



Explore



Virginia Animal Diagnostics Newsletter - May 2021

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Fecal egg count (FEC) in dogs. Is it important?

Fecal egg count (FEC) is a simple procedure and an important tool used in parasite control that allows veterinarians to estimate the number of parasite eggs per gram (epg) of feces. The most common technique used to estimate the FEC is the McMaster test. FEC has many uses in parasite control programs. In horses, for example, the American Association of Equine Practitioners (AAEP) recommends deworming horses according to their egg shedding potential. In small ruminants, FEC is a very useful tool for genetic selection based on the animals' capacity to resist worms. In general, FEC is a widely used tool by large animal veterinarians to monitor pasture contamination and assess anthelmintic efficacy. Although anthelmintic resistance can be detected by molecular testing, the most practical way is performing a fecal egg count reduction test (FECRT), which compares FECs before and after a dewormer is used. According to the percentage of reduction, the veterinarian can conclude if the anthelmintic is effective or if anthelmintic resistance is present. Since the FECRT is based on FECs, results are an estimate and therefore, they are not completely accurate.

But, what about dogs? Is FEC useful in dogs? For the last few years, it has been suggested that some hookworm strains were persistent after anthelmintic treatment. Dealing with recurrent cases of

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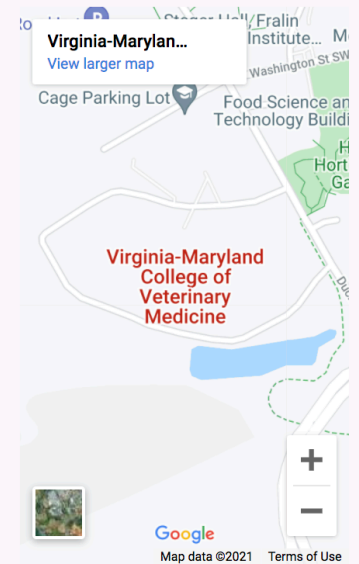
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hookworm infections after treatment is nothing new, especially because of the well documented phenomenon of “larval leak” of *Ancylostoma caninum*, where arrested somatic larvae reactivate their development and migrate to the small intestine and become adults. Every time a clinician deals with a recurrent case of hookworm infection, they first need to rule out larval leakage. However, there is a real problem with multi-drug resistant (MDR) hookworms, that apparently originated in greyhound breeding farms and in racing kennels. The real extent of the problem is unknown so far, but it has been suggested that MDR hookworms could be spreading faster than once thought because many cases have now been diagnosed in breeds other than greyhounds. If a clinician suspects an anthelmintic resistant strain of hookworm, FECRT is the most practical approach. Since the minimum prepatent period of *A. caninum* is 14 days, the post-treatment sample has to be collected between 10 and 14 days after treatment. However, since only 1 dog is usually tested, the interpretation of the FECRT results must be done much more carefully than in large animals. This cannot be stressed enough. In order to reduce variability and increase accuracy, two separate FECs on each of the pretreatment and post-treatment samples is recommended. The interpretation of FECRT for hookworms in dogs has been suggested as follows:

PERCENTAGE	INDICATION
>95% reduction	suggestive of effective treatment (larval leakage is likely the cause of persistent egg shedding)
90%-95% reduction	suggestive of reduced efficacy (persistent egg shedding could be due to either resistance or larval leakage)
75%-89% reduction	suggestive of resistance (larval leakage is unlikely to be the cause of persistent egg shedding)
<75% reduction	indicative of resistance (larval leakage is highly unlikely to be the cause of persistent egg shedding)

Although the McMaster technique can be used, the most efficient, sensitive, and accurate method to evaluate anthelmintic efficacy in

dogs is the mini-FLOTAC technique. Mini-FLOTAC allows the simultaneous diagnosis of eggs, larvae, oocysts, and cysts. The most important difference between mini-FLOTAC and McMaster is in the analytical sensitivity: whereas the sensitivity of McMaster is 50 epg, the sensitivity of mini-FLOTAC is 5 epg. If in-clinic testing is not an option, a specialized laboratory should be contacted. This test will soon be offered to clinicians in the state through ViTALS.

Roger Ramírez-Barrios, DVM, PhD, Parasitology Section Head
ViTALS

The Office of Laboratory Services welcomes the new program manager!



Dr. Jessica Walters is the Program Manager for the Virginia Department of Agriculture and Consumer Services' (VDACS) Office of Laboratory Services. She oversees all four of the veterinary diagnostic labs in the Commonwealth. Dr. Walters is a boarded poultry veterinarian who previously served both as the Poultry Diagnostician for VDACS and as the Director of both the Harrisonburg

Regional Animal Health Laboratory and Lynchburg Regional Animal Health Laboratory for the Office of Laboratory Services. She is also an Official State Agent representative for the National Poultry Improvement Plan. She received her PhD in poultry infectious diseases in 2014, and her DVM in food animal medicine in 2016- both from the Virginia-Maryland College of Veterinary Medicine. She is a Diplomate of the American College of Poultry Veterinarians. Dr. Walters is a native of Powhatan, Virginia with an agricultural background working with horses and beef cattle, but she became interested in poultry production and health while obtaining an undergraduate degree at Virginia Tech. In her current and previous roles, she enjoys management, participating in the education of backyard producers, teaching students about poultry medicine, and working hands-on with the commercial industry. She has a passion for flock health, production management, and disease diagnostics. In her free time, Dr. Walters enjoys international volunteer work, horseback riding, hiking, and traveling. She and her husband

welcomed their daughter Sara Beth in March of 2020. They, along with their menagerie of animals reside in Rockingham County.

Equine and Camelids:



Streptococcus equi ssp equi (Strangles) outbreak

In late 2020, a barn of approximately 70 horses had an isolated case of Strangles. The barn was self-quarantined (voluntarily by the barn manager; Strangles is not a “regulatory” disease, and is thus not subject to an official quarantine by VDACS) and following established AAEP guidelines of vaccination, all horses received two doses of the intranasal *Streptococcus equi ssp equi* (Strangles) vaccine. The barn was able to open again with no issues.

A few months following the initial self-imposed quarantine, an adult horse (horse 1) developed mild nasal discharge and cultured positive for *Streptococcus equi ssp equi*. The barn was again self-quarantined and staff began monitoring all horses for temperatures and/or clinical signs. Any horse with a temperature or clinical signs was isolated, tested and treated. Approximately 24 horses were identified as positive for *S. equi ssp equi*. Most horses had minimal to mild signs of a temperature and/or nasal discharge with only rare horses developing swelling of the lymph nodes. Amongst the affected horses was a 19-year-old, female horse (horse 2) that was bought at a sale prior to the outbreak. The horse was supposed to have been quarantined for 2 weeks upon arrival to the farm, but was instead placed with other horses, including horse 1, after only 4-5 days following arrival to the farm. During the outbreak, this horse was tested weekly for *S. equi ssp equi* by PCR and culture.

Test 1: PCR +, culture +

Test 2: PCR -, no culture

Test 3: PCR +, no culture

Test 4: PCR +, culture +

Test 5: PCR +, culture -

After obtaining the information regarding a break in proper quarantine protocols, it was determined that horse 2 was a carrier for *S. equi* ssp *equi* and likely initiated the strangles outbreak. The horse was eventually euthanized due to continued clinical signs and positive PCR/culture results, despite treatment. At necropsy there was purulent fluid within the frontal sinus and evidence of inflammation in ethmoid conchae and guttural pouch. *S. equi* was isolated only from the sinus.

This case reiterates the importance of isolation of new arrivals and self-quarantines when indicated in preventing the spread of diseases within a population. Interestingly, most of the affected horses only had minimal to mild clinical signs. It is thought that the vaccines administered after the first case was detected may have contributed to the less severe clinical signs.

Jaime Weisman, DVM, MS, RAHL Warrenton

Disseminated neuroendocrine neoplasia in an alpaca

An 8-year-old male alpaca was presented for weight loss and possible abdominal mass. Upon necropsy the abdomen contained several liters of fluid. The liver, diaphragm, and mesocolon had multiple firm, tan masses up to 7 cm diameter. A similar mass was present bilaterally at the angle of the jaw. Histopathology revealed sheets of cells with subdivision into packets separated by a fine fibrovascular stroma. All masses were similar, and one was present in the pancreas. The mass was consistent with a disseminated endocrine tumor, most likely of pancreatic origin.

Phillip Sponenberg, DVM, PhD, Virginia Tech

Ruminants:



Cerebral actinobacillosis in a sheep

An adult ewe was presented for necropsy after being euthanized due to progressive neurologic disease, with signs including head

pressing, dull mentation, trembling, and weakness. Necropsy revealed a large cerebral abscess that extended into the frontal lobe of the brain. On histopathology, the abscess effaced the cerebral gray matter of the left frontal lobe. Abscess contents were submitted for aerobic culture and *Actinobacillus lignieresii* was isolated in pure culture. *A. lignieresii* is normal flora of the oral cavity of cattle and sheep and is best known for causing infections of the tongue following trauma. However, it can also cause opportunistic infections at other sites, such as the nasal cavity, cervical lymph nodes, esophagus, forestomach, lungs, and subcutaneous tissue. This case is an unusual manifestation of *A. lignieresii*: a cerebral abscess that led to severe neurological disease.

Tessa LeCuyer, DVM, PhD, DACVM, Virginia Tech

Lead poisoning in a steer

In a period of three days, 4/25 steers were found dead without premonitory signs. Animals had access to a junkyard. No significant findings were identified during the necropsy. Histologically, numerous eosinophils were identified around vessels of the meninges and brain, plus occasional areas with neuronal necrosis in the cerebral cortex. With this finding, a fresh fragment of liver was submitted to Michigan State University for heavy metals screening. Lead level was 23.11 ug/g (ref. range 0 - 3 ug/g). In cattle, liver levels of 10 ug/g or more are confirmatory for lead poisoning. The fact that animals had access to a junkyard fits very well with this kind of toxic events. It is possible that animals had clinical signs, but were not observed by the owners. Clinical signs of acute lead poisoning appear 24 to 48 hours after ingestion, and include ataxia, blindness, salivation, hyperesthesia to touch and sound, spastic twitching of eyelids, bruxism, muscle tremors and convulsions. Eosinophilic meningoencephalitis is not frequently observed with lead poisoning in cattle, but when present, it is highly suggestive of this condition. It has been observed in calves and cows on a farm receiving land application of sewage sludge. Other findings may include acid fast inclusions in the renal tubular epithelium, which were not identified in this case.

Francisco R. Carvalho, DVM, DSc, DACVP, Virginia Tech

Pigs and birds:



Water deprivation/salt toxicity in a pig

A juvenile male Juliana pig was purchased at a market with no previous medical history. Upon arrival at its new home the pig was moribund, did not eat or drink, and had approximately 50 seizures over the weekend. On admission to the Veterinary Teaching Hospital significant examination findings included: delayed conscious proprioception in the forelimbs, dilated and fixed pupils, and bilaterally absent menace and pupillary light response. Due to lack of response to therapy, the pig was humanely euthanized and submitted for necropsy. No significant gross abnormalities were detected. Microscopically there was laminar cortical necrosis of the middle to deep cerebral cortical gray matter with accompanying perivascular lymphocytic and eosinophilic meningoencephalitis. These findings supported a diagnosis of water deprivation/salt toxicity syndrome in this pig, likely related to water deprivation in this case. Samples of brain submitted to the Virginia Department of Health were negative for rabies.

Thomas Cecere, DVM, PhD, DACVP, Virginia Tech

Transmissible viral proventriculitis in a chicken

A one-year-old Silkie rooster presented to a veterinarian with clinical signs of upper respiratory disease followed by inappetence. On physical examination the bird was minimally responsive and painful on palpation of the caudal coelom. The bird was euthanized and submitted for necropsy. Significant gross findings included marked pectoral muscle atrophy, fibrinous pericarditis, and diffuse thickening of the proventriculus with fetid watery green digesta in the proventriculus and ventriculus. Microscopically there was marked hypertrophy and hyperplasia of proventricular glands, necrosis of glandular and ductal epithelium, and lamina propria inflammation composed primarily of lymphocytes and plasma cells. These findings were compatible with a diagnosis of transmissible viral proventriculitis (TVP) with concurrent colibacillosis. The causative agent of TVP is chicken proventricular necrosis virus, a birnavirus.

Companion/Exotic Animals:



Atherosclerosis in a dog

An 11-year-old male Rottweiler dog was submitted for necropsy with a history of sudden anorexia, vomiting and little diarrhea. The necropsy revealed transmural

gastric ulceration, with septic peritonitis. Histologic examination of internal organs discovered disseminated atheromatosis in arteries of most organs, characterized by numerous cholesterol clefts and foamy macrophages within the sub intima and/or above the adventitia of the medium and small caliber arteries. Some arteries, particularly in the gastric wall, were completely occluded with this inflammatory reaction. Atherosclerosis and secondary gastric rupture with septic peritonitis was the final diagnosis. In veterinary medicine, hypothyroidism is the most significant syndrome associated with atherosclerosis. Dogs with hypothyroidism have been shown to have increased very low-density lipoproteins (VLDL), LDL, and HDL, so they are highly predisposed to develop atheromatous plaques. In the stomach, the arterial lesions likely induced areas of ischemia/hypoxia, with infarct and the resulting gastric rupture.

David Brown, DVM, RAHL Harrisonburg

Ethylene glycol intoxication in a dog

A five-year-old spayed female mixed breed dog presented to the VMCVM emergency service for hemorrhagic diarrhea, vomiting and seizures. Significant clinical chemistry abnormalities included severe renal azotemia (BUN 140 mg/dL, CRT 8.91 mg/dL), hyperphosphatemia (P 21.3 mg/dL) and an elevated anion gap (43.1 mEq/L, RR: 12.3-18.5 mEq/L). A Kacey ethylene glycol test was positive, and the dog was humanely euthanized and submitted for necropsy. The kidneys were diffusely pale and slightly swollen, and microscopically there was diffuse tubular necrosis with myriad tubular calcium oxalate crystals supporting a diagnosis of ethylene glycol toxicosis. Ethylene glycol intoxication is a common cause of

acute tubular necrosis in dogs and cats. It is a component of antifreeze and has a sweet taste, favoring voluntary ingestion.

Thomas Cecere, DVM, PhD, DACVP, Virginia Tech

Laboratory News:

VITALS

I am thrilled that Dr. Jessica Walters has accepted the position of Program Manager of the Office of Laboratory Services for VDACS. I look forward to strengthening the collaborative efforts that were started by Dr. Garvin. Welcome Dr. Walters! I'm excited to be working more closely with you! I would also like to welcome Jennifer Clifford, LVT and Katherine Harshman, LVT to the VITALS team. Ms. Clifford and Ms. Harshman will be stationed in Laboratory Central Receiving and will be sharing courier duties with the retirement of our courier, Steve Lyles. Also leaving us is our histotechnologist, Katherine Trainque, who will be joining the Office of the Vice President for Research. We wish Steve well in retirement, and Kate well in her new her new position. We'll miss you both!

Tanya LeRoith, DVM, PhD, DACVP, VITALS Director

Office of Laboratory Service

The Office of Laboratory Services is excited to continue to consolidate and grow! Our new fee schedule, located at <http://www.vdacs.virginia.gov/animals-fees-for-testing-procedures.shtml> now provides cost, location, preferred specimen type, and approximate turnaround time for all of our system wide diagnostic tests. We hope that by consolidating testing, we can keep costs low and expedite your results by decreasing shipment times. Our goal is to concentrate testing to laboratories that are most utilized by certain commodity groups. Certain testing will still be completed at all labs (i.e. Coggins, cultures and sensitivities, and fecal parasite examinations). As always, you are free to submit samples to whichever lab is easiest for you and we will make sure we get them where they need to go! We are always open to feedback and ideas for testing to further assist our agricultural community. It is our goal to make this lab system the best it can be to further serve our clients. Whether by utilizing data for reports or enhancing testing capabilities, we look forward to continuing to work with all of

**Jessica Walters, DVM, PhD, DACPV Program Manager,
Office of Laboratory Services, AFIS**



Laboratory Locations

RAHLS: Regional Animal Health Laboratory System

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ViTALS Virginia Tech Animal Laboratory Services

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