrogenic effects (Labov, 1977). The term phytoestrogens is used to describe both types of compounds. Phytoestrogens are widely distributed and may have profound effects on wild animal populations.

Reproductive success in California quail, *Lophortyx californicus*, was negatively correlated with the presence of phytoestrogens in forage plants. During dry years when food supplies were scanty, many forbs contained high levels of phytoestrogens. Egg production and hatching success was low (Leopold et al., 1975). This appeared to be a natural population regulation mechanism to control fertility during years when food was scarce.

Some studies have correlated poor reproduction in rodents with estrogenic plant compounds. An early question was posed as to whether the plant effect resulted from a nutritional deficit rather than hormonal stimulation. Subsequent field and laboratory investigations support the thesis of direct hormonal activity. In montane voles, *Microtus montanus*, retarded ovogenesis and decreased uterine size was noted as a result of ingestion of phenolic plant compounds late in the summer season, especially during dry years (Berger et al., 1977).

Reports of rodent studies point out that litter size varies with habitat. In the Japanese vole, *Microtus montebelli*, the number of embryos averaged 6.0 in voles living in fallow rice fields in Japan whereas litters of the same species living in a cypress plantation nearby averaged only 3.8 (Kaneko, 1978). Differences in litter size were not caused by malnutrition or genetic defects, but were determined to be caused by the effects of phytoestrogens in the plants consumed in the cypress plantation.

No studies have been conducted on large wild herbivorous mammals to determine whether or not reproduction is enhanced or inhibited by consumption of phytoestrogens. The thesis that a type of population control involving phytoestrogens may be in operation in wild populations is intriguing but untested. Phytoestrogens may also alter behavioral patterns which in turn may alter reproductive performance.

Fluoroacetate

Resistance to plant toxicity has been used as a genetic marker to trace the evolutionary history of Australian mammals (Oliver et al., 1977, 1979; King et al., 1978). The native habitat of the western grey kangaroo, *Macropus fuliginosus*, and the brushtailed possum, *Trichosurus vulpecula*, is in areas of southwestern Australia where plants of the genera *Gastrolobium* and *Oxylobium* grow. These plants produce fluoroacetic acid which is acutely toxic to nonadapted mammals. Both of the foregoing species have innate resistance to this toxicant. Subspecies of western grey kangaroos, which have been isolated from fluoroacetate-containing plant areas, remain resistant to the toxin. Eastern grey kangaroos, *M. giganteus*, historically lived in areas lacking fluoroacetate-producing plants, and are highly susceptible to its toxic effects (Oliver et al., 1977, 1979). Other examples of subspecific variation in tolerance to fluoroacetates include the tammar, *Macropus eugenii*, and brush rat, *Rattus fuscipes* (Oliver et al., 1977). In resistant species, parenterally administered fluoroacetate was detoxified as readily as that orally administered, indicating that fluoroacetate is not detoxified by gastric microorganisms (Oliver et al., 1979).

WILD ANIMAL MORTALITY CAUSED BY POISONOUS PLANTS

Usually, when wild animals die from the effects of poisonous plants, an ecological balance has been disrupted. Frequently the cause is determined to be lack of suitable forage. Following are some reports of poisoning incidents.

Rocky Mountain elk are affected by locoweeds which are the first green plants to appear in the spring on one range in Colorado. The plants persist throughout the summer. Rumen content studies verify that the elk eat locoweeds (Adcock and Keiss, 1969) and locoweed poisoned elk have been observed showing weakness, depression, muscular tremors, stumbling and incoordination. At necropsy, microscopic lesions were typical of the locoweed toxicity seen in horses and cattle (swelling and fine vac-

uolation of the cytoplasm of neurons, accompanied by axonal dystrophy) (Adcock and Keiss, 1969).

Sika deer, *Cervus nippon*, forced to eat needles, bark and roots of jack pine trees (*Pinus banksiana*) during a drought, were poisoned by essential oils (Hayes and Shotts, 1958). A case of hydrocyanic acid poisoning was reported in Missouri. White-tailed deer had consumed the leaves and grain of *Sorghum vulgare* (Case and Murphy, 1962). White-tailed deer, as well as livestock species, are susceptible to the toxic effects of bluegreen algae accumulations in lakes and ponds (Brandenburg and Shigley, 1947).

Bongo, *Boocercus eurycerus*, a large, bamboo forest antelope of central and east Africa, was reported to be poisoned by setyot vine, *Mimulopis solmsii*, family Acanthaceae (Glover et al., 1966). After flowering every seventh year, the setyot vine dies back. According to tradition, during the second year after the die-back the setyot causes heavy mortality in bongo herds. Field biologists have noted diarrhea and death in bongo and giant forest hogs, *Hylochoerus meinertzhageni*. Preliminary toxicological studies indicated that extracts of the plant collected during the "second year" were also lethal to mice (Simon, 1962).

Koala, *Phascolarctos cinereus*, have been reported to be poisoned by cyanide from leaves of eucalyptus species not commonly eaten by the koala (Everist, 1974). Lesions similar to those of Harding grass (*Phalaris tuberosa*) poisoning in sheep have been noted when red kangaroos graze pastures containing this plant (Hartley, pers. comm.).

CONCLUDING REMARKS

The foregoing presentation is of value only if the information can be applied to management practices that will minimize deaths caused by poisonous plants. Just as wild animals are translocated to alien habitats, so may poisonous plants be introduced to new locations. Native wildlife are then exposed to SPC to which they have neither resistance nor experience to avoid. *Halogeton glomeratus* was introduced to the Great Basin of western United States prior to 1934, causing severe losses in sheep on winter range. It is not known how many deer (*Odocoileus* spp.) or bighorn sheep (*Ovis canadensis*) were affected before an ecological balance was achieved (Kingsbury, 1964).

Changes in ranch management may have a profound effect on the potential for plant poisoning. Pronghorns in one area of Texas were known to migrate from the Marfa Flats to nearby hills to seek browse when drought conditions destroyed the forbs on the flat. Ranchers changed fences from barbed wire to netted fencing, essentially incarcerating the pronghorns to specific ranches, or even to specific pastures. During a drought in 1964–1965, 60% of the 484 pronghorns died from a combination of starvation and consumption of tarbush (*Fluorensia cernua*) (Hailey et al., 1966).

Overstocking and deterioration of ranges enhances the risk of poisoning for both livestock and wild species. Agencies concerned with range management regulations should develop contingency plans for solving this serious problem.

Wildlife biologists must appreciate the more subtle potential effects of SPC. An unexplored facet of plant poisoning in wild animals is the possibility that teratogenic effects on the fetus may cause resorbed feti, abortion or deformed young. Cattle, sheep, horse, and swine feti are affected in varying degrees by ingestion of poison hemlock (*Conium*), lupine (*Lupinus*), corn lily (*Veratrum*), tobacco (*Nicotiana*) and sudan grass (*Sorghum*) (Keeler, 1978). Are wild mammals immune or do they not feed on these plants or ingest a sufficient quantity to cause deleterious effects? Studies to determine such facts are virtually impossible to conduct except in a laboratory feeding trial. It's difficult, if not impossible, to reproduce a set of circumstances duplicating or approaching natural conditions which might result in ingestion of a particular poisonous plant. In addition, in many instances wild species will starve before consuming the plant to be studied.

Native wild animals are better able to cope with native poisonous plants than are livestock or introduced wild animals. Nonetheless, even the most efficient detoxification or degradation system may be overpowered in an animal forced to consume poisonous plants because of starvation or other nutritional imbalances. The potential for plant poisoning and other naturally occurring toxins should be considered along with nutrition and infectious and parasitic dis-

eases when developing wildlife management plans.

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