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Treating Recalcitrant Equine Asthma

The Stepwise Guide to the
Equine Hoof

Lifetime Cost of Horse
Ownership

Tech Update:
Stabilizing a Foal Born by
Emergency C-Section



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¶*Overall, horses in the treatment group had a statistically significant decrease in pain score (2.87 decrease compared to a 0.72 decrease in the control group) from the beginning to the end of the trial (44 days total).

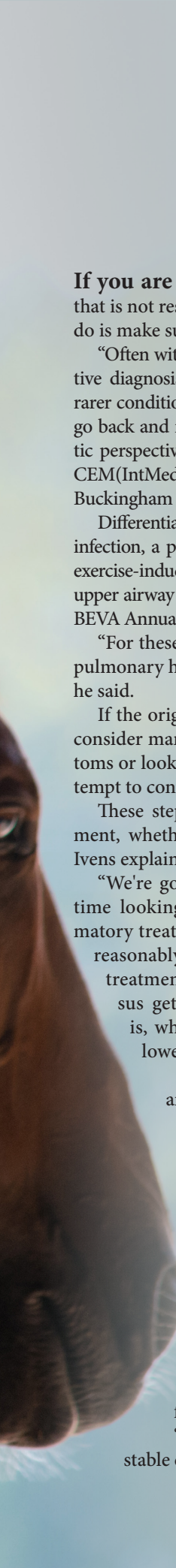
¶Thermal imaging also demonstrated a significant impact on temperature readings both at the time of the trial as well as after exercise, and application of Performance Ultra. Near the end of 30 days, horses in the treatment group had a lower average back temperature.

¶**Reference:

¶Walter, K.W., Altman, J. & Haussler, K. (2023) Reducing chronic back pain and inflammation in horses using a commercial herbal liniment. Equine Veterinary Education, 35, e499-e506.

How to Treat an Asthmatic That is Not Responding

By Marie Rosenthal, MS



If you are dealing with an asthmatic horse that is not responding to treatment, the first thing to do is make sure it actually has asthma.

“Often with asthma, we make a relatively presumptive diagnosis, which is often correct. But there are rarer conditions out there, and sometimes, we have to go back and reevaluate where we are from a diagnostic perspective,” said **Philip A.S. Ivens**, MA VetMB, CEM(IntMed), DECEIM, MRCVS, EBVS, owner of Buckingham Equine Vets, in the United Kingdom.

Differential diagnoses include a viral or bacterial infection, a parasite, such as lungworm, which is rare, exercise-induced pulmonary hemorrhage, neoplasia or upper airway disease, Dr. Ivens pointed out at the 2023 BEVA Annual Congress, held in Birmingham.

“For these performance horses, exercise-induced pulmonary hemorrhages are definitely a possibility,” he said.

If the original diagnosis is confirmed as asthma, consider management: Did you just treat the symptoms or look at reducing inflammation? Did you attempt to control the environment?

These steps are at the core of asthma management, whether it is mild, moderate or severe, Dr. Ivens explained.

“We’re going to spend a reasonable amount of time looking at symptomatic versus anti-inflammatory treatment, I think still in practice and very reasonably, we tend toward more symptomatic treatment in terms of bronchodilators versus getting to the core of what this disease is, which is an inflammatory disease of the lower airway,” he said.

The key differentiation of mild, moderate and severe asthma—the current classification of the disease—is that severe asthma has tachypnea/dyspnea at rest, whereas the mild cases appear clinically normal apart from maybe a cough or nasal discharge, with underlying airway limitation and impaired gaseous exchange. Not all mild cases have cough, however, he said.

There are many environmental factors that inflame the airways of a horse with asthma, including molds and spores, their toxins, particularly in forage; plant debris and inorganic dust.

“These are the inflammatory agents in a stable environment,” he said. “The stable envi-

ronment is naturally a very dusty place. And microbiologic products, endotoxin, peptidoglycan, bacterial DNA even, and mold and fungal products that produces toxins and beta glucans [are often present].

“Why is this important? We just need from a therapeutic perspective to keep a wider view on things—not all of what we’re dealing with is due to allergy,” he said.

2023 was a bad year in his Birmingham practice in terms of summer-pasture-associated asthma. “I’ve certainly had cases in the very extreme hot weather for the UK that had been very well controlled up until now, and a lot of these cases were next to busy yards,” he said.

“In human asthma, pollution is a big confounding factor with asthma exacerbation. I was wondering whether this was part of this sort of irritant effect on what is already underlying disease,” Dr. Ivens said.

Environmental control is the “bedrock of controlling all equine asthma cases,” Dr. Ivens said. “Environmental controls are often underestimated, and if you’ve got a non-responder, assuming that you are comfortable with your diagnosis, it’s the first place I would look.”

He called it imperative to visit the yard, walk around the environment and breathe the air yourself, and if possible, bring the client with you. He told the story of a Thoroughbred who had been winning quite a few races and was moved to a stable next to the trainer’s office, so it could be closely monitored. It started performing badly, and it turned out there were more air particles next to the trainer’s office, because there were more people going in and out and kicking up dust.

“Just that footfall was aerosolizing the dust in the environment, and therefore, causing performance issues in that horse,” he said.

After environmental controls, many veterinarians add bronchodilators, and then, as the clinical signs worsen, reach for corticosteroid treatment. As the disease gets under control, they then work down that pyramid eliminating medications.

“But I think the thing that has changed our understanding of equine asthma is that symptomatic relief with bronchodilators is probably, in most cases, not enough,” he said. The steroid can decrease the inflammation and improve the horse’s quality of life.

“I think as therapeutics evolve, more of these horses would benefit from more sustained and more prolonged corticosteroid treatment,” Dr. Ivens said.

However, corticosteroids are not without side effects, so it is important to practice the “yin and yang”

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Image courtesy of Boehringer Ingelheim

Using the Aservo EquiHaler to treat a horse with asthma.

of balancing steroids with the potential risk for laminitis. In athletic horses, this can be less of a problem because most of them are in better body condition. Pleasure horses today tend to be less conditioned and maybe a little overweight, which would already put them at risk for laminitis.

“I think it's really important to diagnose your laminitis risk,” he said. Do a glucose challenge test to see the degree of hyperinsulinemia and check for any underlying endocrine disease, such as PPID, to make sure that steroids won't aggravate any of these conditions.

And remember all steroids are not created equal, and the inhaled corticosteroids are intrinsically more potent than dexamethasone.

The challenge is getting that inhaled steroid to the site where we need the therapeutic effect. “So even though they are relatively more potent, it's that delivery to the site of inflammation that is the challenge,” Dr. Ivens said.

Approved in 2020, the Boehringer Ingelheim Aservo EquiHaler, which delivers ciclesonide inhalation spray, in a soft mist, was the first FDA-approved inhalant therapy for horses with severe equine asthma. The inhaler includes an ergonomic handle and dosing lever that makes it easy to use, and a nostril adaptor that fits inside the nostril of the horse, al-

lowing the horse to easily inhale the medicated mist into its lungs.

“In human medicine, there has been soft mist technology for quite some time. And we recently licensed an equine product that uses this,” he said. “With a soft mist that then can be inhaled, you lose much less drug in that upper airway.”

Nebulizers are also used, as are meter dose inhalers, but they fire the drug at a high velocity, and only about 20% of the medication is getting into the trachea and the lower airway.

Regardless of your delivery system, corticosteroids treat the inflammation, which is better than just providing symptomatic relief, Dr. Ivens said, which is especially important in non-responders.

“If a case is not responding, reevaluate the diagnosis and once you're certain that this is an equine asthma case, treat the underlying inflammation; Don't provide just symptomatic relief,” he recommended. “Improve the environment where possible while you're getting the disease under control, because in the long term to de-escalate your therapeutic intervention, that's going to be key.

“Finally, think how best to deliver the corticosteroids and which corticosteroid to use depending on the individual patient's circumstances.”

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¹ Data on file.

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³ Burba DJ, Collier MA, DeBault LE, Hanson-Painton O, Thompson HC, Holder CL: In vivo kinetic study on uptake and distribution of intramuscular tritium-labeled polysulfated glycosaminoglycan in equine body fluid compartments and articular cartilage in an osteochondral defect model. *J Equine Vet Sci* 1993; 13: 696-703.

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The STEPWISE GUIDE TO THE HOOF

By Paul Basilio

Although it can be overlooked as an examination target, a sizeable portion of equine lameness starts at the hoof.

“The foot will tell us a lot of information very quickly, because it is a structure that can change very quickly,” said **Craig Lesser**, DVM, CF, of Rood & Riddle Equine Hospital in Lexington, Ky.

“I encourage people to take a second look at the foot during lameness or prepurchase exams to see if an issue is coming about sooner rather than later.”

During a Burst session at the 69th AAEP Annual Convention in San Diego, Dr. Lesser gave a quick rundown of his thorough foot examinations.

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Stepwise Hoof Guide *(continued from page 9)*

STEP 1: LOOK AT THE HORSEHOOF

“It all starts with just watching the horse stand there,” said Dr. Craig Lesser.

Dr. Lesser will take note of whether the horse is doing any unusual weight shifting, holding itself in an altered posture or standing camped under.

“A lot of hindlimb lameness is related to a negative palmar angle,” he explained. “Look at the overall horse, because that’s going to play into what we’re seeing with the foot.”

Pay special note of how the horse moves the foot as well—not only for signs of lameness, but also to see where it lands, whether it breaks over or if the stride might be altered. All of these things could help direct treatment.

STEP 4:

TEST THE HOOF

“No matter how many advanced imaging techniques we have, the hoof tester is still my best tool,” he said. “It helps me tell what hurts and what doesn’t hurt, and helps me decide where we’re going to load and what we need to protect.”

Hoof testers can vary significantly from manufacturer to manufacturer, so Dr. Lesser said that it’s important to find one that fits your hands and your examination needs.

“Finally, don’t be afraid to take radiographs,” he said. “A few radiographs can go a long way.” **MeV**

STEP 2: TOUCH THE FOOT

“When we actually get down and put our hands on the foot, there’s a lot we can see,” Dr. Lesser said. “We’re looking for layers of distortions, and whether they are due to conformation or if we just left a shoe on a foot for longer than we should have.”

Sheared heels, for example, can lead to many other issues, and they can be difficult to pick up unless you get down there and feel for it.

“Additionally, we can pick up on toe cracks earlier on [with a proper exam],” he said. “A lot of our toe cracks are clinical signs of acute laminitis, and quarter cracks could mean an imbalance. We’ll also see bruising before we start to see radiographic changes. A lot of the hoof changes we can see early on will help us get ahead of the game.”

One of the most important things to check, according to Dr. Lesser, is the coronary band.

“It’s not something that I was taught to do right away,” he added. “Now it’s one of the most important parts of my exam.”

Running your fingers along the coronary band can reveal any ledges, sensitivity, or swelling. You may also feel scars or cracks that would have gone unnoticed without the palpation.

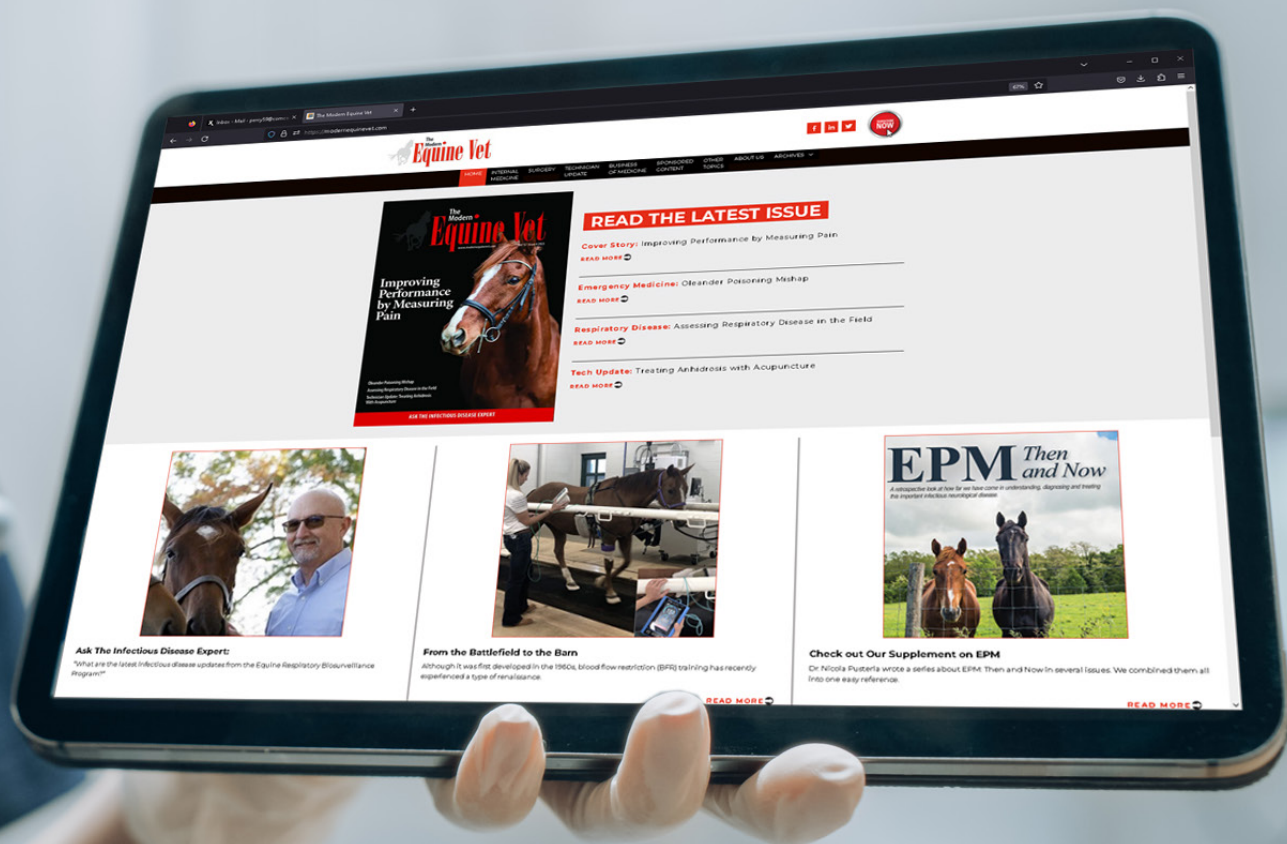
“In a lot of our laminitic horses, we’ll start to notice a ledge formation [on palpation],” he said. “The horse might have some signs of sinking that you wouldn’t see radiographically at that point.”

STEP 3: SEARCH YOUR SOLE

“The sole tells us so much” Dr. Lesser explained. “We can start by looking at the frog, which expands and contracts like any muscle. If it’s atrophied, it’s not being engaged. That can indicate that we need to change the shoeing strategy.”

The bar is an extension of the wall. If they’re crushing or rolled underneath, that can lead to lameness and abscess.

“It’s amazing how many things you’re going to see if you take the time to take your wire brush to the foot and just looking at the bottom of it.”



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Stabilizing a Foal Born Via Emergency Cesarean-Section



By Ali Harman, RVT, VTS-EVT

In February 2023, a 7-year-old Friesian mare presented to the William R Pritchard Veterinary Medical Teaching Hospital (VMTH), at the UC Davis, School of Veterinary Medicine, for evaluation of fever and diarrhea lasting 3 days. Differential diagnoses for the mare included a large colon impaction or right dorsal displacement but a differential for the fever (102° F) was not immediately known. The mare was transferred to the isolation unit for treatment of enterocolitis and endotoxemia.

Upon presentation, the mare was approximately 320-330 days in foal based upon the owner's estimation as a last breeding date was not provided. A consult with the equine theriogenology service revealed a strong and active fetus with a heart rate of 88 beats per minute (bpm) (reference range [RR] 80-120 bpm in utero), combined thickness of uterus and placenta (CTUP) within normal limits at 0.8 mm, RR 0.8-1.0 cm) and a tightly closed cervix.

After 48 hours, the mare's condition declined, and her pain could not be managed medically. A repeat rectal examination was more compatible with a right dorsal displacement of the colon. The foal was also showing symptoms of stress, such as decreased activity on ultrasound and bradycardia (60 bpm). The owner elected surgical delivery of the foal and exploration of the cause of the mare's colic symptoms.

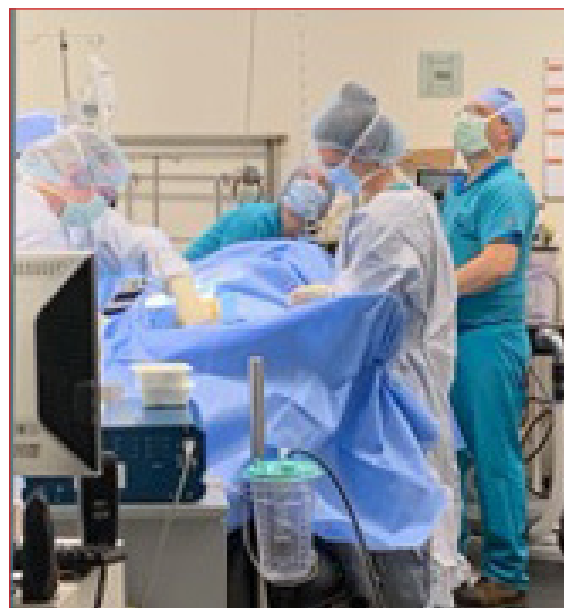
A black colt weighing approximately 50 kg was successfully delivered by the surgical team and was immediately transported to a room adjacent to the surgical suite for evaluation and treatment.

Upon delivery, the foal appeared to be anesthetized due to the surgical medications given to the dam. The foal was bradypneic with a respiratory rate of 20 breaths per minute (brpm) (RR 60-80 brpm immediate post-foaling), bradycardia with a heart rate of 60 bpm (RR 60-120 bpm immediately post-foaling) and an oxygen saturation (SpO₂) of 90%. The patient's mucous membranes (MM) were mildly cyanotic; no adventitious lung sounds were appreciated on auscultation, and the rectal temperature was 99.9° F (RR 99.5-102.5° F).

Immediately after delivery, an 8 mm endotracheal (ET) tube was passed and placement in the trachea was confirmed via auscultation of the lungs and the use of an end tidal carbon dioxide detector. Oxygen was administered via the endotracheal tube at 10 L/min. After 1 minute the foal began spontaneous respiration with a

rate of 60 brpm and an SpO₂ of 94%. The patient's temperature remained normal, and a proper body temperature was maintained with a blanket warmer.

While the ET tube was being placed, the left jugular vein was clipped and prepped using aseptic technique for placement of an emergency access intravenous catheter. An over-the-needle 16 gauge by 3½ inch catheter



The mare at the start of the surgical procedure.



Image courtesy of Ms. Harman

The patient is seen receiving colostrum by an indwelling nasogastric tube. The colostrum is allowed to pass through the tube via gravity

was placed in the left jugular vein and sutured in place with 2-0 prolene.

A venous blood sample was obtained for analysis 3 minutes after delivery which revealed a respiratory acidosis (blood pH 7.260, RR 7.32-7.42 pH units) and a partial pressure of carbon dioxide ($p\text{CO}_2$) of 46.5 mmHg, RR 38-42 mmHg), hyperkalemia (5.0mEq/L, RR of 3.1-5.0 mEq/L), azotemia (creatinine 10.0 mg/dL, RR less than 2 mg/dL), hyperlactatemia (10 mmol/L, RR <2 mmol/L) and leukopenia

(3,460/uL, RR 8000-10,000 WBC per uL).

A 20 mL/kg bolus of crystalloid fluids were given with 50 mL of dextrose 50% added for a final concentration of 2.5% dextrose. After the bolus, a second liter of fluids with a final concentration of 5% dextrose was administered at approximately 10 mL/kg/hr. The foal's heart rate increased to 80 bpm but respiration was still being assisted by the ET tube; SpO_2 only measured at 96%, so oxygen delivery was increased to 13 L/min.

Nine minutes after delivery, the foal began deliberately moving its head and its vitals were stable (84 bpm, 60 brpm, and 100° F) so the ET tube was removed. Oxygen supplementation was transitioned to intranasal delivery through a 14 French Airlife catheter in the left nostril. The foal was then maintained at a rate of 10 L/min for the remainder of his stabilization. The foal made multiple attempts to stand but was kept in sternal recumbency by the clinical team as evaluation of the cuboidal bones had not yet been performed.

Umbilical care was initiated 20 minutes after delivery. The umbilicus was rinsed using a mixture of sterile water and chlorhexidine (1 part chlorhexidine to 3 parts sterile water) and the structure was clamped using straight Kelly forceps. The first clamp was placed 5 cm distal to the abdomen and a second clamp was placed 2.5 cm distal to the first clamp; 2 minutes later the umbilicus was cut between the 2 clamps. The structure was again rinsed with the chlorhexidine mixture.

Due to high risk for sepsis, the foal was started on antimicrobial therapy including sodium ampicillin (30 mg/kg, slow IV) and ceftiofur sodium (5 mg/kg, IV). To support immune and muscle function, vitamin E and selenium (E-SE 2.5 mg) was administered intramuscularly (IM) in the right semimembranosus muscle after cleaning the area with chlorhexidine and alcohol. A 0.3 mg/kg dose of hydrocortisone was given intravenously as a central nervous system stimulant.

Placement of a long-term catheter was performed in the right jugular vein. An area over the right jugular vein was clipped and prepped using aseptic technique and a 16 gauge by 20 cm over-the-wire catheter was successfully placed and sutured in place with 2-0 ethilon.

Forty minutes after delivery, a repeat blood gas analysis was performed. The hyperkalemia resolved (3.6 mEq/L) and the other parameters previously mentioned were showing response to treatment (pH 7.274, creatinine 8.4, mg/dL and lactate of 7.5 mmol/L). A commercial pediatric enema was administered via rectum, which did not yield any fecal output.

An indwelling nasogastric tube (14 French by 125 cm) was placed in the left nare and placement in the



The image on the left shows radiographs being taken of the carpi to evaluate ossification of the cuboidal bones.



A staff member waits for the foal to be brought to the isolation room.

Teaching Points

As the only technician available for the procedure, I wore many hats to help ensure the success of the patient.

The first challenge that I faced was preparing for receiving the foal. Most of the surgical delivery of neonates occurs in a surgical suite that is adjacent to the neonatal ICU (NICU).

Because the dam had been housed in isolation, and we had not yet received laboratory results, results about infections, yet, the procedure about infections, the procedure had to be performed in a different area of the hospital, so we did not contaminate the NICU.

Due to the change in venue, all the equipment, supplies and medications that were needed for the foal had to be moved to a new location. This required that I understood the delivery process, possible complications and treatments for such complications to have the needed items close at hand. Additionally, as the procedure was an emergency, there was only 20 minutes available to gather and set-up the receiving area.

After delivery, it was my responsibility to prepare the site for catheterization, taking notes of times and treatments, and initiating treatments such as fluid and antimicrobial therapy.

A key feature to the success of this delivery, was clear and productive communication among staff. There were multiple services working to perform the procedure (anesthesia, theriogenology, medicine and surgery), which required that all staff members be aware of the chain of events and expectations for the procedure.

For the medicine team receiving the foal, it was clearly communicated who would be responsible for specific tasks to ensure that all needed areas of support were covered and treatments could be initiated simultaneously.

The open line of communication also allowed for collaboration within the medicine team. The clinician placing the endotracheal (ET) tube encountered difficulty in initial attempts at placement. Being experienced with placing ET tubes, I suggested a change in positioning of the patient's head to allow easier access to the trachea. The change in position was accepted, and the ET tube was easily placed.

Understanding the disease processes the foal faced, understanding laboratory values and careful monitoring of the foal allowed me to quickly communicate any concerns regarding the patient.

stomach was confirmed radiographically. The foal was administered 120 mL of donor colostrum.

Lateromedial and dorsopalmar radiographs were taken of both carpi and lateromedial and dorsoplantar views were taken of both tarsi to evaluate the degree of ossification of the cuboidal bones. Review of these studies revealed adequate ossification of both the carpi and tarsi. Although the foal was physically able to stand, the patient was not yet coordinated enough to walk, and his transpiration back to isolation was facilitated with a forklift.

The continued treatment plan for the foal included physical exam and blood gas analysis every 4 hours, continuing ceftiofur sodium at 5 mg/kg, IV every 6 hours, sodium ampicillin at 30 mg/kg, slow IV every 6 hours, constant rate infusion of crystalloid fluids at 3 mL/kg/min, intranasal O₂ therapy at 5 L/min, sucral-fate 1 gram, orally every 12 hours, and hydrocortisone 0.3 mg/kg IV every 6 hours. Feeding orders included

checking for reflux every 2 hours and feeding the foal 400 mL mare's milk (from frozen milk bank) via nasogastric tube if reflux measured less than 50 mL. The foal was permitted to stand every 2 hours.

Nine hours after delivery the foal became tachypneic with a respiration rate of (60 brpm), an increase in pCO₂ at 58 mmHg; due to hypoventilation, intranasal oxygen was increased to 7 L/min. One liter of high gamma plasma (EquiPlas Plus Equine IgG) was given at a rate of 3 mL/kg/hr (IV fluid therapy was discontinued during this time, and dextrose 50% was administered at 3 mg/kg/min during plasma administration).

At 17 hours old, the colt had not yet urinated and was given a 0.15 mg/kg dose of furosemide IV, after which the foal was able to produce urine while in lateral recumbency. He was then unable to pass urine on his own. The penis was prepped using aseptic technique and a closed urinary catheter system was placed.

The foal responded well to treatment within the first



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24 hours. The single dose of frozen colostrum and hyperimmune plasma transfusion provided an adequate response and the foal's immunoglobulin G levels measured greater than 800 mg/dL. Blood gas values continued to improve with the hyperkalemia resolved (3.8 mEq/L) and the hyperglycemia, hyperlactatemia and azotemia resolving with values of 126 mg/dL, 5.8 mmol/L and 6.49 mg/dL respectively. The respiratory acidosis persisted.

As the owners were unable to provide a last breeding date, the true gestational age of the foal was unknown. The foal displayed signs of prematurity/dysmaturity including floppy ears, laxity in all 4 limbs and excessive eponychium. Fetal cortisol levels increase prior to parturition (RR 12-14 µg/dL) and decline to steady for the first five days of life to 5-7 µg/dL. The patient's cortisol levels were measured 24 hours after delivery and were found to only measure at 2.4 µg/dL, which confirmed that the patient was premature.

After that point, the foal's status began to decline, and he showed an increasing number of symptoms consistent with Neonatal Maladjustment Syndrome (NMS). Symptoms included generalized weakness, reduced ventilation, gastrointestinal dysfunction and depression. These signs are associated with the normal pathophysiology of NMS, which was an anticipated complication due to the nature of the foal's delivery. NMS is often seen in foals that have experienced an hypoxic ischemic event-such as respiratory depression seen with anesthetic agents. The respiratory acidosis and hyperlactatemia were all indicative of the foal experiencing a hypoxic event during delivery. The foal was also at an increased risk for NMS due to the inflammatory response seen in the dam (dam had a serum amyloid A level of 744 µg/dL (RR 0-20 µg/dL). As the foal was born alive, direct examination of the CNS was not available, so this



Excessive eponychium was noted on the hooves which can be correlated to prematurity or dysmaturity.

syndrome is diagnosed symptomatically.

The foal did experience several setbacks and required additional care. Respiratory (doxapram, 0.01 mg/kg/min, CRI) and CNS stimulants (hydrocortisone, 0.3 mg/kg, IV every 6 hours) were provided intravenously. The foal failed to pass meconium and was treated with an acetylcystein retention enema, which was productive.

IV fluid therapy continued with crystalloid fluids and dextrose 50% CRI at 1 mg/kg/min. The development of pneumonia was treated with nebulization with amikacin sulfate and sterile water.

The foal responded positively to all treatments and supportive therapy and was discharged at 9 days of age.

Most foals delivered via c-section at the VMTH are non-viable and are typically the result of a dystocia. Receiving a viable neonate was an excellent exercise in emergency medicine, neonatology and understanding of the treatment that would be needed.

Monitoring and management of a critical equine neonate requires a specialized skillset and the ability to note any change in the foal's condition because they are prone to rapid declines.

As a result of the high level of care this foal received, it was discharged at 9 days of age, where foals of similar condition referred in from outside facilities have an average hospitalization stay of 14 days. **MeV**

About the Author

Ali Harman earned a Bachelor's of Science degree in Equine Sciences from UC Davis in 2009. She continued to pursue a career in the equine veterinary industry as a technician. She gained her RVT in 2017, and most recently her Veterinary Technician Specialty in Equine Veterinary Nursing. She is currently the Large Animal Critical Care Assistant Supervisor at UC Davis.

For more information:

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How Eastern Equine Encephalitis Virus Invades Brain Cells

By Tamara Schneider

It may be possible to set up a decoy and prevent eastern equine encephalitis (EEE) from entering the brain.

Scientists predict that as the planet warms and lengthens the mosquito populations' seasons and geographical reach, risk of infection will grow among horses and humans.

Co-senior authors **Michael S. Diamond**, MD, PhD, the Herbert S. Gasser Professor at Washington University, and **Daved H. Fremont**, PhD, a professor of pathology & immunology, investigated how the virus attaches to 1 of its key receptors—very low-density lipoprotein receptor (VLDLR), which is found on the surface of cells in the brain and other parts of the body.

Co-first author Lucas Adams, an MD/PhD student in the Fremont and Diamond laboratories, used cryo-electron microscopy to reconstruct the virus binding to the receptor in atomic-level detail. The results turned out to be unexpectedly complex. The molecule is composed of 8 repeated segments, called domains, strung together like beads on a chain. Usually, a viral protein and its receptor fit together in one very specific way. In this case, however, 2 or 3 different spots

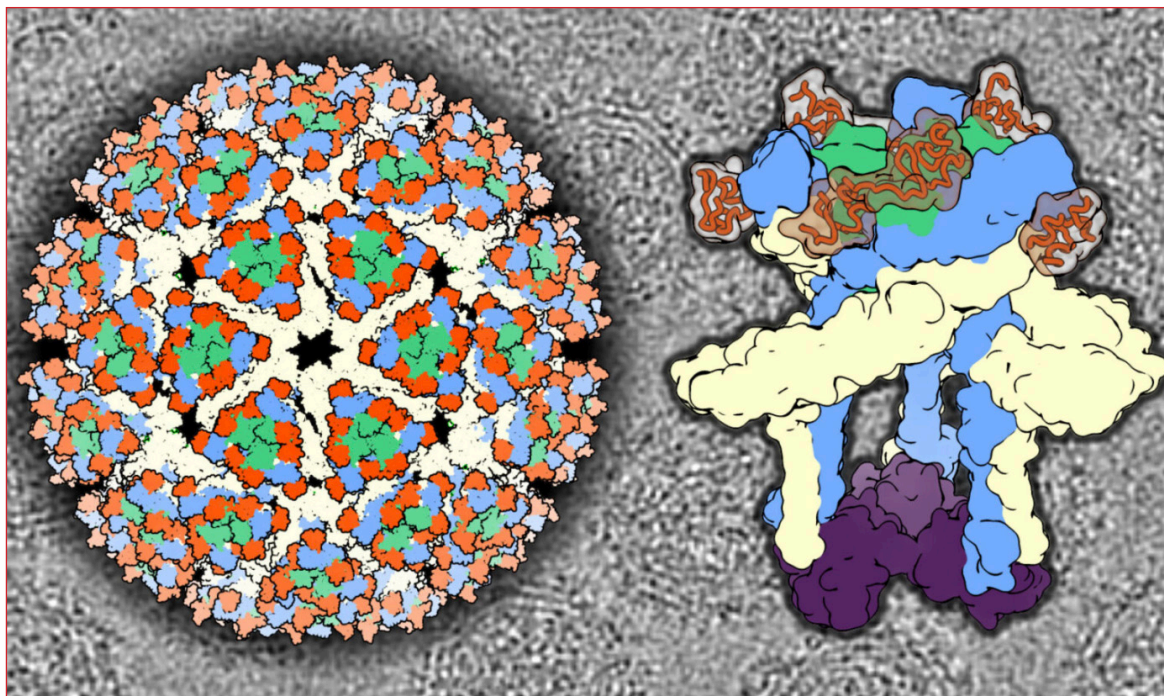
on the viral surface proteins could attach to any of 5 of the molecule's 8 domains.

"What's really striking is that we find multiple binding sites, but the chemistry of each of the binding sites is similar and also similar to the chemistry of binding sites for other viruses that interact with related receptors," said Dr. Fremont, who is also a professor of biochemistry & molecular biophysics and of molecular microbiology. "The chemistry just works out well for the way viruses want to attach to cell membranes."

The domains that make up this molecule also are found in several related cell-surface proteins. Similar domains are found in proteins from across the animal kingdom.

"Since they're using a molecule that naturally has repetitive domains, some of the alphaviruses have evolved to use the same strategy of attachment with multiple different domains in the same receptor," said Dr. Diamond, who is also a professor of medicine, of molecular microbiology, and of pathology & immunology. Alphaviruses include EEE virus and several others that cause brain or joint disease. "There are sequence differences in the VLDLR receptor over evolution in different species, but since the virus has this flexibility in binding, it is able to infect a wide variety of species

EEE virus attaches to a receptor it uses to enter and infect cells. The whole virus is shown on the left and a magnified view of the viral structural proteins on the right. The findings lay the groundwork for a receptor decoy molecule that protects mice from encephalitis caused by the virus.



Images by Lucas Adams

including mosquitoes, birds, rodents and humans.”

To block attachment, the researchers created a panel of decoy receptors by combining subsets of the 8 domains. The idea was that the virus mistakenly would bind to the decoy instead of the receptor on cells, and the decoy with the virus attached could then be cleared away by immune cells.

Co-first author **Saravanan Raju**, MD, PhD, a postdoctoral researcher in the Diamond lab, evaluated the panel of decoys. First, he tested them on cells in vitro; many neutralized the virus. Then, he turned to mice. Dr. Raju pretreated mice with a decoy or saline solution, as a control, 6 hours before injecting the virus under their skin, a mode of infection that mimics natural infection via mosquito bite. Three decoys were tested: 1 known to be unable to neutralize the virus; 1 made from the full-length molecule; and 1 made from just the first 2 domains.

All the mice that received saline solution, the non-neutralizing decoy or the full-length decoy died within eight days of infection. All the mice that received the decoy made from the first two domains survived without signs of illness.

Certain aspects of its biology give Eastern equine encephalitis virus the potential to be weaponized, making it particularly important to find a way to protect against it. In a subsequent experiment in which the mice were infected by inhalation, the de-

coy made from the first 2 domains was still effective, reducing the mice's chance of death by 70%.

“Through a combination of the structural work and the domain deletion work, we were able to figure out which domains are the most critical and create a quite effective decoy receptor that can neutralize viral infection,” Dr. Fremont said.

Researchers at Washington University School of Medicine in St. Louis said their findings should advance the understanding of the complex molecular interactions between viral proteins and their receptors on animal cells. The findings lay a foundation for treatments and vaccines for viral infections.

“Understanding how viruses engage with the cells they infect is a critical part of preventing and treating viral disease,” Dr. Diamond said. “Once you understand that, you have the foundation for developing vaccines and drugs to block it. In this study, it took us a long time to sort out the complexity associated with the particular receptor-virus interaction, but once we acquired this knowledge, we were able to design a decoy molecule that turned out to be very effective at neutralizing the virus and protecting mice from disease.” **MeV**

The story was original published on the Washington University website. It has been edited for style. <https://medicine.wustl.edu/news/study-reveals-new-clues-to-how-eastern-equine-encephalitis-virus-invades-brain-cells/>

For more information:

Adams LJ, Raju S, Ma H, et al. Structural and functional basis of VLDLR usage by eastern equine encephalitis virus. *Cell*. 2024 Jan 3; doi: 10.1016/j.cell.2023.11.031 <https://dx.doi.org/10.1016/j.cell.2023.11.031>

Feelings About Emotions Equal Better Welfare

Working equids whose owners believe in their capacity to feel emotion have significantly better health and welfare outcomes than those whose owners do not, according to research by the University of Portsmouth and The Donkey Sanctuary, in the Isle of Wight (*J Appl Anim Welf Sci*. 2023;1 doi: 10.1080/10888705.2023.2228029 <https://bit.ly/48UzOxw-MeV>).

Researchers visited equid-owning communities in Egypt, Mexico, Pakistan, Senegal, Spain and Portugal, where they carried out welfare assessments, which included a questionnaire about owner's beliefs, values and attitudes toward their animals; and a detailed assessment of the equids' welfare.

Animals whose owners believed they felt emotions

or who had an emotional bond with them, were in better health and had higher body condition scores than those whose owners did not, or who focused on how profitable or useful they were. Similarly, animals whose owners believed they could feel pain were much less likely to be lame.

These relationships were evident across multiple countries, with varying economic incomes.

“Our findings highlight the importance of the relationship between owners and their animals, and its significant impact on animal health and welfare,” said **Emily Haddy**, PhD, a postdoctoral research fellow at the University of Portsmouth's Centre for Comparative and Evolutionary Psychology. **MeV**

Feral Horses' Effect on Climate

By Sarah Gates

One might think of a feral horse as being somewhat carbon neutral, but a new study found carbon emissions from Australian alpine peatlands to be much higher in areas disturbed by feral horses.

Peatlands are incredibly effective at capturing carbon. While peatlands only cover up to 3% of the Earth's land surface, they store an estimated 30% of the world's soil carbon. This is twice the amount of carbon stored in every forest on the planet.



Credit: DPE

Collapsed stream bank in Kosciuszko National Park shows damage caused by wild horses.

Protecting these environments is an important step toward addressing global warming, according to **Sarah Treby**, PhD, an environmental scientist at RMIT University, in Australia.

The researchers measured peatland CO₂ emissions using a portable greenhouse gas analyzer, and found they were significantly higher at sites degraded by feral horses compared with sites where horses were not present. They looked at 12 alpine and subalpine *Sphagnum* moss-dominated bogs in Kosciuszko National Park, which were sampled over a 7-day period in March 2022—7 sites with feral horses present and 5 without. Data collected from sites with and without feral horses showed lowered water and soil quality where feral horses were present.

Horses also cause loss of soil carbon through waterways, known as fluvial carbon loss. Hard horse hooves trample and erode soils, which can end up in creeks and rivers, instead of in the peatland. This carbon is then transported downstream and, ultimately, released as carbon dioxide.

Degraded peatlands, such as those trampled by horses, can release large stores of carbon.

“The evidence suggests the damage resulting from horse grazing and trampling could have negative consequences for the long-term carbon storage of affected peatlands,” Dr. Treby said.

Horses were introduced to Australia in 1788 with the arrival of the first Europeans. Today, about 400,000 feral horses roam the country, according to the Invasive Species Council—more than any other country, with more than 18,000 in the Kosciuszko alone.

Co-author and soil scientist, **Samantha Grover**, PhD, explained these non-native feral horses are a destructive pest in the uniquely vulnerable Australian ecosystems.

Alpine and subalpine *Sphagnum* peatlands are listed as endangered and listed under the Environment Protection and Biodiversity Conservation Act 1999, due to their restricted geographic distribution, biodiversity declines and challenges in their restoration and regeneration.

Pest population management is carried out routinely in Australia for large hooved animals, including deer, pigs, goats, camels and water buffalo. Feral horse population management has been undertaken in many places around the country with a goal of reducing the population to 3,000 by 2027. However, in some areas, control methods that include culling have been controversial, constraining management programs.

“Our research on these peatlands in the Victorian Alps has shown that, in good condition, they can be strong carbon sinks, removing carbon dioxide from the atmosphere every year,” Dr. Grover said.

Australia's peatlands also provide critical habitat and resources for rare and unique species, as well as water for hydroelectricity, drinking and irrigation. **MeV**

For more information:

Original story appeared on the RMIT website. Content was edited for style and length.

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VET Stats

LIFETIME COST OF HORSE OWNERSHIP

**\$300,000 TO
> \$900,000.**

Basic costs, not including event
participation or operating expenses:

Competitive: \$575,000

Recreational: \$295,000

Backyard/pasture: \$215,000

With those costs:

Competitive: \$924,270+

Recreational: \$412,001+

Backyard/pasture: \$288,992+

90%
of horse owners will keep their
horses for life, which could be at
least **25 YEARS.**



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