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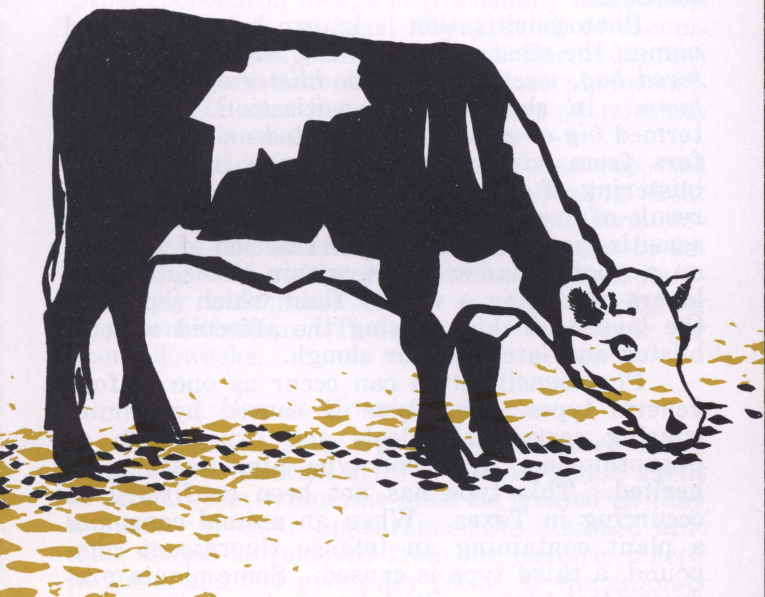
PHOTOSENSITIZATION OF LIVESTOCK in Texas



THE AGRICULTURAL AND MECHANICAL
COLLEGE OF TEXAS
TEXAS AGRICULTURAL EXTENSION SERVICE
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Brahman heifers with skin lesions on heads, sides and legs resulting from the blistering effects of photosensitization.

Scar tissue on udder and hocks of a dairy animal. Photosensitization and blistering were caused by grazing annual grasses.

A case of winter photosensitization. Eye, nose and mouth are inflamed. The brisket has accumulated a large amount of watery fluids.

The same animal 15 days later. The areas around the eyes and nose have peeled and some skin is sloughing from the dewlap.

A buckskin horse with the after effects of photosensitization. The scar tissue is sloughing from areas of the muzzle, face and neck.

Photosensitization of Livestock in Texas . . .

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What Is Photosensitization?

Photosensitization, a noncontagious disease of livestock, results from the abnormal reaction of light-colored skin to sunlight after a photodynamic agent has been absorbed through the animal's system. Grazing certain pasture and range vegetation under specific conditions causes photosensitization in Texas.

Many animals including cattle, sheep, goats, horses and swine may be affected by photosensitization.

Photosensitization is known by several local names, the most common being *bag trouble, blistered bag, weed trouble,* the *blistering* and *sandburn.* In sheep, photosensitization usually is termed *big or swell head.* Photosensitization differs from sunburn which is the burning and blistering of the outer layers of skin as a direct result of excessive exposure to sunlight. Photosensitization is caused by the reaction of sunlight on a photodynamic agent within the subdermal layers producing a watery fluid which separates the layers of skin causing the affected area to blister and later peel or slough.

Photosensitization can occur as one of four general types. One type is caused by administering certain chemicals and drugs such as phenothiozine. A second type, pink tooth, is inherited. This type has not been confirmed as occurring in Texas. When an animal consumes a plant containing an intense fluorescent compound, a third type is caused. Some plants producing this type are St. Johnswort, puncture vine, lantana, lechuguilla and sacahuista. The fourth type, which occurs widely in Texas, is caused when animals consume green plant material that produces phylloerythrin in the digestive tract. This is not thrown off as waste through the liver, bile duct and kidneys.

After the toxic substance has been absorbed, it is carried to the capillary networks of the skin. In sunlight, the light-colored areas become reddened. Animals become extremely sensitive to sunlight and spend most of the daytime under shade. The first noticeable signs are kicking, scratching, switching of tail and head, rubbing against objects and licking and biting the affected parts. The reddened areas may ooze a yellowish fluid. At first, the droppings are hard, indicating fever and constipation, but later they may be thin and watery. Some animals pass a dark-brown or cherry-colored urine.

Animals may become lame because of the swelling of the soft tissues of the feet. When forced to walk, the animals move with a high-stepping gait and may shake the foot before putting it down.

Severity varies among animals. Lesions or scabs may develop later in or on the muzzle, nostrils, eyelids, ears, anus, vulva, flanks and udder. The teats may be covered with watery blisters, with later scab formation and peeling. Secondary infections frequently occur in the eyes of light-colored cattle. Internal disturbances of the liver apparently are present in the early stages of the disease before the external areas are affected. The blistering and peeling actually is an outward result of the internal damage. Dark-colored animals are affected with the disease but they usually do not blister and peel. Sensitivity to sunlight and weight loss are the most noticeable signs in dark-colored animals.

Economic Loss

The blistered skin usually sloughs leaving raw areas which are highly susceptible to secondary infections, especially screwworms. Light-pigmented cattle seldom die from the disease, but death loss is more frequent in sheep and dark-colored cattle. Monetary losses in cattle are caused by excessive weight loss, damaged udders and teats, screwworms, secondary infections and eye damage. Stunted calves are common when the mother's udder and teats are sore and the calf is not allowed to nurse. Hooves may slough in severe attacks. Death loss and severity of secondary infections can be reduced with proper care in the early stages of the disease. Individual animals vary in susceptibility to photosensitization.

Where Does it Occur?

Photosensitization has been recognized for many years and its occurrence is world wide. In this country, many states have reported it in varying degrees.

Photosensitization has occurred for many years in all sections of Texas. In the western part, photosensitization is caused by lechuguilla and sacahuista. Big head in sheep has been reported from eating puncture vine and also from grazing lush green small grains. The disease has occurred in cattle grazing moldy common Bermudagrass along with new vegetative growth. The disease has occurred on soils from deep sands to dark-colored clays.

When Does it Occur?

Photosensitization may occur at any time, but outbreaks in spring and summer are larger and more serious. Most of the outbreaks studied

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in the southeastern part of Texas occurred within 2 weeks of rains and rising temperatures. In most cases, the pastures had been grazed short and annual grasses and weeds had made rapid growth. The combination of drouth, rainfall, temperature and increased growth usually prompts photosensitization in South and East Texas. It has never been observed on good condition, native perennial grass ranges, with abundant forage.

When animals consume large quantities of new, watery green plant material without dry roughage their bodies become upset and the toxic photodynamic agents are not eliminated. A few animals or an entire herd may become affected at one time.

Some grasses giving positive photodynamic symptoms in feeding trials were fringeleaf paspalum, prairie cupgrass and grass bur. Symptoms also occurred when most of the vegetation was composed of a mixture of annual grasses. Photosensitization has occurred on pastures with the vegetation composed of either burclover, ryegrass, oats, Sudangrass, rescuegrass, several winter annual grasses or a combination of these plants.

The disease also occurs in late winter or early spring on improved, cured common Bermudagrass pastures following rain and damp weather which causes mold to grow on the old grass. Other cool-season forage plants usually had grown sufficiently for grazing. The grazing of a combination of moldy cured common Bermudagrass and new green material may produce photosensitization. If any one of the factors is missing, the disease probably will not occur.

In Florida, photosensitization was reported to have occurred 3 to 8 weeks following frost which killed the top growth of common Bermudagrass. As the standing dead grass began to deteriorate, mold and green growth appeared simultaneously with some of the animals developing photosensitization. The pastures were toxic for about 8 weeks.

How to Treat the Disease

No specific drug will prevent the occurrence of photosensitization after a photodynamic agent is eaten. The symptoms must be treated as they appear. At their appearance, the diseased animals should be removed from the pasture, placed in the shade and given dry feed. If the animals have blistered and peeled, the affected parts should be painted or sprayed with a 2 percent methylene blue water solution or any other nontoxic water soluble dye. The methylene blue solution prevents rays of the sun from penetrating the skin. It also promotes drying and

aids healing of the affected parts. Salves and oils tend to retard the healing of the affected parts and *should not be used.* Sodium thiosulfate, at the rate of 1 ounce per 100 pounds live weight of the animals, administered intravenously or orally, has alleviated signs of photosensitization in Florida.

The toxic affect appears to last only a few days on closely grazed, poorly conditioned, native pastures in Texas. It appears that when the annual grasses have stopped quick growth and contain a higher content of fiber, the toxic effects of the plants are minimized. On cured moldy common Bermudagrass pastures in Texas, it is not known how long the toxic effect will last. When the new growth has attained the height where cattle graze only the new forage and not the moldy material with the new growth, the toxic effect seems to stop.

Management of Pastures

The time of year during which photosensitization may occur cannot be predicted definitely. When there has been an outbreak, the operator should record the conditions under which it occurred and prepare for future outbreaks. A reserve of good-quality roughage to be fed during outbreaks is advisable. When the lush green pastures have to be grazed following dry periods, feed ample dry roughage so that the animals will have an adequate balance of forage for proper rumination. After the lush green forage has attained sufficient growth to contain fiber, the feeding of hay may be discontinued. On native pastures in poor condition, defer grazing during the growing season to improve the pastures and possibly obtain better perennial native grasses. In all areas subject to photosensitization, proper stocking and moderate utilization to improve the native forage are recommended.

On improved common Bermudagrass pastures, practice rotation grazing to obtain proper use of pasture forage. Growth not needed for grazing should be baled or stored as a feed reserve. Photosensitization may develop when moldy hay is fed. The removal of animals from the cured common Bermudagrass pastures at the critical growth period in the spring may be necessary.

Good range and pasture grazing management practices should eliminate the problem of photosensitization.

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