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Are EIV Boosters Interchangeable?

Diagnostic Challenges of EPM

Do EqHV and EqPV-H Lead to Training Issues?

Do Mosquitoes Have a Natural Pesticide Protector?

Ask The ID Expert: How Is the Risk of Mosquito-Borne Disease Changing?

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SALES: Matthew Todd • Matthew Gerald

EDITOR: Marie Rosenthal

ART DIRECTOR: Jennifer Barlow

CONTRIBUTING WRITERS: Paul Basillo • Adam Marcus

COPY EDITOR: Patty Wall

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ASK THE Infectious Disease Expert

This column, brought to you by Merck Animal Health, features insightful answers from leading minds.

How is the risk of mosquito-borne disease changing & how can I ensure my clients remain vigilant?

West Nile virus (WNV) activity occurs in almost all of the contiguous states and, while the number of annual cases waxes and wanes, between 100 and 500 equine cases of WNV occur every year.[1] Eastern equine encephalomyelitis (EEE) is especially troubling, with 184 cases in 2019, up from 107 the previous year.[2] A trend of intensification in Northern states and along the East coast has also emerged.[2]

I attribute this increased activity not to vaccine failure, but rather to a surprising lack of vaccination, which I see happening for two main reasons: clients misunderstand the encephalitides and they under-estimate their horses' risk of becoming ill with these viruses.

THREE DISTINCT DISEASES

To help clients better protect their horses, I recommend explaining that there are multiple encephalitides, each with different activity. Horse owners often say, "My horse had his encephalitis shot." But they aren't aware of whether the vaccine covered WNV or Eastern/Western equine encephalomyelitis (EEE/WEE), and that's mainly because they mistakenly assume the diseases are one and the same.

While clients don't need to know the nuances of WNV versus EEE/WEE, they do need to understand that each is inherently different and that even multivalent vaccines aren't one-size-fits-all because predicting local outbreaks is almost impossible. Educate clients that you're considering their horse's lifestyle and geographic location when you recommend vaccination frequency and timing.

STATE BREAKDOWN OF 2019 CASES OF EASTERN EQUINE ENCEPHALITIS[2]

Click the image above to see a larger map.
Image source: USDA Animal and Plant Health Inspection Service.

PREDICTABLY UNPREDICTABLE

The encephalitides can pop up just about anywhere, which is one reason the AAEP recommends every horse be vaccinated against WNV and EEE/WEE every year. Still, some horse owners may let their guard down because of where they live. Many horse owners incorrectly consider EEE a Southern disease. In reality, there were as many cases of EEE in Michigan as in Florida in 2019.[2]

Encephalitis activity is ever-changing, due in part to warmer, wetter weather extending mosquito season and weather events like hurricanes shifting mosquito patterns. This leads to consistent encephalitis activity in expected areas with intensifications in unexpected states.

VACCINATION FIRST, TRAVEL SECOND

Many horse owners overlook how travel affects their horse's vaccination needs, especially if they're traveling to the Southern U.S. I commonly see WNV and EEE breaks in these populations:

- Foals and young horses that are unvaccinated or have not had enough booster injections to develop strong immunity.
- Pregnant broodmares who were neither vaccinated during gestation nor before getting on the trailer to foal in southern locales.
- Horses from Northern states that aren't up to date on their WNV or EEE/WEE boosters.

Even horses considered fully vaccinated should get a WNV and EEE/WEE booster two weeks before traveling to areas with year-round activity. If vaccine history is unknown or if there's any doubt about vaccination status, the horse should receive a full series—an initial vaccination followed by a booster in three to four weeks.

TAKE-HOME MESSAGE

The encephalitides are here to stay and, unfortunately, no one can predict when or where any one of them will appear. That's why horses need annual vaccination—sometimes more—no matter where they live. I like to say that WNV and EEE/WEE vaccination is less about vigilance and more about necessity.

1. 2019 Summary of West Nile Virus Equine Cases in the United States. USDA Animal and Plant Inspection Service. https://www.aphis.usda.gov/animal_health/downloads/animal_diseases/2019-wnv-report.pdf. Accessed May 28, 2021.
2. 2019 Summary of Eastern Equine Encephalitis Cases in the United States. USDA Animal and Plant Inspection Service. https://www.aphis.usda.gov/animal_health/downloads/animal_diseases/2019-eee-report.pdf. Accessed May 28, 2021.

ABOUT THE AUTHOR

Maureen T. Long, DVM, PhD, DACVIM (large animal), is a professor in the Department of Comparative, Diagnostic & Population Medicine at the University of Florida College of Veterinary Medicine. She has written and edited several hundred research papers and educational texts, such as the reference book *Equine Infectious Diseases*. Her research focus is infectious diseases of the horse, and she is currently working on two grant projects, one of which is COVID-19: SARS-CoV-2 Surveillance and Investigation in Florida Animals.

WANT TO ASK A QUESTION? EMAIL THE EDITOR.

For more information on WNV or EEE/WEE, visit merck-animal-health-usa.com/species/equine/products/prestige-5-wnv

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Are EIV Boosters REALLY INTERCHANGEABLE?

By Marie Rosenthal, MS

Equine influenza virus (EIV) vaccination remains the best ways to protect horses from this common respiratory disease. Vaccines not only aid with disease prevention, but they also reduce the severity of clinical signs, as well as decrease viral shedding in infected animals that have been previously vaccinated.

Vaccination, isolation of affected animals and strict biosecurity measures remain the most effective ways to prevent EIV outbreaks.

But what happens if boosters are made by a different manufacturer than the original series? Because vaccines have different strains, is giving a booster from a different manufacturer as efficacious as boosting with the original vaccine? And might a different schedule—say 2 doses 30 days apart—be necessary to assure protection against this common equine infectious disease?

"We often see that veterinarians use different products interchangeably during the immunization of some patients," said **Bruno Karam,** DVM, DACVIM (LAIM), who practices at Pilchuck Veterinary Hospital in Snohomish, Washington. "The changing product process implies a change in strain in the animal being vaccinated.

"There are no protocols in place with recommendations for how to approach this issue of immunization once switching manufacturers during the booster series. This study investigated the humoral immune

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[1] Nogradi N, Couetil LL, Messick J, Stochelski MA, Burgess JA. Evaluation of an Omega-3 Fatty Acid Containing Feed Supplement in the Management of Horses with Chronic Lower Airway Inflammatory Diseases. J Vet Intern Med 2015; 29:299-306.
[2] Couetil LL, Cardwell J.M, Gerber V, Lavoie J.-P, Leguillette R, Richard E.A. Inflammatory Airway Disease of Horses. ACVIM Consensus Statement J of Vet Intern Med 2016; 30:503-515 p. 508-510.

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response to EIV boosters from different manufacturers given to previously immunize adult horses as part of their routine vaccination program."

A recent study done by veterinarians at the University of California at Davis, College of Veterinary Medicine found that antibody titers from a horse that received a different booster dose were not different from those receiving the homologous vaccine, according to Dr. Karam.

In addition to determining whether the switch affected the efficacy, they also wanted to know if the switch in the EIV boosters would require at least a 2-dose series, 30 days apart, and if 2 doses triggered a similar or superior response than a single booster.

Horses that received a different booster dose of EIV vaccine from his original series still developed antibodies.

Antigenic Drift

EIV strains cuasing worldwide outbreaks recently belong to the Florida sublinages clad 1 and 2. In North America, outbreaks are caused by the clade 1 strains, but international travel has led to the potential exposure to the clade 2 strains. Because of this, the American Association of Equine Practitioners and the World Organization for Animal Health recommend that vaccines protect against clade 1 and clade 2 strains, Dr. Karam explained.

They studied 64 healthy adult horses from the Center of Equine Health at the UC Davis campus that had been vaccinated against EIV using a killed adjuvant vaccine that contains the Kentucky 97 strain. The horses were randomly assigned to 3 different vaccine groups, each with about 20 horses. The groups were then subdivided even further, and 10 horses in each group were randomly assigned to receive a booster 30 days later. Another 4 horses served as sentinel horses to monitor for naturally occurring influenza disease.

Group 1 horses were vaccinated against EIV using the product that contained the Kentucky 97 strain.

"It is important to note that the Kentucky 97 stains predate the subdivergence for clade 1 and 2," Dr. Karam said. Group 2 was vaccinated with a product that contained clade 1 and 2 strains represented by the Ohio 03 and the Richmond 07 strains. The third group received a vaccine that had both Florida clades represented by the Florida 13 strain and the Richmond 07 strains.

On day 0, blood was drawn from all horses, even the control horses, and the horses in the 3 vaccine groups were vaccinated. Thirty days later, blood was drawn again and the horses designated for a second dose were given their second doses.

Blood was drawn again on all horses on days, 60, 90 and 180. All samples were clotted and centrifuged and stored in -80° C. Samples were blindly tested for antibody titers using a hemagglutination inhibition assay against the Kentucky 14 strain (clade 1) and Richmond 07 (clade 2) strain.

Three horses did not complete the study, but not because of vaccination or the sampling process.

They found no significant differences among any of the 3 vaccine or control groups at day 0. There were significant differences between day 0 blood draws and subsequent blood draws post vaccination, but there was no significant difference in antibody titers against the Kentucky 14 strain among the 3 vaccines at any time. There was also no significant difference between 1 dose versus 2-dose vaccine groups.

"All 3 vaccines generate a similar antibody response and appear to be suitable options for vaccination," Dr. Karam said. "In previously vaccinated horses, the results provided evidence that hemagglutination antibody response in previously vaccinated horses to a different inactivate injectable product is similar to the vaccine that had been previously used."

Dr. Karam noted an important limitation to the study, however. Although hemagglutination is an acceptable way to indicate protection, single radial hemolysis is the accepted test to ascertain protective titers. Still, the antibody response to appears satisfactory so veterinarians should feel comfortable if they have to use a different booster during an outbreak or for some other reason. **MeV**

For more information:

Karam B, Wilson WD, Chambers TM, et al. Hemagglutinin inhibition antibody responses to commercial equine influenza vaccines in vaccinated horses. *Can Vet J.* 2021;62(3):266-272. **https://pubmed.ncbi.nlm.nih.gov/33692582/**

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1 Data on file.

2 Adequan® i.m. Package Insert, Rev 1/19.

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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New Model for INFECTIOUS DISEASE Could Better Predict Future Pandemics

In the midst of a devastating global pandemic of wildlife origin and with future spillovers imminent as humans continue to come into closer contact with wildlife, infectious-disease models that consider the full ecological and anthropological contexts of disease transmission are critical to the health of all life. Existing models are limited in their ability to predict disease emergence, since they rarely consider the dynamics of the hosts and ecosystems from which pandemics emerge.

Smithsonian scientists and partners provided a framework for a new approach to modeling infectious diseases. It adapts established methods developed to study the planet's natural systems, including global warming, ocean circulation and forest growth, and applies them to parasites and pathogens that cause disease.

Increased human-animal interactions lead to the emergence and spread of zoonotic pathogens, which cause about 75% of infectious diseases affecting human health. Predicting where, how and when people and animals are at risk from emerging pathogens—and the best ways to manage them—remains a significant challenge. Risks for spillover include, but are not limited to, habitat encroachment, illegal wildlife trade and bush meat consumption.

Despite incredible advances in the understanding of how infectious diseases are transmitted, the models for which these efforts are based are relatively limited, focusing on specific pathogens and often overlooking how pathogens interact within their hosts. While scientists and global health organizations are putting a lot of effort into studying the diversity of disease-causing organisms, existing models do not link this

Increased human-animal interactions lead to the emergence and spread of zoonotic pathogens, which cause about 75% of infectious diseases in people. Wild Zebras graze alongside farmers.

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This new model will require collaboration across many fields, such as veterinary and human medicine, disease ecology, biodiversity conservation, biotechnology and anthropology.

diversity to their roles within ecosystems.

"Just as a mechanic must understand how a car's components interact and how it's been engineered in order to improve performance, the same applies to our ability to model infectious disease," said first author **James Hassell,** DVM, wildlife veterinarian, epidemiologist and Keller Family Skorton Scholar for the Smithsonian Conservation Biology Institute's (SCBI) Global Health Program. "Applying systems-level thinking to forecast disease emergence requires a fundamental change in how we conceptualize infectious diseases. This presents significant challenges, but in this article, we explain why they're not insurmountable. When you weigh the cost of prevention versus remediation, the investment in our shared global health, particularly the connections between nature and human health, is vital."

Researchers said this new model will require expertise and collaboration across fields such as veterinary and human medicine, disease ecology, biodiversity conservation, biotechnology and anthropology.

"Disease and health are predominantly viewed as a human construct and the role the environment plays in disease is often overlooked," said **Yvonne-Marie Linton,** PhD, research director for the Walter Reed Biosystematics Unit for the Smithsonian's National Museum of Natural History and Walter Reed Army Institute of Research. "The health of other organisms, from parasites and insects to birds and aquatic organisms, can alter the structure of ecosystems. What we're proposing is a new approach to modeling infectious diseases that are circulating in nature, which would allow scientists to simulate the behavior of these pathogens in wildlife populations, how they respond to human activities and better determine the risk that they pose to people."

General ecosystem models are essentially complex models that can predict how food chains are assembled—the processes of energy transfer between plants and animals are what structure ecosystems—and determine the plants and animals that compose an ecosystem. With the new version, general "episystem" models, the paper's authors outline a framework for integrating disease agents (including parasites, viruses and bacteria) into these models. By identifying general rules for how food chains that include disease entities are structured, it should be possible to predict the types of pathogens that are present in any given ecosystem. This would allow scientists to better understand the characteristics of an ecosystem (such as disturbance) that would make it more likely to contain zoonotic pathogens, predict the threat it poses to people who interact with this ecosystem and even permit computer simulation and testing of interventions aimed at reducing these threats.

While the megadata that would be required to create these models is daunting, long-term studies of intact ecosystems where parasite data have been collected are excellent places to initiate these studies. Efforts to refine them more broadly could then leverage large-scale ecological studies that span continents such as the Smithsonian's ForestGEO and MarineGEO programs.

The potential impacts of this new model go beyond reducing the human interface for disease spillover, to economics. "You could use this new approach not only to look at human diseases, but also to look at the best way to conduct aquaculture or raise healthy livestock," said **Katrina M. Pagenkopp Lohan,** PhD, a marine disease ecologist at the Smithsonian Environmental Research Center. "If you're reintroducing a species into the wild, what do you need that ecosystem to look like for you to be successful? We could actually model that. It's mind blowing."

The cost of such a new approach is considerable, they said, and will take the global cooperation and commitment of scientists, communities, non-governmental organizations and nations. In an era of big data and massive advances in technology, this kind of approach is achievable but requires enhanced data collection, sharing and testing at far greater scales than currently occur. **MeV**

For more information:

Hassel JM, Newbold T, Donson AP, et al. Towards an ecosystem model of infectious disease. *Nat Ecol Evol*. 2021 May 17 https://doi.org/10.1038/s41559-021-01454-8 https://www.nature.com/articles/s41559-021-01454-8

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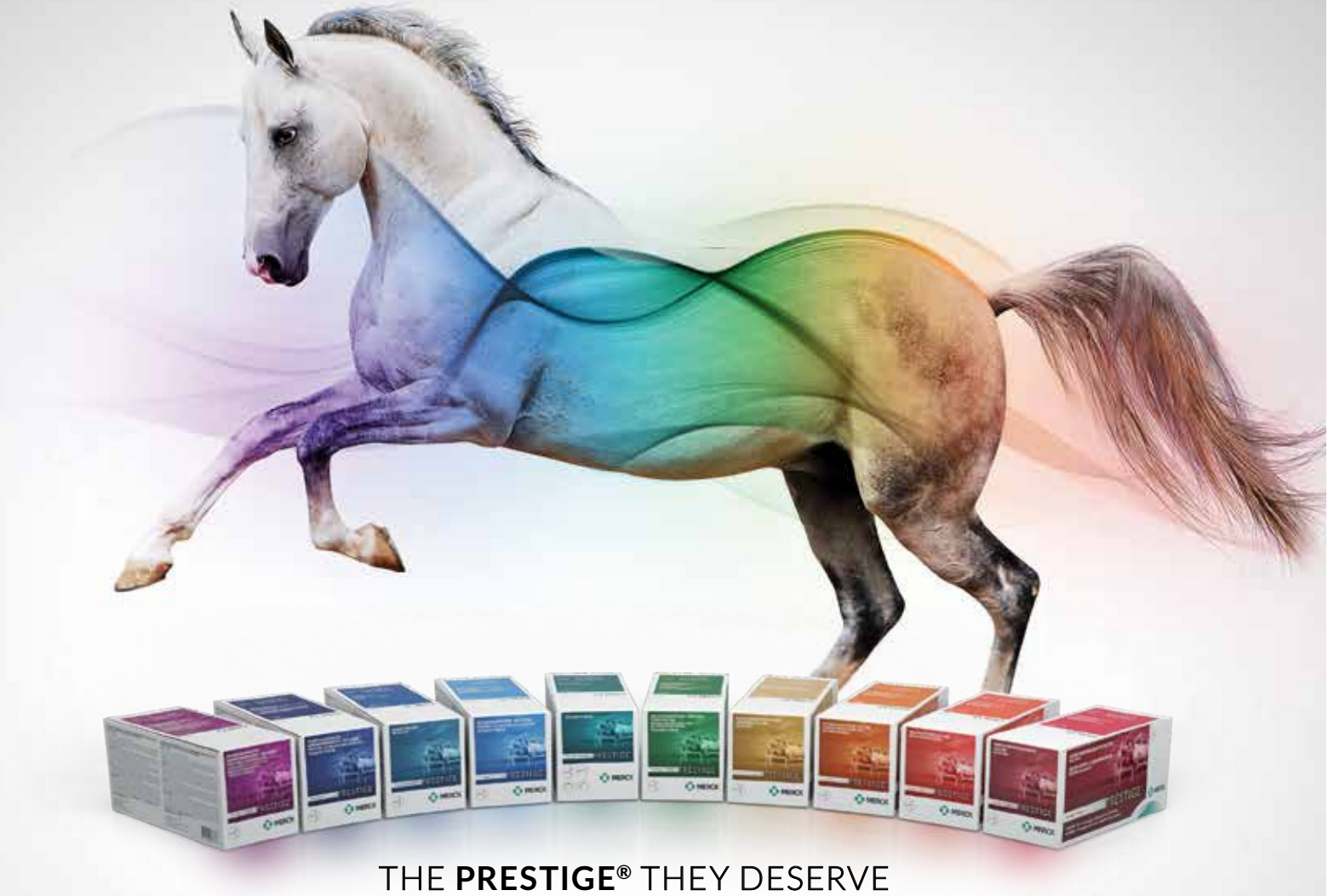

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Navigating the Diagnostic Challenges of EPM

By Nicola Pusterla, DVM, PhD, DACVIM

This is a retrospective on how far we have come in understanding, diagnosing and treating this important infectious neurological disease. In this four-part series, **Nicola Pusterla, DVM, PhD, DACVIM**, guides us through a reflective account of equine protozoal myeloencephalitis (EPM), including advances in testing and diagnosing, as well as treatment and preventive measures. Part 1 of this series provided a brief review of the causes of EPM.

Part 2 of this series.

Equine protozoal myeloencephalitis (EPM) continues to be one of the most challenging maladies practitioners face, particularly when it comes to identification and diagnosis. The temptation to "treat and see" without a complete diagnostic picture can be tough to resist. However, with so many diseases causing clinical signs similar to EPM, it's an urge practitioners must avoid. If a horse has a disease other than EPM, not only have we wasted money on unnecessary treatment, but also time that could have been better used pursuing the true cause of the horse's problem.

Our diagnostic approaches to EPM have come a long way, as has our understanding of the causes. Reliable quantitative testing coupled with practical hands-on examination principles can help practitioners gain confidence in their EPM diagnosis.

What defines an EPM suspect case?

The best initial course of action is a combination of reviewing the horse's health history and performing a thorough physical and neurologic exam. Any region within the central nervous system (CNS) can become parasitized, and the clinical signs may vary depending on which part of the nervous system is affected. Asymmetry is a telltale marker of a suspected EPM case. It is a progressive, multifocal disease, often with muscle atrophy. These signs along with ataxia and dysmetria are the most common clinical signs to watch for in EPM cases.

Let's walk through a very basic 'if-then' scenario to gain a clearer picture of a proper diagnostic workup in a suspect case.

CRITICAL CASE QUESTION: After you've conducted a thorough physical and neurological exam, where does EPM sit on your list of differentials?

Upon physical and neurologic evaluation:

1. Does the horse have a history of EPM or has EPM been diagnosed in the resident population?
   a. Yes
   b. No

2. Is the horse exhibiting asymmetrical weakness and focal muscle atrophy?
   a. Yes
   b. No

3. What is the immune status of the horse?
   a. High-stress, performance, travel, weaning, etc.
   b. Presence of metabolic, endocrine and/or other chronic infectious diseases
   c. Age-related immunosenescence
   d. Normal

HINT: If asymmetric gait and focal muscle atrophy are present, EPM should be considered a top differential.

If the horse does not appear to have any neurological deficits, rather a musculoskeletal condition such as lameness, or clinical signs point to a neurological disorder other than EPM, there is no need to test for EPM. If, however, you answered yes to the questions above and the horse's immune status may be compro-

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The only way to definitively diagnose EPM is on necropsy. Authors of the Updated (2016) ACVIM Consensus Statement on EPM defined the gold standard for diagnosing EPM in a living horse:

Observable neurologic signs consistent with EPM	A serum: CSF ratio that is within the range of established serological tests (e.g., SAG 2,4/3 <100, IFAT ≤ 64), which indicates intrathecal (within the CNS) antibody production against *S. neurona* or *N. hughesi*	Ruling out diseases with similar neurologic signs

mised, antibody testing is recommended to further differentiate EPM from other neurological diseases.

Antibody testing – a cautionary tale

The question I often get asked, "What is the right biological sample to use? Blood or cerebrospinal fluid (CSF)?"

Evidence of intrathecal antibody production is the most accurate way to support an EPM diagnosis. However, blood is a good screening tool. If serum comes back negative, likely there is no infection or recent infection. EPM can generally be ruled out and you can proceed down your list of differentials. (Figure 1) A rare exception would be a very acute onset prior to antibody production, in which case a retest in 10 to 14 days is warranted.

A positive serum test result, however, presents a "gray zone" because it doesn't necessarily equate to an EPM diagnosis. Knowing there is a high seroprevalence to the causative organisms of EPM in healthy U.S. horses, now what?

SERUM POSITIVE? PROCEED WITH CAUTION: 78% of healthy U.S. horses are seropositive to *Sarcocystis neurona* and 34% to *Neospora hughesi*. So, while you can find a seropositive horse in almost every pasture, a positive result doesn't mean that horse has EPM.

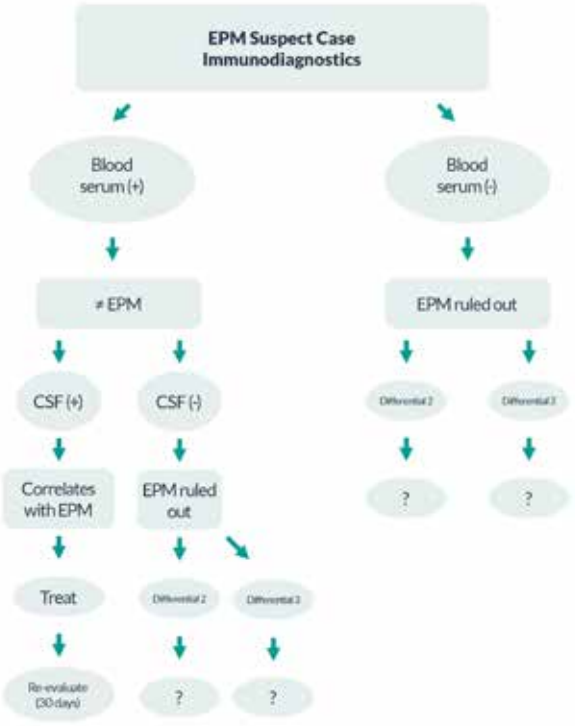
To more definitively rule out (or in) EPM, a CSF tap is recommended. If the CSF sample is negative, EPM is ruled out and the practitioner should proceed to the next differential diagnosis. If the CSF sample is positive, consider it a pretty strong case for an EPM diagnosis. (Figure 1)

Be aware, a positive CSF result can happen for reasons other than antibody production within the CNS. For one, blood contamination. If there is a high blood titer to *S. neurona*, for example, this could give a positive result from blood-derived antibody and not production within the CNS.

Therefore, current best practice consensus is to collect spinal fluid and a blood sample and compare the antibody titers in each to determine if there is evidence of a CNS infection. This is done by evaluating the ratio of antibody titer in serum divided by antibody titer in CSF.

Treating the horse with an antiprotozoal drug for 2 weeks and then reassessing may be a practical approach if you feel strongly this is an EPM case and CSF sampling is not accepted. However, this is ONLY recommended if you can physically reassess the horse in 2 weeks to evaluate progress. At that time, critically evaluate the improvement by repeating the same thorough physical and neurologic work up. A significant improvement must be seen to continue with unfinished treatment (I like to see at least a 25% improve-

FIGURE 1: What is a Proper EPM Diagnostic Work-up?



If EPM rises to the top of your list of differentials after a thorough physical and neurological exam, antibody testing should be used to further differentiate EPM from other neurological diseases. Intrathecal antibody production is the most accurate way to support an EPM diagnosis, while blood is useful for screening.

The only **dual ingredient** injectable corticosteroid approved by the FDA for use in horses

The link between **RAPID ONSET** and **LONG-ACTING RELIEF** of pain & inflammation[1]

BetaVet® (betamethasone sodium phosphate & betamethasone acetate injectable suspension) is indicated for the control of pain and inflammation associated with osteoarthritis in horses. **Learn more at www.betavetequine.com or call 1-800-458-0163.**

Please see Brief Summary of Full Prescribing Information on the following page.

INDICATION BetaVet® (betamethasone sodium phosphate and betamethasone acetate injectable suspension) is indicated for the control of pain and inflammation associated with osteoarthritis in horses. **IMPORTANT SAFETY INFORMATION** For **Intra-articular** (I.A.) use in Horses. **CONTRAINDICATIONS** BetaVet® is contraindicated in horses with hypersensitivity to betamethasone. Intra-articular injection of corticosteroids for local effect is contraindicated in the presence of septic arthritis. **WARNINGS:** Do not use in horses intended for human consumption. Clinical and experimental data have demonstrated that corticosteroids administered orally or parenterally to animals may induce the first stage of parturition when administered during the last trimester of pregnancy and may precipitate premature parturition followed by dystocia, fetal death, retained placenta, and metritis. Additionally, corticosteroids administered to dogs, rabbits and rodents during pregnancy have resulted in congenital anomalies. Before use of corticosteroids in pregnant animals, the possible benefits should be weighed against potential hazards. **Human Warnings:** Not for use in humans. Keep this and all medications out of the reach of children. **PRECAUTIONS:** Corticosteroids, including BetaVet,® administered intra-articularly are systemically absorbed. Do not use in horses with acute infections. Acute moderate to severe exacerbation of pain, further loss of joint motion, fever, or malaise within several days following intra-articular injection may indicate a septic process. Because of the anti-inflammatory action of corticosteroids, signs of infection in the treated joint may be masked. Due to the potential for exacerbation of clinical signs of laminitis, glucocorticoids should be used with caution in horses with a history of laminitis, or horses otherwise at a higher risk for laminitis. Use with caution in horses with chronic nephritis, equine pituitary pars intermedia dysfunction (PPID), and congestive heart failure. Concurrent use of other anti-inflammatory drugs, should be approached with caution. Consider appropriate wash out times prior to administering additional NSAIDs or corticosteroids. **ADVERSE REACTIONS:** Adverse reactions reported during a field study of 239 horses of various breeds which had been administered either BetaVet® (n=119) or a saline control (n=120) at five percent (5%) and above were: acute joint effusion and/or local injection site swelling (within 2 days of injection), 15% BetaVet® and 13% saline control; increased lameness (within the first 5 days), 6.7% BetaVet® and 8.3% saline control; loose stool, 5.9% BetaVet® and 8.3% saline control; increased heat in joint, 2.5% BetaVet® and 5% saline control; and depression, 5.9% BetaVet® and 1.6% saline control. **SHAKE WELL IMMEDIATELY BEFORE USE.** For additional safety information, please see full prescribing information. **CAUTION: Federal law restricts this drug to use by or on the order of a licensed veterinarian.**

References: 1. Trotter GW. Intra-articular corticosteroids. In: McIlwraith CW, Trotter GW, eds. Joint Disease in the Horse. Philadelphia: W.B. Saunders; 1996; 237-256.

BetaVet™
betamethasone sodium phosphate & betamethasone acetate injectable suspension

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BetaVet®

betamethasone sodium phosphate &
betamethasone acetate

INJECTABLE SUSPENSION

BRIEF SUMMARY OF PRESCRIBING INFORMATION
(Betamethasone Sodium Phosphate and Betamethasone Acetate Injectable Suspension) 6 mg betamethasone per mL
For Intra-Articular (I.A.) Use in Horses

CAUTION: Federal law restricts this drug to use by or on the order of a licensed veterinarian.

INDICATION: BetaVet® is indicated for the control of pain and inflammation associated with osteoarthritis in horses.

DOSAGE AND ADMINISTRATION: Shake well immediately before use.

CONTRAINDICATIONS: BetaVet® is contraindicated in horses with hypersensitivity to betamethasone. Intra-articular injection of corticosteroids for local effect is contraindicated in the presence of septic arthritis.

WARNINGS: Do not use in horses intended for human consumption.
Clinical and experimental data have demonstrated that corticosteroids administered orally or parenterally to animals may induce the first stage of parturition when administered during the last trimester of pregnancy and may precipitate premature parturition followed by dystocia, fetal death, retained placenta, and metritis. Additionally, corticosteroids administered to dogs, rabbits and rodents during pregnancy have resulted in cleft palate in offspring. Corticosteroids administered to dogs during pregnancy have also resulted in other congenital anomalies including deformed forelegs, phocomelia and anasarca. Therefore, before use of corticosteroids in pregnant animals, the possible benefits to the pregnant animal should be weighed against potential hazards to its developing embryo or fetus. **Human Warnings:** Not for use in humans. For use in animals only. Keep this and all medications out of the reach of children. Consult a physician in the case of accidental human exposure.

PRECAUTIONS: Corticosteroids, including BetaVet®, administered intra-articularly are systemically absorbed. Do not use in horses with acute infections. Acute moderate to severe exacerbation of pain, further loss of joint motion, fever, or malaise within several days following intra-articular injection may indicate a septic process. Because of the anti-inflammatory action of corticosteroids, signs of infection in the treated joint may be masked. Appropriate examination of joint fluid is necessary to exclude a septic process. If a bacterial infection is present, appropriate antibacterial therapy should be instituted immediately. Additional doses of corticosteroids should not be administered until joint sepsis has been definitively ruled out. Due to the potential for exacerbation of clinical signs of laminitis, glucocorticoids should be used with caution in horses with a history of laminitis, or horses otherwise at a higher risk for laminitis. Use with caution in horses with chronic nephritis, equine pituitary pars intermedia dysfunction (PPID), and congestive heart failure. Concurrent use of other anti-inflammatory drugs, such as NSAIDs or other corticosteroids, should be approached with caution. Due to the potential for systemic exposure, concomitant use of NSAIDs and corticosteroids may increase the risk of gastrointestinal, renal, and other toxicity. Consider appropriate wash out times prior to administering additional NSAIDs or corticosteroids.

ADVERSE REACTIONS: Adverse reactions reported during a field study of 239 horses of various breeds which had been administered either BetaVet® (n=119) or a saline control (n=120) were: acute joint effusion and/or local injection site swelling (within 2 days of injection), 15% BetaVet® and 13% saline control; increased lameness (within the first 5 days), 6.7% BetaVet® and 8.3% saline control; loose stool, 5.9% BetaVet® and 8.3% saline control; increased heat in joint, 2.5% BetaVet® and 5% saline control; depression, 5.9% BetaVet® and 1.6% saline control; agitation/anxiety, 4.2% BetaVet® and 2.5% saline control; delayed swelling of treated joint (5 or more days after injection), 2.5% BetaVet® and 3.3% saline control; inappetance, 3.4% BetaVet® and 2.5% saline control; dry stool, 1.7% BetaVet® and 0% saline control; excessive sweating, 0.8% BetaVet® and 0% saline control; acute non-weight bearing lameness, 0.8% BetaVet®and 0% saline control; and laminitis, 0.8% BetaVet® and 0% saline control.

CLINICAL PHARMACOLOGY: Betamethasone is a potent glucocorticoid steroid with anti-inflammatory and immunosuppressive properties. Depending upon their physico-chemical properties, drugs administered intra-articularly may enter the general circulation because the synovial joint cavity is in direct equilibrium with the surrounding blood supply. After the intra-articular administration of 9 mg BetaVet® in horses, there were quantifiable concentrations of betamethasone (above 1.0 ng/mL) in the plasma.

EFFECTIVENESS: A negative control, randomized, masked field study provided data to evaluate the effectiveness of BetaVet® administered at 1.5 mL (9 mg betamethasone) once intra-articularly for the control of pain and inflammation associated with osteoarthritis in horses. Clinical success was defined as improvement in one lameness grade according to the AAEP lameness scoring system on Day 5 following treatment. The success rate for horses in the BetaVet® group was statistically significantly different (p=0.0061) than that in the saline group, with success rates of 75.73% and 52.52%, respectively (back-transformed from the logistic regression).

ANIMAL SAFETY: A 3-week target animal safety (TAS) study was conducted to evaluate the safety of BetaVet® in mature, healthy horses. Treatment groups included a control (isotonic saline at a volume equivalent to the 4x group); 1X (0.0225 mg betamethasone per pound bodyweight; BetaVet®); 2X (0.045 mg betamethasone per pound bodyweight; BetaVet®) and 4X (0.09 mg betamethasone per pound bodyweight; BetaVet®). Treatments were administered by intra-articular injection into the left middle carpal joint once every 5-days for 3 treatments. Injection site reactions were the most common observations in all treatment groups. Injection site reactions were observed within 1 hour of dosing and included swelling at the injection site, lameness/stiffness of the left front limb, and flexing the left front knee at rest. The injection site reactions ranged from slight swelling (in many horses on multiple days in all treatment groups) to excessive fluid with swelling, pain, and lameness (4x group only). Injection site reactions were observed most commonly on treatment days, and generally decreased in number and severity over subsequent days. The incidence of injection site reactions increased after the second and third injection (number of abnormalities noted on day 10 > day 5 > day 0). In the BetaVet® treated groups the number and severity of the injection site reactions were dose dependent. The 4X BetaVet® group had the highest overall incidence of and severity of injection site reactions, which included heat, swelling, pain, bleeding, and holding the limb up at rest. The control group and 4X group (which received similar injection volumes) had a similar incidence of injection site reactions; however, the severity of reactions was greater in the 4X group. Absolute neutrophils were statistically significantly higher in the BetaVet® treated groups as compared to the control group. Trends toward a decrease in lymphocytes and eosinophils, and an increase in monocytes were identified in the BetaVet® treated groups after the initial dose of BetaVet®. Individual animal values for white blood cells generally remained within the reference range. BetaVet® treated horses also had a trend toward increased blood glucose after the initial dose. Some individual animals showed mild increases in blood glucose above the reference range.

SHAKE WELL BEFORE USING
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To report an adverse event please contact American Regent Animal Health at (888) 354-4857 or email pv@americanregent.com.

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ment). If the horse responds to treatment, you have further evidence to support an EPM diagnosis. If the horse does not respond, it's back to the drawing board. This is a critical communication point with the owner to help them understand next steps and avoid potential frustration of treating for EPM with no improvement. (See sidebar on client communication tips.)

Tricky disease. Tricky diagnosis.

EPM got you scratching your head? Take heart. Each clinical presentation is different, and the best way to outwit this disease mimicker is with a solid dose of due diligence. There are no shortcuts when it comes to doing a proper EPM diagnostic work up. Look for hallmarks of clinical disease in your physical exam and don't shy away from the need for immunodiagnostics to get to the root cause.

The good news: If EPM is diagnosed, there are safe and effective EPM treatment options. Come back for the third in our four-part series as we dive into the latest treatment advancements. **MeV**

For more information:

James KE, Smith WA, Conrad PA, et al. Seroprevalences of anti-Sarcocystis neurona and anti-Neospora Hughesi antibodies among healthy equids in the United States. *JAVMA*. 2017;250(11) :1291-1301 **https://doi.org/10.2460/javma.250.11.1291**

Reed SM, et al. Equine protozoal myeloencephalitis: An updated consensus statement with a focus on parasite biology, diagnosis, treatment and prevention. *J Vet Intern Med*. 2016;30:491–502. **https://onlinelibrary.wiley.com/doi/10.1111/jvim.13834**

NAHMS. Equine Protozoal Myeloencephalitis (EPM) in the U.S. In: USDA:APHIS:VS, ed. Centers for Epidemiology and Animal Health. Fort Collins, Colorado: *NAHMS*; 2001:1–46. **https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/monitoring-and-surveillance/nahms/nahms_equine_studies**

TIPS FOR TALKING EPM DIAGNOSTICS WITH CLIENTS

As practitioners, we often face the eager client who confidently walks in and says, "Doc, I need this horse tested for EPM," or "I'm pretty sure it's EPM, can you get him started on an EPM treatment?"

1) Remind owners that EPM can be the master of disguise and mimic many neurologic diseases. It's important to evaluate all potential causes of the horse's illness before rushing to EPM testing and/or treatment.

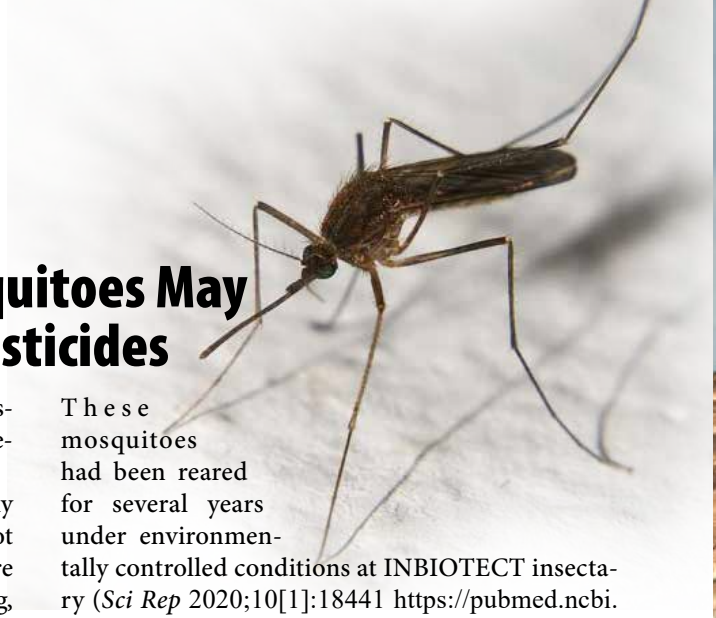
2) More than two-thirds of healthy U.S. horses are carrying antibodies to *S. neurona*—the primary causative organism of EPM—without showing clinical signs. Only a very small percentage (<1%) of horses succumb to clinical disease.

3) If the horse does not have EPM, it will not respond to an EPM treatment and you've wasted time and money without getting to the root cause of the issue. This could prolong suffering for the horse.

4) Increase client confidence in your diagnostic work up by explaining the importance of a thorough history on the horse, as well as a physical and neurological examination along with proper antibody testing to rule EPM in or out. We cannot rely on one without the other.

5) Emphasize the importance of continued vigilance with regular veterinary examinations to ensure treatment success and ongoing care and management of the horse with EPM.

6) Reinforce early veterinary intervention for any horse showing signs of neurological disease. The earlier disease is caught, and treatment begins, the better the outcome.



Bacteria Carried by Mosquitoes May Protect Them Against Pesticides

A common bacterial species naturally infecting mosquitoes may actually be protecting them against specific mosquito pesticides, a study has found.

Wolbachia is a bacterium that occurs naturally and spreads among insects, but scientists were not sure if there was a symbiotic relationship, but there appears to be. Scientists at the University of Reading, and the INBIOTEC-CONICET and the National University of San Juan in Argentina, studied the effect of *Wolbachia* on a common mosquito species and found those carrying the bacteria were less susceptible to widely used pesticides.

This shows the importance of looking more closely at how bacteria in mosquitoes and pesticides interact, especially at a time when new plans are being formulated for which [control] methods to use, where to use them and which species to target," explained **Alejandra Perotti,** PhD, associate professor in invertebrate biology at the University of Reading, in the United Kingdom, and a co-author of the study.

In the new study, the researchers looked at *Culex quinquefasciatus*, the southern house mosquito. These mosquitoes had been reared for several years under environmentally controlled conditions at INBIOTECT insectary (*Sci Rep* 2020;10[1]:18441 https://pubmed.ncbi.nlm.nih.gov/33116256/).

This is 1 of the most widespread species in hotter climates. The mosquito transmits several diseases, including a range of viruses such as the West Nile Virus (WNV), the San Luis Encephalitis Virus (SLEV) and the Venezuelan Equine Encephalitis Virus, and in addition a variety of parasites (filarial worms) in Central and South America, Africa and Asia.

The team found that the mosquito larvae naturally infected by an Argentinian native strain of *Wolbachia* were less susceptible to 3 bacterial pesticides—*Bacillus thuringiensis israelensis, Bacillus wiedmannii biovar thuringiensis* and *Lysinibacillus sphaericus*—2 of which are commercially available and used in many countries to control mosquito populations. **MeV**

EqHV and EqPV-H Unlikely Reasons for Training Issues

Equine hepacivirus (EqHV) and equine parvovirus-hepatitis (EqPV-H) infections are unlikely to be the primary cause for the High-serum γ-glutamyl transferase (GGT) syndrome seen in Thoroughbred racehorses.

GGT activity has been associated with and thought to be a marker of maladaptation to training and possibly poor performance in racehorses, but the cause is unknown. Researchers wanted to evaluate the role of oxidative stress, cholestasis, liver injury and infection with EqHV or EqPV-H infection in racehorses with increased serum GGT activity to see if they were the cause.

The work consisted of a pilot study with 8 horses with elevated serum GGT activity (≥50 U/L) and 8 controls with normal GGT activity, followed by a larger study with 27 case-control pairs from 3 different yards. The researchers performed serum liver chemistries, selenium measurements, viral polymerase chain reaction (PCR) and metabolomics.

With both studies combined, the overall prevalence of EqHV was 9% and the overall prevalence of EqPV-H was 36%.

There was no difference in prevalence or copy numbers between cases and controls for either virus. Cases had decreased serum selenium concentrations, higher serum glutamate dehydrogenase and alkaline phosphatase activities and higher serum bile acid concentrations compared with controls. Metabolomic findings were not consistent between the two studies, with different metabolites emerging from each analysis.

The researchers concluded that the mechanism underlying high GGT activity in this population is likely complex and multifactorial, involving hepatic injury and possible cholestasis as well as oxidative stress. **MeV**

For more information:

Mann S, Ramsay JD, Wakshly JJ, et al. Investigating the pathogenesis of high-serum gamma-glutamyl transferase activity in Thoroughbred racehorses: A series of case-control studies *Equine Vet J*. 2021 Feb 8 **https://doi.org/10.1111/evj.13435**

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