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A Horse Owner's Guide to Botulism

By Alyssa Biscoglio, DVM Student (Class of 2020) Edited by Stacy H. Tinkler, DVM, MPH, Dipl. ACVIM-LA

What is botulism?

Botulism is a paralytic disease affecting equines world-wide, and is caused by neurotoxins produced by the bacterium *Clostridium botulinum*. This bacterium is typically present in soil, decaying plant matter and animal carcasses. *C. botulinum* is a spore forming bacteria that thrives in anaerobic (where there is little to no oxygen) and basic (pH levels are > 6) environments. There are several types of *C. botulinum* toxins and they are classified as types A through G. Clinical botulism in horses has been attributed to *C. botulinum* types A, B, C, and D. Most cases of equine botulism in the United States occur in Kentucky and the Mid-Atlantic states, with types B and C most commonly reported. Type A toxin is reported more frequently in the Western United states in sporadic outbreaks, and Type B is the most commonly reported serotype in the whole US (>85% of cases).

How does a horse contract botulism?

Horses acquire *C. botulinum* toxin via three main routes: ingestion of spores (toxicoinfectious), wound infections, and through ingestion of preformed toxins in decaying plant matter, improperly preserved haylage or feed contaminated with decaying animal carcasses. The toxicoinfectious route commonly occurs in foals who ingest *C. botulinum* spores, and is sometimes referred to as Shaker Foal Syndrome due to the observed muscle tremors caused by the neurotoxin. The ingested spores become active bacteria in the animal's gut and begin producing toxins. Toxicoinfectious botulism is most common in foals 1 to 3 months of age, although it has been observed in foals as young as 7 days of age. Wounds may become infected with *C. botulinum* if not kept clean and dry. Deep puncture wounds may create anaerobic environments that foster bacterial growth and subsequent toxin formation.

How does botulism affect my horse?

Botulinum toxin causes paralysis because it blocks the transmission of electrical impulses from nerves to muscles via binding to the junction where these impulses are transferred. Once the communication between nerves and muscles is lost, muscles become weak and/or paralyzed. Once the toxin is bound to these junctions there is no way to remove it. Therefore, the only way for an animal to heal is for new junctions to form. Regeneration of these junctions may take around 7 to 10 days but return to full strength may take an affected animal up to 1 month. During earlier stages of disease horses typically demonstrate weakness with an intolerance to exercise, a short stiff gait, and inability to swallow. Horses may appear sleepy, their third eyelid may protrude, and their tongues may hang out of their mouths or be slow to retract. Horses will often drool and "play with their food/water" due to their inability to swallow. Foals exhibit similar signs and are typically noted dribbling milk from their mouth and nostrils. Colic may also be observed in the early stages of disease due to decreased gastrointestinal motility. Weakness observed in horses may progress to muscle tremors and increased time spent laying down. In the final stages of disease, horses may become unable to rise as full paralysis sets in. Ultimately, the muscles that allow horses to breathe become paralyzed, resulting in death. *(continued on page 2)*

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Botulism (continued from cover)

How is botulism diagnosed and treated?

Definitive diagnosis of botulism is challenging. In the majority of cases, diagnosis of botulism is made using the horse's history and clinical signs. Other neurologic diseases such as rabies, sleeping sickness, EPM, or viral encephalitis must also be ruled out. Toxin gene detection by using a PCR test after culture of *C. botulinum* from GI contents or feed is now the preferred test for diagnosis.

Treatment of equine botulism may be very costly due to the intensive care required. Treatment also comes with many risks, as even well managed down horses may suffer complications such as colic, pressure sores, and pneumonia. The treatment for botulism consists of early administration of anti-toxin as well as supportive care. The anti-toxin works by binding circulating toxin, and preventing toxin binding to the nerve cell surface receptor. Horses that are treated with anti-toxin have a much higher survival rate than those that are not. Supportive care includes feeding and oral fluids through a nasogastric tube or intravenous nutrition as well as IV fluids in animals that are unable to swallow. Additionally, horses may need to wear a muzzle to prevent intake and aspiration of shavings used as bedding. The stall should be heavily bedded if the horse is down for long stretches of time. Horses unable to rise may need additional padding and to be rotated every few hours to prevent development of pressure sores. Some horses may need a urinary catheter placed, and horses unable to blink will need artificial tears. Severe cases may require a ventilator if the horse is unable to breathe on its own. Horses mildly affected by botulism have a fair prognosis with proper medical management. Horses unable to rise have a very poor prognosis and foals have a good prognosis if the disease is caught early on and anti-toxin is administered. Horses typically require 10 to 14 days to gradually recover. A longer recovery may be required if large amounts of toxin are ingested.

How can botulism be prevented?

Botulism may be prevented through proper vaccination, wound care, and administration of quality feed. Vaccination is only effective for Type B toxin, which is responsible for the majority of horse related botulism cases. Vaccination is recommended in endemic areas, or animals at greater risk for exposure, such as those on cured forages or round bales. Pregnant mares in endemic areas should be vaccinated and given a booster 4-6 weeks before foaling. Foals from unvaccinated mares can be transiently and passively protected using antitoxin or vaccinated with the type B toxoid but it is not labeled for foals.

All feed should be inspected for foreign material, decaying plant material, poisonous plant material, and decaying animal carcasses before it is fed. Avoid feeding round bales as they may contain decaying plant or animal material that may go un-noticed. Square hay bales are best as they may be inspected as they are fed. Keeping wounds clean and dry may help prevent infection. Puncture wounds, castration incision sites, and foal navels that have not been dipped may be at greater risk of *C. botulinum* infection.

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Equine Melanomas: "Not your ordinary lumps and bumps"

By Gillian Hasslinger, DVM Student (Class of 2020) Edited by Stacy H. Tinkler, DVM, MPH, Dipl. ACVIM-LA

Ever wonder what could possibly be the cause of your horse's lumps and bumps? Although not the most common cause of skin masses, equine melanomas are just as important to be aware of, especially in grey horses. A melanoma is a tumor of the pigment-producing cells in the skin, called melanocytes, and thought to be due to a genetic mutation rather than solely the result of high ultraviolet light levels.^{1,2} While melanomas may develop in any horse breed or color, around 80% of grey horses develop melanomas at 15 years of age or older. Of these, two-thirds of melanomas will metastasize to other regions of the body.³ Melanomas may first appear as benign, small, firm, black nodules underneath the tail, perineum, prepuce, head, neck, lips, or eyelids, and may be spotted while grooming or during your horse's annual veterinary physical exam.^{4,5}

Types of melanoma

Melanomas can be broken down into 4 separate types, including melanocytic nevi, dermal melanomas, dermal melanomatosis, and anaplastic malignant melanomas. A melanocytic nevus is a single benign nodule in the top skin layers that occurs in horses of any color and age at a location uncommon for melanoma formation. Dermal melanomas are small, singular masses located in the deeper skin layers at any location, from which both benign and malignant melanomas can arise. Dermal melanomatosis describes multiple, merging skin lesions found at locations common for melanoma development, especially in grey horses 15 years of age or older. Dermal melanomatosis can be distinguished from dermal melanomas by appearance, but would be identical if comparing the tissues themselves. These two categories comprise most of the melanomas diagnosed in grey horses. And finally, anaplastic malignant melanomas are commonly found in older, non-grey horses, but can still metastasize 1 year after formation.^{1,2,5}

Clinical signs and diagnosis of melanoma

Over time, melanomas can slowly become malignant and spread to other body systems.⁴ Once melanomas have spread, horses may begin to behave abnormally and display weight loss, colic, discomfort eating or drinking, difficulty defecating, and neck turning.4,5 If these signs are displayed, a veterinarian should be contacted for further examination and to perform further diagnostic tests. Diagnosis of melanomas involves not only a physical examination, but sampling of the tissues to determine whether they are benign or malignant.⁴ Although it is not possible to accurately determine whether melanomas will spread by tissue appearance alone, further research is currently being developed to find a reliable genetic marker to predict malignancy.3 Oftentimes veterinarians will also perform a rectal exam to determine whether there are any melanoma masses that may cause discomfort while defecating. Other diagnostics, including imaging or bloodwork are not typically pursued unless the clinical signs cannot be explained by the presence of melanomas.⁴ Before officially diagnosing a horse with melanomas, other neoplastic and non-neoplastic causes for skin masses should be considered (sarcoid, mast cell tumors, habronemiasis).2

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News & Notes



Rita Hilt, RVT

graduated from Purdue's Veterinary Technology program in 1990. I started out my career doing overnight patient care in the Large Animal Hospital (just when tracking for the senior students was getting started). I spent 6 years alternating with another technician between third shift doing patient care and day shift, helping with the surgery section. I worked in Clinical Pathology for about 4 years before leaving for a research position in West Lafayette. I was away from Purdue for about 15 years. During this time, I spent several years working for the USDA with livestock behavior research to help improving livestock health and well-being for better production.

In 2014, I found the opportunity to return to the Purdue Large Animal Hospital, again doing patient care on second shift. I love the work that I get to do with the patients, students and clients, but I especially love the hours as I am not a morning person at all! I have 5 horses that range from 12-35 years old and a pack of "tiny dogs" (2 Jack Russell's and 3 rescued Chihuahuas) that keep me well-entertained. I'm a member of the Red Hats and Purple Chaps and I participate in many parades with our ladies in Indiana, Louisville and Chicago.



Hello! My name is Brandi Maxie and I am an RVT who joined the Large Animal team at Purdue in July 2018. I was born and raised in central Ohio where I earned my RVT from Columbus State Community College in 2002. I worked for 5 years at The Ohio State University Veterinary Teaching Hospital in the Equine surgery, medicine and ICU departments. I spent the next 12 years at OSU Large Animal Services (Ambulatory) where I enjoyed an incredible variety in my duties that challenged me and helped me grow.

Brandi Maxie, RVT

The collegiality and support here at Purdue has smoothed the transition. I am expanding my skills and am challenged every day in the best way. The way that the technicians are utilized here is very impressive and they are truly passionate about their patient care, career path, and student development.

In my spare time, I enjoy showing, lure coursing, and doing therapy work with my 5 Rhodesian Ridgebacks. I also enjoy hiking nature trails with the dogs, traveling, trail-riding my horse and cooking.

LA Techs...



Carolyn McLaughlin, RVT

My name is Carolyn McLaughlin, and I am one of the Large Animal Medicine Technologists. I graduated from the Vet Tech Institute at Fox College in August of 2017, and was hired by Purdue University in December 2017. I really enjoy working here, I feel like I learn something new from every case I help with! I enjoy spending my free time kayaking, cycling, and hanging out with friends and family.



Above are two different presentations of melanomas. https://www.sporthorsevets.com/equine-melanoma/ https://thehorse.com/16830/canine-melanoma-vaccine-testing-in-horses-underway/

A Villain in Your Backyard – Anaplasmosis and Tick Prevention

By Levi Smith, DVM Student (Class of 2020) - Edited by Stacy H. Tinkler, DVM, MPH, Dipl. ACVIM-LA

Equine granulocytic anaplasmosis (EGA), often referred to as Anaplasmosis, is a disease caused by an infection from bacteria called Anaplasma phagocytophilum. EGA has also been previously referred to as equine ehrlichiosis or equine granulocytic ehrlichiosis. Anaplasma bacteria live in the gut of Ixodes ticks, similar to the bacteria that causes Lyme disease, and is transmitted to horses through a tick bite. The bacteria then enter the circulation, and once the bacteria enter the blood stream, they invade the white blood cells that normally protect the body from these types of infections. After invading the white blood cells in the blood, the Anaplasma organism alters white blood cell function, and the horse loses the capability of fighting off other infections. The organism also affects the bone marrow production of red blood cells, white blood cells and platelets. The mechanism for the red cell and platelet changes is not completely understood, but an immunemediated cause has been proposed. In addition, the presence of the bacteria is thought to induce profound inflammatory reactions causing damage to organs like the spleen, liver, lungs, and leaky blood vessels leading to edema.

Clinical Signs - Studies state that it takes less than 14 days after the tick bite for the animal to begin showing signs of Anaplasma infection. The course of the disease depends on the age of the horse and how long the infection produces illness. Horses over 4-years-old tend to develop a fever, stop eating, take on a yellowish color, have small pin-point areas of bleeding on their gums (Figure 1), have swelling in their lower limbs, are often lethargic, and may seem wobbly or not want to move at all. Younger horses do not tend to develop as severe clinical signs, and horses less than 1-year-old may not show many signs at all. Because the body is unable to protect itself from other infections, animals infected with Anaplasma may also demonstrate signs associated with secondary infections or may injure themselves from being wobbly and uncoordinated. These clinical signs may point your veterinarian to rule out other diseases like liver disease, equine infectious anemia (EIA), immune-mediated destruction of red cells and platelets, and other viral diseases.

Diagnostics – Anaplasmosis can be diagnostically challenging and several different blood tests may be helpful but not all are definitive. A stained blood smear examined microscopically may show multiple bluish-gray inclusions of the immature stages of Anaplasma in the white blood cells (Figure 2), but there is only a short-window of time that the Anaplasma may be maturing in those cells (typically days 3-5 post-infection), and the presence of the inclusions cannot be relied upon for diagnosis. A PCR test has been shown to correctly and accurately identify the presence of Anaplasma phagocytophilum DNA in samples that may not have sufficient amounts of the organisms in the blood cells to identify under the microscope. A complete blood cell count can be done to identify decreased numbers of blood cells, and, while helpful or suggestive of infection, is not a definitive diagnostic test. Lastly, paired antibody titers collected several weeks apart can also be used but would not aid in a timely diagnosis.



Figure 1. Horse with pinpoint bleeding of the gums from *A. phagocytophilum.* (Source: Pusterla and Madigan 2013).



Figure 1. Morulae of *A. phagocytophilum* in horse white blood cell on blood smear. (Image courtesy of Dr. Joanne Messick.)

Treatment and Prognosis – Luckily, Anaplasmosis can be treated and has an excellent prognosis. An antibiotic, oxytetracycline, can be administered through an IV catheter for a few days for effective treatment, or a combination of IV and oral antibiotics, oxytetracycline and doxycycline, also have been shown to work well. The addition of anti-inflammatories like flunixin meglumine (Banamine) help in making horses feel better while recovering from the infection and reduce any systemic inflammation that may be occurring. Typically, there will be clinical improvement within 12-24 hours. If the horse goes untreated, the disease should improve on its own within 2-3 weeks; however, the weight loss, limb swelling, and wobbliness could lead to complications. In a hospital setting, other treatments like fluids through an IV cath*(continued on page 6)*

I'll bet you a dime, your horse doesn't have Lyme!

Co-authored by Stacy H. Tinkler, DVM, MPH, Dipl. ACVIM-LA and Erin Yanoviak, DVM (Class of 2018)



Lyme disease is a tick-borne disease caused by an overactive immune response to the bacterium *Borrelia burgdorferi* (Figure 1). Lyme disease affects multiple organ systems, and in humans commonly infects the skin and joints. In the United States, black-legged ticks (*Ixodes scapularis*) infected with *B. burgdorferi* can transmit this bacterium to animals and humans. Even though greater than 50% of *Ixodes* ticks in prevalent areas such as the northeastern states and Wisconsin are infected with *B. burgdorferi*, only 2% of humans bitten by these ticks are likely to develop Lyme disease. Indiana is an emerging risk area for Lyme disease because it is located between Lyme disease endemic areas (Figure 2). This disease not only affects humans, but also our furry companions including dogs and horses; however, true *B. burgdorferi* infection with resulting Lyme disease in horses is not common.

Most humans are infected by the immature form of the tick known as the nymph. Nymphs are difficult to see because they are less than 2mm in size. Nymphs and tick larvae typically feed on small to medium sized animals, whereas adult ticks typically feed on larger herbivores such as deer, sheep, cows, and horses (Figure 3). The Lyme disease bacteria lives in the tick's gut and is transferred to the animal during blood meals. Once the tick attaches to a host, the Lyme disease bacteria travels from the gut to the tick's salivary glands. This bacteria is then introduced into the host through the tick's saliva when bitten. Borrelia burgdorferi has 3 outer surface proteins, OspA, OspC and OspF, and these surface proteins help the bacteria hide from the immune system. For example, by displaying OspA, B. burgdorferi is able to remain in the gut of the tick, and by displaying OspC, it can adhere to the tick's salivary glands. The tick must be attached for at least 36-48 hours for the host to be infected.

MYTH: My horse is lame and has a swollen joint so it has Lyme disease.

FACT: There is much that we do not know about Lyme disease in horses. Symptoms are variable but some documented syndromes attributed to Lyme disease in horses include: neurologic disease (equine neuroborreliosis), eye disease (uveitis), and skin disease (cutaneous pseudolymphoma). Unlike human Lyme disease,

excess fluid around the joints has been minimal in most Lymesuspect horses. Infection with other tick-borne organisms such as *Anaplasma phagocytophilum* is a much more common cause of tick-borne disease in horses.

MYTH: My horse was positive on a blood test for Lyme disease therefore it has Lyme disease.

FACT: Diagnosing Lyme disease in humans as well as horses is a challenge, and there are no definitive antemortem (in the live animal) tests in the horse. Serum tests for antibodies or for the *B. burgdorferi* outer surface proteins Osp A, C and F document exposure to *B. burgdorferi*, meaning that a tick with *Borrelia* bit the animal being tested and that it was exposed to or infected with *B.burgdorferi*, not that it is necessarily suffering from Lyme disease. Many clinically normal horses living in areas where Lyme is present often have detectable antibody levels to *B. burgdorferi*, and the disease is often over-diagnosed. Ruling out other more common diseases that might explain a horse's symptoms should be done first, and is most important.

MYTH: You must always treat when horses have a positive blood test for Lyme disease.

FACT: Treatment of horses not showing signs of disease but with a positive blood test will result in the unnecessary treatment of many horses resulting in extra expense, increased risk of possible adverse reactions, and inappropriate use of antibiotics. Horses with symptoms, and in which all the other more common causes of eye, neurologic and skin disease have been ruled out should be the only animals treated with antibiotics. Treatment for Borrelia includes the tetracycline antibiotics, such as oxytetracycline, doxycycline, and minocycline. In comparison to treatment of Lyme disease in humans, treatment of the disease in horses is complicated by the difficulty in confirming the diagnosis, poor bioavailability of oral antibiotics commonly used for treatment, and the longer duration of infection in horses prior to beginning treatment. The appropriate duration of antibiotic treatment is still unknown in horses but should be based on clinical response to therapy and, to a lesser degree, a decrease in serum antibody level. Treatment with steroids is not recommended unless the horse has acute and severe eye or



Figure 1. Eng, E. M. (n.d.). Lyme Disease Bacteria Borrelia burgdorferi [Digital image]. Retrieved May 25, 2017, from http://www.bayarealyme.org/about-lyme/whatcauses-lyme-disease/borrelia-burgdorferi/



Figure 2. Centers for Disease Control and Prevention (2017, May 23). Lyme Disease. Retrieved from https://www.cdc.gov/lyme/stats/maps.html



(continued on page 6)

Figure 3. Centers for Disease Control and Prevention (2017, May 23). Lyme Disease. Retrieved from https://www.cdc.gov/lyme/index.html

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Lyme (continued from page 5)

neurologic disease as their use has been associated with harmful outcomes in some cases. More research is needed in this area.

MYTH: The prognosis for recovery for all Lyme disease in horses is good.

FACT: The prognosis for horses treated for neuroborreliosis is reportedly poor, with only a single case of successful treatment reported in the literature. The prognosis for horses with Lyme-induced uveitis is also poor for recovery of vision. Antibiotic treatment resulted in an excellent outcome in one horse case with Lyme pseudolymphoma in the literature. Differences in prognosis, poorer in horses versus humans with confirmed Lyme syndromes, are likely related to duration of infection before treatment, and species differences in bioavailability of the antibiotic treatments.

MYTH: There is no way to prevent Lyme disease and treatment is the best way to combat the disease.

FACT: The best way to prevent equine Lyme disease is to prevent tick exposure and prolonged tick attachment. Late summer, fall, and early winter are the most common times for adult ticks to attach so during these times removing ticks should be a priority.

How to remove a tick (Figure 4)

- **1.** Use a fine-tipped tweezer to grasp the tick as close to the skin surface as possible
- **2.** Pull upward with steady, even pressure
- 3. Clean the bite area with soap or rubbing alcohol

Clipping and maintaining pastures is also a good way to prevent exposure to ticks. There is no horse specific Lyme vaccine, but multiple canine-approved Lyme vaccines are available and may result in the production of protective antibodies in horses but these vaccine-derived antibodies may make Lyme diagnosis even more challenging. Permethrin-based insecticides are approved for use in horses but require frequent application.

Take Home Points

Lyme disease in horses remains a challenge to diagnose because testing for *Borrelia* is difficult and there is a large variation in clinical signs when this disease does occur in horses. It is possible for a horse with vague clinical signs of Lyme disease to test positive and to not actually have the disease. More common causes of such clinical signs must be ruled out before making a diagnosis of Lyme disease. Since Indiana is an emerging risk area for Lyme disease, it is important to keep this disease in the back of your mind but understand that true cases of disease are still rare, and diagnosis and treatment are complicated and controversial in horses. An ounce of prevention is still worth a pound of cure, and this is especially true as it relates to equine Lyme disease. The most important way to prevent Lyme disease is to prevent exposure to



Figure 4. Centers for Disease Control and Prevention (2017, May 23). Lyme Disease. Retrieved from https://www.cdc. gov/lyme/removal/index.html

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Anaplasmosis (continued from page 4)

eter, standing leg wraps, and stall confinement help improve the recovery time for those with Anaplasmosis. Unfortunately, there is still the chance of relapse of infection within 30 days, even in treated animals.

Tick Prevention – Utilizing tick repellents and preventatives are the best way to prevent equine granulocytic anaplasmosis, as well as other tick-borne diseases like Lyme disease. Some ways to help prevent your horse from being bitten by ticks is to keep areas where your horses are pastured dry, regularly disturbed, and well-exposed to the sun. Other preventative management measures include mowing pastures, clearing debris and leaves from the pastures, and if possible, deterring and keeping deer away. Topical products are available for horses that help prevent tick exposure. Products with permethrin have been well-associated with high success of repelling ticks. Products are available as wipe-on, pour-on, spot-on, and spray-on application that all seem to have similar effectiveness when applied appropriately. It is important to remember that dirt, sweat, and water can shorten the length of effectiveness of the tick repellents. It is important to consult your veterinarian about the best product for your specific horses' needs.

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Is Thyroid Supplementation Right for My Horse?

By Janice Kritchevsky, VMD, MS, Dipl. ACVIM-LA

If you have spent any time in a racing or performance horse stable, chances are you have seen a tub or two of thyroid supplement medication. A large number of performance horses receive thyroid medication daily. You might ask, "Is hypothyroidism really that common in the horse world"? The answer to that question

is no. Next you might ask, "So what's the deal with all the horses on thyroid supplement?" The answer to that is, "It's complicated." There are a large number of reasons why horses are placed on thyroid medication—some good and some not-so-good.

Thyroid hormones are important for maintaining resting metabolic rate and energy metabolism. Like people, the thyroid gland in a healthy horse produces all the thyroid hormone the body needs. If the thyroid gland doesn't make enough hormone, hypothyroidism occurs.

Unlike people, true hypothyroidism is extremely rare in adult horses. When it does occur, clinical signs include cold intolerance, dry eye, and a roughened hair coat. Your veterinarian can diagnose hypothyroidism by performing a thyroid gland stimulation test. Just measuring blood thyroid hormone concentrations is not sufficient to determine whether or not a horse is hypothyroid. This is because there are a huge number of factors that can lower resting blood thyroid hormone levels for a short period of time, but the thyroid gland is fine and will respond to stimulation when more thyroid hormone is needed.

At times, a horse's thyroid gland is normal, but an increased dose of thyroid hormone supplement is prescribed in order to

Melanomas (continued from page 2)

Treatment options

Treatment options for metastatic melanomas are limited, but equine melanoma remains an ever-growing field of research. Surgical removal is curative in some cases (melanocytic nevi, dermal melanomas), but ineffective for others (dermal melanomatosis) due to the high prevalence of internal spread, invasiveness, and difficult surgical correction. In addition, many tumors previously removed may return, and may even increase in size and number.⁵ Chemotherapeutic agents, such as cisplatin, may be effective for smaller tumors (<3 cm) when implanted directly, but have had inconsistent results in treatment of larger, metastatic tumors or those already treated with other therapies.^{2,4} Electrochemotherapy, or delivery of electrical pulses, has been used as a supplemental therapeutic option to help absorption of cisplatin into the melanoma tissue, but is less effective than other treatment options.⁴ Cimetidine, a histamine receptor antagonist, was previously considered an effective therapy, but following recent studies has been deemed an unreliable treatment option.⁵ With so few viable options available, there is a growing need for new therapies to treat horses with metastatic melanomas.

New treatments on the horizon

In recent years, a new therapy option has spread to the equine community following treatment of canine melanomas with vaccine therapy. A DNA vaccine called Oncept[™] has been circulating in research studies and shows promise for use in treating equine melanomas. A recent study involved the administration of Oncept[™] to horses using the same vaccination protocol as used in dogs (4-biweekly injections, followed by a 6 month booster).⁶ Results are

increase a horse's metabolic rate. This is done when a horse is suffering from Equine Metabolic Syndrome and has higher than normal blood insulin concentrations even after when placed on a low-carbohydrate diet. The signs of Equine Metabolic Syndrome include having a high body condition score, increased fat long the top line and a cresty neck, and, most importantly, laminitis. Adding thyroid supplement to the treatment plan of a horse with Equine Metabolic Syndrome promotes fat utilization and weight loss. Talk to your veterinarian if you are worried about Equine Metabolic Syndrome in your horse. You can discuss whether including thyroid hormone treatment is a good choice.

Finally, some place horses on thyroid supplement if they feel the horse is backing off feed, losing energy, or showing any of a host of non-specific issues. In these cases, thyroid hormone is used as a non-specific "pick-me-up." There are two problems with taking this approach. First, a serious medical problem might be covered up or missed. Any horse that loses weight or becomes depressed needs to be evaluated. Secondly, giving hormone takes away the need for horse's own thyroid gland to make hormone, and the gland will shut down. It will not be able to respond normally to the stimuli that would usually cause more hormone secretion.

Thyroid hormone can be a life-saving therapy, helping to prevent or manage laminitis in horses with metabolic disease. But it should never be given "just because." It causes changes to every cell in the body; it should only be given if you understand exactly why the horse needs to receive it under the guidance of your veterinarian.

promising for use of this vaccine in horses, but it is still undergoing clinical trials at this time before becoming an officially approved treatment.^{2,6} Another new and upcoming option is a plasmid DNA vaccine called ImmuneFXTM that contains a gene from the bacteria *Streptococcus pyogenes*. The vaccine is injected into tumors, and the tumors then express the gene which causes a strong anti-tumor response in the body⁷ and effectively reduces tumor burden by 40-50%. Lastly, keep an eye out for betulinic acid for melanoma chemotherapy. It's derived from birch bark and is reported to have strong anti-inflammatory properties! Although only minimal research has been done in this area so far, these three options hold promise for treating equine melanomas.

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