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Davis-Thompson Foundation

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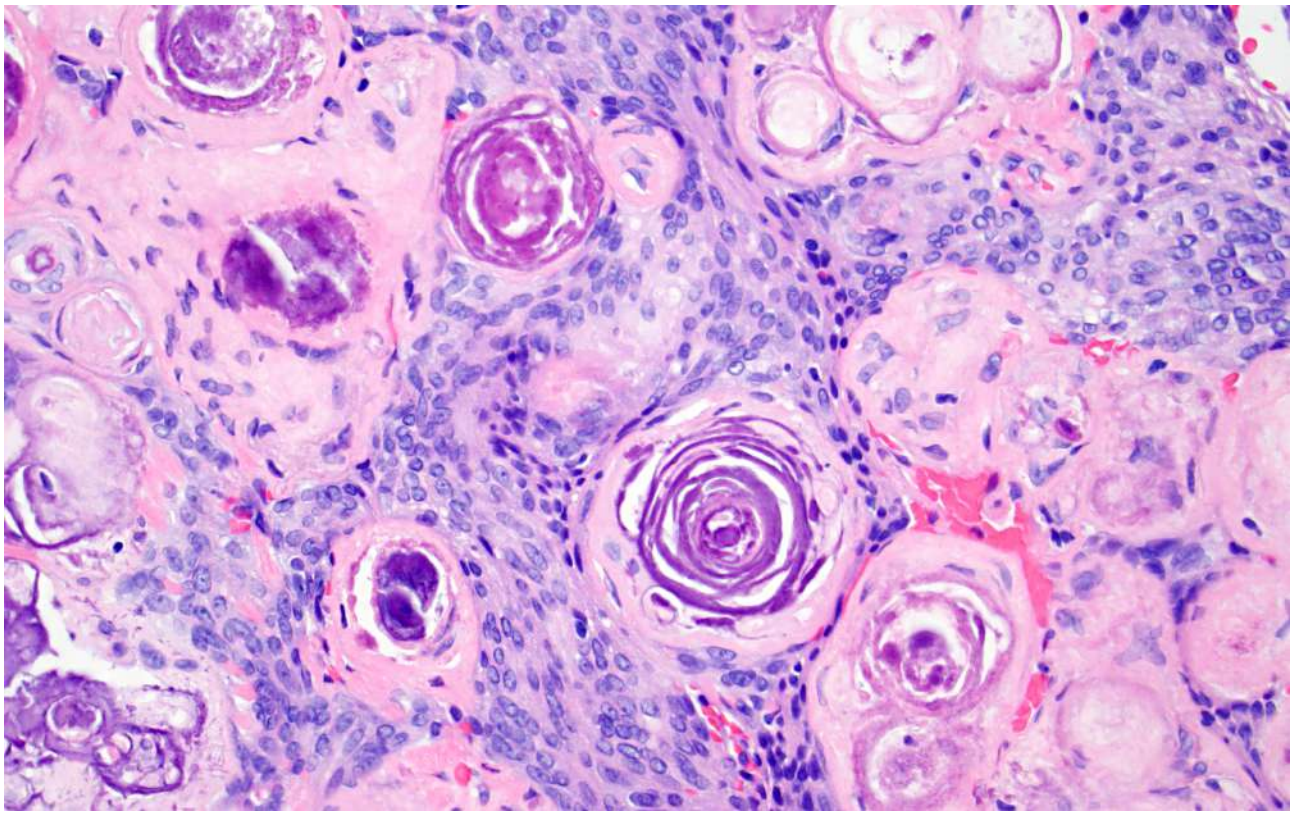
For the advancemet of veterinary and comparative pathology

THE DAVIS-THOMPSON FOUNDATION NEWSLETTER

M a y

VOL. 54



Meningiomas are thought to arise from the:

A. Dura mater
B. Arachnoid mater
C. Pia mater
D. Mesothelium

Monthly cover photograph winners:
Amelia I. Andersson and Michael A. Talavera
Mississippi State University

Answer: B. Arachnoid mater

An eight-year-old, neutered male, mixed breed canine with psammomatous type meningioma containing multifocal basophilic, concentric concretions consistent with psammoma bodies.

-Dr. Katherine D. Watson - Cover Image Editor
-Dr. M. Donald McGavin - Cover Image Composition Analyst

MESSAGE FROM THE CEO

Dear colleagues

It is my pleasure to welcome you to the May issue of the Davis-Thompson Foundation newsletter, with the compliments, as usual, of our outstanding managing editors Jeann Leal and Javier Asin.

As usual, this issue comes with information on myriad training opportunities around the world, in several languages. Remember that two of our "Big 4", CLIIC and POLA are coming soon and registration is open. POLA and our Descriptive Veterinary Pathology Course are also offered in Europe this year. Please see below the words of the Foundation's President, Dr Jey Koehler about our Descriptive Veterinary Pathology course:

"This year, our beloved Descriptive Veterinary Pathology course will be transformed into Foundations of Pathology. The course will have a three-day virtual component in September and two half days of in-person instruction at the ACVP meeting in Seattle. While some of the material will remain the same, there will be new material as well and all will be aimed at the early stage trainee. We highly recommend that this course be taken by first-year residents. Topics will include introduction to evaluating and describing gross, microscopic, and ultrastructural lesions, interpreting IHC/ISH and molecular testing, as well as study techniques, structuring study during your residency, networking with other trainees, understanding the role of self-assessments, and more!

Due to reduced travel costs for lecturers, we will be offering this course for much less than the in-person DVP. The tuition will be a flat rate of $400, which will cover both the virtual and in-person components (sorry, no, they cannot be separated). Scholarships will be available, with priority placed on those in training programs in under-resourced countries. Attendance at the in-person component is limited to 40 people due to prior arrangements with the meeting hotel. Be sure to watch our social media accounts, the website events page, and of course the newsletter and listserv announcements to know when registration will begin! We are so excited to bring this new course to you."

Looking forward to seeing everyone in one of our seminars.

Kind regards,

Francisco (Paco) Uzal
Chief Executive Officer
Davis-Thompson Foundation

JVDI IN FOCUS

Our May focus is an open-access article appearing in the May issue: "**Betanodavirus meningoencephalitis in an Atlantic blue marlin**" by Kirstin A. Cook, John P. Hawke, David B. Groman, Tobia Pretto, Anna Toffan, Larry A. Hanson, D. Nguyen, Lorelei Ford, Wes A. Baumgartner.

J Vet Diagn Invest 2024;35(3). **https://journals.sagepub.com/doi/abs/10.1177/10406387231218223**

Viral nervous necrosis (viral encephalopathy and retinopathy) is caused by piscine nodavirus (*Nodaviridae, Betanodavirus*). Since 1986, this highly infectious virus has caused mass mortalities of up to 100% in farmed saltwater and freshwater fish around the world (with the exception of South America and Antarctica), affecting >60 species across 10 orders. The Atlantic blue marlin (*Makaira nigricans* Lacépède, 1802) is a top-level predator found throughout the tropical waters of the Atlantic and Indo-Pacific oceans. Despite their popularity as a sportfish, relatively little is known about the Atlantic blue marlin and other billfish. We describe here chronic betanodavirus infection in a juvenile Atlantic blue marlin, which is, to our knowledge, the first report of disease in *M. nigricans*.

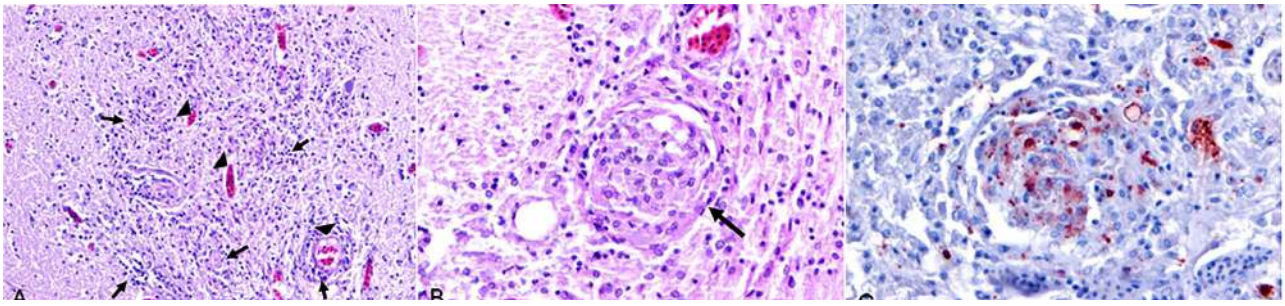


Figure 1. Betanodavirus meningoencephalitis in an Atlantic blue marlin. **A.** The neuropil is infiltrated by large numbers of macrophages (arrows); blood vessels are moderately congested (arrowheads). H&E. **B.** A granuloma (arrow) composed of concentric layers of epithelioid macrophages. H&E. **C.** The cytoplasm of several macrophages in and around a granuloma has red (positive) signal for viral antigen. Betanodavirus immunohistochemistry.

The Journal of Veterinary Diagnostic Investigation is the official journal of the American Association of Veterinary Laboratory Diagnosticians. The mission of the Journal is to educate by informing readers of progress in veterinary laboratory medicine and related fields of endeavor. The key objectives of the JVDI are to promote the science of veterinary laboratory medicine and the betterment of animal and public health. JVDI fully supports diversity, equity, and inclusion in our publishing activities.

Editor-in-chief, Dr. Grant Maxie / https://journals.sagepub.com/home/VDI

DIAGNOSTIC EXERCISE

Case #: 232; **Month:** March; **Year:** 2024

Contributors: Lauren Thielemann[1], Clinson Lui[1], Wesley McAda[2], Alexis Jennings[3], Michelle C. Coleman[4], Laura Bryan[1].
[1]School of Veterinary Medicine and Biomedical Sciences, Department of Veterinary Pathobiology, Texas A&M University
[2]McAda Veterinary Clinic, Hallettsville, Texas
[3]Boren Veterinary Medical Teaching Hospital, Oklahoma State University, Stillwater, OK USA
[4] Department of Large Animal Medicine, College of Veterinary Medicine, The University of Georgia, Athens, GA USA
Corresponding author: lbryan@cvm.tamu.edu

Clinical History: A 16-year-old paint mare presented to the Texas A&M equine emergency medicine service for chronic weight loss despite an adequate appetite, inappropriate mentation, and hair loss. Clinical signs started three months prior with a history of gradual weight loss that did not respond to prophylactic oral deworming, diet change, or dental float. The horse then began displaying signs of pruritus with self-inflicted patchy alopecia primarily localized to the pigmented skin followed by rapid epaxial and gluteal muscle atrophy and fluctuant swelling around the head and neck (Fig. 1). Palpable, firm skin nodules appeared in the cervicothoracic, pectoral, axillary, and inguinal regions (Fig. 2). The mare also began exhibiting signs of colic, decreased borborygmi and dry fecal matter. Despite an initial two-day improvement with gastric decompression, anti-inflammatory medications, and supportive care, the horse became acutely neurologic with abnormal mentation and right front limb lameness/paresis. See video: https://youtu.be/NbiVcdOOkg0

Neurologic signs significantly worsened over the next three days to pyrexia, weakened tail tone, abnormal right hindstride, generalized ataxia, head pressing, and violent outbursts. Hematology revealed a stress leukogram with a moderate mature neutrophilia (11,537 cells/µL), mild lymphopenia (695 cells/µL), and mild eosinophilia (1,390 cells/µL). Chemistry analysis revealed hyponatremia (128 mmol/L), hyperphosphatemia (5.6 mg/dl), hyperglobulinemia (4.4 g/dl), hyperbilirubinemia (4.9 mg/dl), elevated ALP (534 U/L), elevated AST (722 U/L), elevated lactic acid (32 mg/dl), and elevated creatinine kinase (1293 U/L).

DIAGNOSTIC EXERCISE

Ultrasound evaluation showed pleural effusion, a heterogenous liver, and an enlarged lymph node within the cecal band. Physical exam further revealed multifocal ulcerations on the buccal mucosal surfaces and severe muscle wasting. Due to the diffuse and severe nature of the disease and poor prognosis, the horse was humanely euthanized.

Necropsy Findings: Multiple thickened areas of alopecia with superficial crusting were distributed across the abdomen, pectoral, and inguinal areas, with preference to pigmented skin. On cut section, these thickened areas were markedly firm, mottled pale tan to red, and extended into the underlying panniculus and skeletal muscle along with expansion by edema and hemorrhage (Fig. 3). The cervical, thoracic, paraspinal, shoulder, and pectoral skeletal muscles were pale and dry (Fig. 4). Additional hemorrhage and ulcerative lesions were within the oral and urinary bladder mucosae. Diffuse lymphadenomegaly was noted throughout the body, and the retroperitoneal and peritoneal space showed marked effusion. Many firm, pale tan, rounded nodules measuring up to 1 cm diameter were found infiltrating the wall of the jejunum, omentum, and on the greater curvature of the stomach. There were 1 mm, pale tan, firm, nodules disseminated throughout the parenchyma of all liver lobes. Histologic lesions were noted in the skin, skeletal muscle (Fig. 5), liver, meninges (Fig. 6), and bone marrow (Fig. 7).

Figure 1. Alopecia on the poll

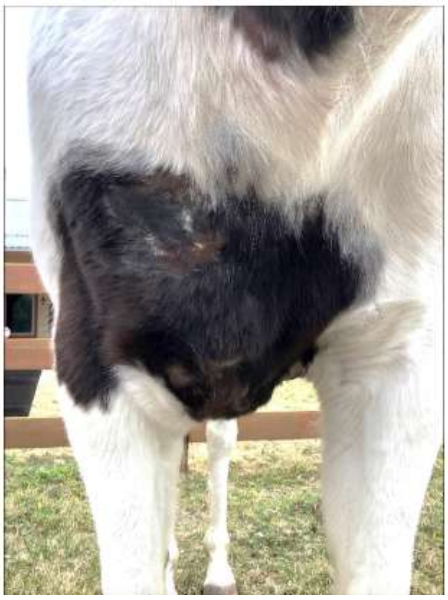


Figure 2. Abrasions and swelling in pectoral region.

DIAGNOSTIC EXERCISE

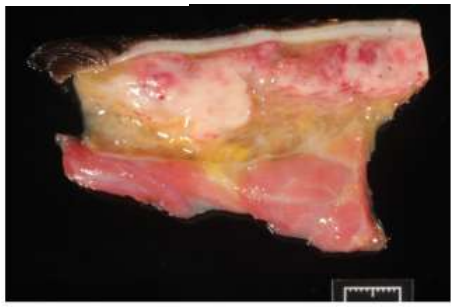


Figure 3. Multifocal tan, firm areas extend from the dermis into the subcutis.

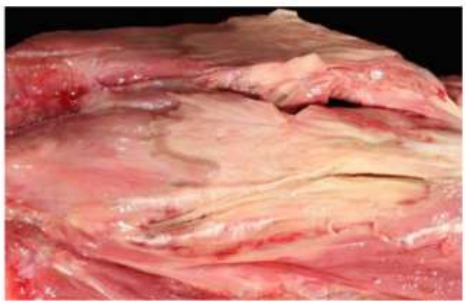


Figure 4. Multiple skeletal muscle fascicles are pale yellow and dry.

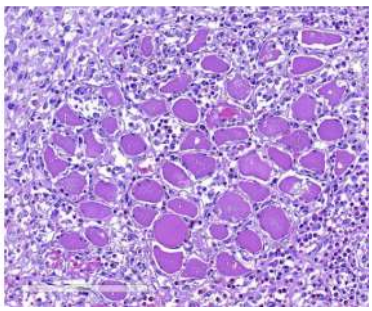


Figure 5. Skeletal muscle, H&E, 200x: myofibers are necrotic and surrounded by numerous eosinophils and macrophages.

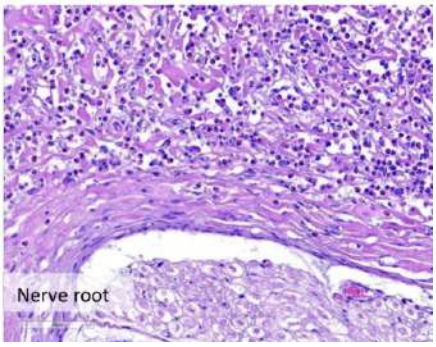


Figure 6. Spinal cord meninges, H&E, 200x: Eosinophils and macrophages are dispersed throughout the meninges and around nerve roots.

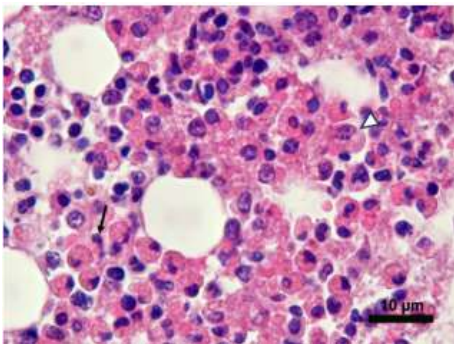


Figure 7. Bone marrow, H&E, 1000x: Numerous mature eosinophils (arrow) efface the marrow and macrophages occasionally contain erythrocytes (arrowhead).

Follow-up questions:

- Microscopic description
- Morphologic diagnosis
- Differential diagnoses
- Name of the condition
- Diagnosis

Click here for answers

Associate Editor for this Diagnostic Exercise: Raquel Rech
Editor-in-chief: Claudio Barros

SEMINAR REVIEWS

Davis-Thompson Foundation

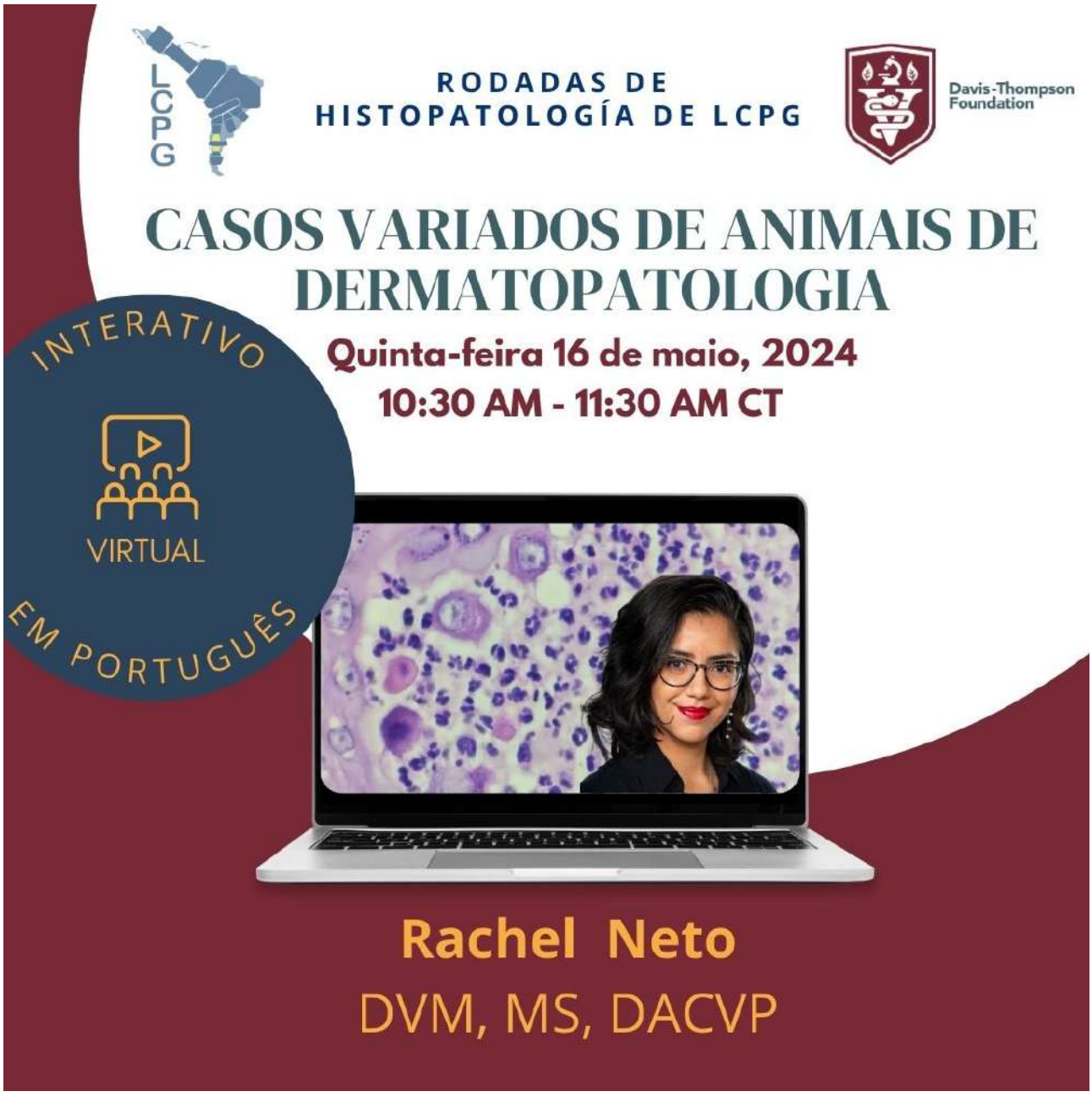
PennVet
University of Pennsylvania

Necropsy Course

On April 20-21, foreign graduate veterinarians on track to obtain their license to practice in the USA joined Dr. De Negri and Dr. Helgert for a weekend of necropsy practice in preparation for their Clinical Proficiency Exam administered by the AVMA Educational Commission for Foreign Veterinary Graduates.
This was the DTFoundation's 4th course of its kind and the first one to be held at the University of Pennsylvania School of Veterinary Medicine, New Bolton Center.

WEST COAST CONFERENCE

RODADAS DE HISTOPATOLOGIA



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EASTERN EUROPEAN MEETING

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CLIIC

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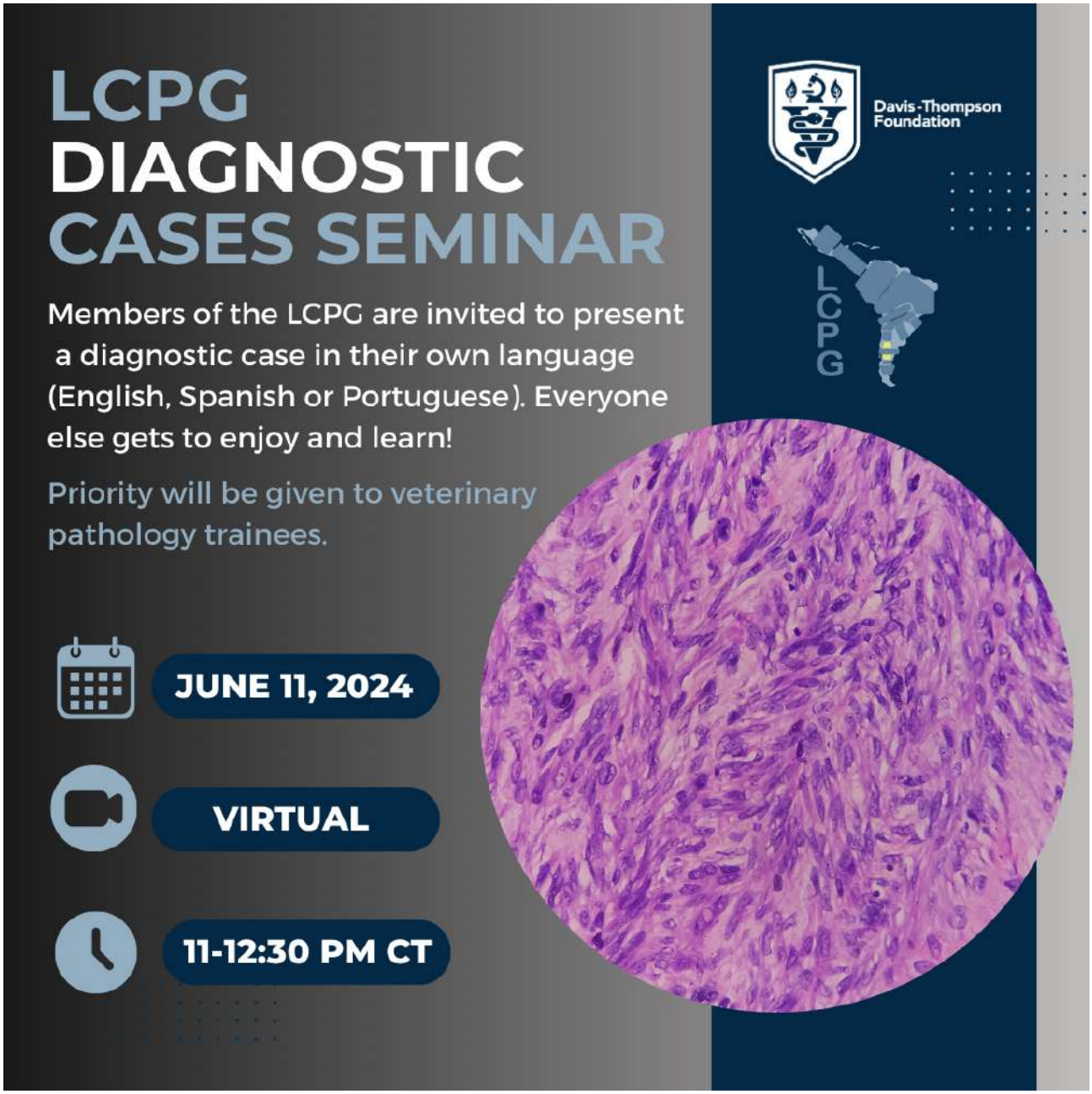
LCPG DIAGNOSTIC CASES SEMINAR



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EUROPEAN DESCRIPTIVE PATHOLOGY COURSE

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CLASS & POLA

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EUROPOLA

More information coming soon

EUROPEAN SYMPOSIUM ON DERMATOPATHOLOGY

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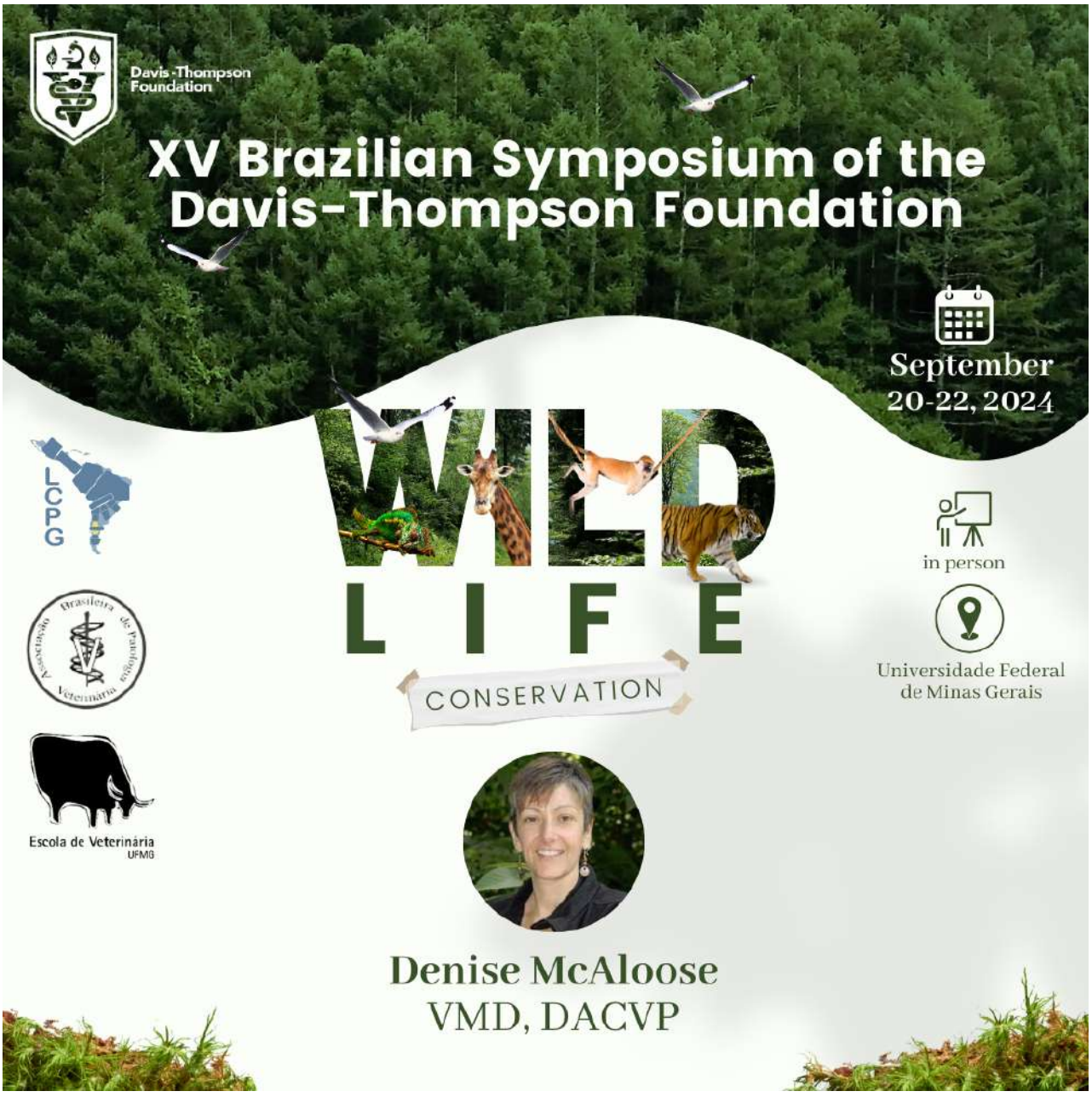
BRAZILIAN SYMPOSIUM OF THE DTF



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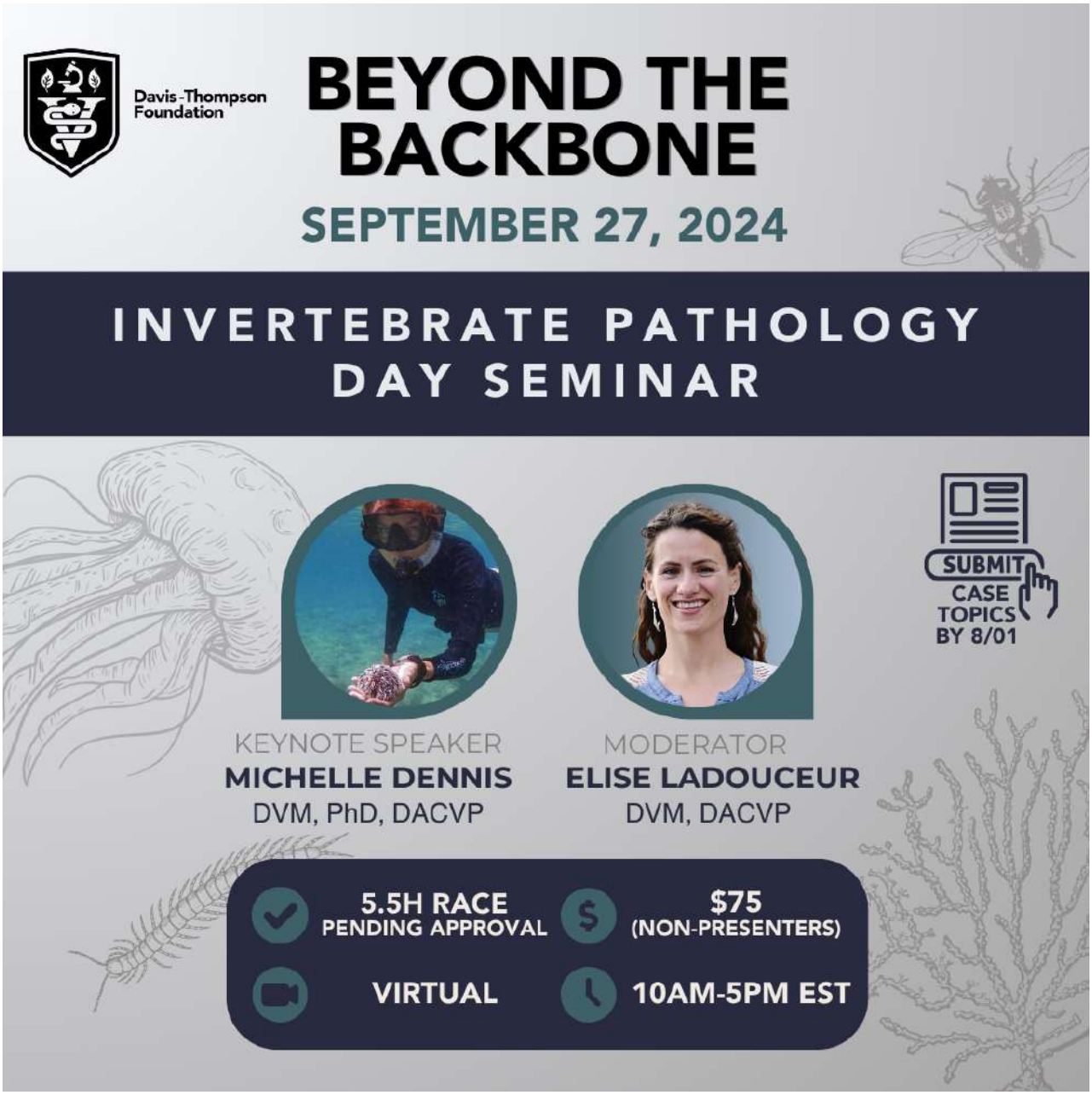
INVERTEBRATE PATHOLOGY DAY SEMINAR



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SOUTHCENTRAL DIVISION MEETING

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WESTERN ROUND ROBIN CASE

CONTRIBUTING LABORATORY:
Oregon Veterinary Diagnostic Laboratory, OSU

Clinical history: 2-day-old female, Gypsy Vanner foal with severe muscular enzyme increase and standing difficulty.

Gross findings: Skeletal muscles throughout the diaphragm (Fig. A), appendicular skeleton (Fig. B), tongue, and intercostal muscles are also mildly to moderately pale with yellow and/or white streaks. There is mild fascial edema in the proximal aspect of the left pelvic limb. The urine is light brown, and the urinary bladder is distended.

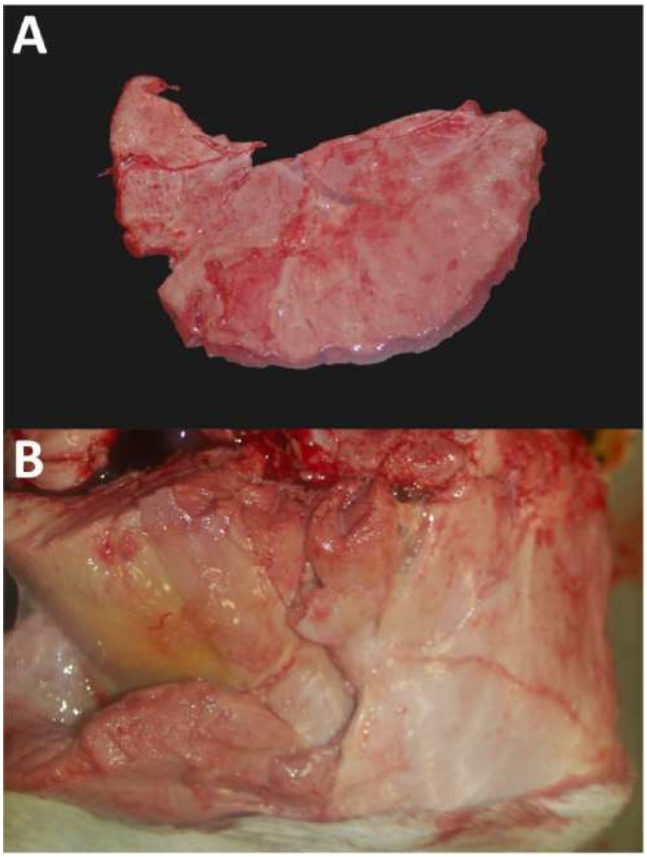
Histopathology: Skin: Skeletal muscle (thigh, diaphragm, tongue, intercostal muscle, extraocular muscle): Severe diffuse coagulative necrosis, vacuolar degeneration, and swelling of the myocytes; severe multifocal mineral deposition in the myocytes; multifocally contraction bands are hypereosinophilic, shrunken or fragmented (contraction band necrosis); myofibers replaced by karyorrhectic debris, moderate numbers of macrophages, and few fibroblasts; multifocally few myofibers have basophilic cytoplasm, infrequent rowing of the nuclei, nuclear internalization and large nuclei with prominent nucleoli (regeneration).

Diagnosis: Skeletal muscle (pelvic limb, diaphragm, tongue, intercostal muscle, and extraocular muscle): Severe,multifocal, acute to sub-acute, monophasic degeneration, necrosis and mineralization with minimal myofiber regeneration

Etiology:
- Selenium (liver): 0.23 µg/g dry. (Ref range: 0.5-2.10 µg/g dry)
- Zinc (liver): 611.21 µg/g dry. (Ref range: 90-450 µg/g dry)

Comments:
Nutritional myopathy was diagnosed in this foal based on the muscle lesions and low concentration of selenium in the liver. Nutritional myopathy usually occurs in young animals on diets deficient in selenium and/or vitamin E. The usual age range is 1 day to 12 weeks in horses, and it may be present at birth.

WESTERN ROUND ROBIN CASE



However, in severely selenium-deficient areas, such as the Pacific Northwest, selenium deficiency myopathy can occur in horses of any age, and selenium supplements to the animal's ration must be provided. Vitamin E and selenium is an important antioxidant. Silver, copper, cadmium, cobalt, vanadium, tellurium, zinc, and possibly other metals can induce an increased requirement for vitamin E and/or selenium, in some way binding selenium or preventing its participation in free radical protective activities. In the current case, excess zinc may lead to greater demand for selenium and/or vitamin E.

The most severely affected muscles are those that have the highest workload including cardiomyocytes. In foals with severe, acute myopathy leading to death or euthanasia, lesions are at the stage of massive muscle necrosis and mineralization with minimal macrophage infiltration (monophasic). In animals that have lived longer, the lesions are polyphasic, and active necrosis, macrophage infiltration, and regeneration are present. In the current case, it is thought that the demand for antioxidants increased rapidly, mainly in the muscles of the thigh and respiratory muscles, which required a rapid workload after birth and showed severe muscle necrosis rather than the tongue.

The diagnosis of nutritional myopathy is confirmed by detecting deficient concentrations of selenium or vitamin E in the blood of live animals or liver samples obtained at necropsy. Differentials for muscle necrosis in horses include white muscle disease, capture myopathy, exertional rhabdomyolysis, toxic myopathy due to toxic plants and ionophores, polysaccharide storage myopathy, and glycogen branching enzyme defect. Nutritional myopathy typically shows multiple polyphasic muscle necrosis, but it should be noted that nutritional myopathy can show monophasic necrosis as well in an acute onset.

WESTERN ROUND ROBIN CASE

References

1. Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, vol 1 6th ed. Elsevier Limited, Philadelphia,PA

2. Pathologic Basis of Veterinary Disease, 7th ed. Elsevier Limited, Philadelphia, PA

Submitters: Drs. Mari Inohana and Duncan Russell

Click here to see this slide in Noah´s slidebox

NOAH'S SLIDEBOX

BSTP CORNER

BRITISH SOCIETY OF TOXICOLOGICAL PATHOLOGY

Notice of Future Meetings

Virtual Continuing Education Symposium 9: Digestive System
20th – 29th February 2024
Tuesday, Wednesday and Thursday
13.00 – 17.00 (GMT+0, London/UCT+0/ET-5)

CES 9 will be held over two weeks - on the afternoons of Tuesday 20th, Wednesday 21st, Thursday 22nd, Tuesday 27th, Wednesday 28th and Thursday 29th February 2024, from 13.00 – 17.00 (GMT+0, London/UCT+0) each day.

REGISTRATION IS NOW OPEN WITH AN EARLY BIRD DEADLINE OF FRIDAY 26th JANUARY 2024

This CES will give you the opportunity to have an overview of the normal anatomy and physiology of the digestive system; repair and regeneration mechanisms; spontaneous lesions of the rodent, rabbit, and non-human primate GI tract; toxicology and carcinogenesis of the exocrine pancreas; health monitoring of laboratory rodent colonies; pathology of infectious GI diseases of rodents, rabbits and non-human primates; anatomy, physiology, histology and pathology of the teeth. Other topics to be covered include spontaneous pathology and infectious disease in the canine and minipig digestive system; rodent models of inflammatory bowel disease; from biomarkers to AI; bioaccumulation of therapeutic drugs.

Reduced fee funding opportunities are also available for trainee/early career pathologists as well as a number of free registration bursary places.

If you would like further information, have any queries, or would like to reserve a place, please contact the Hg3 Conferences Ltd - events@hg3.co.uk

This symposium will be organised by Hg3 Conferences Ltd, who have been appointed by the Council of the BSTP to take over the administrative organisation of all BSTP events – events@hg3.co.uk

or visit: **https://www.bstp.org.uk/events/ces-9-digestive-system/**

Virtual Continuing Education Symposium 10: Urinary System
9th – 18th July 2024
Tuesday, Wednesday and Thursday
13.00 – 17.00 (GMT+1, London/UCT+1)

CES 10 will be held over two weeks – on the afternoons of Tuesday 9th, Wednesday 10th, Thursday 11th, Tuesday 16th, Wednesday 17th and Thursday 18th July 2024, from 13.00 – 17.00 (GMT+1, London/UCT+1) each day.

This CES will give you the opportunity to learn about the urinary system. There will also be roundtable/share knowledge discussions and questions.

Updated information about this symposium will be posted on the BSTP website and BSTP group LinkedIn pages as it becomes available.

If you would like further information, have any queries or would like to reserve a place, please contact the Hg3 Conferences Ltd - events@hg3.co.uk

This symposium will be organised by HG3 Conferences Ltd, who have been appointed by the Council of the BSTP to take over the administrative organisation of all BSTP events – events@hg3.co.uk

or visit: **https://www.bstp.org.uk/events/ces-10-urinary-system/**

For registration and more information about the events, visit the BSTP website:
https://www.bstp.org.uk/events/bstp-events/

IDEXX CASECONNEXX CORNER

Signalment: 8-year-old, male, neutered, Papillon dog

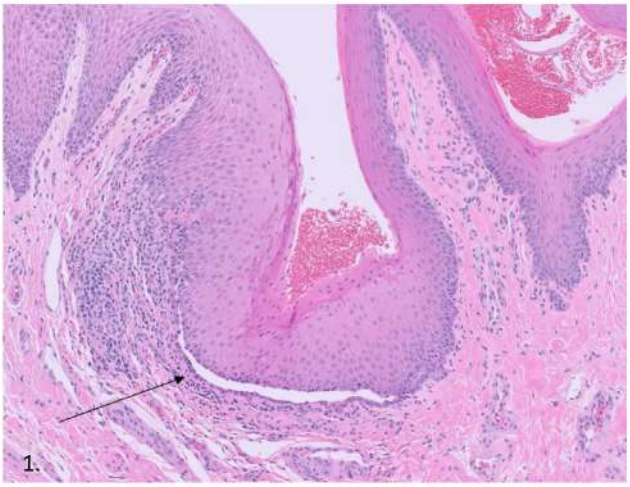
Source/ History: Patient presented for drooling. Severe ulceration of tongue (particularly affecting the ventral surfaces), bilateral mandibular lymphadenopathy, and moderate dental calculus noted during clinical examination.

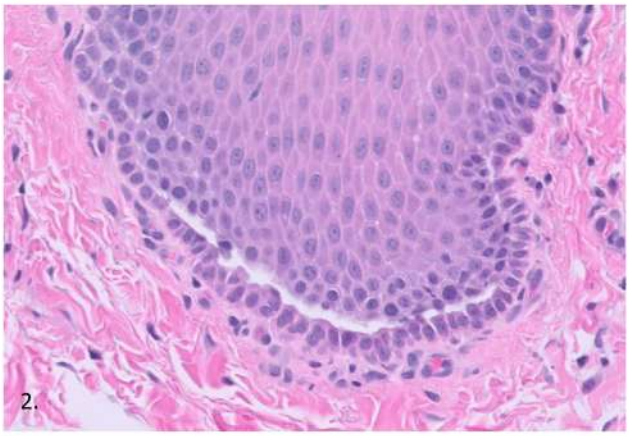
Histopathologic Description:
Oral mucosa, tongue: The stratified squamous mucosal epithelium of the tongue demonstrates variable hyperplasia, often with a complex papillary histoarchitecture, and multifocal erosion and ulceration. Frequently, there are areas of suprabasal clefting/separation, accompanied by variably severe mucosal epithelial edema (spongiosis), mild to marked mixed inflammatory leukocyte exocytosis and submucosal mixed inflammation. In areas of suprabasal clefting and mucosal erosion/ulceration, there is often preservation of basal layer epithelial cells, with a characteristic "tombstone" architecture. There are multifocal random scattered or occasionally clustered foci of apoptotic mucosal epithelial cells.

Interpretation:
Oral mucosa, tongue: Erosive to ulcerative glossitis / stomatitis, with suprabasal clefting and pleocellular inflammation, consistent with pemphigus vulgaris

Comments:
Pemphigus vulgaris (PV) is a rare and severe vesiculobullous and ulcerative autoimmune disease of the dog and cat and is characterized by autoimmune targeting of desmoglein-3 (strongly expressed in the suprabasal keratinocytes of the oral mucosa). German Shepherd dogs and Collies may be over-represented, with a median age of onset of 6 years; although, disease in dogs less than 1 year of age has been reported. In dogs, there appears to be a slight male predilection, where as age, breed, or sex predilections have not been noted in cats. Most canine and feline cases have lesions affecting the oral cavity and mucocutaneous junctions, characterized by coalescing ulcers on the tongue, palate and gingiva, cases of mucosal PV may evolve to a mucocutaneous phenotype. Phenotypes that also affect haired skin may target alternative desmosomal cadherin antigens such as desmoglein-1. Progression of clinical phenotype may be a result of epitope spreading in which previously sequestered antigens are exposed by the initial autoimmune inflammatory process. A variant affecting only the nasal planum has been reported. Cases of drug induced PV are described in man and have been observed sporadically in the companion dog (e.g. sulfasalazine therapy).

References: Gross, Thelma Lee, et al. (2005), Skin diseases of the dog and cat: clinical and histopathologic diagnosis, 32-35





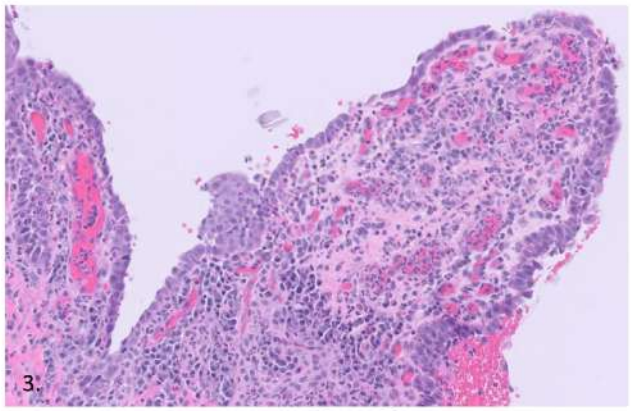


Figure 1. Hyperplastic mucosal epithelium with prominent suprabasal clefts (black arrow) **Figure 2.** Higher power image of suprabasal clefts resulting in abrupt separation of stratum spinosum from the stratum basale. **Figure 3.** Multifocal regions of erosion and ulceration, often lined by a single remnant layer of "tombstoned" stratum basale epithelial cells. Mixed inflammation, edema, and neovascularization in the subepithelial stroma.

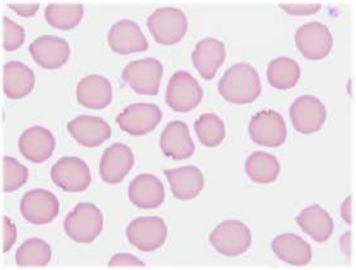
Case by:
Luke Haydock BVSc (Dist.) DVSc DACVP

IDEXX

CaseConnexx
Visit CaseConnexx.com to Register

ACVP ERRORS IN PATHOLOGY

Have you seen a Pathology Error published in a scientific journal?



Help us determine the extent of the problem

Submit to the ACVP Errors in Publications Web Portal

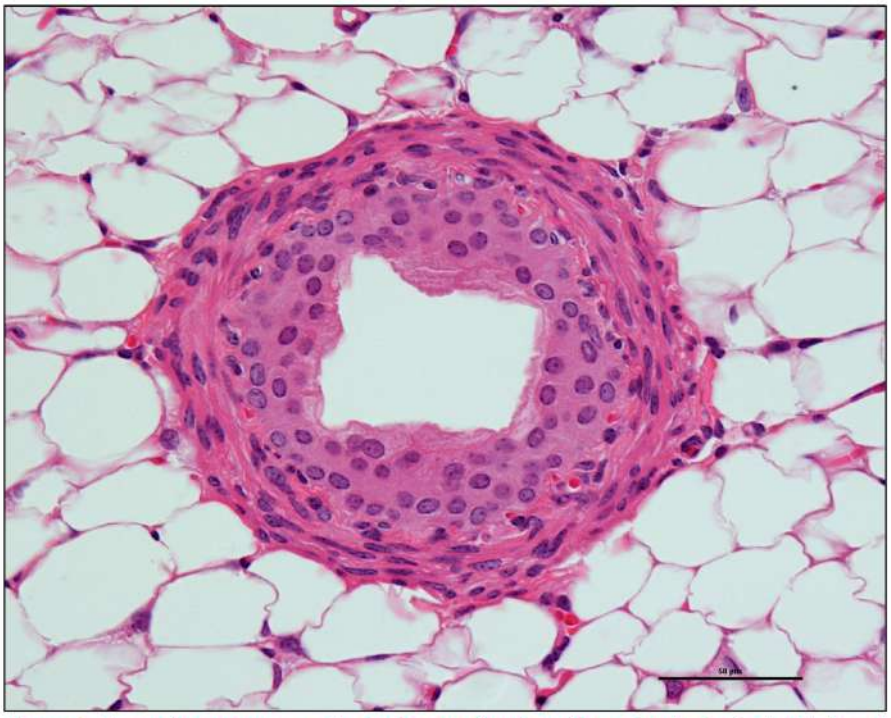


Figure 1. Tissue from a mouse. *The **tunica media** of **medium-sized arteries** is thickened by concentric rings of excessive smooth muscle that is characteristic of **hypertensive arteriosclerosis****

*This is a **hypothetical example** of a pathology error which **misdiagnoses** a normal microanatomic/histologic structure (**mouse ureter**) as a lesion. Errors, omissions, or "false claims" in pathology data such as this are becoming **increasingly common in scientific journals**, especially those that **do not routinely include pathologists** in their peer review process. The ACVP Advocacy and Policy Committee and Board of Directors have therefore developed a **web submission portal** that will allow professionals with advanced pathology training to report concerns on substantiative errors in publications.

We need your help!

When you come across a pathology error published in a scientific journal, please take 5-10 minutes to fill out the anonymous survey on the **ACVP Errors in Publication web submission portal**. The information gathered from submissions to this web portal will be collated and used to compile the data needed to develop and promote publication standards and review procedures to improve the accuracy of published pathology data. Any data obtained will be maintained strictly in **anonymous** form and will greatly benefit the advancement of this initiative.

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ASVP CORNER

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EARLY BIRD DISCOUNTS AVAILABLE!

13-15 SEPTEMBER, 2024

HOTEL GRAND CHANCELLOR
ADELAIDE, SA

2024

ASVP & DTF SCIENTIFIC CONFERENCE

THE URINARY SYSTEM

We are delighted to invite you to attend the ASVP & Davis-Thompson Foundation 2024 Scientific Conference at the Hotel Grand Chancellor, Adelaide, on 13-15 September 2024.

This year's theme is the Urinary System and we are excited to announce the engagement of high-calibre international and national speakers that cover the specialities of internal medicine, clinical pathology and anatomical pathology.

MARY NABITY
Keynote Speaker
Associate Professor
Texas A&M University

RACHEL CIANCIOLO
Keynote Speaker
Anatomic Pathologist
Zoetis Inc.

MARY THOMPSON
Keynote Speaker
Associate Professor in Small Animal Medicine, The University of Sydney

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- an outstanding scientific program
- a diversity of delegates
- a supportive environment for members, students and early career pathologists to present and learn from colleagues
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- social dinner with colleagues
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A selection of cases from the March 2024 edition of *The Scope*, the newsletter of the Australian Society for Veterinary Pathology.

Outbreak of meningoencephalitis in calves caused by bovine herpesvirus 5

Significant calf deaths occurred in a mob of 50 Angus heifers located in the Central West region of NSW. The owner had been finding 2-3 week old calves dead in the paddock. Those seen alive before death reportedly showed neurological signs including lethargy, panting, hypersalivation, circling and seizures. Twenty calves had died at the time of submission of samples from one calf.

Histopathology revealed a non-suppurative meningoencephalitis, which was most severe in the rostral cerebrum (Figure 1). There was associated multifocal neuronal necrosis. Bovine herpesvirus 5 infection typically preferentially affects the rostral cerebrum. Infectious bovine rhinotracheitis qPCR on fresh brain was positive. The virus was typed as BoHV-5.

BoHV-5 is antigenically related to BoHV-1 (the cause of infectious bovine rhinotracheitis). Both viruses are detected by the IBR real time PCR, although BoHV-1 rarely causes encephalitis . BoHV-5 encephalitis occurs as a sporadic disease or sometimes as outbreaks in calves and yearlings (Favier et al, 2012). Despite the virus having been initially described in Australia (Johnston et al, 1962), we diagnose it relatively uncommonly in cattle in NSW.

Calodium hepaticum granulomatous hepatitis in a black and white ruffed lemur

Chronic liver disease was identified in a black and white ruffed lemur in a zoo. It had been a poor doer all of its life with worsening weight loss over one month. An abdominal aspirate produced bright yellow clear fluid with a SG of 1.020.

The lemur was euthanased and necropsy examination revealed widespread multifocal, irregular, coalescing white sunken areas through the parenchyma of the liver, visible through the capsule. The intervening parenchyma was yellow and raised above the

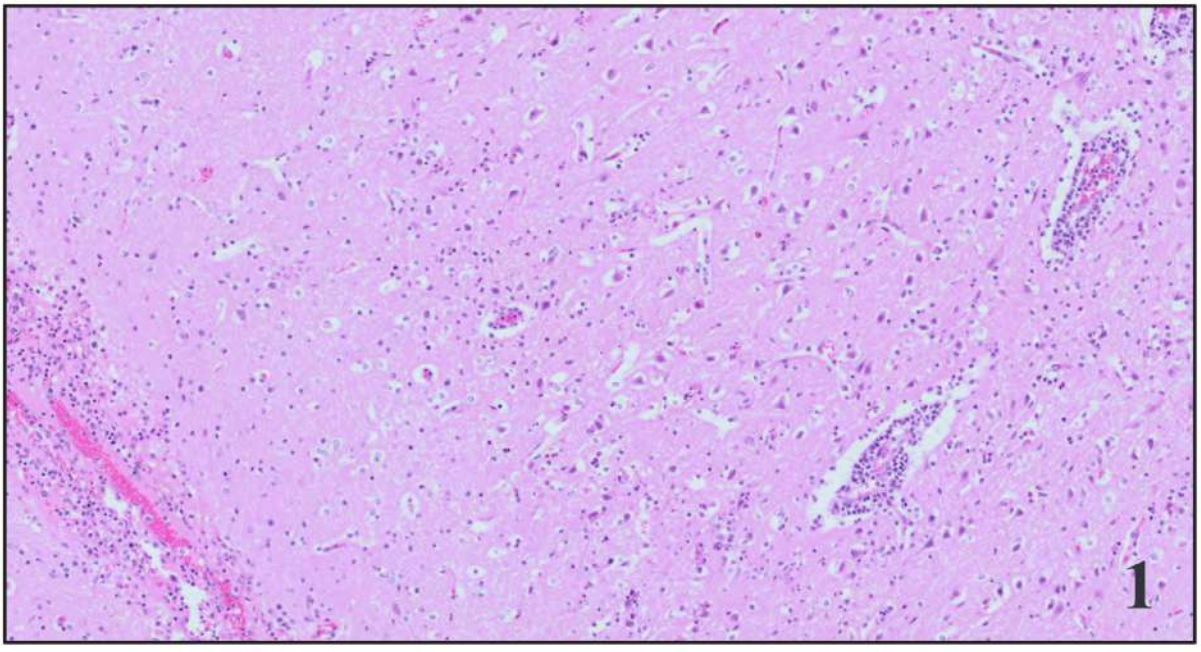

Figure 1: Bovine calf. Rostral cerebrum. BoHV-5 associated meningoencephalitis, non-suppurative, acute, severe with multifocal neuronal and glial cell necrosis and satellitosis. HE.

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pale foci (Figure 2). A provisional diagnosis of chronic inflammation, primary or secondary neoplasia was made.

Microscopically, the white foci seen grossly were multifocal random extensive areas of macrophage and multinucleate giant cell infiltration with fewer focal areas of PMNs and fibrous encapsulation with accompanying lymphoid infiltrates (Figure 3). Giant cells were organised around thick walled approx 50 x 20µm ova, each with bipolar plugs and a multinucleate zygote. Ova capsules had two layers, the outer layer with prominent radial striations (Figure 4). Hepatocellular parenchyma was nodular (hyperplasia) and many hepatocytes contained both intracytoplasmic lightly eosinophilic sharp-edged inclusions (apoptotic bodies or ingested plasma) to about 20 µm and hypereosinophilic hyaline inclusions to 20µm. Many hepatocytes also contained microvesicular lipid like vacuoles concurrently with the other inclusions. Some nuclei contained occasional large clear vacuoles and/or smaller eosinophilic round inclusions (cytoplasmic intrusions). Numerous canalicular bile plugs were present and globules of bile-like material

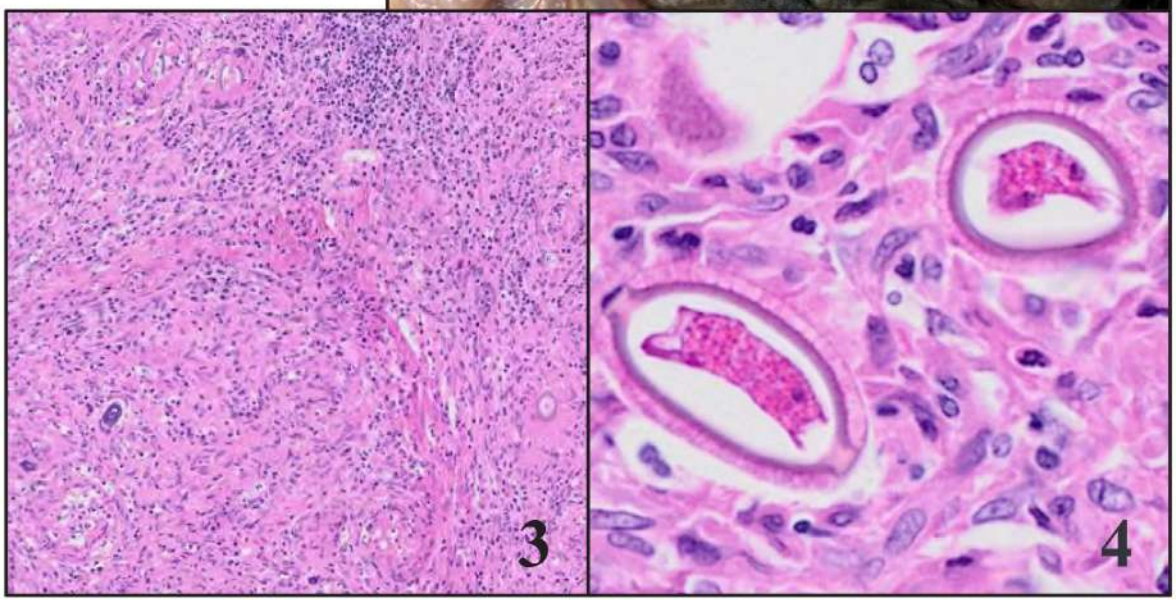

Figure 2: Lemur. Periotoneum. The liver is enlarged with pale yellow discolouration and multifocal to coalescing firm white nodules, interpreted as multifocal granulomatous hepatopathy.

Figure 3: Lemur. Liver. Hepatic necrosis and granulomatous inflammation with fibrosis. HE.

Figure 4: Lemur. Liver. Granulomatous hepatitis with intralesional ova. The ova have bipolar plugs and a bilayered outer capsule with prominent striations. HE.

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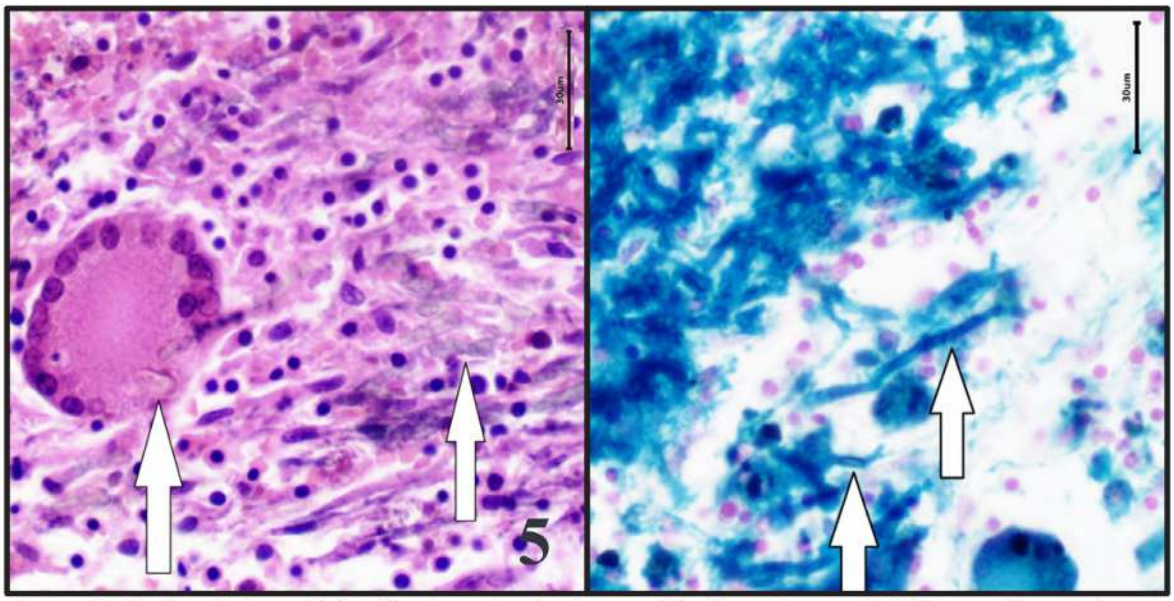

Figure 5: Canine. Spleen. Refractile elongate structures within the spleen and within a multinucleate giant cell. HE. Bar 30µm.

Figure 6: Canine. Spleen. Gamna-Gandy bodies stain blue, indicating iron content. Perls Prussian blue. Bar 30µm.

The trichurid ova were distinctive enough to allow identification as *Calodium hepaticum* (*Capillaria hepatica*) even in the absence of adult worms. Adult worms are short lived but lay many eggs in their travels through the liver. *Calodium hepaticum* has a unique lifecycle where the infected animal has to be eaten or die and decompose in order to liberate the eggs from the liver when they need from 6 weeks to 5 months in an oxygenated damp environment to embryonate and become infective. Rats are the definitive host and cannibalism the most common means of sustaining the life cycle (Fuehrer, 2014).

Gamna-Gandy bodies: a potential histopathology pitfall

Structures resembling fungal hyphae, later deemed to be Gamna-Gandy bodies, were detected in the histopathology sections of a spleen belonging to a 10-year-old Staffordshire bull terrier cross. Accurate identification of pseudo-fungi in histology specimens is imperative to avoid unnecessary treatment.

Histopathology confirmed a splenic haematoma with mild, multifocal, chronic haematopoietic nodular hyperplasia and mild, focal, chronic lymphoid nodular hyperplasia. However, multifocally, within the trabeculae and red pulp cords, structures resembling fungal hyphae were found. These structures were approximately 20x3 micrometres in dimension, moderately refractile, sometimes branching and occasionally surrounded by inflammatory cells (Figure 5). The structures were negative for Grocott's methenamine silver (GMS) stain and positive for Perl's Prussian blue stain (Figure 6) and were suspected to be Gamna-Gandy bodies.

Gamna-Gandy bodies are collagenous fibres encrusted with iron and calcium deposits (Ryseff et al., 2014). They are usually encountered within the spleen, however, they may also be found in other tissues such as the lymph node, or within tumours (Moore et al., 2017). Gamna-Gandy bodies are also referred to as pseudo-fungi due to their resemblance to true fungal elements microscopically (Lyapichev et al., 2016). Gamna-Gandy bodies frequently stain strongly to iron sensitive stains such as Perl's Prussian blue and variably to calcium stains such as Alizarin red S and von Kossa (Lyapichev et al., 2016). These histochemical stains can aid in differentiation of Gamna-Gandy bodies from true fungi which are negative for Perl's

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Prussian blue, von Kossa, Alizarin red S and positive for Grocott's methenamine silver (Lyapichev et al., 2016).

As pathological disorders of the spleen frequently result in congestion and haemorrhage, it is useful to remember that excessive iron deposition on collagenous fibers within the spleen could result in Gamna-Gandy bodies on histology specimens.

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Acknowledgements

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The ASVP thanks the Davis-Thompson Foundation for the opportunity to share casework.

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Paco Uzal appointed "Academic in the United States" by the ANAV

by Federico Giannitti and Leonardo Minatel

The one and only "Paco Uzal" was appointed "Academic in the United States" by the National Academy of Agronomy and Veterinary of Argentina (ANAV) last April 19.

At the inauguration ceremony, Paco gave a lecture titled "40 years around the world; the journey of a veterinary pathologist", which was broadcasted via Zoom and can be watched on youtube: https://www.youtube.com/watch?v=yih6tE1w4SA

Congratulations on this achievement Paco!!

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Davis-Thompson Foundation

Seminar Series in Spanish 2024

seminarios en español 11:00am-12:30pm CDT

ABR 17 COMPLETED

Neoplasias de mastocitos
Valeria Grieco, DVM, PhD

JUN 20

Fotografia macroscopica en la sala de necropsias
Lluís Lujan, DVM, PhD, Dipl. ECVP

AGOS 15

Patologia forense veterinaria: Generalidades y el caso de la asfixia
Carlos Gonzalez, MV, MPhil, PhD

SEPT 12

Sarcomas de tejidos blandos: del patólogo al oncólogo
Federico Cifuentes, MV, MSc, DrCs, DACVP
Rodrigo Crossley, MV, MS

NOV 28

Casos interactivos de dermatopatología
Joaquin Ortega, DVM, PhD, DACVP

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2024 LCPG & DTF ACTIVITIES IN LATIN AMERICA

Country	Name of Seminar	Dates	Place/University	Speakers	Organizers
Argentina	XVIII Seminar of the Argentinean Subdivision of the Davis-Thompson Foundation and XII Forum on Teaching Veterinary Pathology.	August	Rosario, Argentina. Facultad de Ciencias Veterinarias, Universidad Nacional de Rosario	TBD	Leonardo Minatel
	Latin American roadshow: Gastrointestinal pathology	Oct 24-25	Buenos Aires, Argentina. Universidad de Buenos Aires.	Francisco Uzal	Leonado Minatel
Brazil	2024 Brazilian Symposium of the DTF - Pathology of zoo and wildlife	September 20-22	Universidade Federal de Minas Gerais - Belo Horizonte, MG, Brazil	Denise McAloose	Renato de Lima Santos / Ayisa Rodrigues de Oliveira
Chile	Pathology of wildlife	August 8-9	Valdivia, Chile. Universidad Austral de Chile	Mauricio Seguel, Enrique Paredes, Mauricio Navarro, Manuel Moroni.	Mauricio Navarro
Colombia	Latin American roadshow: Gastrointestinal pathology	Nov 1-2	Barranquilla, Colombia. Universidad San Martin	Francisco Uzal	Paola Barato
Costa Rica	Workshop in freshwater fish medicine and pathology in Latin America	Mar 22-23	San Jose, Costa Rica. Escuela de medicina y cirugia veterinaria San Francisco de Asís	Esteban Soto, Paola Barato	Roberto Olivares
Guatemala	Latin American roadshow: Gastrointestinal pathology	Nov 4-5	Ciudad de Guatemala, Guatemala. Universidad de San Carlos.	Francisco Uzal	Deborah Rodriguez
México	IV on-line necropsy course	Apr 8-19	México (On-line)	Elizabeth Rodriguez, Maria del Carmen Carmona, Alfredo Perez, Mario Bedolla, Carlos Gonzalez, Elizabeth Morales, Gerardo Salas, Mireya Juarez, Luis Garcia-Marquez, Diana Galvan, Ruben Lopez, Laura Romero, Francisco Carvallo.	Ruben Lopez
	V seminar of the Mexican subdivision of the Davis-Thompson Foundation	September	Tamaulipas, México. Universidad Autónoma de Tamaulipas	TBD	Ubicelio Martin
	Workshop in freshwater fish medicine and pathology in Latin America	November 21-22	Faculta de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de Mexico.	Esteban Soto, Paola Barato	Ruben Lopez
Paraguay	Latin American roadshow: Gastrointestinal pathology	Oct 28-29	Asunción, Paraguay. Universidad Nacional de Asunción.	Francisco Uzal	Leila Maidana, Mirtha Suarez
Uruguay	Latin American roadshow: Gastrointestinal pathology	Oct 21-22	Montevideo, Uruguay. Universidad de la Republica.	Francisco Uzal	Jose Manuel Verdes
Venezuela	II Seminar of the Venezuelan Subdivision of the Davis-Thompson Foundation	July	TBD	Francisco Uzal	Yaritza Salas

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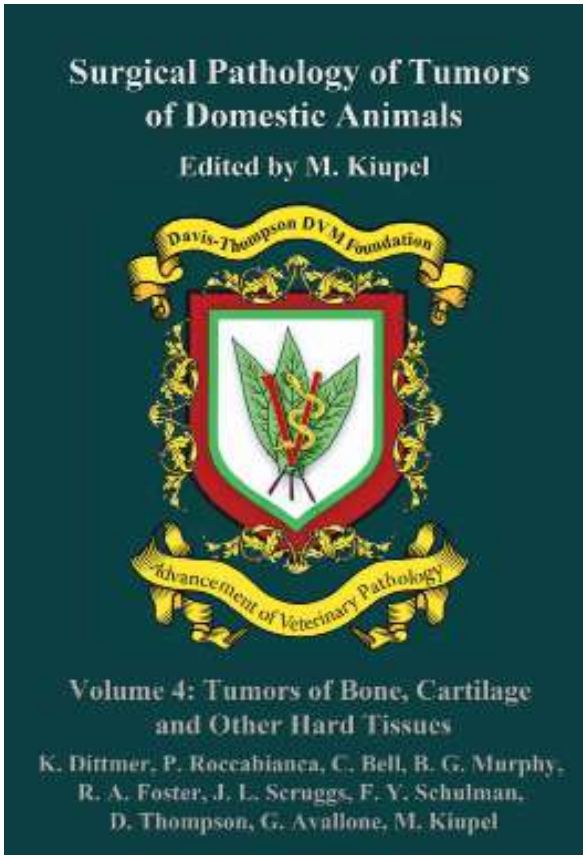
MISCELLANEOUS ANNOUNCEMENTS



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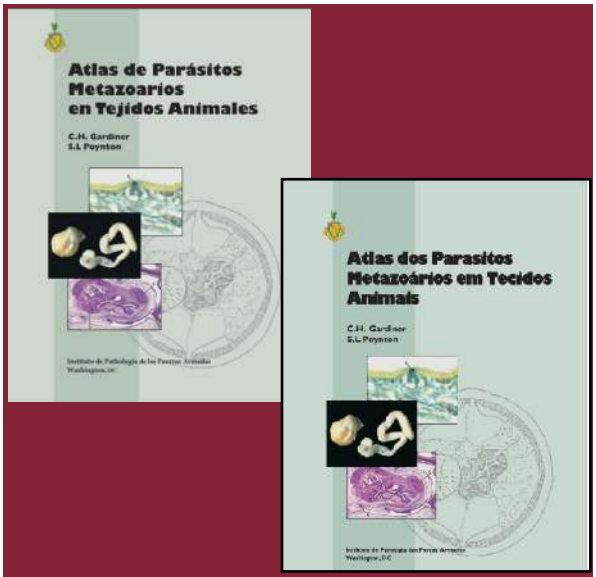
Davis-Thompson Foundation Pathology Externship

Since 1980, the Davis-Thompson Foundation lab sites have hosted more than 125 veterinary students at 8 participating diagnostic laboratories. These students usually have a strong interest in pathology itself or zoo or poultry medicine that require a strong pathology background. The Foundation is always interested in having veterinary students apply for an externship and we would like to add more externship sites that do not usually have veterinary students, to help increase their interest and knowledge of pathology with some off-campus experience. For more information, contact Dr. Jim Britt, **jobritt@sbcglobal.net;** 501-912-1449.



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