Northeast Florida Livestock Agents' Group

Livestock & Forages Field Day

July 26, 2018

Santa Fe River Ranch
29220 NW 122 Street
Alachua, Florida 32615

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NORTH FLORIDA
LIVESTOCK AGENTS GROUP

2018 Livestock & Forages Field Day

July 26, 2018

Agenda

- 8:00am -8:45am Registration
- 8:45am Educational Rotations (Pick 4 to attend)
  - Pesticide Calibration - Dr. Kevin Korus & Dan Fenneman
  - Weed Control - Clay Cooper & Luke Harlow
  - Mineral Supplementation for Cattle - Paulette Tomlinson
  - Importance of Vaccinations for Beef - Jane C. Griffin
  - Importance of Vaccinations for Horse - Alicia Halbritter
  - Forage Systems - Dr. Marcelo Wallau
- 8:45am – 9:15am Round 1
- 9:20am -9:50am Round 2
- 9:55am – 10:25am Round 3
- 10:30am – 11:00am Round 4
- 11:00am – 11:15am Sponsor Break
- 11:15am – 11:45am Equipment Demonstrations
- 11:45am- Lunch
- 12:15pm- Weed & Herbicide Update- Dr. Brent Sellers

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Broadcast Boom Sprayer Calibration [1]

Thomas W. Dean and Frederick M. Fishel[2]

This document details all steps needed to estimate accurately in gallons-per-acre (GPA) how much a properly calibrated boom sprayer will apply.

Introduction

One of the most important tasks for a pesticide applicator is making sure the correct amount of pesticide is applied to the target site. Calibration is the process by which the amount of pesticide being applied per a unit of area is determined. Problems in calibration can lead to the application of too little or too much pesticide. Studies have shown that pesticides are often applied at rates the pesticide applicator does not intend. If, however, too little pesticide is applied, the pest may not be controlled as intended. Sometimes it's possible to repeat the application. In other cases, a repeat application may cause an overdose. Using more product than label directions recommend is illegal, costly, and will not control pests more effectively, but it will potentially leave harmful residues.

The section of the label "Directions for Use" will list the rate of application effective for control of the target pest(s). Often the effective level of application will be listed as a range of rates, depending upon various conditions, such as environmental factors and the target pest's stage of growth (Figure 1).

LEGUME VEGETABLES

Crops of Crop Group 6 (except soybean, dry) including:

Edible Podded and Succulent Shelled Pea and Bean and Dried Shelled Pea and Bean

Bean (*Lupinus* spp., including grain lupin, sweet lupin, white lupin, and white sweet lupin)

Bean (*Phaseolus* spp., including field bean, kidney bean, lima bean, navy bean, pinto bean, runner bean, snap bean, tepary bean, wax bean)

Bean (*Vigna* spp., including adzuki bean, asparagus bean, blackeyed pea, caljang, Chinese longbean, cowpea, Crowder pea, moth bean, mung bean, rice bean, Southern pea, urd bean, yardlong bean)

Pea (*Pisum* spp. including dwarf pea, edible-pod pea, English pea, field pea, garden pea, green pea, snow pea, sugar snap pea)

Other Beans and Peas (Broad bean (fava), Chickpea (garbanzo bean), Guar, Jackbean, Lablab bean (hyacinth bean), Lentil, Pigeon pea, soybean (immature seed), Sword bean)

PESTS CONTROLLED	Rate fluid ounces/Acre	Rate lb ai/A
Aphids Whiteflies	4.0 - 5.0	0.06 - 0.08

Notes and Restrictions

Pre-Harvest Interval (PHI): 1 day (edible podded and succulent beans and peas); 7 day (dry shelled beans and peas)

Minimum interval between applications: 7 days

Maximum MOVENTO allowed per crop season: 10 fluid ounces/Acre (0.16 lb ai/A)

Figure 1. Example of rates listed in a pesticide labels "Directions for Use" section.
Credits: Crop Data Management Systems

Getting Started

Because not all nozzles output the same amounts, EDIS Publication PI23 *Boom Sprayer Nozzle Performance Test* (http://edis.ifas.ufl.edu/PI015) explains how to correct sprayers for variations in nozzle output. Before calibrating your broadcast boom sprayer, perform a nozzle uniformity check (Figure 2).

Basic Calibration Principles

Output of a broadcast boom sprayer is based on several variables:

- Pressure that forces liquid through the nozzle tip;

1. This document is PI-24, one of a series of the Agronomy Department, UF/IFAS Extension. Original publication date April 1998. Revised June 2008 and January 2014. Reviewed March 2017. Visit the EDIS website at http://edis.ifas.ufl.edu.

2. Thomas W. Dean, former assistant Extension scientist, Pesticide Information Office, Food Science and Human Nutrition Department; and Frederick M. Fishel, professor, Agronomy Department, and director, Pesticide Information Office; UF/IFAS Extension, Gainesville, FL 32611.

- Nozzle orifice (tip opening) size;
- Ground speed; and/or
- Spacing of nozzles on the boom or width of spray pattern.

Basic tools needed to calibrate a broadcast boom sprayer are the following: graduated measuring bucket or cylinder, stopwatch or watch with a second hand, two flags or stakes to mark a premeasured distance, and a tape measure or measuring wheel (Figure 3).

Figure 2. Checking the uniformity of output from a broadcast boom sprayer.
Credits: UF/IFAS Pesticide Information Office

Figure 3. Basic tools for sprayer calibration.
Credits: UF/IFAS Pesticide Information Office

Calibration Step-by-Step

Sprayer output is most commonly referred to in terms of gallons-per-acre (GPA). Determining GPA consists of two basic principles—amount of area covered by the sprayer in a given amount of time and amount of output from an average nozzle on the sprayer's boom. The following steps describe how a broadcast boom sprayer can be calibrated by collecting the liquid delivered from the average nozzle that is applying to 1/128th of an acre. Using this method, the number of ounces of liquid collected from the average nozzle will be equal to the number of gallons the sprayer's boom will apply to one acre, since there are 128 fluid ounces in one gallon. The steps in this process follow below.

Step 1. Determine swath-width-per-nozzle (in feet) as follows:

$$\frac{\text{Nozzle spacing in inches}}{12} = \text{swath width in feet}$$

Step 2. Since 1/128th of an acre = 340 ft^2, calculate the distance needed by your boom's nozzle spacing to make a test run that will treat 1/128th of an acre.

$$\frac{340\ \text{ft}^2}{\text{Swath-width-per-nozzle (in feet)}} = \text{test-run distance (in feet)}$$

Step 3. Select a place with enough space for your equipment to make a "rolling-start" approach at operating speed. Based on the calculation in Step 2, measure the test-run distance needed. Use flags, stakes, or some markers to clearly mark the beginning and end of the test-run area.

Step 4. Choose a gear setting, engine speed, and pump pressure. Record these values for future reference.

Step 5. Fill the sprayer's tank with clean water, then approach the test-run area at operating speed with the boom shut-off valve open (sprayer applying clean water).

Step 6. Using a stopwatch or a watch with a second hand, begin timing the moment the sprayer enters the test-run area. As precisely as possible, stop timing when the sprayer passes the end flag of the test-run area. For added precision, repeat the test and average the times. It's easier to have a second person conduct the timing (Figure 4).

Step 7. Record the number of seconds elapsed during the test run. If there is any doubt about the time, repeat Step 6 until you are confident of an accurate time.

Figure 4. Conducting a timed test run over a premeasured length.
Credits: UF/IFAS Pesticide Information Office

Step 8. Park the sprayer and set the brake, but keep the engine rpm at the same setting used on the test course. Continue to run the pump and keep the boom shut-off valve open, so the sprayer is still applying clean water.

Step 9. From the nozzle-uniformity check, select a nozzle whose output closely represents the average calculated for all nozzles on the boom. This calibration nozzle will serve as a good indicator of overall boom performance.

Step 10. Obtain a clean, graduated container marked in fluid ounces and a stopwatch or a watch with a second hand.

Step 11. For the same amount of time as it took to drive the test course, collect the spray output (water) from the calibration nozzle. Record the number of fluid ounces collected from the calibration nozzle during the test time.

Step 12. Interpret the results of your calibration nozzle's output based on the number of fluid ounces of water collected from the calibration nozzle during the test time. This number indicates the GPA being applied by the boom sprayer (Figure 5). For example, if you caught 20 fluid ounces, then operating the sprayer at the selected settings will apply 20 GPA. Likewise, collecting 47.2 ounces would mean an equivalent GPA of 47.2.

Figure 5. The number of ounces collected from the calibration nozzle is equivalent to the GPA.
Credits: UF/IFAS Pesticide Information Office

Additional Information

Fishel, F.M. 2005. *Boom Sprayer Nozzle Performance Test.* Gainesville: University of Florida Institute of Food and Agricultural Sciences. http://edis.ifas.ufl.edu/PI015

Ferrell, J. A., Sellers, B. A., and Leon, R. 2005. *Calibration of Herbicide Applicators.* Gainesville: University of Florida Institute of Food and Agricultural Sciences. http://edis.ifas.ufl.edu/WG013

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General Principles of Weed Management [1]

Vernon V. Vandiver, Jr. and David H. Teem[2]

In any discussion of weed control, there must first be a definition of a weed. Many definitions have been used; however, for the purposes of this guide a weed is defined as: any plant that is a hazard, nuisance, or causes injury to man, his animals or his desired crops. It should be obvious from this definition that almost any plant can be a weed under certain circumstances. For example, corn and soybeans are both crops and neither is generally considered a weed; however, if you are producing soybeans, and corn is present in your field it could then be defined as a weed. Likewise, a small mixture of Bragg soybeans in a field of Cobb soybeans is of little consequence if the beans are to be sold for oil; however, if you are attempting to produce certified Cobb soybeans, then the Bragg variety is a serious weed problem. Also, sugarcane would normally never be considered a weed; however sugarcane growing in the drainage ditches of a sod farm would have to be managed as an aquatic weed.

Whether you live on the farm or in the city, weeds either directly or indirectly influence your everyday life. Weeds reduce yields, quality, and interfere with efficient harvest. These reductions are eventually passed to the consumer, either in increased prices or in poor quality products. Weeds interfere with recreational activities in aquatic areas or in parks and playground areas. Weeds, such as poison-ivy and poison-oak, cause misery to many people and others, such as ragweed, result in hay fever, thus causing discomfort and increased medical expenses.

It is estimated that the current total losses to Florida agriculture due to weeds are approximately 400 million dollars per year. In the United States, it is projected that the current use of herbicides comprises more than 60% of the total pesticide sales. With this magnitude of agricultural losses and herbicide usage, this aquatic weed control guide has been prepared to aid in more efficient and effective weed control. Since weed control consists of more than using herbicides, this guide refers to other methods of weed control in addition to herbicides.

Classification of Weeds

Weeds may be classified as grasses, sedges, and broadleaf weeds. In addition, for the purposes of this guide, algae will be considered weeds. Weeds may be further classified by the length of their life cycle. The three basic life cycles of weed plants are annuals, biennials, and perennials. The classification of weeds based on the length of their life cycle may not be obvious on visual inspection, but may have a great

1. This document is SS AGR 123, one of a series of the Department of Agronomy, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida. Revised: November 1997. Reviewed: May 2002. Please visit the EDIS Web site at http://edis.ifas.ufl.edu.

2. Vernon V. Vandiver, Jr., Associate Professor and Extension Aquatic Weeds Specialist, Fort Lauderdale Research and Education Center, Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida; and David H. Teem, former Extension Agronomist, University of Florida, Gainesville, FL 32611.

impact on the selection and success of control procedures.

Grass Weeds

True grasses have hollow, rounded stems and nodes (joints) that are closed and hard. The leaf blades are alternate on each side of the stem, have parallel veins, and are much longer than they are wide. Some examples are crabgrass, goosegrass, crowfootgrass, sandbur, annual bluegrass, torpedograss, and vaseygrass.

Broadleaf Weeds

Broadleaf weeds are a highly variable group of plants, but most have showy flowers and net-like veins in their leaves. They are easy to separate from grasses due to their generally different leaf structure and habits of growth. Some examples of broadleaf weeds are cudweed, creeping charlie, henbit, spurges, burning nettle, pennywort, creeping beggarweed, cocklebur, sicklepod, and Florida beggarweed.

Sedges

Sedges are an important group of "grass-like" weeds; however, they are not true grasses and are characterized by a solid, triangular-shaped stem with leaves extending in three directions. There are annual sedges (some are often called water grass), and the predominant and difficult to control perennial sedges. Of the latter group, yellow nutsedge is yellow-green in color and reproduces by seed, rhizomes (underground stems), and tubers. The rhizomes radiate from the plant, with a single bulb or tuber at the end, which may produce a new plant. Purple nutsedge is usually smaller in growth habit than yellow nutsedge, has reddish purple seed heads and produces a series of bulbs on radiating rhizomes called "tuber chains".

Algae

The algae are a diverse group of plants that occur in a wide range of environmental habitats. They are photosynthetic plants that contain chlorophyll, have simple reproductive structures, and their tissues are not differentiated into true roots, stems, or leaves. They range from unicellular, or single cells, to fairly complex multicellular organisms. Some algae have such a complex growth form that they are mistaken for vascular plants - Chara would be one such example. The size of individual algal plants range from microscopic, unicellular species, which are approximately 0.000039 in. (0.0010 mm) in diameter to large filamentous marine algae that obtain lengths of over 100 ft.(30 m).

Annual Weeds

Annual weeds, as the names implies, complete their life cycle within one year. They germinate from seed, grow, mature, produce seed and die in 12 months or less. They may be annual grasses, sedges, or broadleaf weeds. In addition, their life cycle may begin at different seasons of the year. Thus, summer annuals emerge in the spring, mature, produce seed, and die before winter of each year. Weeds such as crabgrass and cocklebur are typical of summer annuals. Similarly, winter annual weeds sprout from seed in the fall, and complete their life cycle before summer of the next calendar year. Sowthistle, henbit, annual bluegrass, and chickweed are examples of winter annual weeds.

Biennial Weeds

Compared to annual weeds, biennial weeds are few in number. These weeds have a 2-year life cycle. They germinate from seed in the fall, and develop large root systems and a compact cluster of leaves during the first year. The second year they mature, produce seed and die. Examples of biennial weeds are cudweed, Carolina falsedandelion, wild carrot, and bull thistle.

Perennial Weeds

Weeds that live more than two years are perennials. They reproduce by vegetative parts such as tubers, bulbs, rhizomes or stolons (above-ground stems). Some also produce seed in addition to vegetative reproduction. During the winter season most live-over in a dormant state and many lose their above-ground foliage and stems. With the beginning of spring they regenerate from food reserves in their root systems. Torpedograss, nutsedge and various vines are members of this group of weeds. Florida betony is a perennial weed that under a sub-tropical climate, initiates its growth in the fall, grows during

the winter months and goes dormant during the heat of summer.

Perennial weeds may be further divided into groups based on the type of root system and their reproductive process:

- Simple perennials reproduce by seeds but root pieces disturbed by cultivation or other mechanical means will produce new plants. Florida betony, and certain trees and shrubs are characteristic of this group;
- Bulbous perennials reproduce by seed and above or below-ground bulbs. Yellow nutsedge and wild onions have their bulbs below ground while wild garlic has an above ground bulb.
- Creeping perennials which produce seed but also produce rhizomes or stolons. Bermudagrass, torpedograss and purple nutsedge produce these specialized stems (rhizomes and stolons) that act as food storage organs and can initiate growth at each node along the stem.

The perennial weeds are the most difficult to control because of their great reproductive potential and persistence.

In annual cultivated row crops, annual weeds, such as crabgrass, goosegrass, cocklebur and ragweed are the most prevalent. However, perennial weeds such as nutsedge, johnsongrass and common bermudagrass may also be severe problems. In pastures and rangeland, perennial weeds tend to dominate among the perennial forage crops. Weeds such as smutgrass, dogfennel, waxmyrtle and pricklypear are very competitive but would not survive in a cultivated, row crop situation. Similarly in fruit crops where the desirable plants are also perennial, weeds such as guineagrass, vaseygrass, and milkweed vine are perennial problems.

Methods of Weed Control

There are several methods of weed control, including mechanical crop competition, crop rotation, biological, fire, and chemical. Best weed control is usually achieved by a combination of two or more of these methods. Many times this combination of weed control methods is called an integrated weed control program.

Mechanical

One type of mechanical control is burial. This method is most effective on annual weeds in which all the growing points can be buried. Burial is usually less effective on perennial weeds which have underground stems and roots and are capable of regrowth from these underground storage organs. Another method of mechanical control is cultivation. The main objective in cultivation is to cut the root system of the weeds; deep cultivation should usually be avoided due to damage to the crop roots. Deep cultivation may also bring more weed seed to the surface where they will germinate. Most studies have shown that when weeds can be controlled without cultivation, there is no advantage to cultivating. In fact, there may be disadvantages such as drying out the soil surface, bringing weed seed to the surface, and disturbing the root system of the crop.

Mowing is another method of mechanical control. Mowing is usually most effective on tall growing annuals, and not as effective on short growing plants or perennials. The growth habit of the plant usually indicates how effective mowing will be. Since grasses grow from basal meristems, mowing is not usually an effective method of controlling these weeds. Perennial weeds which regrow from underground storage organs require frequent and usually long-term mowing for control. This is because the leaf area must be removed continually and not allowed to regenerate any carbohydrates for storage in order to starve the underground plant parts. Annual weeds are usually mowed to prevent seed production and to allow the crop a better competitive advantage.

Draglines or backhoes are frequently used for mechanical control of aquatic weeds in sites such as canals and ditches. Aquatic weed removal may be done when the canal or ditch is cleaned, removing silt and sediments to restore the original profile of the waterway, or as a separate operation using a special "weed bucket" on the equipment. A variety of floating, cutter and harvester machines are manufactured which can be used in many aquatic sites. The requirements needed for operating such

equipment are an access area to launch the boat, sufficient space to operate in the body of water, and sufficient water depth to float the equipment in the area of the proposed operation.

Plant and Crop Competition

Crop competition is usually one of the most economical and best methods of weed control; however, it is often one of the most overlooked methods. Weeds compete with crops for space, light, moisture, nutrients, and carbon dioxide. Usually the plant which starts first and is growing under ideal conditions will have the competitive advantage. Factors such as planting date, row spacing, seeding rate, planting depth, soil moisture, soil fertility, and soil pH have an influence on the competitive advantage of the crop or weed.

Most of our crop plants have been developed under conditions which were as near optimum as possible for that crop; therefore, everything that can be done to simulate these conditions for the crop plant should be in its favor. Since weeds have not been developed by plant breeders for specific conditions, they are often more tolerant of a wide range in conditions. Usually only one crop species is planted; however, there are many weed species available to compete with this crop. For example, as soil pH becomes higher or lower than its optimum for the crop there is usually a weed species which is tolerant of that pH. This is also true for factors such as fertility, soil moisture, and depth of emergence. Planting the crop at the optimum soil temperature, depth, soil moisture, soil fertility, and soil pH will allow it to emerge most rapidly, grow, and cover the row middles, thus reducing much of the weed competition by shading.

Plant competition is used successfully to control aquatic weeds in certain situations. For example, Chara has been planted and managed in South Florida canals to limit and suppress the regrowth of Hydrilla.

Crop Rotation

If the same crop is planted in the same field year after year there usually will be some weed or weeds which are tolerant and favored by the cultural practices and herbicides used on that crop. By rotating to other crops many of the cultural practices and herbicide programs are changed. This often will reduce the population of specific weeds which were tolerant in the previous crop.

Biological Control

Biological weed control as a practical tool has not been utilized to a great extent in controlling weeds. There have been certain instances of successful biological control programs; however, these have been infrequent. This is an area in which present and future research appears promising. Insects, disease, and nematodes do naturally suppress growth of certain plants, a continual process in the field. One area of weed science which should be recognized is how the use of fungicides, nematicides, and insecticides influence weed populations. If a plant, which is not a serious problem, is naturally suppressed by one of these organisms, will that plant become a weed if that organism is controlled?

There has been considerable work in the biological control of aquatic weeds. Three insects have been introduced into the United States, including Florida, for control of water-hyacinth. Additional insects have now been introduced to control other aquatic weeds in Florida. In addition, the Florida Game and Fresh Water Fish Commission has a permit system which enables the public to use grass carp for control of submersed, emergent, and other aquatic weeds. A permit must be obtained from the Florida Game and Fresh Water Fish Commission before anyone in Florida may purchase, possess, or use the grass carp for aquatic weed control purposes.

Fire

Fire is an old method of weed control, and in certain instances can be used to favor selectively certain species over others. Controlled burning can be useful to remove weeds from ditch banks, roadsides and other waste areas. Fire has been used for many years to favor the growth of pine seedlings over hardwoods. Special equipment for flaming is available. Fire is usually more effective on annual weeds than on perennial weeds and usually does not kill weed seed in the soil.

Chemical Control

Time of Application

The time of application is usually divided into three areas: preplant, preemergence, and postemergence.

Preplant

Preplant refers to applications made before the crop is planted. Currently, in most cases, these materials are incorporated into the soil and are called preplant incorporated treatments (ppi). The great advantage of these incorporated treatments is that the herbicide is placed in the zone where weed seed germinate and is not dependent on rainfall to move the herbicide into this zone. This type of treatment adds to the cost of incorporation and requires that the crop be tolerant of the herbicide, as the crop seed and the herbicide will be in contact. Examples of such herbicides are trifluralin, profluralin, benefin, and vernolate.

Preemergence

Preemergence treatments usually refer to applications made after the crop is planted but before it emerges. However, strictly speaking, preemergence may apply to other situations such as preemergence to the crop, preemergence to the weeds, or preemergence to both crop and weed. These preemergence applications are usually applied to the soil surface and require rainfall or irrigation to move the herbicide into the soil. If the herbicide is not moved into the soil where the weed seed are located it will not be effective. If left on the soil surface, these herbicides are often lost due to photodecomposition and vaporization.

Postemergence

Postemergence treatments are applied following emergence; however, as with preemergence it should be specified as to postemergence to the crop or weed. If the crop has emerged but no weeds are present then the application is postemergence to the crop but preemergence to the weeds and would be applied to the soil surface. If the crop has emerged and the weeds have emerged, then the application is postemergence to both weed and crop and would be applied to the foliage of the weeds.

Area of Application

The area to which chemicals are usually applied are described as band, broadcast, directed, and spot treatments, although some individuals may give more specific descriptions such as semi-directed, directed broadcast, and directed spray and recovery.

Broadcast Applications

As the name implies, broadcast applications cover the entire area. These treatments, while requiring the largest amount of chemical and highest cost per acre, usually result in the best weed control.

Band Applications

Band applications usually refer to treating a narrow strip directly over the row. This reduces the amount of chemical required and the cost per acre; however, with this type application the area between the rows is not treated and usually will require cultivation or chemical treatment later in the season.

Directed Applications

Directed applications are applied to a particular area or part of the plant. These applications are usually directed to the base of the crop plant and away from the leaves. The ability to use directed sprays usually depends on a height differential between the crop and the weed. If the crop is taller than the weeds then drop nozzles can be used to direct the spray treatment over the weeds but below the leaves of the crop.

Directed sprays are very useful in late season control of weeds and usually follow a preplant or preemergence application. In many cases preplant or preemergence applications do not persist long enough to control late germinating weed seed or may not be used on certain soil types. In such cases directed sprays are used to obtain effective weed control and improve harvest efficiency.

Spot Treatment

Spot treatments are used for weeds which are localized in certain areas but are not widespread over the entire area. When only isolated areas of weeds are present, this is the most economical and best method to control and prevent their spread to other areas.

Prevention

If effective weed control has been achieved using the previously discussed methods, one further step should be considered. This is preventing weeds from re-infesting the area.

Knowledge of how weeds enter the field is important. Weed seed may be distributed in crop seed, hay, straw, by wind, water, animals, machinery and other ways.

Fence rows, ditches, and ditch banks are often neglected when controlling weeds in crops. Weed seed produced in these areas may move into the field and start new weed infestations. As it is difficult to prevent weed seed from blowing into the field, or being carried by birds or water, if the weeds can be stopped from growing and producing seed in these adjacent areas, it will reduce the possibility of infestation or re-infestation.

Care should be taken not to move aquatic weeds from one body of water to another in boats, on boat trailers, and on mechanical equipment such as draglines. Many aquatic weeds have the potential to establish populations rapidly and cause problems in adjacent waters. Do not use mechanical equipment to control an infestation of a plant such as Hydrilla that covers only a small percentage of a body of water, as fragmentation of the plant can cause rapid infestation of the remainder of the water body.

Certified, registered, and foundation seed, or clean planting material cannot be over emphasized in preventing weeds from infesting fields. It is also important to clean equipment before entering fields or when moving from one field to another. Soil on tractor tires or other areas of equipment may contain large numbers of weed seed. Cultivators and mowers should be cleaned to prevent the movement of vegetative plant parts such as rhizomes and stolons from different areas in the fields or from field to field. Although a few plants remaining in the field or in fence rows, or in a body of water, may not appear significant it should be remembered that many weeds produce extremely large quantities of seed. For example, one redroot pigweed plant may produce as many as 117,000 seed or one mullein plant may produce over 220,000 seed. Hydrilla may produce over 10 million reproductive propagules (turions) per acre (0.405 ha).

Herbicides

Herbicides may be classified in several ways depending on where and how they are applied and their action in or on the plant. Herbicides may be either foliage applied or soil applied. They may kill by contact or may be translocated through a plant.

Herbicides may also be selective or non-selective. Some herbicides may be effective either foliage applied or soil applied. Whether a herbicide is selective or non-selective may depend on several factors, such as the crop or weed present, time of application, the method of application, and rate of application.

For example, paraquat would usually be described as a foliage applied, contact, non-selective herbicide; while atrazine would usually be described as a soil applied, translocated, selective herbicide.

Foliage Applied Herbicides

Foliage applied herbicides may be applied to the leaves, stems or shoots of plants. Herbicides that kill only those parts of the plants which the spray touches are contact herbicides.

The herbicide may be taken into the plant and moved through-out the plant, resulting in plant injury or death. These are translocated herbicides. For example, if a drop of paraquat were applied to the leaf tip of a young tomato plant then only that leaf tip would be killed; however, if a drop of 2,4-D were applied to the leaf tip of a young tomato plant then other areas of the plant would express symptoms due to translocation of the 2,4-D throughout the plant.

For foliage applied herbicides to be effective they must enter the plant and this entrance may be influenced by factors, such as the shape or orientation of the leaf, roughness of the leaf surface, pubescence (hairs) on the leaves, presence of wax, or the formulation of the herbicide. For example, it is difficult to obtain good coverage of plants with narrow upright leaves such as wild onion since the herbicide bounces or runs off, while plants with prostrate leaves such as wild mustard are much easier to cover.

The presence of pubescence on the leaf may result in a herbicide not reaching the leaf surface but remaining suspended on the hairs. In such cases, the addition of a surfactant may be useful in reducing the surface tension of the water droplet and allowing it to spread through the hairs onto the leaf surface where it may be absorbed.

On waxy leaf surfaces the formulation of the herbicide may be important. For example, the amine formulations of phenoxy herbicides are not highly soluble in wax and are not as effective on plants with waxy leaves as are ester formulations which are soluble in wax.

Contact herbicides are most effective on annual weeds but are not usually effective on perennials. Translocated herbicides may be effective on annuals or perennials.

Soil Applied Herbicides

Soil applied herbicides primarily enter plants through the root system and many factors influence the effectiveness of soil applied herbicides. Factors such as tolerance of the crop or weed to the herbicide, depth of weed and crop seed, amount of sand, silt, clay and organic matter of the soil, amount and time of rainfall, and temperature are a few factors which may influence effectiveness.

One example of herbicide tolerance is the use of atrazine in corn. When atrazine is applied to a field of corn, it may enter both the corn and the weeds; however, corn has the ability to detoxify atrazine, whereas many weed species cannot, thus resulting in selective weed control.

Although soil applied herbicides must be moved into the soil to be effective, the amount of movement in the soil can be used to achieve selectivity. For example, if the problem weeds are in the upper 1 inch of soil and the crop seed can be planted at a depth of 2 inches, then a herbicide which does not move rapidly in the soil, or can be incorporated to a depth of 1 inch, can be used, thus resulting in the crop seed remaining below the zone of herbicide.

Persistence of herbicides is extremely important in the duration of weed control, and also in determining what crop can be planted later in the season. The persistence of herbicides applied to the soil may be influenced by numerous factors such as microbial decomposition, chemical decomposition, adsorption, leaching, volatilization and photodecomposition.

Soil type is extremely important. Soluble herbicides applied to sandy soils may be rapidly leached out of the zone containing weed seed and into the zone of germination or roots of the crop thus resulting in poor weed control and damage to the crop. In fine textured soils or organic soils, herbicides may be so tightly adsorbed that they are not available for weed control or they may be released so slowly that they are not in a concentration great enough to kill the weeds. This lack of release may also increase the persistence of herbicides into the following crop in fine textured or organic soils.

Herbicides which specify incorporation into the soil may do so to improve the contact of the herbicide with the seed and also to minimize the loss of the herbicides by volatilization and photodecomposition. Some herbicides, if not incorporated soon after application may be lost from the soil surface.

One of the major factors influencing herbicide persistence in the soil are microorganisms. These organisms may use the organic herbicides as a source of carbon for their food supply. The specific herbicide, microorganism present, temperature, water, oxygen, and mineral nutrients are all important in the persistence of the herbicide.

Generally the fastest degradation of herbicides is achieved in warm, moist, well aerated, fertile soils at pH ranges favorable to crop growth. The greatest

persistence of herbicides may be expected if the soil is cold, dry, poorly aerated, and generally unfavorable to the growth of the microorganisms.

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SS-AGR-405

Brunswickgrass or *Paspalum nicorae*: A Weed Contaminant in Southern Pastures and Bahiagrass Seed Production Fields[1]

Ann Blount, Marcelo Wallau, Brent Sellers, Dennis Hancock, Leanne Dillard, Jose Dubeux, Cheryl Mackowiak, Joao Vendramini, and Clay Cooper[2]

Introduction

Brunswickgrass (*Paspalum nicorae* Parodi) is becoming a problematic weed in summer perennial grass pastures in the Southeast. This plant is native to southern Brazil, northern Argentina, Paraguay, and Uruguay. It was introduced into the US as a soil conservation plant for erosion control and as a potential forage crop. Brunswickgrass is well-adapted to moderately acidic, sandy soils, but it also grows well in sandy loam and well-drained, light to medium clay-based soils. The plant is competitive with bahiagrass and bermudagrass. Since it is less palatable, it can eventually dominate a perennial grass pasture. Brunswickgrass has become naturalized and has reportedly contaminated bahiagrass seed fields and pastures in the southeastern states, including some of the important counties for seed production in Florida, such as Gilchrist, Levy, Alachua, Citrus, and Sumter.

Cattle will consume brunswickgrass when it is young and tender. However, the plant quickly becomes rank and loses its palatability, and cattle avoid it. It proliferates when the more desirable forages have been overgrazed. As it thrives under reduced competition, it spreads and becomes more difficult to eradicate. Pastures contaminated with this grass will appear to have tufts or hills of plants where cattle refuse to graze (Figure 1). Because of the rhizomatous habit of the plant, those patches tend to increase in size year after year and eventually dominate the pasture.

Figure 1. Closely grazed bahiagrass pasture with patches of brunswickgrass in late September (toward the end of the growing season) in Levy County, FL.
Credits: Marcelo Wallau, UF/IFAS

1. This document is SS-AGR-405, one of a series of the Agronomy Department, UF/IFAS Extension. Written in cooperation with Johnny Melton, Jack Melton Family, Inc. Original publication date April 2018. Visit the EDIS website at http://edis.ifas.ufl.edu.

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During the seed cleaning process, brunswickgrass seed does not readily separate from Pensacola bahiagrass seed: both seeds are close in size and shape. This has made it difficult for bahiagrass seed processors to effectively eliminate brunswickgrass in order to meet total weed seed specifications (2.0%) for saleable seed. It is believed that brunswickgrass is more readily removed from Argentine bahiagrass due to differences in seed size. There has been an increasing number of reports of brunswickgrass infestations in pastures around the state, and certain measurements should be taken to reduce spread. The most effective way is to avoid infestation by using certified seeds when establishing new pastures. No herbicide management is currently recommended. However, in cases of high infestation, total systemic herbicide application followed by cultivation and crop rotation could be needed.

Appearance

Brunswickgrass is a perennial summer grass with a growing season and appearance similar to those of bahiagrass (Figure 2), especially during the vegetative phase. Brunswickgrass often has three to four racemes per seed head (Figure 3, left), while Pensacola bahiagrass (*P. notatum var. saurae* Parodi) typically has two to three racemes (Figure 3, right) (Hitchcock 1971).

Figure 2. Bahiagrass seed field contaminated by brunswickgrass. Species are similar, but can be differentiated by seed head.
Credits: Marcelo Wallau, UF/IFAS

Brunswickgrass has a deep and aggressive rhizome system that looks different from bahiagrass rhizomes. Brunswickgrass rhizomes occur below the soil surface (with a depth of approximately 4 in or 10 cm) and spread laterally (Figure 4 B), while bahiagrass rhizomes, which are sometimes referred to as stolons, spread along the soil surface (Figure 4 A).

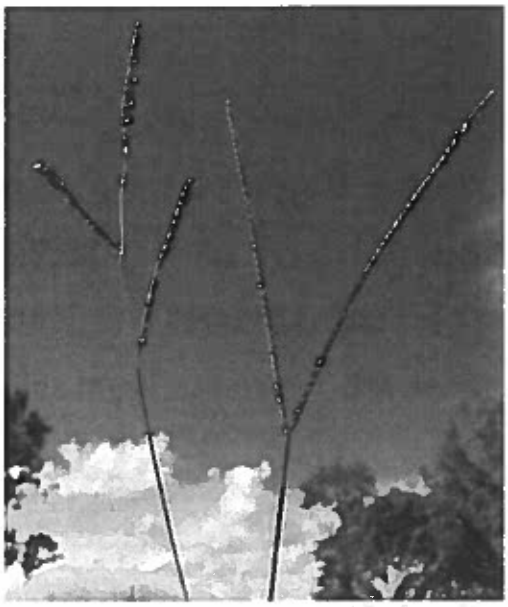

Figure 3. Seed heads of brunswickgrass (left) and bahiagrass (right).
Credits: Marcelo Wallau, UF/IFAS

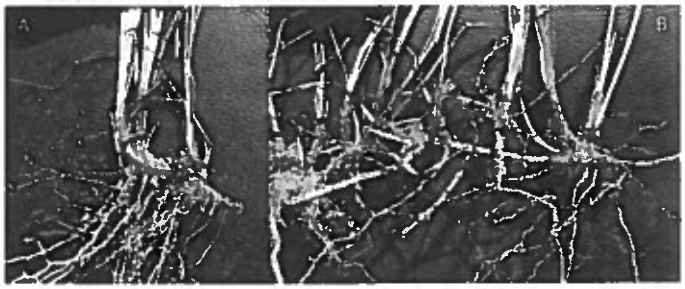

Figure 4. Rhizome comparison of brunswickgrass (right A) and Pensacola bahiagrass (left B).
Credits: Marcelo Wallau, UF/IFAS

Seeds are slightly smaller than those of Pensacola bahiagrass. The seed coat has a dark, chestnut brown center that varies somewhat in size, depending on the variety. The seeds are noticeably convex in shape compared to the relatively flat, tan seeds of Pensacola bahiagrass (Figures 5 and 6). Seed may average about 200,000 per pound, based on our estimates.

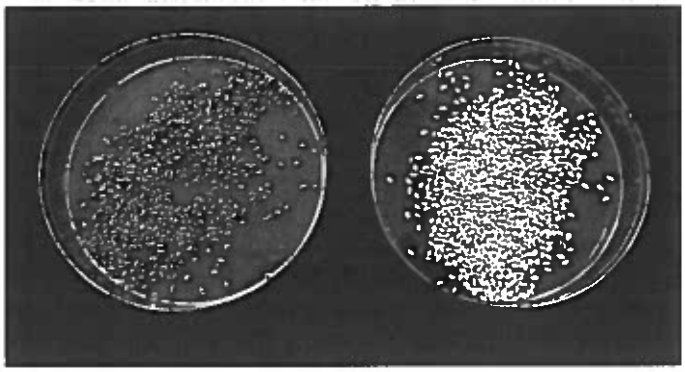

Figure 5. Seeds of brunswickgrass (left) and Pensacola bahiagrass (right).
Credits: Ann Blount, UF/IFAS

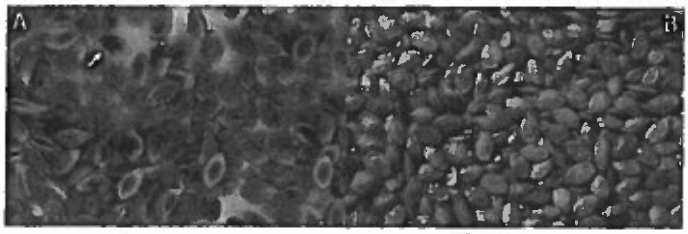

Figure 6. Close-up of brunswickgrass (A) and Pensacola bahiagrass (B) seeds. Note the brown-colored coat of the brunswickgrass seeds when removing the glumes (arrow on A).
Credits: Marcelo Wallau, UF/IFAS

Variety/Germplasm

Brunswickgrass (*Paspalum nicorae* Parodi) is synonymous with *P. plicatulum* Michaux. var. *arenarium* Arechav. It is sometimes referred to as *P. lepton* Shult. (Oliveira and Valls 2008). Two seed sources were released and promoted for conservation plantings by the Soil Conservation Service (presently Natural Resources Conservation Service—NRCS) from Plant Materials Center in Americus, GA (Belt and Englert 1999; NPGS GRIN Global 2016). 'Amcorae' (Origin: Argentina, Source: PI 202044, CPI 21370, ATF 1040) is a bluish-green, vigorous introduction released in 1969. A later release, 'Doncorae' (Origin: Brazil, Source: PI 310131, CPI 125877, ATF 1028), occurred in 1993. It has rapid seedling establishment, vigorous growth habit, and winter hardiness.

Management

Brunswickgrass is tetraploid, similar to Argentine-type bahiagrass. Control of this grass with herbicides will likely be more difficult because of its higher ploidy level. To our knowledge, no herbicides currently exist that will selectively remove brunswickgrass without severely injuring or killing the desirable pasture grass. Several recent herbicide treatments on brunswickgrass-infested Florida pastures do show some promise of possible chemical control. However, these are preliminary results that require further confirmation of successful eradication or reduced brunswickgrass stands. Systemic herbicides should be the most effective in killing brunswickgrass, due to the plant's strong rhizome system. High rates of glyphosate will likely be required to kill the pasture as the first step of total field renovation. Mechanical cultivation alone may not solve the problem; it may exacerbate the spread of brunswickgrass through rhizome segments. A combination of mechanical cultivation, herbicides, and crop rotation may provide successful control of brunswickgrass, since seed survival in the soil seed bank is not believed to be long-term.

The best preventive actions a producer can take to avoid further distribution of this grass are to refrain from harvesting contaminated production fields and to always use certified seeds when establishing new pastures. Certified ("blue tag") seed has been produced under strict production guidelines that minimize the risk of weed contamination. It is important to remember that large quantities of bahiagrass seed are sold without any field inspections for purity, resulting in the sale of some contaminated seed for use in new pasture plantings. Plan to purchase certified seed from a reliable seed source.

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Table 1. Comparison chart of brunswickgrass and Pensacola bahiagrass.

Characteristic	Brunswickgrass	Pensacola Bahiagrass
Growing season	April to October	April to October
Flowering	July to September	July to September
Height	8–28 in	4–24 in
Leaf shape	Linear, lanceolate, white mid-rib	Linear, lanceolate, crowded at the base with overlapped keeled sheaths
Leaf size	8–14 in long, 0.25 in wide, but highly variable	1–20 in long, 0.1–0.5 in wide
Leaf pubescence	Generally smooth, but can be hairy	Smooth leaves and sheath
Seed head	3 to 4 alternate racemes	2 racemes, Y-shaped
Seeds	Brown-coated, convex, hairy glumes when present	Tan-colored, relatively flat
Seed weight	Estimated 200,000 seeds/lb	Estimated 250,000–275,000 seeds/lb
Root system	Long, thin rhizomes	Short, thick, J-shaped superficial rhizomes

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SS-AGR-363

Identification and Control of Johnsongrass, Vaseygrass, and Guinea Grass in Pastures[1]

H. Smith, J. Ferrell, and B. Sellers[2]

Johnsongrass is a common perennial grass that grows throughout the South and Midwest. It is so common and well known as a troublesome weed that any large undesirable grass is often called johnsongrass. This is problematic because it is one of three perennial grasses found in pastures. Vaseygrass and guinea grass are often misidentified as johnsongrass but they have very different herbicide recommendations. Calling a plant johnsongrass when it is really vaseygrass or guinea grass can result in the wrong recommendation and lead to an expensive herbicide failure.

Identification: Johnsongrass, Vaseygrass, Guinea Grass

All three grasses have a prominent white midrib that extends the length of the leaf. But few similarities exist beyond this characteristic.

Growth Habit

All three grasses are perennial, but only johnsongrass has a creeping rhizome system and grows in patches rather than in individual bunches. Vaseygrass and guinea grass are both bunch-type grasses without a significant rhizome system. Additionally, vaseygrass is most commonly found in wet fields or along drainage ditches. Johnsongrass and guinea grass prefer dryer sites.

Seedhead

Johnsongrass and guinea grass have an open panicle seedhead that is angular. Color and size are the key differences between johnsongrass and guinea grass seedheads. Johnsongrass seeds are much larger and have a red/black mottled color, while the guinea grass seeds are smaller and somewhat green. Vaseygrass has a very different seedhead with alternating spikelets forming silky hairs around the seeds. Seeds are produced along the entire length of the seedhead branch, which does not occur in johnsongrass or guinea grass seedheads.

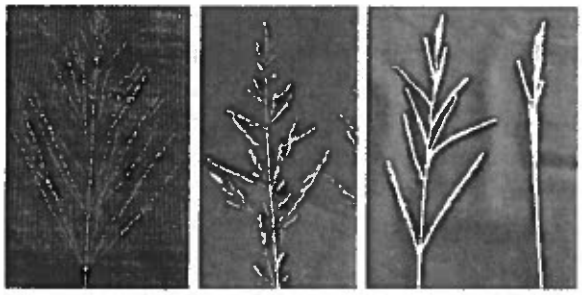

Figure 1. From left to right, guinea grass seedhead (Credits: Hunter Smith); johnsongrass seedhead (Credits: Brent Sellers); vaseygrass seedhead.
Credits: Brent Sellers

1. This document is SS-AGR-363, one of a series of the Agronomy Department, UF/IFAS Extension. Original publication date August 2012. Reviewed October 2015. Visit the EDIS website at http://edis.ifas.ufl.edu.

2. H. Smith, graduate assistant; J. Ferrell, professor, Agronomy Department; and B. Sellers, associate professor, Agronomy Department, Range Cattle Research and Education Center; UF/IFAS Extension, Gainesville, FL 32611.

Seeds

Guinea grass has small, oval, light green seeds, which often have wrinkles. Vaseygrass seeds have similar characteristics but are flatter, with the presence of hairs. Johnsongrass has much larger, pointed seeds that develop a reddish/brown tint as they mature.

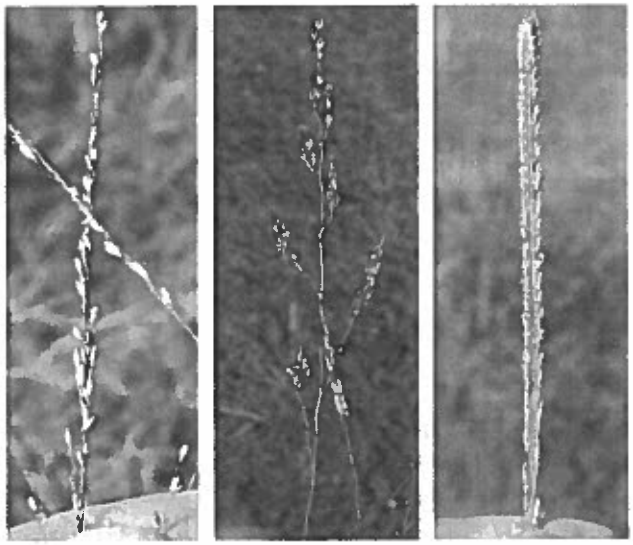

Figure 2. From left to right, guinea grass seedhead branch (Credits: Brent Sellers); johnsongrass seedhead branch (Credits: Hunter Smith); and vaseygrass spikelet.
Credits: Brent Sellers

Stems

The stems of johnsongrass and guinea grass can look very similar. Inspection of the stems will show scattered but abundant hairs along the stem of guinea grass. Stem hair on guinea grass varies because of the different biotypes. Johnsongrass stems are totally smooth with no hairs. Vaseygrass stems have hairs where the leaf meets the stem or on the stem toward the base of the plant. This is because vaseygrass will generally lose stem hairs as the stems elongate.

Leaves

Johnsongrass leaves have a large white midrib and a smooth, glossy appearance. Guinea grass leaves have a less prominent white midrib, and the undersides are rough with stiff hairs. Vaseygrass leaves are long and narrow with an indented midrib and crinkled leaf margins.

Roots

A fifth and final identification method is to pull or dig up the roots. All three of these grasses are perennial, but johnsongrass has large white rhizomes that are easily seen if the plant is well established. Vaseygrass and guinea grass have smaller, more fibrous root structures compared to johnsongrass.

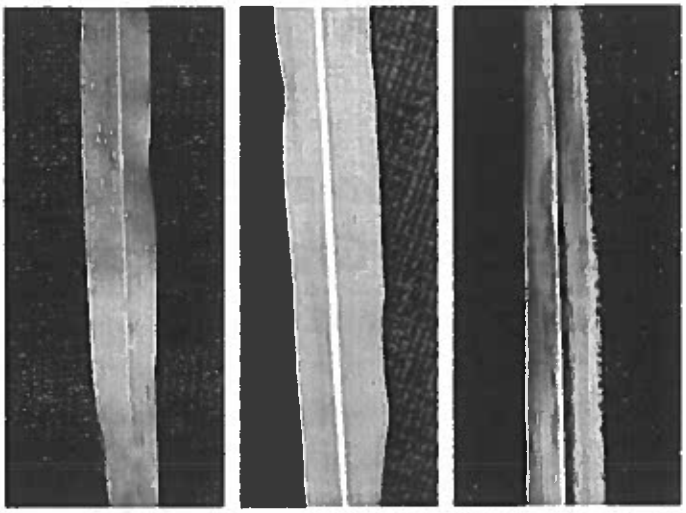

Figure 4. From left to right, guinea grass leaf blade; johnsongrass leaf blade; vaseygrass leaf blade.
Credits: Hunter Smith

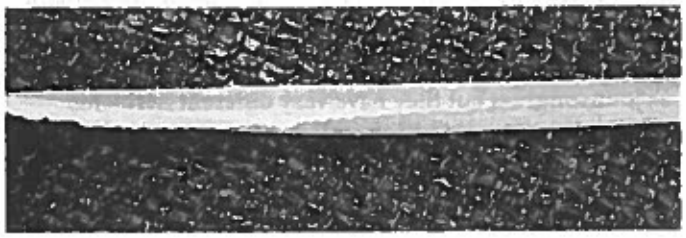

Figure 5. Vaseygrass leaf margin.
Credits: Hunter Smith

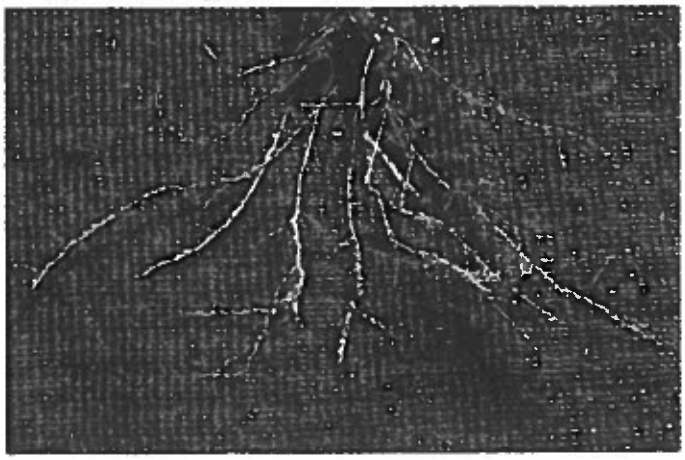

Figure 6. Guinea grass root structure
Credits: Hunter Smith

Control

Johnsongrass

Outrider: For best johnsongrass control, apply 1.33 ounces per acre when grass is actively growing and is at least 18–24 inches tall, up to the heading stage.

Impose (bermudagrass only): Use 4–6 ounces per acre on johnsongrass less than 24 inches. Higher rates can be used, but unacceptable injury on bermudagrass will likely occur.

Although 4 oz of Impose can control johnsongrass, some regrowth should be expected on older stands that are large at the time of application.

Pastora (bermudagrass only): Use 1 oz/A on seedling johnsongrass (rhizomes < 18") and 1.5 oz/A on mature stands. Bermudagrass injury will occur with Pastora, but will be less than that observed with Impose. Maximum application rate of Pastora is 2.5 ounces per acre per year.

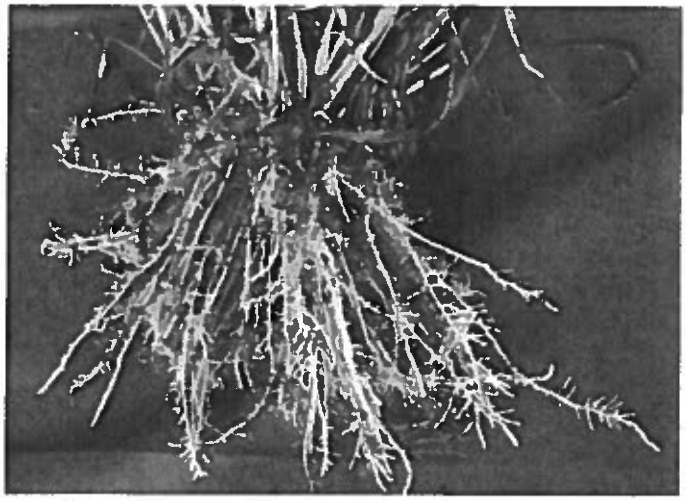

Figure 7. Vaseygrass root structure
Credits: Brent Sellers

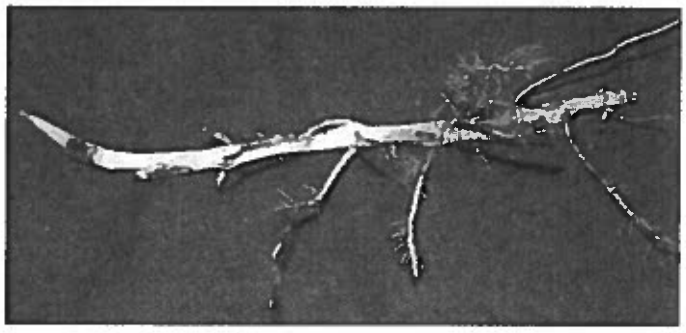

Figure 8. Johnsongrass rhizome
Credits: Brent Sellers

Vaseygrass

Impose (bermudagrass only): Vaseygrass control can be accomplished by using 6–8 ounces per acre. This rate of Impose will be highly injurious to bermudagrass and one cutting of hay will likely be lost. This injury can be minimized if the application is made immediately after hay removal and before the bermudagrass leaf-out. Additionally, do not apply Impose until after the first hay cutting when rainfall is common.

Glyphosate: Spot spraying with 1% solution (1.2 oz/gal) can be effective. Care should be taken to avoid contact with desirable grasses.

Guinea Grass

Glyphosate: Spot spraying with 1% solution (1.2 oz/gal) can be effective. Care should be taken to avoid contact with desirable grasses.

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SS-AGR-18

Smutgrass Control in Perennial Grass Pastures[1]

Brent Sellers, Neha Rana, and José Luiz C.S. Dias[2]

Introduction

Smutgrass (Figure 1)—an invasive bunch grass, native to tropical Asia—is a serious weed of improved perennial grass pastures, roadsides, natural areas, and waste areas in Florida. Results of a survey conducted by the South Florida Beef Forage Program in 2003 indicated that smutgrass ranks as the second-most problematic weed species in Florida pastures, behind tropical soda apple (which is the most problematic weed). However, because practices to control tropical soda apple have been widely adopted in Florida since that survey was conducted, it is likely that smutgrass has by now become the most problematic weed species in Florida pastures.

Two smutgrass species are found in Florida—small smutgrass (*Sporobolus indicus*; Figure 2) and giant smutgrass, which is also known as West Indian dropseed (*Sporobolus jacquemontii*; Figure 3). Small smutgrass was once the predominant smutgrass species throughout Florida. By the 1990s, however, giant smutgrass had become the most common smutgrass species throughout central and south Florida. Giant smutgrass continues to move northward in Florida.

Figure 1. Smutgrass infestations are common in bahiagrass pastures throughout Florida.
Credits: B. Sellers, UF/IFAS

Mature smutgrass plants are unpalatable to livestock, but some grazing of mature smutgrass does occur. New regrowth of smutgrass, similar in quality to that of bahiagrass, is grazed for two to three weeks after burning or mowing. However, it is difficult to graze cattle on smutgrass due to the need to rotate cattle among smutgrass-infested paddocks so that growth of the smutgrass does not reach a stage where cattle will not graze the plants.

1. This document is SS-AGR-18, one of a series of the Agronomy Department, UF/IFAS Extension. Original publication date February 2000. Revised April 2011, January 2015, and February 2018. Visit the EDIS website at http://edis.ifas.ufl.edu.

2. Brent Sellers, associate professor, Department of Agronomy; Neha Rana, former graduate research assistant, Department of Agronomy, UF/IFAS RCREC, Ona, FL; and José Luiz C.S. Dias, graduate research assistant, Department of Agronomy; UF/IFAS Range Cattle Research and Education Center, Ona, FL. Original authors included M. B. Adjei, associate professor and P. Mislevy, professor, both formerly of the Agronomy Department based at the UF/IFAS Range Cattle Research and Education Center, Ona, FL.

Figure 2. Small smutgrass, once the most troublesome smutgrass species in Florida, is still found throughout the state.
Credits: B. Sellers, UF/IFAS

Figure 3. Giant smutgrass first became a problem in south Florida and is spreading north through the state. In central and south Florida, giant smutgrass is more problematic than small smutgrass.
Credits: B. Sellers, UF/IFAS

Biology

Both smutgrass species—small and giant—are perennial bunch grasses. Average bunch size of small smutgrass is approximately 8–10 inches in diameter while giant smutgrass diameter is approximately 12–18 inches.

Small smutgrass has a compact seedhead (Figure 4) with the panicle branches touching the panicle. The small smutgrass seedhead is almost always infected with a black fungus. Small smutgrass plants produce approximately 1,400 seeds per seedhead and 45,000 seeds per plant.

Figure 4. Small smutgrass seedheads are appressed near the raceme, giving the seedhead a cylindrical appearance.
Credits: B. Sellers, UF/IFAS

In contrast, giant smutgrass has an open seedhead with panicle branches directed somewhat upward (Figure 5). The seedhead of giant smutgrass is usually not infected with the black fungus, but giant smutgrass plants are sometimes infected with this fungus. Little information exists concerning seed production of giant smutgrass, but some indications suggest this plant may be a more prolific seed producer than small smutgrass.

Seed production of both species occurs throughout the growing season, and new seedheads are produced shortly after mowing or burning. The seeds, which are red to orange in color, remain attached to seedheads for some time after maturing and are spread by adhering to livestock and machinery or by movement via water and wind. Natural seed germination has been shown to average less than 9%, and seed are thought to remain viable in the soil for at least two years.

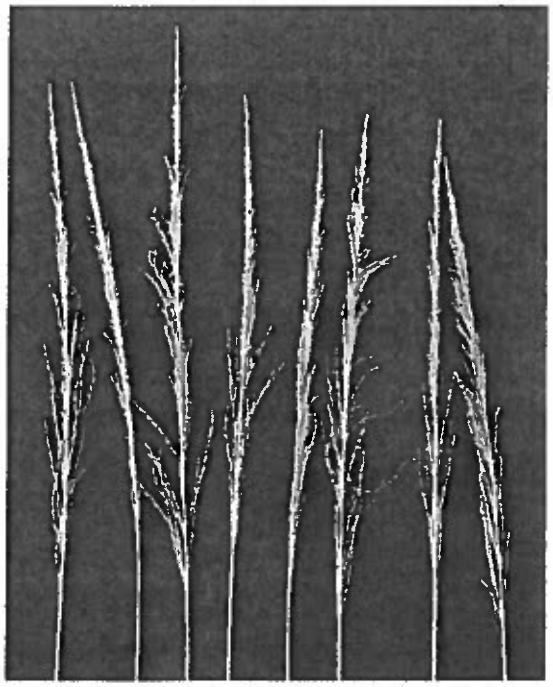

Figure 5. Giant smutgrass seedheads are open and generally not infested with the black smut fungus that typically infests small smutgrass, but sometimes smut fungus is also found on giant smutgrass.
Credits: B. Sellers, UF/IFAS

Control

Cultural practices to control smutgrass species have not been successful to date. Mowing decreases the diameter of the clumps, but often results in increased density. Burning is thought to increase the germination of seeds in the soil seed bank. However, both burning and mowing allow for approximately two to three weeks of grazing. Smutgrass forage quality during this two- to three-week window is often equal to or slightly greater than bahiagrass.

Chemical control of smutgrass includes applying hexazinone at 1.0 lb/acre (Velpar/Tide Hexazinone at 2 qt/acre; Velossa at 1.67 qt/acre) to small and giant smutgrass. A surfactant may be added to Velpar (Velossa contains a surfactant), but recent research has indicated that a surfactant is not necessary since the herbicide works primarily by root uptake. Mowing smutgrass prior to hexazinone application does not increase control. Hexazinone should be applied from June through September, when rainfall is typically sufficient to move the herbicide into the root zone for uptake. There is little foliar activity from hexazinone on smutgrass. If rainfall does not occur within a one-week period after application, the herbicide treatment will likely fail. The same can be said for rainfall that exceeds 3 inches within one week after application. There are no grazing restrictions for hexazinone if the application rate is below 1.13 lb/acre. However, there is a 38-day haying restriction.

Hexazinone is a highly effective herbicide but is also expensive. Experiments were recently conducted to determine when hexazinone should be applied to maximize smutgrass control, especially in light of the best timing for application to realize return on the herbicide investment. An economic analysis indicated that hexazinone should not be applied until smutgrass density is approximately 50 percent of the area of a pasture. Applications of this herbicide prior to this level of infestation will not result in enough additional bahiagrass biomass (i.e., ability to increase stocking rate) to justify the cost of application. However, in terms of preventing smutgrass infestation, it may be economically justifiable to spray highly infested areas of a pasture, even before 50% of the entire pasture is infested.

Oak trees are extremely sensitive to hexazinone, and care should be taken to stay at least 100 ft away from oak trees. If smutgrass is present under or near oak trees, spot applications of 3% glyphosate are effective.

Forage Grass Tolerance

Bahiagrass will turn slightly yellow about 15–20 days after spraying with hexazinone at the recommended rates. However, bahiagrass will recover and turn dark green within about 40 days. This green color will be darker than the non-treated pastures. Bermudagrass will turn yellow with some necrosis for approximately 30 days before new green growth occurs.

Recommendations

General

- Do not apply hexazinone within 100 feet of oak trees because application within this range may cause death of the tree.
- Read the Velpar, Tide Hexazinone, or Velossa label for complete instructions on reapplication interval, safety, grazing, and haying restrictions.
- Cattle may graze treated pastures if applications are less than 4.5 pt/acre Velpar or Tide Hexazinone and 3.75 pt/acre Velossa.
- To realize economic gains from hexazinone application, smutgrass infestation should be approximately 50 percent of pasture.
- If the initial smutgrass density covers more than 80 percent of the pasture area (if 8 out of 10 regular steps

touch the base of smutgrass plants), complete renovation of the pasture should be considered.

Bahiagrass/Bermudagrass Pastures

- Graze pasture in the spring until the beginning of the rainy season.
- Apply 2.0 qt/acre Velpar/Tide Hexazinone (1.67 qt/acre Velossa) during the summer rainy season but not later than the end of September. Apply when plants are actively growing and rainfall is dependable and consistent.
- Fertilization after hexazinone application will increase forage production and allow the desirable grass to quickly fill the open areas created by dying smutgrass.

Floralta Limpograss

- Hexazinone is not currently labeled for smutgrass control in limpograss.

Stargrass

- Hexazinone is not currently labeled for smutgrass control in stargrass.

Mulato

- Hexazinone is not currently labeled for smutgrass control in Mulato as it will be severely injured—DO NOT USE.

Pasture Renovation

In highly infested bahiagrass pastures where smutgrass groundcover exceeds 70%–80%, pasture renovation should be considered. Spray the entire pasture with 4 qt/acre glyphosate and begin tillage practices no earlier than three weeks after application. Repeated tillage will destroy newly emerged smutgrass and will aid in depleting the soil seedbank. The final seedbed should be a smooth, flat surface devoid of vegetation. For additional information on bahiagrass varieties and seeding rates, see EDIS publications AG342/SS-AGR-332, *Bahiagrass (*Paspalum notatum*): Overview and Management* (http://edis.ifas.ufl.edu/ag342) and AG107/SS-AGR-161, *Forage Planting and Establishment Methods* (http://edis.ifas.ufl.edu/ag107).

Even with repeated tillage following glyphosate application, smutgrass will likely emerge with bahiagrass, and smutgrass seedheads will be present by the following summer growing season. One year after seeding and during the rainy season, apply 0.5 lb/acre hexazinone (Velpar/Tide Hexazinone at 32 oz/acre or Velossa at 27 oz/acre). Recent research has suggested that hexazinone application one year after seeding resulted in >90% control of smutgrass for two years after application. However, the newly renovated pasture should be scouted the following year, and a second application of hexazinone may be warranted if smutgrass densities remain high.

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SS-AGR-52

Cogongrass (*Imperata cylindrica*) Biology, Ecology, and Management in Florida Grazing Lands[1]

B. A. Sellers, J. A. Ferrell, G. E. MacDonald, K. A. Langeland, and S. L. Flory[2]

Cogongrass is found on every continent and is considered a weedy pest in 73 countries. In the U.S., cogongrass is found primarily in the Southeast. It was accidentally introduced into Alabama in the early 1900s, and purposely introduced as a potential forage and soil stabilizer in Florida (and other states) in the 1930s and early 1940s. However, soon after investigations began it was realized that cogongrass could be a weedy pest. Since its introduction, cogongrass has spread to nearly every county in Florida. In some cases, it has completely taken over pastures so that it is the only species present. This is a common thread where cogongrass invades; it quickly displaces desirable species and requires intensive management.

There are many reasons why cogongrass is such a prolific invader. It is a warm-season, perennial grass species with an extensive rhizome root system. In fact, at least 60% of the total plant biomass is often found below the soil surface. In addition to the rhizome root system, cogongrass adapts to poor soil conditions, and its fires burn so hot that they eliminate nearly all native species. Cogongrass is drought tolerant and has prolific wind-dispersed seed production. Additionally, it can grow in both full sunlight and highly shaded areas, although it is less tolerant to shade.

Cogongrass spreads through its creeping rhizome system and seed production. The rhizomes can penetrate to a depth of 4 feet, but most of the root system is within the top 6 inches of the soil surface. The rhizomes are responsible for long-term survival and short-distance spread of cogongrass. Long-distance spread is accomplished through seed production. Seeds can travel by wind, animals, and equipment. Seed viability is significant in north Florida and other states of the Southeast; however, there are no confirmed cases of viable seed production in central and south Florida.

An established cogongrass stand invests heavily in its perennial root system. These infestations are capable of producing over 3 tons of root biomass per acre. This extensive network of rhizomes is capable of conserving water while the top growth dies back during prolonged drought. This is essentially a survival mechanism to keep the rhizome system alive. Another key to cogongrass invasion is that the root system may produce allelopathic chemicals that reduce the competitive ability of neighboring plants.

Identification

Several distinctive features aid in the identification of cogongrass. First, cogongrass infestations usually occur in circular patches. The grass blades tend to be yellow to green in color (Figure 1). Individual leaf blades are flat and

1. This document is SS-AGR-52, one of a series of the Agronomy Department, UF/IFAS Extension. Original publication date April 2002. Revised August 2012. Reviewed October 2015. Visit the EDIS website at http://edis.ifas.ufl.edu.

2. B. A. Sellers, associate professor, Agronomy Department, Range Cattle Research and Education Center; J. A. Ferrell, professor; G. E. MacDonald, professor; K. A. Langeland, professor; and S. L. Flory, assistant professor, Agronomy Department; UF/IFAS Extension, Gainesville, FL 32611.

serrated, with an off-center prominent white midrib (Figure 2). The leaves reach 2–6 feet in height. The seed head (Figure 3) is fluffy, white, and plume-like. Flowering typically occurs in spring or after disturbance of the sward (mowing, etc.). Seed heads range from 2 to 8 inches in length and can contain up to 3,000 seeds. Each seed contains silky-white hairs that aid in wind dispersal. When dug, the rhizomes (Figure 4) are white, segmented (have nodes), and are highly branched. The ends of the rhizome are sharp pointed and can pierce the roots of other plants.

Figure 1. Cogongrass plants are yellow to green in color. Note that the edges of the leaf tend to have more yellow than green.
Credits: G. Keith Douce, University of Georgia, www.forestryimages.org.

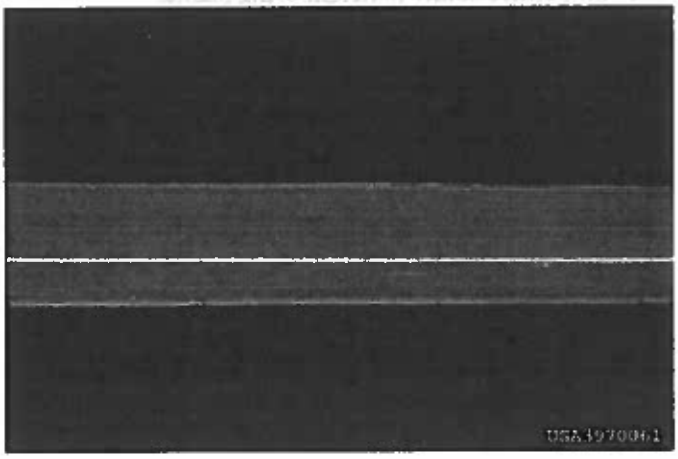

Figure 2. Cogongrass leaves have serrated edges and a prominent, white, off-center midrib.
Credits: L. M. Marsh, Florida Department of Agriculture and Consumer Services, www.forestryimages.org.

Figure 3. Cogongrass seed heads are fluffy and white. Each plant produces nearly 3,000 seeds.
Credits: John D. Byrd, Mississippi State University, www.forestryimages.org.

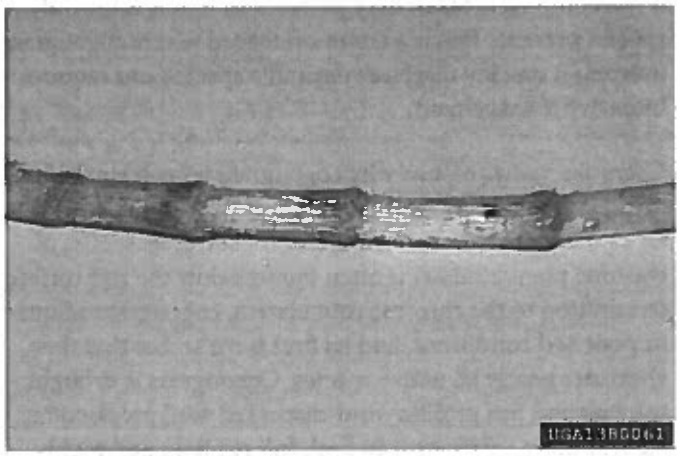

Figure 4. Cogongrass rhizomes are segmented (have nodes) where new shoots are able to grow.
Credits: Chris Evans, River to River CWMA, www.forestryimages.org.

Forage Value

Cogongrass has been used in Southeast Asia as forage because it is the dominant vegetation on over 300 million acres. In these areas it was found that only very young shoots should be grazed or cut for hay. At this stage, the leaves lack sharp points and razor-like leaf margins. For about four weeks following a prescribed burn, crude protein of regrowth is comparable to bahiagrass. Crude protein of mature stands rarely attains the minimal 7% level needed to sustain cattle, making supplementation essential for livestock production. Cogongrass yields are relatively

low, even under heavy fertilization, and usually do not exceed 5 tons per acre.

Management

For many years researchers all over the world have studied cogongrass control. During this time nearly all available herbicides have been tested, but few effective products have been found. For example, all of the commonly used pasture herbicides such as metsulfuron, 2,4-D, triclopyr, Velpar, and other combinations have little to no activity on cogongrass. Only glyphosate (Roundup, etc.) and imazapyr (Arsenal, Stalker, etc.) herbicides have been found to be effective, but long-term control is rarely achieved.

Imazapyr is an extremely effective herbicide that controls a variety of weeds, from herbaceous to woody species. One or two applications of imazapyr (0.75 lb/acre) will often effectively control cogongrass for 18–24 months. However, there are several disadvantages to using this herbicide. First, imazapyr will severely injure or kill forage grasses such as bermudagrass and bahiagrass. It also has a long soil half-life and will remain in the soil for several months after application. This often leads to "bare ground" for up to 6 months in the application area because of the non-selective nature of this herbicide. Imazapyr also has the potential to move down slopes during periods of rainfall, killing or injuring other species in the runoff area (oaks and other hardwood trees are especially sensitive). Second, imazapyr can only be used as a "spot-treatment" with no more than 10% of the pasture area treated per year.

Similarly, glyphosate is also a non-selective herbicide that effectively controls a variety of weeds. Unlike imazapyr, glyphosate possesses very little to no soil activity. Non-target effects caused by runoff during high rainfall events are not likely. Since glyphosate has no soil activity, it does not take very long for weeds or desirable grasses to reinfest the treated areas. Cogongrass will likely reinfest the area if only one application of glyphosate is applied during the same year. Research in Alabama has revealed that it takes approximately three years of two applications per year to reduce cogongrass rhizome biomass by 90%.

Small Infestations

Early detection of cogongrass in any setting is extremely important. A young infestation will be much easier to treat and eradicate than established infestations. In this case, we would define a small patch as 20–30 feet or less in diameter. Even for a small patch, monitoring is required after the initial application to ensure that any re-sprouting is quickly treated. See Table 1 for specific timelines and suggested herbicide rates.

Large Infestations

Large infestations are 30 feet or larger in diameter. These types of infestations can be considered as established and likely have a large, intact root system. This will require more herbicide treatments to completely eradicate cogongrass. See Table 2 for specific timelines and suggested herbicide rates.

Integrated Management

Herbicide inputs alone are rarely successful in eradicating perennial species like cogongrass. In these cases, we need to use all of the tools we have to remove an unwanted species to reestablish a desirable species. This type of strategy is best employed in an area where cogongrass has long been established and is the predominant species present. See Table 3 for specific timelines and suggested herbicide rates.

In general, burn the area infested with cogongrass in August to September. One to four months later, treat the burned area with a mixture of imazapyr and/or glyphosate. Take soil samples prior to spring tillage the next growing season to ensure that the soil pH is adequate for your desirable forage species. Till the treated area the following spring to a depth of at least 6 inches and prepare a seedbed.

Consult with your local county Extension agent to consider your options for forage cultivars and fertility recommendations. Getting a good start on the desirable forage will help limit cogongrass reinfestations in your pasture. Continue to monitor this area in six-month intervals until the fourth year. Spot treat with glyphosate when necessary to remove any new cogongrass growth.

Table 1. Herbicide suggestions for small infestations of cogongrass in grazing areas. This includes both improved and native rangeland. These concentrations are good for mixing in small (3–30 gallon) sprayers. Please read the entire label of the suggested products prior to treating existing cogongrass stands.

	Timing	Herbicide Rate	Application Notes
1st year	Fall (August–November)	1% Arsenal/Stalker + 0.25% non-ionic surfactant	Treat only 10% of the area to be grazed. No grazing restrictions, but do not cut for hay for 7 days. Read the herbicide label for mixing instructions.
		3% glyphosate	No grazing or haying restrictions. Read the herbicide label for mixing instructions.
		0.5% Arsenal/Stalker + 2% glyphosate + 0.25% non-ionic surfactant	Treat only 10% of the area to be grazed. No grazing restrictions, but do not cut for hay for 7 days. Read the herbicide label for mixing instructions.
2nd year	Spring (monitor regrowth)	2–3% glyphosate	See above.
	Fall (monitor regrowth)	2–3% glyphosate	See above.
3rd year–until eradicated	Spring–Fall (monitor regrowth)	Spot treat at the above rates for the 2nd year.	

Table 2. Herbicide suggestions for large cogongrass infestations in grazing areas, including both improved and native rangeland. These suggestions are intended for large (>1000 gallon) sprayers. Please read the entire label of the suggested products prior to treating existing cogongrass.

	Timing	Herbicide Rate	Application Notes
1st year	Fall (August–November)	48 oz/acre Arsenal/Stalker + 0.25% non-ionic surfactant	Treat only 10% of the area to be grazed. No grazing restrictions, but do not cut for hay for 7 days. Read the herbicide label for mixing instructions.
		3 to 4 qt/acre glyphosate	Do not graze for 8 weeks. Read the herbicide label for mixing instructions.
		24 oz/acre Arsenal/Stalker + 2 qt/acre glyphosate + 0.25% non-ionic surfactant	Treat only 10% of the area to be grazed. No grazing restrictions, but do not cut for hay for 7 days. Read the herbicide label for mixing instructions.
2nd year	Spring (monitor regrowth)	2–3% glyphosate	No grazing or haying restrictions.
	Fall (monitor regrowth)	2–3% glyphosate	No grazing or haying restrictions.
3rd year–until eradicated	Spring–Fall (monitor regrowth)	Spot treat at above rates for the 2nd year.	See above.

Table 3. Control of cogongrass using an integrated approach. Adjust your timelines based on your location in Florida. For example, burning should be performed earlier in north Florida than in south Florida because of the first onset of a potential killing frost. Please read all herbicide labels prior to treating cogongrass for restrictions and mixing instructions.

	Timing	Herbicide Rate	Application Notes
1st year	Summer—Fall (August–November)	1. Burn	Cogongrass fires burn extremely hot. Be sure to have firebreaks in place before attempting to burn cogongrass.
		2. Apply herbicide: 24 oz/acre Arsenal/Stalker + 2 qt/acre glyphosate + 0.25% non-ionic surfactant	Treat only 10% of the area to be grazed. No grazing restrictions, but do not cut for hay for 7 days. Read the herbicide label for mixing instructions.
		3. Take soil samples.	Have the soil pH tested at a reputable laboratory. Amend the soil as needed to grow desirable forage.
2nd year	Spring	1. Tillage	Prepare a seedbed for desirable forage species. Repeated tillage will help to desiccate any remaining cogongrass rhizomes.
		2. Plant desirable forage.	Please consult your local Extension agent for up-to-date recommendations on forage cultivars and fertility recommendations.
3rd year	Spring (monitor regrowth)	2–3% glyphosate	No grazing or haying restrictions.
	Fall (monitor regrowth)	2–3% glyphosate	No grazing or haying restrictions.
4th year–until eradicated	Spring–Fall (monitor regrowth)	Spot treat at the above rates for the 3rd year.	See above.

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SE MINERAL 12-12

MINERAL FEED FOR CATTLE ON PASTURE
CAUTION USE ONLY AS DIRECTED

GUARANTEED ANALYSIS

Calcium (Ca) (Min)	10.80 %
Calcium (Ca) (Max)	12.96 %
Phosphorus (P) (Min)	12.0 %
Salt (NaCl) (Min)	3.50 %
Salt (NaCl) (Max)	4.50 %
Magnesium (Mg) (Min)	1.0 %
Potassium (K) (Min)	1.00 %
Manganese (Mn) (Min)	3500 ppm
Cobalt (Co) (Min)	84 ppm
Copper (Cu) (Min)	2500 ppm
Iodine (I) (Min)	190 ppm
Selenium (Se) (Min)	54.00 ppm
Zinc (Zn) (Min)	6600 ppm
Vitamin A (Min)	200000 IU/LB
Vitamin D3 (Min)	20000 IU/LB
Vitamin E (Min)	20 IU/LB

INGREDIENTS

Dicalcium Phosphate, Monocalcium Phosphate, Processed Grain By-Products, Calcium Carbonate, Salt, Sodium Selenite, Molasses Products, Soybean Oil, Magnesium Oxide, Potassium Chloride, Copper Sulfate, Zinc Sulfate, Colored with Iron Oxide, Vitamin D3 Supplement, Vitamin A Supplement, Cobalt Carbonate, Manganese Sulfate, Ethylenediamine Dihydriodide, Basic Copper Chloride, Vitamin E Supplement

327H LKC-G 2

DIRECTIONS

Feed this product free choice to cattle receiving rations composed largely of grass hay or to cattle grazing range or grass pastures. Optimum intake is 2 ounces per head daily.

IMPORTANT

Follow these management practices:
1. Cattle receiving phosphorus deficient diets may over-consume this product when it is first offered.

See Reverse Side For Precautionary Statements

8 04273 05018 8

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FREE-CHOICE CATTLE MINERAL

GUARANTEED ANALYSIS

Calcium (Ca) Min	12.00%
Calcium (Ca) Max	14.80%
Phosphorus (P) Min	7.00%
Salt (NaCl) Min	16.00%
Salt (NaCl) Max	18.00%
Fluorine (F) Min	0.07%
Magnesium (Mg) Min	3.00%
Potassium (K) Min	1.00%
Sulfur (S) Min	1.20%
Manganese (Mn) Min	3000 PPM
Zinc (Zn) Min	4000 PPM
Copper (Cu) Min	1500 PPM
Cobalt (Co) Min	60 PPM
Iodine (I) Min	70 PPM
Selenium (Se) Min	26.5 PPM
Vitamin "A" Min	220,000 USP UNITS/LB
Vitamin "D-3" Min	45,000 USP UNITS/LB
Vitamin "E" Min	220 INT UNITS/LB

INGREDIENTS

Monocalcium Phosphate, Dicalcium Phosphate, Calcium Carbonate, Salt, Magnesium Oxide, Potassium Chloride, Potassium Sulfate, Magnesium Sulfate, Zinc Oxide, Zinc Sulfate, Manganous Oxide, Manganese Sulfate, Ferrous Sulfate, Iron Carbonate, Iron Oxide, Sodium Selenite, Copper Sulfate, Ethylenediamine Dihydriodide, Cobalt Carbonate, Vitamin A Supplement, Cholecalciferol (source of vitamin D-3), Vitamin E Supplement, Cottonseed Meal, Dried Cane Molasses, Processed Grain By-Products, Cane Molasses, Natural and Artificial Flavoring.

FEEDING DIRECTIONS

Feed free choice in a covered trough at different locations in pastures. Remove all other sources of salt and minerals.
Four ounces of this mineral will supply 3 milligrams of supplemental Selenium. Keep plenty of clean, fresh water before cattle at all times. When mixing, mix at the rate of 3 to 4 ounces per head per day.

CAUTION! This mineral contains Copper.
Do Not Feed To Sheep!

CAUTION! This mineral contains added Selenium. Do not permit excessive consumption. Intake of supplemental Selenium from this mineral should not exceed 0.3 PPM on a complete ration basis or 3 milligrams per head per day for beef and dairy cattle.

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Macromineral Requirements in Beef Cattle

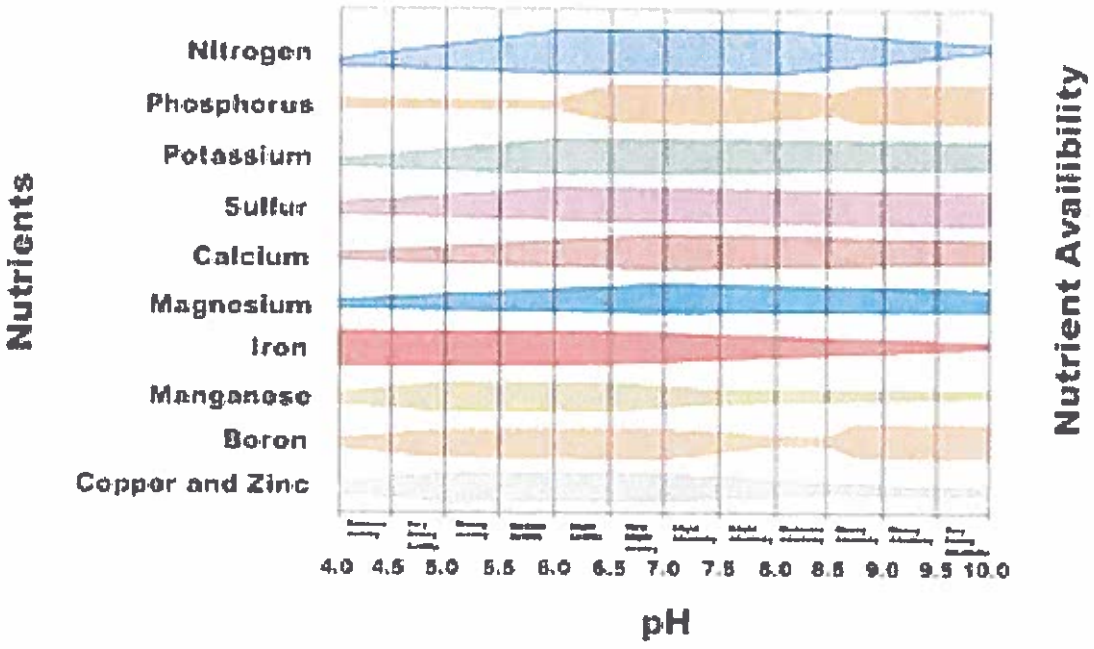
Mineral*, %	Requirement			
	Growing and Finishing Cattle	Stressed Calves**	Dry, Gestating Cows	Lactating Cows
Calcium	0.31	0.6-0.8	0.18	0.58
Magnesium	0.10	0.2-0.3	0.12	0.20
Phosphorus	0.21	0.4-0.5	0.16	0.26
Potassium	0.60	1.2-1.4	0.60	0.70
Sodium	0.06-0.08	0.2-0.3	0.06-0.06	0.10
Sulfur	0.15	0.15	0.15	0.15

*Research data are inadequate to determine chlorine requirements

**Suggested range

Source: NRC, 2000. Adapted from NRC Nutrient Requirements of Beef Cattle, 7th revised edition.

Influence of pH on Availability of Plant Nutrients



(S.S.S.A.P., 1946, 11:305.)

Mineral Concentrations in Grazed Cool-Season Annual Grass Pastures in North Florida[1]

Bob Myer, G. Chelliah, Lee McDowell, Nancy Wilkinson, Ann Blount, and Cheryl Mackowiak[2]

Minerals make up a small portion of an animal's diet; however, they play an important role in health, growth, and reproduction.

While free-choice mineral supplementation is common for beef cattle on pasture, pasture forage is still the main source of many nutritionally essential minerals. In the southeastern US when permanent warm-season pastures are dormant, cool-season annual grasses, such as oats, rye, and annual ryegrass, are commonly planted to provide forage for grazing by beef cattle during the late fall to spring period. These forages are highly digestible and are high in energy and protein; however, there is limited information about concentrations of various nutritionally important minerals.

North Florida Grazing Study

A four-year grazing study was conducted at the UF/IFAS North Florida Research and Education Center (NFREC) Beef Unit, which is located near Marianna in northwest Florida. The study evaluated two cool-season pasture establishment methods (sod-seeding into dormant warm-season pasture or planting into a clean tilled prepared seed-bed) and two forage treatments (mono-crop vs. a mixture of forage species) for grazing by growing beef cattle. A mineral study was a component of this grazing study. The purpose of the mineral study was to measure monthly concentrations of selected minerals in forage from the various pastures used in the grazing study during the late fall-winter-spring grazing season in north Florida. The minerals measured were the macro minerals calcium (Ca) phosphorus (P), sodium (Na), potassium (K) and magnesium (Mg), and the trace minerals copper (Cu), iron (Fe), zinc (Zn), manganese (Mn), cobalt (Co), and selenium (Se).

The pasture soils were well-drained acidic, sandy soils (fine loamy, kaolintic, thermic Kandiudults) typical of the Southern Coastal Plain. Prior to planting each year, pastures were fertilized and, if needed, dolomite lime applied based on soil analyses by a commercial laboratory. All pastures over the four year period were grown under dry land conditions. The pastures were top dressed twice, with 75 lb N/ac., within each year.

Mineral Concentrations

The overall average concentrations obtained of the minerals measured from the four-year study are presented in the Tables 1 and 2. Very little effect due to annual cool-season pasture forage treatment or pasture establishment method was noted on concentrations of the minerals evaluated. Some year-to-year variation was noted for all minerals except sodium. Magnesium varied the most, almost two-fold from year-to-year.

1. This document is AN224, one of a series of the Animal Sciences Department, UF/IFAS Extension. Original publication date August 2009. Revised August 2012. Reviewed October 2015. Visit the EDIS website at http://edis.ifas.ufl.edu

2. Bob Myer, professor emeritus, Department of Animal Sciences, North Florida Research and Education Center; G. Chelliah, former graduate student, Department of Animal Sciences; Lee McDowell, professor emeritus, Department of Animal Sciences; Nancy Wilkinson, former chemist, Department of Animal Sciences; Ann Blount, professor, Department of Agronomy, NFREC; and Cheryl Mackowiak, associate professor, Department of Soil and Water Science, NFREC; UF/IFAS Extension, Gainesville, FL 32611.

Month of grazing season, however, had greatest influence on concentrations of minerals evaluated (see Figures 1, 1a, and 2). There was a large month-to-month variation in concentrations of potassium, phosphorus, iron, and manganese, little variation for calcium, magnesium, copper, and cobalt, and essentially no variation for sodium and selenium. Forage concentrations of phosphorus and potassium were greatest during the winter and declined during spring with lowest levels noted in May; magnesium was lowest in early spring. Concentrations of iron decreased and manganese increased as the grazing season progressed. Due to high costs, only few samples were analyzed for cobalt and selenium and thus limited monthly data. Nonetheless, there was evidence that cobalt increased as the grazing season progressed (from 0.05 ppm early on to 0.10 ppm in May); selenium did not vary much from month to month (0.05 to 0.06 ppm). Please note that some important essential minerals such as iodine and chlorine were not measured. Iodine (iodized salt) and chlorine (ordinary salt) are present in typical cattle mineral supplements.

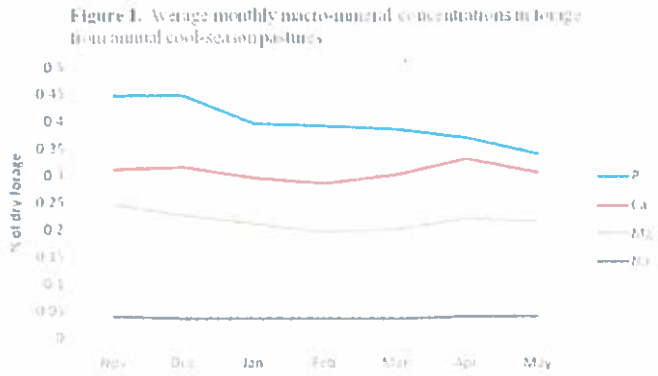


Figure 1. Average monthly macro-mineral concentrations in forage from annual cool-season pastures.

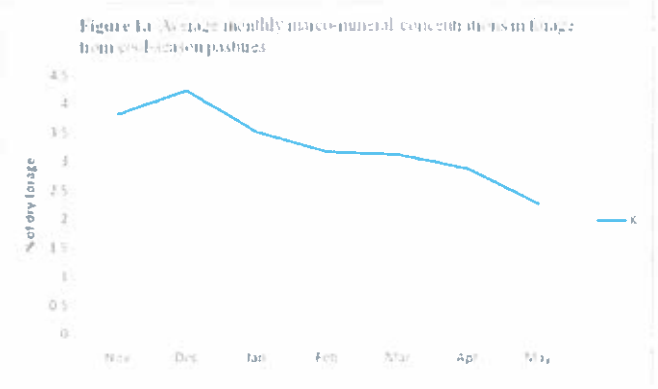


Figure 1a. Average monthly macro-mineral concentrations in forage from cool-season pastures

Overall average concentrations of calcium and magnesium from our study were at the low end of ranges of concentrations previously published for ryegrass, rye, and oat forage grown in other parts of the US; phosphorus and potassium were at the high end and sodium in the middle (Table 1). Concentrations of iron and manganese were lower than previously reported, zinc higher, and copper and selenium were similar (Table 2). However, large variations in concentrations were noted in our study as mention above.

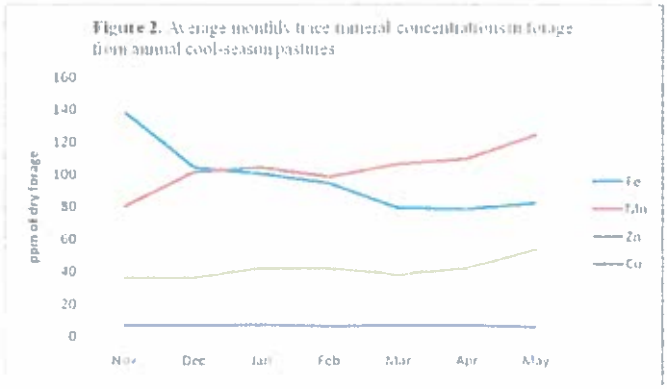


Figure 2. Average monthly trace mineral concentrations in forage from annual cool-season pastures.

Implications of Findings

From our findings and compared to requirements, sodium would be very deficient; copper, selenium, and cobalt would be deficient; calcium would be slightly deficient, phosphorus, magnesium, and zinc would be marginally deficient; iron and manganese would be adequate, and potassium would be in excess for beef cattle grazing cool-season pastures in the southeastern USA (Tables 1 and 2). Fortunately, most free-choice cattle mineral supplements will more than make up for these deficiencies **provided that mineral supplement is present and that the cattle are consuming it.**

The marginally low forage magnesium concentrations noted in this study, combined with the high potassium concentrations, may be a potential magnesium deficiency problem for beef cattle, which can result in grass tetany (hypomagnesemia), especially during the early spring months. This reinforces that specially formulated high magnesium (hi-mag) mineral supplement should be offered at this time. Further information about grass tetany and its prevention can be found in the UF/IFAS Extension publication *Grass Tetany in Cattle* (SS AGR-64/DS137), which can be found on the EDIS website (http://edis.ifas.ufl.edu).

Table 1. Macro-mineral concentrations of grazed cool-season annual grasses in north Florida (dry weight basis).

Mineral	Concentration, %	Requirement[a], %	Reported[b], %
Calcium	0.31 ± 0.05	0.3 to 0.5	0.32 to 0.65
Phosphorus	0.38 ± 0.04	0.2 to 0.4	0.23 to 0.41
Sodium	0.04 ± 0.01	0.1	0.01 to 0.11
Potassium	2.9 ± 0.3	0.6	1.7 to 3.4
Magnesium	0.21 ± 0.03	0.1 to 0.2	0.20 to 0.35

[a]Taken from Nutrient Requirements of Beef Cattle, National Research Council (2000).
[b]Previously reported concentrations in fresh ryegrass, oat, and rye forage (dry weight basis, from Ensminger et al., 1990, *Feeds and Nutrition*, Ensminger Publishing Co., Clovis, CA, US and NRC, 2000.

Table 2. Trace mineral concentrations of grazed cool-season annual grasses in north Florida (dry weight basis).

Mineral	Concentration, ppm	Requirement[a], ppm	Reported[b], ppm
Copper	5.8 ± 0.8	10	4 to 8
Iron	83 ± 14	50	101 to 367
Zinc	40 ± 5	30	25 to 30
Manganese	110 ± 14	40	42 to 66
Cobalt	0.06 ± 0.01	0.1	--
Selenium	0.05 ± 0.01	0.1	0.07

[a]Taken from Nutrient Requirements of Beef Cattle, National Research Council (2000).
[b]Previously reported concentrations in fresh ryegrass, oat, and rye forage (dry weight basis; from Ensminger et al. 1990, *Feeds and Nutrition*, Ensminger Publishing Co., Clovis, CA, US and NRC, 2000.

The Interaction of Nutrition and Health in Beef Cows[1]

Matt Hersom[2]

Introduction

In cattle, all physiological processes in the body, including the immune system, are influenced by the cattle's nutritional status (Carroll and Forsberg 2007). Therefore, the past and present nutritional status serves as an immune function modulator in cattle that can influence the performance and reproduction of beef cattle.

Immunity

Immunity is generally classified (Figure 1) as either innate (natural) or acquired (specific) in cattle. Innate immunity includes physical barriers (skin, mucus, saliva), chemical barriers (stomach acid), and blood-borne systems, including the complement cascade and phagocytes. These systems are naturally present at birth as the first line of defense, and they are not influenced by any prior exposure to disease (Chandra 1997). The acquired system includes T-cell immunity (cell-medicated immunity), which provides defense against intracellular pathogens such as viruses that invade host cells and tumors, and B-cell immunity (humoral immunity), which fights extracellular microbial infections. The acquired system is adaptive to the specific aspects of each pathogen and is developed by exposure to microorganisms and antigens; thus, vaccination works in this system.

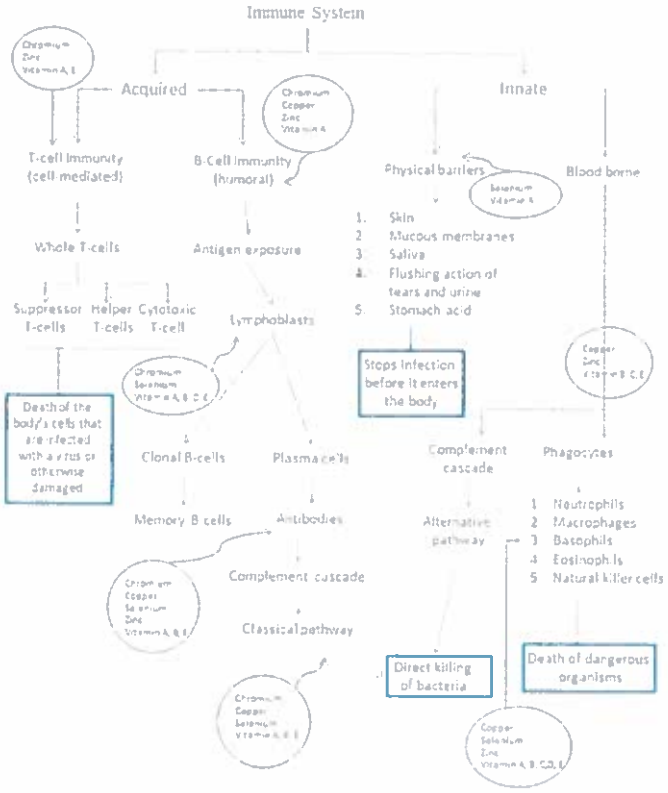


Figure 1. Interaction of the immune system and micronutrients.
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Diet Effects on Immunity

Diet intake is the first way that nutrition affects health and immunity. Low intake, and thus low nutrient intake, negatively affect immunity. The animal's diet supplies the substrates that are required for the development, maintenance, and function of the immune system (Klasing 2002).

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The nutrients that support the immune system include energy, protein-amino acids, minerals, vitamins, and some specific fats that have a direct effect on the immune system. The nutrients that allow the proper function of the immune system and the mechanisms they support are outlined in Table 1. The immune system requires substrates to support the anabolic and proliferative processes associated with immune cell function, antibody production, and liver protein production. Immune function is just one metabolic function within the body and likely has different dietary requirements for some substrates compared to growth or reproduction. As a result, the dietary requirement for some nutrients may be greater when the immune system is stimulated compared to a minimally active immune system.

Nutritional immunity is the process when the body controls the concentration of nutrients available for pathogens to decrease their rate of replication. Chronic feed restrictions decrease several functions of the acquired immune system, whereas moderate restrictions can increase immune function. Additionally, a protein-energy ratio and feeding pattern can affect hormone concentrations that influence immune system function. The hormones that respond to diet and affect immune function include insulin, glucagon, glucocorticoids, and insulin-like growth factors. The diet also minimizes the extent to which the immune system affects healthy tissues. Antioxidants are dietary-derived molecules that inhibit the oxidation reactions and formation of free radicals, which damage healthy cells. The supply of antioxidants to protect healthy cells and tissues localizes the damage of infection. The immune system uses substances at the infection site to kill pathogens that are detrimental to healthy tissues.

Nutritional Inputs

ENERGY

The maintenance of the immune system is a daily metabolic cost to cattle that is incorporated into the cattle's maintenance energy cost. Likewise, activation of the immune system and the fever response incurs an energy cost to cattle (Carroll and Forseberg 2007). Estimates of a 10% to 13% increase in energy requirements are associated with an immune response for every degree increase in body temperature. In addition to the energy expenditure associated with the increase in body temperature, there is an energy cost associated with production of the antibodies and liver proteins. The redirecting of energy resources to support an immune response means energy is not available for productive purposes associated with body weight gain, reproduction, or lactation.

Therefore, in situations of sickness when diet intake is decreased, nutrient supply is limited and energy expenditure is increased; cow energy stores found in fat tissue and lean muscle tissue become important. The reserve of energy available from fat and muscle underscore the necessity of maintaining cows in adequate body condition particularly in times of physiological stress. When cows are in a decreased body condition, in which energy and protein reserves are limited, the animal's immune system may be impaired (Chandra 1997). Although little research exists, it is certainly feasible to consider that limited energy supplies can negatively affect the integrity of the body's physical barriers, mucus, and other innate immune functions (Chandra 1997).

PROTEIN

Many components of the innate and acquired immune system require protein and amino acids for synthesis and function. As such, during disease or infection, proteins and amino acids are diverted from normal functions to support the synthesis of immunoglobulins and T-cell- and B-cell-mediated immunity, and they are catabolized for energy production (Scrimshaw and SanGiovanni 1997). Similar to the energy discussion, protein stores in lean muscle are an important substrate resource to support the immune system. Inadequate protein nutrition impairs cell-mediated immunity and immunoglobulin production. Additionally, poor protein nutrition leads to compromised production of immunoglobulins that support colostrum quality, which is essential for calf performance.

MICRONUTRIENTS

The micronutrients in the diet (trace minerals and vitamins) can have profound effects on the immunity and health of cattle. Figure 1 integrates the immune system and important micronutrients. Four general concepts are related to micronutrient and immunity (Chandra 1997): (1) Changes in the immune response occur early in the decrease in micronutrient intake; (2) the decreased immune response is dependent upon the nutrient, interactions with other nutrients, the deficiency severity, infection presence, and animal age; (3) immune system abnormality predicts the outcome and risk of infection; and (4) excessive intake is also associated with impaired immune function. In fact, Cole (1993) suggested that for young, stressed cattle trace mineral requirements are no greater than for healthy unstressed cattle. Likewise, Olson et al. (1999) reported decreased reproductive performance in 2-year-old cows fed twice the NRC recommendation of copper, cobalt, manganese, and zinc compared to normal mineral recommendations.

MINERALS

Trace minerals affect immunity and health, primarily through their function in important enzyme activity associated with energy metabolism, cellular protein synthesis, and DNA replication. Inconsistent data has been reported in literature regarding the beneficial effects of mineral supplementation beyond what is considered needed for normal physiology (Carroll and Forsberg 2007).

Zinc

Zinc is reported to be a cofactor for more than 300 different enzymes that modulate many physiological processes (Carroll and Foresberg 2007), which indicates the vast importance this trace mineral has in cattle's metabolism. In multiple reports, zinc is identified as serving several immunological functions, including both innate and acquired immune systems, tissue integrity, protein synthesis, and inflammation (Carroll and Foresberg 2007; Erickson et al. 2000). The immune system is a rapid response system that relies on many enzymes to function, and zinc is an important component. Potential limitations of zinc in the body could lead to limitations of enzyme activity and the response potential of the immune system. In ruminants, the effect of zinc status may have less importance in immune function for healthy cattle but have definitive positive impact in stress cattle. Research with stressed calves has demonstrated that zinc supplementation can decrease bovine respiratory disease associated deaths by 52% (Carroll and Foresberg 2007), but other research has reported no effect on performance or health. The impact of zinc may be dictated by the specific immune stimulus, animal's mineral status, concentration and bioavailability of the supplemental zinc, and the animal itself.

Copper

Copper is considered an essential nutrient and is important in many physiological and metabolic processes. However, supplemental copper's direct effect on immunity is not fully compelling (Duff and Galyean 2007). Mechanistically, copper is key for the enzyme superoxide dismutase, which is vital for phagocytes when they engulf pathogens (Suttle and Jones 1989). Copper's immunological role has been reported to serve in antibody production, inflammation, and neutrophil phagocytosis (Erickson et al 2000; Schrimshaw and Sangiovanni 1997, Carroll and Forsberg 2007). Copper supplementation has not been reported in summary literature to improve cattle performance or morbidity (Galyean et al. 1999, Carroll and Foresberg 2007, Duff and Galyean 2007).

Selenium

Conclusive deficiencies of selenium exist regionally, particularly in Florida. Selenium is an important component of the antioxidant system through the glutathione peroxidase enzyme. Deficiency of selenium can contribute to oxidative stress in animals. Oxidative stress is detrimental to cellular metabolism and can damage DNA and affect cell membranes and integrity (Carroll and Forsberg 2007). Supplemental selenium has been documented to increase or enhance neutrophils and macrohphage phagocytosis (Duff and Galyean 2007; Erickson et al. 2000). Other research has demonstrated that supplemental selenium has enhanced antibody response and production of specific antibodies against *Escherichia coli* (Carroll and Forsberg 2007). Transfer of positive immune function from dam to calf has been documented by Reffett Stabel et al (1989). However, supplemental selenium has not been demonstrated to increase cattle performance, measured by body weight gain.

Chromium

Generally considered an essential mineral for carbohydrate metabolism through maintenance of normal blood glucose and a cofactor of insulin, chromium has a definitive metabolic function (Carroll and Forsberg 2007). In beef cattle, chromium has been reported to positively affect growth performance, feed efficiency, morbidity rate, and immune response. Generally, the immune system stimulation conferred by chromium has been in cattle subjected to a stressor. Feeder calves have demonstrated increased serum immunoglobulin M and G and have enhanced antibody response when supplemented with high-chromium yeast (Moonise-Shageer and Mowat 1993). Chromium may function to alleviate the stress-induced suppression of the immune system; thus, the beneficial effect is in times of stress, but has no effect in non-stress situations.

VITAMINS

Vitamin deficiencies have long been known to induce immune system disorders. Nearly every aspect of the immune system has a dependency on an adequate supply of one or multiple vitamins. Vitamins have essential roles for blood formation, maintenance and production of white blood cells, natural killer cells, and antibody production (Carroll and Forsberg 2007). Vitamins are also key components of the antioxidant system because they inactivate reactive oxygen species. Reactive oxygen species can destroy cellular membranes, cellular proteins, and DNA. Protection against reactive oxygen species is important for all body tissues and especially immune cells. During high-stress periods and greater stimulation of the immune system, the body's ability to eliminate reactive oxygen species can be overwhelmed;

thus, the antioxidant system of vitamins and minerals is important.

Vitamin A

Identified as retinol or beta-carotene, vitamin A is an important nutrient regarding the immune system. Vitamin A does not occur naturally, but rather as various forms of carotene in plant materials. In the body, the carotenes are converted to retinol, and cattle are less efficient at this conversion compared to monogastric animals (NRC 1996), but because cattle generally consume greater amounts of forage, vitamin A levels are maintained. Vitamin A is a fat-soluble vitamin and is stored in the liver, and therefore requires months for deficiencies to manifest. However, vitamin A levels are dynamic, and many physiological and nutritional factors affect cows' vitamin A status. Vitamin A is understood to be essential for skin maintenance and mucus membranes in the respiratory and gastrointestinal tract (Carroll and Forsberg 2007; Scrimshaw and Sangiovanni 1997). Inadequate integrity of membranes allows for greater opportunity for bacterial and viral invasion. Deficiency of vitamin A also decreases the functional ability of natural killer cells and decreases the response in white blood cells. Supplemental vitamin A to correct a deficiency is more effectively corrected through injectable sources, rather than feed sources, because vitamin A is extensively destroyed in the rumen and abomasum. Supplementation of vitamin A has only been shown to be beneficial in deficient or marginally deficient animals, whereas no response is observed in animals that are considered adequate.

Vitamin E

Identified as tocopherol, vitamin E is another fat-soluble vitamin that is very important to the functional ability of the immune system. Vitamin E functions as part of the antioxidant mechanism, often with selenium, to protect against free-radical formation and damage. Vitamin E also functions to maintain the immune system, DNA repair, and is an important constituent of all cellular membranes (Carroll and Forsberg 2007). Because of the aforementioned functions, vitamin E is found in great concentration in immune cells. Vitamin E supplementation has been demonstrated to augment the acquired immune system. Vitamin E supplementation has been reported to increase the inflammatory process, which as a result has increased humoral/antibody concentration (Duff and Galyean 2007). Studies that have examined the effect of supplemental vitamin E in immunity/stress-challenge situations generally concluded that vitamin E can reduce the severity and duration of the challenge, but it does not directly enhance animals' growth performance (Carroll and Forsberg 2007; Duff and Galyean 2007). Increases in growth performance are a result of a shortened stress challenge and subsequent resumption of feed intake and growth.

Vitamin B

In general practice, deficiency of vitamin B12 is not a production concern for cattle receiving a nutritionally balanced diet (Duff and Galyean 2007). Additionally, deficiency of vitamin B12 is difficult to distinguish from a cobalt deficiency because vitamin B12 contains cobalt (NRC 1996). Vitamin B12 is formed in the rumen of healthy cattle through the action of the microorganism, but it is cobalt dependent. As a functional vitamin, B12 is essential to cattle for the metabolism of proprionate (an energy source produced through ruminal fermentation), cellular replication through nucleic acids, and protein metabolism. As a result, deficiency of vitamin B12 negatively affects antibody formation and white blood cell replication (Scrimshaw and Sangiovanni 1997).

Vitamin D and C

As a practical matter, supplementation of vitamins D or C to affect cattle health and immunity is uncertain. Vitamin D is important for bone formation, calcium and phosphorus regulation, and some immune function. Little storage of vitamin D occurs in cattle, but because of exposure of cattle to sunlight and consumption of sun-cured forages, deficiencies are rare. Vitamin C is a component of the antioxidant system, and vitamin C acts to protect the body against free radicals and interacts with vitamin E. However, most vitamin C is degraded in the rumen; therefore, cattle must rely on tissue formation of vitamin C rather than supplemental sources.

Conclusion

The overall nutritional status of cattle has important implications for productive outputs including growth, lactation, and reproduction. Nutritional status, which is determined simply as adequate feed intake or specifically as vitamin status, profoundly affects multiple immunological functions. Maintenance of physical barriers, antigen production, and cellular-based immunity is controlled and influenced by nutrition. Adequate energy, protein, trace minerals, and vitamin status are nutritional variables that are quantitatively affected by cattle producers through provisions of an adequate nutritional environment. Attention to pasture, stored forage, energy-protein supplementation, and vitamin-mineral supplementation programs are a means to affect cattle immune function.

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Table 1. Nutritional modulators of immune mechanisms

Nutrient	Mechanism
All nutrients	Supply substrates for immune system function
Energy, protein, feeding pattern	Regulate immunity through altering balance of hormones
Fatty acids, vitamins A, D, E	Direct regulatory effect on immune system cells
Trace minerals	Important components of enzymatic reactions
Iron, biotin, manganese	Deprave pathogens of nutrients
Antioxidants	Reduce damage to host cells caused by immune response
Non-starch polysaccharides, lectin, sugars	Physical and chemical actions of feeds in the intestine

Adapted from Klasing (2002)

ALABAMA A&M AND AUBURN UNIVERSITIES

ALABAMA COOPERATIVE Extension SYSTEM

ANR-0968

Vaccinations for the Beef Cattle Herd

Vaccination programs for beef cattle herds are designed to protect the animals from diseases caused by infectious organisms such as viruses, bacteria, and protozoans. Vaccines stimulate an animal's immune system to produce a protective response against an organism. The immune system will then "remember" how to produce a response against the organism if it ever is infected with that organism. Vaccines cannot prevent exposure to infectious organisms, but they do increase an animal's ability to fight off an infection or lessen the severity of the disease if it occurs.

The majority of cattle vaccines are injected, although some may be given by other routes, such as intranasal and oral. Although antibiotics are also often administered via injection, treating an animal with one of these drugs is not a vaccination but rather a treatment once an infection has occurred.

Animal health product manufacturers go to great expense to obtain approval for vaccines from the U.S. Department of Agriculture. They must prove that the vaccine is safe and that it will do what the label claims it will do. See Extension publication ANR-1416, "Understanding Protection Claims on Vaccine Labels," for more information.

Vaccine trade names can be confusing; however, the label will always specify which diseases and microorganisms the vaccine provides protection against. Sometimes different terms used in the name can be confusing as well. Terms such as 4-way, 5-way, 7-way, or 8-way do not refer to any particular type of vaccine, but rather to the number of different subtypes of a microorganism in a vaccine. These terms are most often used for leptospirosis or clostridial disease vaccines, which contain several subtypes of the *Leptospira* or *Clostridium* organisms.

Types of Vaccines

Most vaccines contain either modified live, killed, or chemically altered organisms. If a vaccine is used correctly, whether it is modified live, killed, or chemically altered, it will increase an animal's resistance to disease, but each type of vaccine does have its limitations as well.

Modified live vaccines (MLV) contain a small amount of virus or bacteria that has been altered so that it does not cause clinical disease when used according to product label directions. However, the virus or bacteria can still replicate in the vaccinated animal resulting in a controlled infection. Recognition of the replicating organism by the animal's immune system stimulates an effective immune response. MLVs are mainly available for diseases caused by viruses, such as bovine herpes virus 1, the causative agent of infectious bovine rhinotracheitis (IBR), bovine viral diarrhea virus (BVDV), bovine respiratory syncytial virus (BRSV), and parainfluenza-3 virus (PI_3). Some MLVs are safe for use in pregnant cows **if you follow all label directions**.

Modified Live Vaccines	
Advantages: • One initial dose may be sufficient, but boosters are sometimes required. • Typically stimulate more rapid, stronger, and longer-lasting immunity than killed vaccines. • Less likely than killed vaccines to cause allergic reactions and post-vaccination lumps. • Usually less expensive than killed vaccines.	Disadvantages: • Risk of causing abortion or transient infertility; therefore, MLV should generally be administered 6 to 8 weeks prior to the breeding season (read label directions). • Must be mixed on-farm and used within about 30 minutes.

Figure 1. Advantages and disadvantages of MLVs

However, if not used according to label directions, MLVs can cause abortion in pregnant cows (Figure 1). In addition, some MLVs are not approved for use in calves nursing pregnant cows because of the slight possibility that the calves could temporarily shed the vaccine virus and infect the cows. However, some MLVs can be safely used in calves nursing pregnant cows if the cows have been properly vaccinated according to label directions. MLVs are also safe to use in weaned calves, including replacement heifers.

Killed vaccines (KVs) and toxoids contain organisms or subunits of organisms that do not replicate or reproduce themselves in the animal following administration. KVs usually contain adjuvants, or added substances, that further stimulate the immune system to respond to the vaccine challenge. KVs are safe to use in any animal, including pregnant cows (Figure 2).

Chemically altered vaccines (CAVs) contain modified live organisms that are grown in chemicals that cause specific mutations of the organism. An example of chemically altered vaccine technology is temperature-sensitive (TS) vaccine organisms that cannot replicate at an animal's normal body temperature but can grow at the temperatures associated with the ocular (eye) or nasal mucosa. Because there is no systemic replication with TS vaccines, they are safe for use in pregnant animals (Figure 3).

Killed Vaccines and Toxoids	
Advantages: • Available for many diseases. • No risk of the vaccine organism spreading between animals. • Minimal risk of causing abortion. • No on-farm mixing required.	Disadvantages: • More likely to cause allergic reactions and post-vaccination lumps. • Two initial doses required. • Slower onset of immunity. • Immunity is usually not as strong or long-lasting as MLV products. • Usually more expensive than MLV products.

Figure 2. Advantages and disadvantages of KVs and toxoids

Although vaccines will not cause the disease they are supposed to protect against, some animals may have a fever temporarily after vaccination. Some animals also may have swelling and soreness at the sight of injection. In some cases, animals may go off feed and decrease milk production for a few days.

Booster Vaccinations

For young animals being vaccinated for the first time, a second, or booster, vaccination is often required a few weeks after the first, or primary, vaccination. A booster vaccination is definitely required for killed vaccines to provide optimal protection. The label directions will indicate when and if a booster vaccination is required. Failure to give the booster at the proper time could result in an incompletely protected adult animal even if that animal is vaccinated every year thereafter.

The time between the primary and booster vaccinations is of interest to beef producers. Management considerations might make it difficult for some producers to give booster vaccinations within the time span called for on the label, which is often from 3 to 6 weeks after primary vaccination. When the USDA approves a vaccine, it does so only for the label directions that were tested by the manufacturer. Exceeding the label-recommended time span between the primary and booster vaccination may not make a vaccine ineffective, but it might make the vaccine less effective. Follow label instructions as closely as possible to facilitate development of maximum immunity in response to vaccination.

Chemically Altered Vaccines	
Advantages: • Share many of the advantages of MLV products. • Safety is similar to KV products. • Minimal risk of causing abortion.	Disadvantages: • Two initial doses required. • Slower onset of immunity than MLV products. • Immunity is usually not as strong or long-lasting as MLV products. • Usually more expensive than MLV products. • Must be mixed on-farm and used within about 30 minutes.

Figure 3. Advantages and disadvantages of CAVs

Proper Handling of Vaccines

The best vaccine program will fail if the product is damaged by improper handling. For example, if the label says to store a vaccine at 35 to 45 degrees F, the vaccine should be refrigerated. Vaccines should not be allowed to freeze, nor should they be stored in direct sunlight.

Most MLVs must be reconstituted by adding sterile water to a dehydrated "cake" in a separate sterile vial. Once the water is added, the vaccine organisms are fragile and will be "live" for only a short time. As a rule of thumb, only reconstitute enough vaccine to be used in 30 to 45 minutes, and use a cooler or other climate-controlled storage container to protect reconstituted vaccines from extremes of cold, heat, and sunlight.

Keep needles and syringes clean to avoid infections at the site of injection. DO NOT use disinfectants to clean needles and syringes used to administer vaccines, especially MLVs. Even a trace or film of disinfectant in a syringe or needle can kill the live organisms and make the vaccine worthless. Follow product guidelines for cleaning multi-use vaccine syringe guns, but in general, after use, rinse thoroughly with hot water to clean the injection equipment, and then sterilize it using boiling water.

DO NOT mix different vaccines together in one syringe or combine other injectable drugs into the same syringe with vaccines. Although this method has been advocated as a method of reducing the number of injections, it could inactivate the vaccine because of incompatibilities with the other compounds.

Method of Injection

The only acceptable site for injection is in the neck, both for intramuscular (IM; in the muscle) and subcutaneous (SQ; under the skin) injections (See figure 4). Intramuscular injections of some products can cause significant muscle damage, so it is necessary to avoid injecting anything in the top butt or rump of the animal. Injection site reactions can cause damage to valuable beef product, and this muscle damage costs the beef industry millions of dollars a year from lost product and lower calf prices. All injections should be administered IM or SQ in the neck (Figure 4). Some product directions allow for IM or SQ administration, in which case SQ is the preferred method. Use only 18- or 16-gauge needles, ½ or ¾ inch long, to administer an SQ injection. Use only 18- or 16-gauge needles, 1 to 1½ inches long, to administer IM injections. Refer to ANR-1280, "Alabama Beef Quality Assurance: Administer Drugs Properly," for more information related to proper drug administration.

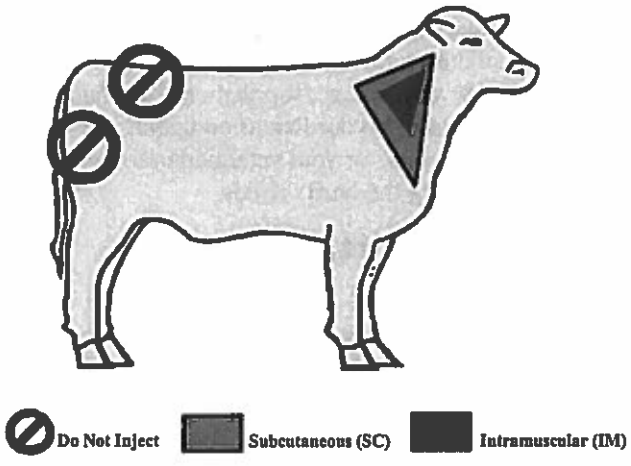


Figure 4. Use neck for injections. Do not inject in rump or leg.

Importance of Nutrition

Vaccination alone will not guarantee a healthy herd. In order for a vaccine to work, the animal's immune system must be able to respond to it, and for an immune system to respond, an animal must receive proper nutrition. Proper nutrition includes energy and protein as well as mineral supplementation.

Some calves that have been properly vaccinated with excellent vaccines have still died in the feed yard because their preshipment mineral nutrition was deficient. Minerals such as copper, selenium, and zinc are required in very small amounts in the diet; however, if the forage is deficient in some of these elements and they are not supplemented in a diet or a free-choice mineral mix, the immune system may not function correctly. Review your forage, supplement, and mineral nutrition programs with your Extension agent, Extension nutrition specialist, or your veterinarian to ensure that you are meeting the herd's needs.

Vaccinating the Right Animal at the Right Time

To determine the best time to vaccinate animals in your herd, first write down the breeding and calving seasons, and then schedule vaccinations and other management events. Most recommended vaccines are best given at specific ages and/or at specific times as related to management and reproductive cycles. For example, blackleg is a rapidly fatal disease of calves. Calves should be vaccinated for blackleg by 3 to 4 months of age when the temporary immunity from the dam has declined and the calf's immune system can respond to the vaccine.

When protecting cows against reproductive diseases, it is often best to vaccinate at least 6 to 8 weeks prior to the breeding season to allow time for development of a protective immune response. However, if vaccinating cows to increase the amount of antibodies in colostrum against diseases such as calf scours, you may need to vaccinate 1 to 4 months prior to calving. Vaccine timing varies from product to product, so always follow vaccine label directions with respect to vaccine administration timing to maximize product efficacy.

Vaccinating for Diseases That Are a Routine Threat

Vaccines are available for many disease conditions. However, many diseases are not a routine threat to most beef herds, and some vaccines are not sufficiently effective to justify their use. Therefore, only a few vaccines are included in a routine vaccination schedule. The glossary of conditions and terms at the end of this publication lists both routine and not-so-routine infectious diseases and vaccines for them.

Vaccinations for Different Animals in the Herd

Every cattle operation will have unique vaccination requirements based on individual herd goals, so the following guidelines for vaccinating cattle may not be applicable in all situations. The best use of these guidelines is as a starting point to develop an effective vaccination protocol with your herd-health veterinarian and/or Extension agent. When appropriate, ensure that products are safe for pregnant animals and for calves nursing pregnant cows. Properly store and administer vaccines according to label directions; adhere to designated meat withdrawal times; booster primary vaccinations when recommended, and follow all Beef Quality Assurance (BQA) guidelines.

Nursing calves:

- 7-way clostridial (blackleg)
- IBR/BVD/PI_3/BRSV
  - IBR = infectious bovine rhinotracheitis
  - BVD = bovine viral diarrhea
  - PI_3 = parainfluenza3
  - BRSV = bovine respiratory syncytial virus
- Calfhood vaccination against brucellosis for 4- to 10-month-old heifers if recommended by herd veterinarian.
- Consider a leptospirosis 5-way vaccine for future replacement heifers and bulls.

Preconditioned feeder calves:

- IBR/BVD/PI3/BRSV
- 7-way clostridial (blackleg)
- *Mannheimia haemolytica*
- *Pasteurella multocida*

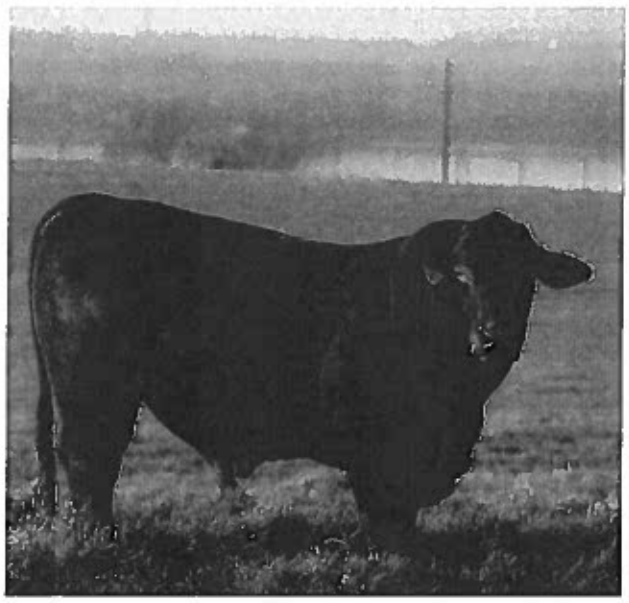
Breeding animals (replacement heifers, cows, and bulls should generally be vaccinated at least 6 to 8 weeks prior to the breeding season so that immunity is high during the breeding season):

- IBR/BVD/PI3/BRSV
- Leptospirosis 5-way
- Vibriosis (*Campylobacter fetus*)

Remember, these guidelines are just a starting point for developing an effective vaccination protocol with your herd-health veterinarian and/or Extension agent. You must still decide which product to use based on understanding a particular vaccine's expected level of protection (see ANR-1416, "Understanding Protection Claims on Vaccine Labels") and the different types of vaccines available (KVs, toxoids, MLVs, or CAVs). Your veterinarian and Extension agent will have the best advice for your particular operation, but this information will assist you in developing and monitoring your herd's vaccination program.

Conclusion

Work closely with your veterinarian and/or Extension agent to customize a vaccination program for your cow herd. A veterinarian may add or delete vaccinations that are generally recommended for most herds in your location. In addition, a veterinarian can offer objective advice on specific vaccine products. By being involved in the design of the herd-health production calendar, a veterinarian will be better able to help prevent disease and deal with it if it occurs. Remember to always properly store and administer vaccines according to label directions, adhere to designated meat withdrawal times, and follow all other Beef Quality Assurance (BQA) guidelines.



Glossary of Conditions and Terms

Anaplasmosis
An often fatal infectious disease of cattle caused by a microscopic parasite of red blood cells, spread by ticks or horsefly bites or by reusing needles or instruments between animals. A vaccine is available in some states with a conditional USDA license, but unless the risk is high, a routine vaccination for anaplasmosis is not recommended.

Bacterin
A bacterial vaccine.

Blackleg
A highly fatal disease of young cattle caused by one type of *Clostridium* bacteria. See Clostridial disease.

BRSV (Bovine Respiratory Syncytial Virus)
A virus that can cause severe, acute respiratory disease, especially in young cattle.

Brucellosis:
An infection resulting in abortion in females and inflammation and damage to the testicles in males, caused by the bacterium *Brucella abortus*. Also known as Bang's disease. See Calfhood vaccination.

BVD (Bovine Virus Diarrhea)
A disease caused by bovine viral diarrhea virus (BVDV), resulting in numerous problems, such as damage to the digestive and immune systems, pneumonia, abortions, calf deformities, and others. Incomplete vaccination programs, such as those omitting a needed booster vaccination, have led to BVD outbreaks in some herds.

Calfhood vaccination (official calfhood vaccination)
Vaccination against *Brucella abortus* for heifers between approximately 4 and 10 months old (the exact age for official calfhood vaccination is state specific). Calfhood vaccination must be administered by a federally accredited veterinarian (most large animal veterinarians are federally accredited). Calfhood vaccination against *Brucella abortus* is not mandatory in most states. Animals given the official calfhood vaccination are marked in the right ear with an official orange ear tag and a special tattoo. The tattoo consists of an R designating the strain of the vaccine (RB51), a V-shaped shield in the middle, and then a number denoting the last digit of the year of the vaccination. For example, calves vaccinated against *Brucella abortus* in the year 2012 would have RV2 tattooed in the middle of the inside of the right ear. The decision to have heifer calves vaccinated for brucellosis should be based on the advice of the herd's veterinarian and depends on the marketing plan of the herd.

Clostridial disease
Fatal disease of young cattle caused by one of the *Clostridium* bacteria. Blackleg is the most well known, but other clostridial diseases are also highly fatal. Currently, the most commonly used clostridial vaccination in cattle is the 7-way type, which protects against *Clostridium chauveoi* (blackleg), *Clostridium septicum*, *Clostridium sordelli* (malignant edema), *Clostridium novyi* (black disease), and three types of *Clostridium perfringens* (enterotoxemia).

Coronavirus
A virus that can cause diarrhea (scours) and dehydration in young calves. Some scours vaccines given to pregnant females will contain coronavirus. Vaccination of the pregnant dam raises the level of antibodies to coronavirus in her colostrum, the first milk she produces which is suckled by the calf after it is born. Antibodies from colostrum provide the calf's immunity for the first few weeks and months of life.

E. coli
A bacterium that can cause a life-threatening infection and diarrhea (scours) in newborn calves. Some scours vaccines given to pregnant females will contain *E. coli*. Vaccination raises the level of antibodies against *E. coli* in the dam's colostrum milk suckled by the calf after it is born. Antibodies from colostrum provide the calf's immunity for the first few weeks and months of life.

Histophilus somni
A bacterium that can cause respiratory, nervous system, and reproductive diseases.

IBR (Infectious Bovine Rhinotracheitis)
A disease caused by a herpes virus, resulting in respiratory signs, reproductive failure, and abortions. The disease is sometimes called red nose and often initiates the shipping fever complex. Many IBR vaccines include additional respiratory viruses such as BVDV, BRSV, and PI3.

Intramuscular (IM)
Injection in the muscle, that is, with a needle penetrating directly into the muscle, usually at least 1 inch.

Leptospirosis (5 varieties)
A bacterium causing kidney disease, abortion in pregnant females, and sickness in calves. The five varieties of *Leptospira* most commonly found in cattle include *hardjo, Icterohaemorrhagiae, canicola, pomona*, and *grippotyphosa*. Breeding animals should be vaccinated with a 5-way lepto vaccine once a year before the breeding season. Leptospirosis vaccine is often combined with vibriosis vaccine.

Mannheimia haemolytica
A bacterium causing shipping fever pneumonia, often after infection with one of the respiratory viruses such as IBR, PI3, BRSV, or BVDV. Newer vaccines containing the leukotoxoid portion of Mannheimia haemolytica are more effective than the older vaccines, which did not provide adequate protection.

Pinkeye (Infectious Bovine Keratoconjunctivitis, or IBK)
An infection of the eye caused by infection with the bacterium Moraxella bovis, spread by flies. Higher incidence of pinkeye may occur in herds not vaccinated against IBR virus. Pinkeye vaccines are available.

PI_3 (Parainfluenza-3 virus)
A virus that can cause respiratory disease.

Rotavirus
A virus that can cause diarrhea (scours) and dehydration in young calves. Some scours vaccines given to pregnant females will contain rotavirus. Vaccination of the pregnant dam raises the level of antibodies to rotavirus in her colostrum, the first milk she produces which is suckled by the calf after it is born. Antibodies from colostrum provide the calf's immunity for the first few weeks and months of life.

Subcutaneous (SQ or subq)
Injection under the skin, not deep into the underlying muscle. If approved as a route of injection on the vaccine label, subcutaneous injection is just as effective as the intramuscular route and is the preferred route to avoid muscle damage.

Toxoid
A vaccine that protects against a bacterial toxin.

Trichomoniasis
A protozoal disease caused by *Tritrichomonas foetus* and transmitted during breeding, resulting in failure of early pregnancy, an extended breeding season as females come back into heat, and abortion. A vaccine is available to raise the resistance against *Tritrichomonas foetus* in the breeding herd.

Vibriosis:
A bacterial disease caused by *Campylobacter fetus* subspecies *venerealis*, resulting in failure of early pregnancy and an extended breeding season as females come back into heat. Vaccinate breeding bulls and females at least once a year before the breeding season. Vibriosis vaccine is often combined with leptospirosis in one vaccine.

ALABAMA COOPERATIVE Extension SYSTEM

ANR-0968

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Equine Vaccination and Parasite Control: Current Recommendations

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UF UNIVERSITY of FLORIDA
IFAS Extension

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Overview

- General overview
- Physical examination
- Why vaccinate?
- How do I vaccinate?
- What diseases should I vaccinate for?
- Parasite Control

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Annual Physical Examination

- Important part of wellness program
- Know normals for your horse
- Veterinary-client-patient relationship
- Critical for emergency situations – your vet knows your horse

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Why Vaccinate?

- Critical component of a horse health maintenance program
- Primes the immune system for a quick response when exposed to infection
- Prevent life-threatening diseases
- Minimize or eliminate contagious diseases that affect performance or herd health

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Why Vaccinate? Prevent some FATAL diseases

- Rabies
- Tetanus
- Encephalomyelitis (EEE/WEE)
- West Nile Virus

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Aids in the Prevention of Others:

- Rhinopneumonitis (Equine Herpesvirus)
- Influenza
- Strangles
- Botulism
- Potomac Horse Fever
- Rotavirus
- And more….

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Vaccination can NOT guarantee disease prevention in all cases

- Vaccination should be used in conjunction with good nutrition, parasite control, pasture management, and minimizing stress in your herd

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How do I vaccinate?

- No standard vaccination program for every horse, but ALL horses should receive EWT, Rabies, and WNV vaccination
- Work with your veterinarian on what is best for your horse/herd
- A primary series of vaccine and a booster dose should be given prior to exposure

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How?

- Most vaccines are given IM (in muscle)
- Some are available to give IN (intranasal)
  - Influenza
  - Strangles

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Adverse Reactions

- Are uncommon
- Muscle soreness
- Swelling
- Fever
- Lethargy
- Swollen legs, vasculitis, colic

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Vaccine Reminders

- Safety and efficacy data are not available on the concurrent use of vaccines
- Administration of killed and MLV vaccines at same site may inactivate the MLV

Maternal Antibodies

- Protect the foal but inhibit the foal's response to vaccination – *maternal antibody interference*
- Adequate passive transfer leads to similar antibody titers in the foal as are in the mare at the time of foaling

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Maternal Antibody Advantage

- Take advantage of maternal antibodies and boost all mares 4-6 weeks prior to foaling
- Ensure foal consumes adequate colostrum

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Vaccination of Foals

- Start most foals at 5-6 months of age
  - Foals in the SE region should start a 3 dose series for EEE and WNV at 3-4 months old
- For most diseases give 3 dose series instead of 2

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What should I vaccinate for?

- ALL HORSES
  - Tetanus
  - Encephalomyelitis
  - West Nile Virus
  - Rabies
- MOST/PERFORMANCE
  - Influenza
  - Equine Herpesvirus
- SOME
  - Strangles
  - Potomac Horse Fever
  - Botulism
  - Rotavirus

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Tetanus

- Caused by *Clostridium tetani*
- Present in intestine, manure, and soil
- Spores can exist for years in soil
- Spores enter through wounds, lacerations, umbilicus

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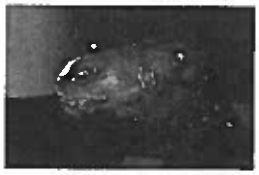
Clinical Signs of Tetanus

- Muscle stiffness
- Sawhorse stance
- Third eyelid prolapse
- Lockjaw
- Flaring nostrils
- Hypersensitive to touch



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Tetanus

- Not contagious
- Mortality rate is high
- All horses should be vaccinated annually
- Tetanus toxoid vaccine is safe and provides good protection
- Tetanus antitoxin is for previously unvaccinated horses
  - Risk of liver disease

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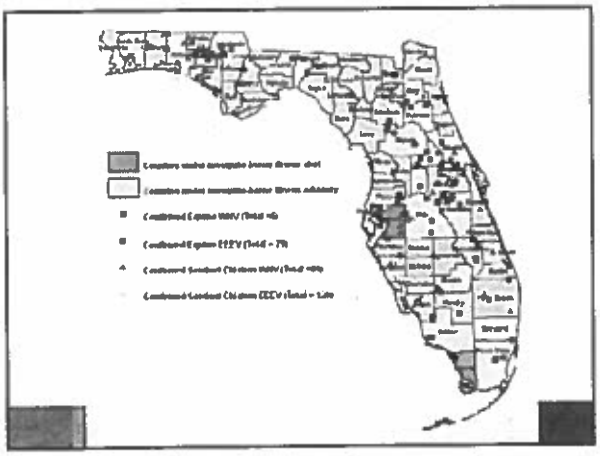
When to vaccinate - Tetanus

- Not typically seasonal, vaccinate any time of year
- Most horses in spring, in combination with EEE/WEE
- Boost if wound or surgery >6 months from last dose

Encephalomyelitis

- Often referred to as "sleeping sickness"
- Vaccines available for Eastern Equine Encephalomyelitis (EEE), Western Equine Encephalomyelitis (WEE), and Venezualen Equine Encephalomyelitis (VEE)
- Viruses are transmitted by mosquitos



Encephalomyelitis

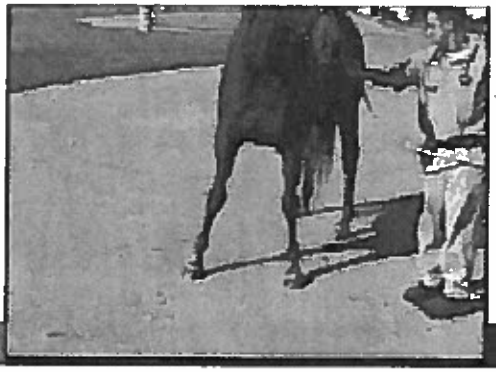
- WEE seen throughout North America
- EEE seen mostly in the East and Southeast, prevalent in Florida
- VEE not in US for many years – reportable foreign disease

Clinical Signs of Encephalomyelitis

- Result from inflammation of the brain and/or spinal cord
- Fever
- Depression
- Staggering gait (ataxia)
- Paralysis
- Seizures

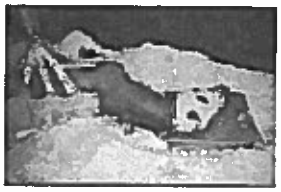
Abnormal Mentation

Abnormal Gait - Ataxia



Encephalomyelitis

- EEE/VEE – mortality rate 70-90% die
- WEE – mortality rate about 50%
- Vaccination is safe and generally very effective against these diseases



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When to Vaccinate – EEE/WEE

- ALL horses should be vaccinated annually in the spring, prior to mosquito season
- In Florida, recommend boosting every 4 months for EEE/WEE
- Foals should receive first dose at 3-4 months of age, and 2 additional doses one month apart

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Vaccination Recommendations

- Adult horses >3 years of age.
  - Vaccinate a minimum of **two times** yearly
  - Vaccinate first in February or very early March
  - Revaccinate in August/September
  - Your veterinarian may recommend vaccination 3X year based on the local activity. Some counties/townships have continuous activity
    - Citra
    - Volusia
    - Ocala National Forest
    - Panhandle
    - Some areas outside of Jacksonville

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Vaccination Recommendations

- ALL New Arrivals into Florida
  - Vaccinate 4-6 wks before shipping
  - If not vaccinated before shipping:
    - Give full initial series (two doses, 3 wks apart) if minimally vaccinated for EEE
    - Give one dose if vaccinated 4-6 months previously
  - If NO EEE vaccine history, consider placing on 3X/year for at least three years
  - If vaccinated annually increase to 2X/year

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Vaccination Recommendations

- Young horses 1-3 years old
  - Must be vaccinated three times per year
  - Vaccine Schedule
  - January, February
  - June, July
  - September, October

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West Nile Virus

- Another mosquito transmitted virus
- Causes inflammation of the brain and spinal cord
- Not contagious from horse to horse

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Clinical Signs of West Nile Virus

- Can vary
- Fever
- Muscle tremors
- Incoordination/ ataxia
- Hypersensitive
- Facial nerve or other paralysis

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West Nile Virus

- Mortality rate about 33%
- Associated with inability to stand, prolonged recumbency

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When to Vaccinate - WNV

- Multiple vaccines available
  - Fort Dodge/Pfizer West Nile Innovator – killed
  - BI's Vetera
  - Merial's RecombiTek – recombinant vaccine
  - Intervet's killed chimera vaccine
- Annual vaccination recommended
- In Florida and SE states with larger mosquito populations, may boost every 6 months

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Rabies

- 100% FATAL in all cases
- Virus affecting the nervous system
- Horses are exposed through infected wildlife
- Horses can infect people

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Rabies in Horses

- From 2005-2006, rabies cases in horses and mules increased 12.8% *(Blanton et al)*
  - But decreased by 20% in 2007 (42 horses in US)
- Since 2006, 8 horses in Florida have died from rabies
- FATAL in all mammals with clinical signs
  - 2 human survivors with no vaccination
- Death occurs in 3-7 days once signs develop

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Common Risk Factors for Horses

- Live in an endemic area
- No vaccination
- Horse lives outside 24 hours/day
- Young > old
- No breed or gender predisposed
- Typically affects one horse on a farm

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Clinical Signs of Rabies

- Can look like anything!
- Behavioral changes, blindness
- Ataxia and incoordination
- Fever
- Hypersalivation
- Paralysis
- Colic

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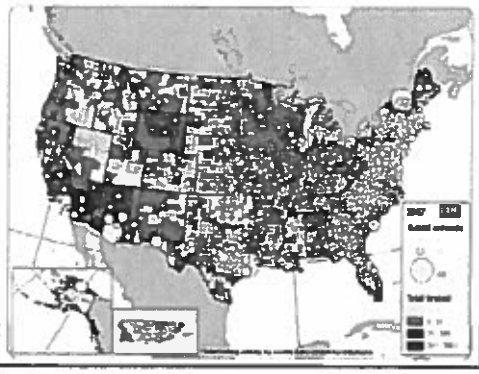
Rabies in a Horse

Other Diseases Can Look Similar

Rabies: CDC Reported Cases



Rabies Vaccination

- The National Association of State Public Heath Veterinarians recommends vaccination for all livestock in frequent contact with humans, and specifically horses that travel interstate
- The AAEP considers Rabies a CORE vaccine for all horses

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When to vaccinate - Rabies

- Incubation period 2-9 weeks
- Death in 3-5 days once signs develop
- Vaccinate once a year
- Vaccine is safe and effective

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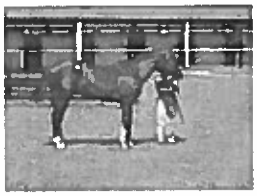
Equine Influenza

- Incubation period 1-3 days
- High fever (1-5 days)
- Young horses are at risk!
- Spread by aerosolized droplets, infected fomites – highly contagious



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Equine Influenza – Clinical Signs

- Cough (several weeks) and fever
- Lethargy, depression, reduced appetite, muscle soreness
- Nasal discharge
- Most horses recover in 10-14 days with supportive care

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When to Vaccinate - Influenza

- Intranasal vaccine every 6 months
- Intramuscular vaccine every 4-6 months
- Start foals at 6-9 months of age

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Equine Herpesvirus (EHV)

- EHV-1 : Abortion, neonatal death, neurologic disease, respiratory disease
- EHV-4 : respiratory disease
- Also known as "Rhinopneumonitis"

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Equine Herpesvirus - Respiratory

- Clinical signs are identical to influenza
- Incubation time 2-10 days
- Shed virus for 2-3 weeks
- Adults 2-4 times a year
- Weanlings and yearlings every 3 months

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Equine Herpesvirus – Neurological Form


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EHV-1 – Neurological Form

- Vaccines are NOT effective at preventing disease
- Outbreaks can occur with the first signs
  - FEVER
  - +/- Respiratory signs
  - Monitor stressed horses closely

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Equine Herpesvirus - Abortion

- Abortion is caused by EHV-1
- Pregnant mares: booster 5,7,9 of pregnancy

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Strangles: *Streptococcus equi spp. equi*

- Transmission: Ingestion or inhalation of infected discharge
  - Horse-horse contact or fomites
  - Highly contagious
- High morbidity, low mortality
- Incubation period 3-5 days

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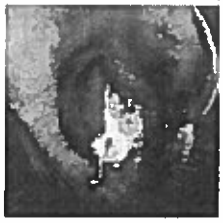
Strangles – Clinical Signs

- Cough, fever, lymph node enlargement
- Respiratory distress due to retropharyngeal lymph node enlargement
- Complications
  - Purpura
  - Metastatic ("Bastard") strangles



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Vaccination for Strangles

- Previously affected farms
- May lessen disease severity
- Vaccine reactions
  - Purpura
  - Abscesses if IM vaccines become contaminated
- Annual booster
- Intranasal vaccine

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Strangles Vaccination in an Outbreak

- Generally not recommended
- Can consider for healthy horses with no signs and NO contact with sick horses
- Consider serology prior to vaccination – Contraindicated if >1:1,600

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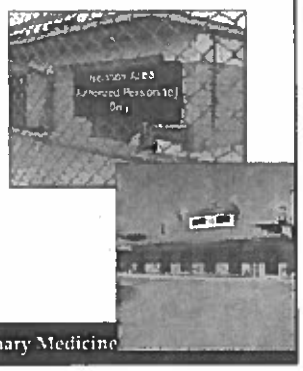
Management Practices

- Quarantine and monitoring of new horses
- Isolation facility and protocol
- Requirements prior to introduction of new horse
- Separation of groups of horses according to use, susceptibility to infection



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Management Practices

- Vector control
- Management of sick horses

Keep good records!

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Management and Internal Parasites

- Parasites can cause extensive internal damage
- Effects range from dull hair coat and unthriftiness to weight loss, colic, and death

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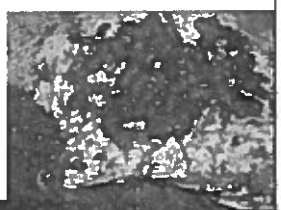
Equine Internal Parasites

- Over 150 species can affect the horse
- The most common are:
  - Large Strongyles
  - Small Strongyles
  - Ascarids (roundworms)
  - Tapeworms
  - Bots
  - Pinworms
  - Lungworms
  - Threadworms



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Clinical Signs of Internal Parasites

- May have none at all
- Dull, rough hair coat
- Loss of condition
- Poor performance
- Unthrifty
- Lethargy/depression
- Colic
- Diarrhea
- Pot belly (young horses)

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Major Internal Parasites

- Large Strongyles
- Small Strongyles (Cyathostomes)
  - Seasonally transmitted
  - Winter in Florida
- Roundworms (Ascarids)
  - Year round infection
- Tapeworms

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Resistance

- When a greater frequency of individuals in a population can tolerate doses of a compound than in a normal population of the same species and is heritable
- Treatment with dewormers selects for resistant genetic alleles over time (because susceptible worms die)

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Refugia

- Critical to limit resistance
- Refugia are the parasites not exposed to the drug at the time of treatment (eggs and larvae on pasture), certain stages in treated horses (depending on drug/dose) and those in untreated horses
- Provide a pool of sensitive parasites

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Resistant Small Strongyles on SE horse Farms
(Kaplan et al 2004)

- 1274 horses tested from 44 large farms in GA, SC, FL, KY and LA
- Resistance testing only for small strongyles
- Percent of farms found to harbor resistant worms:
  - 97.7% for fenbendazole
  - 0% for ivermectin
  - 53.5% for oxibendazole
  - 40.5% for pyrantel pamoate

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Diagnosis of Internal Parasites

- Fecal Egg Counts (FEC) can be very helpful!!!
  - Negative fecal does not always mean no parasites
- FECRT (Fecal Egg Count Reduction Test)
  - Monitors response to dewormer, recheck fecal 14 days after deworming – should be reduced >90% if parasites are sensitive to dewormer
- Monitor multiple horses on farm at same time
- Some parasites are difficult to diagnose - tapeworms

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What do Fecal Egg Counts Mean?

- FEC <200 epg – low egg shedder, horse less likely to have any ill effects of parasite
- FEC 200-500 – moderate egg shedder
- FEC >500 epg – high egg shedding into environment
- FEC do NOT correlate directly with actual intestinal worm burden, but does give an estimate of how much horse is contaminating the environment.
- 20% of your horses shed 80% of the worm eggs!

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Dewormers

- None are 100% effective
- Use a broad spectrum product as basis for control (ivermectin, moxidectin)
- Be sure to treat for tapeworms 1-2 times per year
  - Double dose Strongid®
  - Product containing praziquantel

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Deworming

- Consult your veterinarian
- Rotating dewormers too frequently will promote resistant parasites
- Don't neglect management!

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FL Parasite Control Program Recommended by Dr. Kaplan

- **Oct 1**- Perform FEC on ALL horses. Treat all horses with Ivermectin or moxidectin (+praziquantel)
- **Dec 1** – Treat horses that were treated with ivermectin in October with oxibedaziole and/or pyrantel. All horses with FEC> 500 epg. +/- treat horses with FEC 200-500 epg.
- **Jan 1** – Treat ALL horses regardless of FEC, use ivermectin/praziquantel or moxidectin/praziquantel; perform FEC all horses

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FL Parasite Program Cont'd.

- **April 1** – Treat only high shedders if moxidectin was used in January. Treat with oxibendazole, pyrantel, or power pack
- **May-September** – NO TREATMENT necessary. Too hot for transmission.
- Monitoring FEC is critical for this program

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Recommendations for Foals and Yearlings (< 18 mos)

- Critical to perform FECRT on all drugs used in foals and monitor egg reappearance
- Ivermectin or moxidectin at least every 6 months for large strongyles
- For roundworms – resistance has been documented to ivermectin/moxidectin
  - Start treatment at 2 months old with ivermectin or benzimidazole, pyrantel
  - Monitor ERP – may be as short as every 4-6 weeks and require retreatment.
- Larvacidal treatment for small strongyles between 6 mo – 2 yrs in late spring

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In Summary

- Consult your veterinarian
- Remember basic good management and horse health care
- Semi-annual physical exams, vaccination, and fecal examination
- http://extension.vetmed.ufl.edu/equine-extension/

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Vaccination Program for Horses

By **Thomas J. Lane, BS, DVM, Professor Emeritus, University of Florida, Large Animal Clinical Sciences**

The goal of vaccination is to develop and maintain both individual and herd immunity against infectious diseases. Commercial vaccines are available for rabies, encephalomyelitis (Eastern, Western, and Venezuelan), tetanus, influenza, equine herpesviruses 1 and 4, botulism, equine ehrlichiosis (Potomac horse fever), equine viral arteritis, rotavirus, West Nile virus, and *Streptococcus equi* (strangles). Vaccination programs are formulated based on the animal's age, use, and level of exposure. Broodmare vaccination is important to provide active immunity for the mare and passive immunity for the foal via transfer of colostral antibodies. Vaccination guidelines for foals have been modified because of the interference of maternal antibodies with the initial vaccination response. Sources such as the American Association of Equine Practitioners can provide the most current equine vaccination recommendations for horses in the USA.

The following vaccination recommendations assume that foals are born to vaccinated mares and have absorbed adequate colostral antibodies with IgG levels >800 mg/dL.

Foals with failure of passive antibody transfer (ie, IgG levels <200 mg/dL) and/or foals born to unvaccinated mares can receive their initial vaccination for equine herpesvirus 1 and 4, tetanus, and Eastern and Western equine encephalomyelitis beginning at 3–4 mo of age, followed by a second dose 4–6 wk later and a third dose at 10–12 mo of age. These foals can receive their first dose of rabies vaccine at 3–4 mo of age, followed by a booster at 12 mo. Influenza vaccination can be started at 6 mo of age. Foals born to mares that have never been exposed to or vaccinated against West Nile virus can receive their first vaccination at 3–5 mo of age.

Tetanus:

Clostridium tetani is present in all parts of the world. Tetanus occurs most commonly when wounds become contaminated with the organism from the soil. Vaccination is recommended for all horses and ponies on an annual basis. A horse with an unknown vaccination status that sustains an injury should receive a dose of tetanus antitoxin along with a dose of tetanus toxoid. A second dose of toxoid should be given 4 wk later. Foals from vaccinated mares should be given a three-dose series at 6, 7, and 9 mo of age. Foals from unvaccinated mares should receive tetanus toxoid at 3, 4, and 6 mo of age. Whenever a vaccinated horse experiences a serious wound, a tetanus booster may be indicated.

Equine Herpesvirus 1 and 4 (Rhinopneumonitis):

Two types of equine herpesviruses vaccine are available, EHV-1 and EHV-4. The EHV-1 virus is the primary cause of abortion and neurologic disease in horses. The EHV-4 virus is the primary cause of respiratory infections, especially in young horses. The control of rhinopneumonitis is by a combination of vaccination and good management practices. To prevent abortion, a killed EHV-1 vaccine should be administered to pregnant mares at 3, 5, 7, and 9 mo of gestation. Respiratory disease is best prevented using a combination vaccine of EHV-1 and EHV-4. There is no evidence that vaccination protects against the neurologic form of the disease. Vaccine recommendations for foals are three doses every 4 wk starting at 6 mo of age, and a booster at 1 yr of age. Pleasure and performance horses should be vaccinated every 3–6 mo, depending on the risk of exposure. Broodmares should have an EHV-4 vaccination 2–4 wk before foaling to ensure the availability of colostral immunity.

Encephalitis:

Eastern equine encephalitis (EEE) virus has a wide distribution. This includes the eastern USA, Central and South America, and eastern Canada. In the USA, EEE is seen primarily in the southeastern states but has been reported in all states east of the Mississippi River. EEE in horses is nearly always fatal regardless of treatment. Infected horses usually become comatose, seizure, and die in 36–48 hr.

The virus of Western equine encephalitis (WEE) is primarily located in the western USA. In recent years, reports of WEE have not been common, probably as a result of adequate vaccination programs. The virus is not as pathogenic as EEE, and animals infected with WEE have a greater chance of survival. Mortality is usually 50%.

Venezuelan equine encephalitis (VEE) virus causes outbreaks of disease in horses in Mexico, Central and South America, and occasionally the southern USA. The VEE vaccine is a single vaccine or in combination with EEE and WEE. Its need is limited to those horses traveling to or located in endemic areas.

West Nile virus (WNV) disease is considered a zoonotic disease similar to the encephalitis viruses. The bird population allows for maintenance of the virus, which is then transmitted by mosquitoes to both people and horses. There is very little risk of disease from any infected horses. Vaccination protocols for broodmares, foals, and adult horses are the same as those for Eastern and Western encephalitis. Combination vaccines that include EEE, WEE, and WNV are available.

Influenza:

Influenza is one of the most frequent causes of viral respiratory disease in horses and is highly contagious among susceptible horses. Therefore, vaccination is recommended for all foals, broodmares, and horses at risk of exposure, usually as a result of showing, racing, or shipping. Young foals from vaccinated mares, because of maternal antibodies, should be vaccinated at 9, 10, and 12 mo of age. Foals from unvaccinated mares should be vaccinated earlier. The intranasal vaccine can be used every 6 mo in young, susceptible performance horses 2–4 yr old. A vaccination is recommended every 6 mo for horses exposed to other horses at equine events.

Rabies:

Signs of rabies include the inability to eat or drink, disorientation, and incoordination. Rabies is fatal, and also poses a health risk to those who handle infected horses. Prevention in horses is primarily via vaccination. Broodmares should be vaccinated 4–6 wk before foaling. Foals from vaccinated mares

should be vaccinated at 6 and 7 mo of age and again at 12 mo of age. Foals from unvaccinated mares should be vaccinated at 3, 4, and 12 mo of age. All adult horses should be vaccinated annually.

Potomac Horse Fever:

Potomac horse fever (equine monocytic ehrlichiosis, equine ehrlichial colitis) is caused by *Neorickettsia risticii*. Vaccination is recommended in endemic areas, such as near freshwater streams, rivers, ponds, and heavily irrigated pastures. Annual boosters in the spring are recommended. Pregnant mares should receive a booster before foaling. Although the vaccine may lessen the impact of the disease, it appears not to prevent abortion in pregnant mares.

Botulism:

Equine botulism is mostly seen in the Mid-Atlantic area of the United States, but the disease has also been reported worldwide. In the USA, the most common types are B and C botulism, although type A has been reported in the western USA. The spores of *Colostridium botulinum* are found in the soil and are resistant to light, drying, and heat. The Type B vaccine gives good protection in adult horses. Adult horses and broodmares in areas where botulism is a potential hazard should receive initially three doses at 30-day intervals and then annual boosters. Foals from vaccinated mares in endemic areas should be vaccinated at 2, 3, and 4 mo of age, because colostral immunity does not always occur. Foals from unvaccinated mares should be vaccinated at 2, 4, and 8 wk of age. The use of Type B vaccine in areas where Type C occurs has uncertain results.

Streptococcus equi Infection:

Streptococcus equi infection (equine strangles, equine distemper) can be highly contagious. Use of the IM or intranasal vaccine is recommended only in those situations or on premises where the disease has been a problem. The IM vaccine involves three injections administered 2–4 wk apart, starting at 4 mo of age in foals, with a booster at 12 mo of age. If the intranasal vaccine is used, vaccination can begin at 6–9 mo of age, with a second dose given 3–4 wk later and a third dose administered at 12 mo of age. Adult horses, if in potential disease situations, should receive an annual vaccination. Broodmares on endemic farms should receive an annual booster using the IM vaccine 4–6 wk before foaling. Because of the increased risk of inducing immune-mediated purpura hemorrhagica, horses with titers to the SeM surface protein of *S equi* in excess of 1:1,600 may not need to be vaccinated.

Rotavirus:

Foal diarrhea as a result of rotavirus infection can be a severe problem on some breeding farms. Single cases or a farm outbreak can occur. Therefore, pregnant mares should be vaccinated IM at 8, 9, and 10 mo of gestation. This will increase the amount of colostral immunoglobulins. It is a vaccine for use in particular situations or areas of high endemicity.

Equine Infectious Anemia:

In the acute state, equine infectious anemia (swamp fever) causes severe RBC destruction and anemia. Once infected, a horse can become a carrier for life and is a threat to other horses. The virus is transmitted by bloodsucking insects. Currently, the disease is uncommon, and most horse owners are aware of its dire consequences. A simple blood test, the Coggins test, is available to detect infected

horses. A horse that tests positive cannot cross state lines and is required to be maintained in strict isolation for life. For horses participating in equine activities or being transported across state lines, proof of a negative Coggins test is required. Annual testing of every horse is recommended. No vaccine is available.

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VACCINATIONS FOR FOALS

<u>ALL</u> VACCINATION PROGRAMS SHOULD BE DEVELOPED IN CONSULTATION WITH A LICENSED VETERINARIAN

The two categories below reflect differences in the foal's susceptibility to disease and ability to mount an appropriate immune response to vaccination based on the presence (or absence) of maternal antibodies derived from colostrums. The phenomenon of maternal antibody interference is discussed in the text portion of these guidelines.

<u>CORE VACCINATIONS</u> protect against diseases that are endemic to a region, those with potential public health significance, required by law, virulent/highly infectious, and/or those posing a risk of severe disease. Core vaccines have clearly demonstrated efficacy and safety, and thus exhibit a high enough level of patient benefit and low enough level of risk to justify their use in all equids.

	Foals and Weanlings (<12 months of age) *Of mares vaccinated in the prepartum period against the disease indicated*	**Foals and Weanlings** (<12 months of age) *Of unvaccinated mare*	
DISEASE			**COMMENTS**
Tetanus	3-dose series: 1st dose at 4-6 months of age 2nd dose 4-6 weeks after the 1st dose 3rd dose at 10-12 months of age	3-dose series: 1st dose at 1-4 months of age 2nd dose at 4 weeks after the 1st dose 3rd dose 4 weeks after 2nd dose	
Eastern/Western Equine Encephalomyelitis (EEE/WEE)	3-dose series: 1st dose at 4-6 months of age* 2nd dose at 4-6 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. ***Foals in the Southeastern USA:*** The primary vaccination series should be initiated with an additional dose at 3 months of age due to early seasonal vector presence.	3-dose series: 1st dose at 3-4 months of age* 2nd dose 4 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. ***Foals in the Southeastern USA:*** The primary vaccination series should be initiated at 3 months of age due to early seasonal vector presence.	<u>*Note:*</u> Primary vaccination series scheduling may be amended with vaccinations administered earlier to younger foals that are at increased disease risk due to the presence of vectors. A foal born during the vector season may warrant beginning vaccination at an earlier age than a foal born prior to the vector season.
Rabies	3-dose series: 1st dose at 6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose at 10-12 months of age	3-dose series: 1st dose at 3-4 months of age 2nd dose 4 weeks after 1st dose 3rd dose at 10-12 months of age	
West Nile Virus (WNV)	<u>*Inactivated vaccine**</u> 3-dose series: 1st dose at 4-6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. <u>*Recombinant canary pox vaccine*</u> 3-dose series: 1st dose at 5-6 months of age 2nd dose 4 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. <u>*Flavivirus chimera vaccine*</u> 2-dose series: 1st dose at 5-6 months of age 2nd dose at 10-12 months of age,	<u>*Inactivated vaccine**</u> 3-dose series: 1st dose at 3-4 months of age 2nd dose at 4 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. <u>*Recombinant canary pox vaccine*</u> 3-dose series: 1st dose at 5-6 months of age 2nd dose at 4 weeks after 1st dose 3rd dose at 10-12 months of age, prior to the onset of the next vector season. <u>*Flavivirus chimera vaccine*</u> 2-dose series: 1st dose at 5-6 months of age 2nd dose at 10-12 months of age	<u>*Note:*</u> Primary vaccination series scheduling may be amended with vaccinations administered to younger foals that are at increased risk of exposure due to the presence of vectors. A foal born during the vector season may warrant initiation of the primary vaccination series at an earlier age than a foal born prior to the vector season There is no data for the use of the recombinant or chimera product in foals <5 months of age. If either product is administered to foals at

	prior to the onset of the next vector season. ***Foals in the Southeastern USA:*** Due to early seasonal vector presence, the primary vaccination series should be initiated earlier with the addition of a dose at 3 months of age.	prior to the onset of the next vector season. ***Foals in the Southeastern USA:*** Due to early seasonal vector presence, the primary vaccination series should be initiated at 3 months of age.	<5 months of age, the recommended primary schedule should still be completed.

<u>RISK-BASED VACCINATIONS</u> are those having applications which may vary between individuals, populations, and geographic regions. Risk assessment should be performed by, or in consultation with, a licensed veterinarian to identify which vaccines are appropriate for a given horse or population of horses. The listing of a vaccine here is not a recommendation for its inclusion into a vaccination program. Vaccine scheduling is provided for use after it has been determined which, if any, risk-based vaccines are indicated. Note: vaccines are listed in this table in alphabetical order not in order of priority for use.

DISEASE	Foals and Weanlings (<12 months of age) *Of mares vaccinated in the prepartum period against the disease indicated*	Foals and Weanlings (<12 months of age) *Of unvaccinated mares*	COMMENTS
Anthrax	Not applicable. As it is not recommended to vaccinate mares during pregnancy there will be no foals of mares vaccinated prepartum	No age specific guidelines are available for this vaccine. Manufacturer's recommendation is for primary series of 2 doses administered subcutaneously at a 2-3 week interval.	Antimicrobial drugs must <u>not</u> be given concurrently with this vaccine. Caution should be used during storage, handling and administration of this live bacterial product. Consult a physician immediately should accidental human exposure (via mucus membranes, conjunctiva or broken skin) occur.
Botulism	3-dose series: 1st dose 2-3 months of age 2nd dose 4 weeks after 1st dose 3rd dose 4 weeks after 2nd dose	3-dose series: 1st dose 1-3 months of age 2nd dose 4 weeks after 1st dose 3rd dose 4 weeks after 2nd dose	Maternal antibody does not interfere with vaccination; foals at high risk may be vaccinated as early as 2 weeks of age.
Equine Herpesvirus (EHV)	<u>*Inactivated or modified live vaccine*</u> 3-dose series: 1st dose 4-6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose at 10-12 months of age Revaccinate at 6-month intervals	<u>*Inactivated or modified live vaccine*</u> 3-dose series: 1st dose of 4-6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose at 10-12 months of age Revaccinate at 6-month intervals.	
Equine Viral Arteritis (EVA)	**Colt (male) foals:** Single dose at 6-12 months of age (see comments)	**Colt (male) foals:** Single dose at 6-12 months of age (see comments)	**<u>Prior to initial vaccination, colt (male) foals should undergo serologic testing</u>** and be confirmed negative for antibodies to EAV. Testing should be performed shortly prior to, or preferably at, the time of vaccination. As foals can carry colostral derived antibodies to EAV for up to 6 months, testing and vaccination should <u>not</u> be performed prior to 6 months of age.

Equine Influenza	*Inactivated vaccine* 3-dose series: 1st dose at 6 months of age 2nd dose 3-4 weeks after 1st dose 3rd dose at 10-12 months of age *Modified live vaccine* 2-dose series administered intranasally: 1st dose at 6-7 months of age 2nd dose at 11-12 months of age Revaccinate at 6-month intervals	*Inactivated vaccine* 3-dose series: 1st dose at 6 months of age 2nd dose at 3-4 weeks after 1st dose 3rd dose at 10-12 months of age *Modified live vaccine* 2-dose series administered intranasally: 1st dose at 6-7 months of age 2nd dose at 11-12 months of age Revaccinate at 6-month intervals	An increased risk of disease may warrant vaccination of younger foals. Because some maternal, anti-influenza antibody is likely to be present, a complete series of primary vaccinations should be given after 6 months of age.
Potomac Horse Fever (PHF)	2-dose series: 1st dose at 5 months of age 2nd dose 3-4 weeks after 1st dose	2-dose series: 1st dose at 5 months of age 2nd dose 3-4 weeks after 1st dose	If risk warrants, vaccine may be administered to younger foals. Subsequent doses are to be administered at 4-week intervals until 6 months of age.
Rotavirus	Not recommended in foals	Not recommended in foals	
Strangles ***Streptococcus equi***	*Killed vaccine* 3-dose series: 1st dose at 4-6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose 4-6 weeks after 2nd dose *Modified live vaccine* 3-dose series administered intranasally: 1st dose at 6-9 months of age 2nd dose 3-4 weeks after 1st dose 3rd dose at 11-12 months of age	*Killed vaccine* 3-dose series: 1st dose at 4-6 months of age 2nd dose 4-6 weeks after 1st dose 3rd dose 4-6 weeks after 2nd dose *Modified live vaccine* 3-dose series administered intranasally: 1st dose at 6-9 months of age 2nd dose 3-4 weeks after 1st dose 3rd dose at 11-12 months of age	Vaccination is **not** recommended as a strategy in outbreak mitigation. If risk warrants, the modified live vaccine (MLV) may be safely administered to foals as young as 6 weeks of age. However, vaccine efficacy in this age group has not been adequately studied. If MLV is administered to younger foals, a 3rd dose of vaccine should then be administered 2-4 weeks prior to weaning.

Vaccinations for Foals developed by the American Association of Equine Practitioners Infectious Disease Committee, 2008.

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VACCINATIONS FOR ADULT HORSES

ALL VACCINATION PROGRAMS SHOULD BE DEVELOPED IN CONSULTATION WITH A LICENSED VETERINARIAN

CORE VACCINATIONS protect against diseases that are endemic to a region, are virulent/highly contagious, pose a risk of severe disease, those having potential public health significance, and/or are required by law. Core vaccines have clearly demonstrable efficacy and safety, with a high enough level of patient benefit and low enough level of risk to justify their use in all equids.

	Broodmares	**Other Adult Horses** (>1 year of age) *Previously vaccinated against the disease indicated*	**Other Adult Horses** (>1 year of age) *Unvaccinated or Lacking vaccination history*	
DISEASE				**COMMENTS**
Tetanus	*Previously vaccinated* Annual, 4-6 weeks pre-partum *Previously unvaccinated or having unknown vaccination history:* 2-dose series 2nd dose 4-6 weeks after 1st dose Revaccinate 4-6 weeks pre-partum	Annual	2-dose series 2nd dose 4-6 weeks after 1st dose. Annual revaccination	Booster at time of penetrating injury or prior to surgery if last dose was administered over 6 months previously.
Eastern/Western Equine Encephalomyelitis (EEE/WEE)	*Previously vaccinated:* Annual, 4-6 weeks pre-partum *Previously unvaccinated or having unknown vaccination history:* 2-dose series 2nd dose 4 weeks after 1st dose Revaccinate 4-6 weeks pre-partum	Annual – spring, prior to onset of vector season	2-dose series 2nd dose 4-6 weeks after 1st dose Revaccinate prior to the onset of the next vector season	Consider 6-month revaccination interval for: 1. Horses residing in endemic areas 2. Immunocompressed horses
West Nile Virus (WNV)	*Previously vaccinated:* Annual, 4-6 weeks pre-partum *Unvaccinated or lacking vaccination history:* It is preferable to vaccinate naïve mares when open. In areas of high risk, initiate primary series as described for unvaccinated adult horses.	Annual – spring, prior to onset of vector season	*Inactivated vaccine:* 2-dose series 2nd dose 4-6 weeks after 1st dose Revaccinate prior to the onset of the next vector season *Recombinant canary pox vaccine:* 2-dose series 2nd dose 4-6 weeks after 1st dose Revaccinate prior to the onset of the next vector season *Flavivirus chimera vaccine:* Single dose Revaccinate prior to onset of the next vector season	When using the inactivated or the recombinant product, consider 6-month revaccination interval for: 1. Horses residing in endemic areas 2. Juvenile (<5 yrs of age) 3. Geriatric horses (>15 yrs of age) 4. Immunocompressed horses
Rabies	Annual, 4-6 weeks pre-partum OR Prior to breeding*	Annual	Single dose Annual revaccination	*Due to the relatively long duration of immunity, this vaccine may be given post-foaling but prior to breeding and thus reduce the number of vaccines given to a mare pre-partum

RISK-BASED VACCINES are selected for use based on risk assessment performed by, or in consultation with, a licensed veterinarian. Use of these vaccines may vary between individuals, populations, and/or geographic regions.**
Note: Vaccines are listed in this table in alphabetical order, not in order of priority for use.

****Refer to "Principles of Vaccination" in main document for criteria used in performing risk assessment.**

	Broodmares	**Other Adult Horses** (>1 year of age) *Previously vaccinated against the disease indicated*	**Other Adult Horses** (>1 year of age) *Unvaccinated or Lacking vaccination history*	
DISEASE				**COMMENTS**
Anthrax	Not recommended during gestation	Annual	2-dose series 2nd dose 3-4 weeks after 1st dose Annual revaccination	Do not administer concurrently with antibiotics. Use caution during storage, handling and administration. Consult a physician immediately if human exposure to vaccine occurs by accidental injection, ingestion, or otherwise through the conjunctiva or broken skin.
Botulism	*Previously vaccinated:* Annual, 4-6 weeks pre-partum *Previously unvaccinated or having unknown vaccination history:* 3-dose series 1st dose at 8 months gestation 2nd dose 4 weeks after 1st dose 3rd dose 4 weeks after 2nd dose	Annual	3-dose series 2nd dose 4 weeks after 1st dose 3rd dose 4 weeks after 2nd dose Annual revaccination	
Equine Herpesvirus (EHV)	3-dose series with product labeled for protection against EHV abortion. Give at 5, 7 and 9 months of gestation.	Annual (see comments)	3-dose series 2nd dose 4-6 weeks after 1st dose 3rd dose at 4-6 weeks after 2nd dose	Consider 6-month revaccination interval for: 1. Horses less than 5 years of age. 2. Horses on breeding farms or in contact with pregnant mares. 3. Performance or show horses at high risk.
Equine Viral Arteritis (EVA)	Not recommended unless high risk.	Annual *Stallions, teasers:* Vaccinate 2-4 weeks before breeding season. *Mares:* Vaccinate when open.	Single dose (see comments)	**Prior to initial vaccination, intact males and any horses potentially intended for export should undergo serologic testing** and be confirmed negative for antibodies to EAV. Testing should be performed shortly prior to, or preferably at, the time of vaccination.

Influenza	*Previously vaccinated:* *Inactivated vaccine*: Semi-annual with one dose administered 4-6 weeks pre-partum *Canary pox vector vaccine:* Semi-annual with one dose administered 4-6 weeks pre-partum *Previously unvaccinated or having unknown vaccination history:* *Inactivated vaccine*: 3-dose series 2nd dose 4-6 weeks after 1st dose 3rd dose 4-6 weeks pre-partum *Canary pox vector vaccine:* 2-dose series 2nd dose 4-6 weeks after 1st dose but no later than 4 weeks pre-partum	Horses with ongoing risk of exposure: semi-annual Horses at low risk of exposure: Annual	*Modified live vaccine:* Single dose administered intransally. Revaccinate semi-annually to annually. *Inactivated vaccine*: 3-dose series 2nd dose 4-6 weeks after 1st dose 3rd dose 3-6 months after 2nd dose Revaccinate semi-annual to annually. *Canary pox vector vaccine:* 2-dose series 2nd dose 4-6 weeks after 1st dose Revaccinate semi-annually	
Potomac Horse Fever (PHF)	*Previously vaccinated:* Semi-annual, with one dose given 4-6 weeks prepartum. *Previously unvaccinated or having unknown vaccination history:* 2-dose series 1st dose 7-9 weeks prepartum 2nd dose 4-6 weeks prepartum	Semi-annual to annual	2-dose series 2nd dose 3-4 weeks after 1st dose Semi-annual or annual booster	A revaccination interval of 3-4 months may be considered in endemic areas when disease risk is high
Rotavirus	3-dose series 1st dose at 8 months gestation 2nd and 3rd dose at 4-week intervals thereafter	Not applicable	Not applicable	
Strangles *Streptococcus equi*	*Previously vaccinated:* *Killed vaccine containing M-protein):* Semi-annual with one dose given 4-6 weeks pre-partum *Previously unvaccinated or having unknown vaccination history:* *Killed vaccine containing M-protein):* 3-dose series 2nd dose 2-4 weeks after 1st dose 3rd dose 4-6 weeks prepartum	Semi-annual to annual	*Killed vaccine containing M-protein:* 2-3 dose series 2nd dose 2-4 weeks after 1st dose 3rd dose (where recommended by manufacturer) 2-4 weeks after 2nd dose Revaccinate semi-annually *Modified live vaccine:* 2-dose series administered intranasally 2nd dose 3 weeks after 1st dose Revaccinate semi-annually to annually	Vaccination is **not** recommended as a strategy in outbreak mitigation

Vaccinations for Adult Horses developed by the American Association of Equine Practitioners Infectious Disease Committee, 2008.

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