SOME THEORETICAL IMPLICATIONS OF POISONOUS PLANTS AND SOUTHEASTERN DEER DISEASES*

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Toxicologists generally do not recognize a poison as such, but primary consideration is for the quantity of a specific product which has been acquired. As an example, for many years strychnine has been incorporated in medicinal tonics, but when consumed in excessive quantities this drug is a deadly convulsant. Most city residents breathe carbon monoxide daily without immediate harm, but under circumstances whereby the intake of this gas reaches a certain level, its lethal qualities are certain. It has been established that when common table salt is ingested in large enough quantities, it can be a potent poison. Even a benign substance like water can be toxic if taken in sufficiently large amounts.

Approximately 100 species of poisonous plants are known to flourish in the southeastern United States. Many of these are found in abundance throughout wide areas, and others are relatively scattered in distribution. There is considerable variation in the degree of toxicity of most plants, and the components of each species differ in their toxic principles. The amount of rainfall, the season of the year, and even the types of soils are factors which influence the quantity of poison present in a given plant.

The flora of virtually all white-tailed deer (*Odocoileus virginianus*) habitat is partially comprised of one or more poisonous plants, but these animals remain healthy in their presence. It also is known that wild deer ingest certain amounts of lethal compounds, and the manifestation of harmful after effects does not occur. Not only do deer eat a great variety of known toxic materials, but many plants containing such principles constitute a portion of the natural browse of these animals.

Observations of this type phenomenon often are misleading. In essence, wild deer might appear to be "immune" to many toxic compounds: e.g. cyanide, trematol, gelsemine, andromedotoxin, and others. This is unlikely. In view of the striking similarity of the digestive systems of these animals with those of domestic sheep, goats, and cattle, it is inconceivable that deer should possess a special resistance, while the latter species do not have any aspect of a similar tolerance. In retrospect, the annual loss of livestock in the United States attributed to the ingestion of poisonous plants has been estimated at millions of dollars.

Published accounts on the ecological relationship of poisonous plants and deer are extremely limited, and we may be erroneously assuming that this facet of disease etiology is of little consequence to the field of game management. When more facts become available pertaining to the underlying causes of major deer "die-offs," it is possible that this consensus will be questioned. It is thusly proposed that some hypothetical situations be considered.

JOHNSON GRASS (*Sorghum halepense*)

This weedy grass is troublesome for farm crops and, with exception of the mountainous regions, it is common in most areas of the southeastern United States. It grows on open waste land, along roadsides, around fences and in cultivated fields. Under ordinary circumstances Johnson grass and related plants (Sudan grass) are not poisonous and frequently are used for hay and forage. Only during adverse environmental conditions do these grasses become the

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source of a deadly, rapid-acting cyanogenetic glycoside. Factors such as humidity, temperature and rate of growth influence the amount of toxins in the plants. Droughts, malnutrition, frost and heavy insect attacks markedly increase the toxicity of the sorghums, and as a result of such adversities fatal poisoning is not uncommon in livestock.

Dhurrin is the toxic glycoside of Johnson grass. When this compound is hydrolyzed by an enzyme, parahydroxy benzaldehyde glucose and hydrocyanic acid are formed. The latter compound (hydrogen cyanide) is one of the most lethal of all poisons, but even this can be consumed in small quantities without ill effects to an animal. There is a blood level of hydrogen cyanide, however, which is fatal for any mammal. The compound inhibits certain respiratory enzymes and death ensues suddenly. At necropsy the blood of the subject will be abnormally red.

Deer will ingest *Sorghum* spp. and it is not logical to assume that these animals have a resistance to cyanide poisoning. It thereby seems conceivable that during certain aberrant fluctuations in the plant life within an area, localized deer losses could occur from this type of poisoning. The condition most likely would be precipitated during a long dry season, in the late summer or early fall. Poisoning from this group of plants should be expected in the piedmont and coastal plains, and would be unlikely to occur in the mountain region. Serious and widespread deer "die-offs" from the cyanogenetic glucosides would not be anticipated; however, in restricted areas heavy losses are possible.

BRACKEN FERN (*Pteridium aquilinum*)

This fern grows in a majority of the physiographic provinces of the southeastern United States, but it is most abundant in the open pine forest of the coastal plains. Dry, sterile soils favor the growth of this plant. Although mature bracken fern leaves are not very palatable, in the late summer and fall herbivorous animals often resort to this plant as a source of food. This usually follows a scarcity of good grazing caused by drought in the late spring, and after animals have developed a taste for the fern, they frequently continue eating it even after other forage becomes available. Under such conditions, heavy losses have been numerous in the livestock industry.

Thiaminase has been extracted from bracken fern, however, the toxic constituent of this plant has not been definitely established. Recent work has shown that a water soluble fraction can be obtained from the plant, which will produce clinical signs that are very similar to those associated with the known bracken fern poisoning syndrome. It is thought that the toxic constituent of this fern inactivates thiamine (vitamin B_1), which is an essential component of the respiratory enzyme systems. Poisoning usually is of a chronic nature, and death may not occur for several months after the ingestion of toxic amounts of the plant. The most obvious necropsy finding associated with this type poisoning is bleeding from the natural body openings.

Wild deer would not be expected to eat bracken fern when sufficient browse is available. A scarcity of adequate food caused by drought in the late spring might induce individual animals to acquire a taste for these ferns, and it is very possible that many deer have died as a result. Poisoning from these plants would be most likely to occur in the fall and would probably be restricted to the coastal plains. Serious and widespread deer "die-offs" could theoretically result from this type poisoning.

WHITE SNAKEROOT (*Eupatorium rugosum*)

This perennial herb is abundant in the mountains of the Southeast, and frequently covers vast areas of the woodland floor. It is native and widespread from eastern North America, westward to Minnesota and Texas. The plant grows in ravines and valleys, rarely occurring outside of the forest, and often constitutes over 75 per cent of the herbaceous vegetation in many mountain coves. It is uncommon in the piedmont and rare over much of the coastal plains. In the late summer and autumn, especially when there is a shortage of other forage, domestic animals are known to eat the tops of this plant. Poison-

ing can occur from browsing during a single day or the toxin may accumulate over a period of time.

One of the higher alcohols, trematol, is the toxic ingredient of white snakeroot. Although trematol can be consumed in low quantities without pathological consequences, high blood levels of this compound have been responsible for heavy losses of cattle and sheep. In these animals the disease is known as "trembles" or "milk sickness," and the most noticeable symptom is trembling, especially in the muscles of the nose and legs. Labored respiration, weakness and finally an inability to stand also accompanies the syndrome. The most pronounced necropsy finding is a reddened and hemorrhagic, highly inflamed intestinal tract.

At the time of several major deer die-offs of the Southeast, wild deer were reportedly browsing heavily on white snakeroot. It is therefore possible that poisoning from this plant could be responsible for some of the major deer losses which have occurred in the Appalachian mountain region. This situation could be precipitated by a long, dry summer, and it would be expected to take place in August or September. This type poisoning should be restricted to the mountains and piedmont; it would not be expected to occur in the coastal plains province.

YELLOW JESSAMINE (*Gelsemium sempervirens*)

This high climbing woody vine is commonly found in parts of the piedmont and throughout most of the coastal plains regions, but it is uncommon in the mountains and interior plateau. It grows abundantly in open hammocks, thickets, swamps, cleared fields, along fence rows, around stumps and on rocky bluffs. Most of the leaves remain over winter on the vines of this plant, but they are not very palatable for animals. At times when other forage has been depleted, cattle, sheep, goats, horses and swine have been fatally poisoned by consuming the conspicuously green leaves of this plant.

Gelsemine, gelseminine, and other related alkaloids are the toxic constituents of the flowers, leaves and roots of yellow jessamine. These alkaloids are powerful depressors of the motor neurons of the brain and spinal cord, and death is caused by respiratory arrest. Necropsy lesions associated with gelsemine poisoning are not pathognomonic and chemical tests for determining the presence of the alkaloids are difficult.

When adequate feed is available, wild deer would not be expected to eat large quantities of yellow jessamine. During years of poor mast, however, it is conceivable that these animals might consume toxic amounts of this plant. Poisoning from these vines would be most likely in the late fall or early winter. Although the toxins are lethal, widespread deer "die-off" would be anticipated with this type poisoning. Losses would be expected only in areas heavily populated with deer, and particularly following a poor mast crop.

MOUNTAIN LAUREL (*Kalmia latifolia*)

This evergreen shrub is abundant in the woodlands of the mountains in eastern North America and it extends in scattered localities along hillsides and bluffs to within approximately 50 miles of the seacoast. The leaves of this plant are tough and leathery, and animals seldom ingest them if more palatable forage is available. In the late winter and early spring, however, when laurel is one of the few conspicuously green plants available, both livestock and wild deer will consume the twigs and leaves of this shrub. As a consequence, serious poisoning has occurred in sheep, goats, cattle and horses.

Andromedotoxin is the toxic principle of mountain laurel and related members of the Heath Family. This compound possesses powerful sympathomimetic properties and also acts as a hypnotic. Toxic doses of andromedotoxin are associated with profuse salivation, convulsions followed by paralysis, and death is caused by an inhibition of the respiratory centers. Poisoned animals have been known to recover after two days from the initial manifestation of clinical signs. Pulmonary edema is the major necropsy finding which is fairly prevalent in animals poisoned by this plant.

White-tailed deer browse *Kalmia* in varying degrees, but under most circumstances it is not taken in areas where rhododendron is plentiful. Sprout growths are more commonly eaten than the less succulent portions, and deer rarely use more than a few twigs and leaves. It is known that both laurel and rhododendron are poisonous to deer, but that they do not seem to eat, of their own free will, enough of either to exceed their tolerance for them. One investigator concluded that, "Rhododendron does not seem to visibly affect deer even when it represents their main diet; but mountain laurel is suspected of indirectly causing numerous deaths among younger deer." It resulted in weakening the deer which probably made them more susceptible to a variety of pathogens and to dog predation. How long deer can endure this intake of these toxins has not been determined. Deaths of this type would be expected to occur in late winter.

COMMENTS

With reference to both plants and the circumstances which have been described, the theoretical aspects of this presentation should be reiterated. There are no confirmed accounts of poisoning in white-tailed deer caused by any of the toxic components which have been considered. In fact, very little work has been conducted to either prove or disprove the significance which plant poisons may have relative to major deer "die-offs."

From food studies, impressions have been gained that wild deer possess an inherent resistance to practically all naturally occurring toxic materials. A wealth of information is available, however, which incriminates many plants as being the etiology for heavy losses to the livestock industry. In testing over 300 drugs, the authors have consistently found white-tailed deer to be less resistant than domestic goats to poisonous products.

During the past 50 years, thousands of white-tailed deer in the Southeast have succumbed to recurrent diseases of an undetermined origin. Although various ideas have been proposed and sporadic searches have been conducted to incriminate an infectious organism, these efforts have been of little avail. Neither have poisonous plants been incriminated in causing "die-offs" among deer. It is therefore logical that these should receive maximum consideration, and whenever an occasion again arises, attention should be given to the ecological role that poisonous plants might contribute to deer mortality. This will require long and intensive studies, but when enough data have been compiled, it seems feasible that many major deer "die-offs" can be predicted, and under some circumstances even preventative measures are conceivable.

FISHERIES SESSION

THE NATIONAL SCIENCE FOUNDATION AND RESEARCH IN FISHERIES

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The opportunity for discussing the activities of the National Science Foundation as related to fisheries is indeed welcome. Federal support of this field is an old story to many of you who are either engaged directly in government supported research or in the administration of such research projects. The greater proportion of financial assistance from other agencies is specified for the applied aspects or management of commercial and recreational resources. Funds available from them for fundamental biological investigations are limited or earmarked especially for projects of lengthy duration.

The National Science Foundation is unique among government agencies in that the research which it can support must be basic, not applied, research.