Dr. William B. Epperson graduated cum laude from The Ohio State University College of Veterinary Medicine in 1985 and received his Master of Science degree from the OSU Department of Veterinary Preventive Medicine in 1990. His areas of study included bovine infectious disease outbreak investigation, epidemiology, biochemistry, and applied ruminant nutrition. Bill is a board certified Diplomate with the American College of Veterinary Preventive Medicine with a subspecialty certification in Epidemiology. In 1996 he was awarded the Meritorious Service Award for Extension Specialists.

Prior to joining OSU, Dr. Epperson spent 4 years in private practice and 3 years as a Senior Research Veterinarian at American Cyanamid. He is originally from Galion, Ohio and is a third generation veterinarian.

Dr. Epperson will have a 0.65 FTE appointment in Extension. For dairy extension needs, please contact Dr. Epperson at (614) 292-9453 or epperson.1@osu.edu. Dr. Kent Hoblet has been the Dairy Extension Veterinarian since 1984. Dr. Hoblet will continue to hold an extension appointment for work on special projects, but at a reduced level (0.35 vs present 0.50 FTE).

**Welcome New Extension Veterinarian**

Dr. Bill Epperson graduated cum laude from The Ohio State University College of Veterinary Medicine in 1985 and received his Master of Science degree from the OSU Department of Veterinary Preventive Medicine in 1990. His areas of study included bovine infectious disease outbreak investigation, epidemiology, biochemistry, and applied ruminant nutrition. Bill is a board certified Diplomate with the American College of Veterinary Preventive Medicine with a subspecialty certification in Epidemiology. In 1996 he was awarded the Meritorious Service Award for Extension Specialists.

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**Welcome New Extension Veterinarian**

Dr. William B. Epperson

We are pleased to announce that Dr. William B. Epperson has accepted the position of Associate Professor and Extension Veterinarian for Dairy in the Department of Veterinary Preventive Medicine. After 10 years of service, he will leave his position of Extension Veterinarian at South Dakota State University where he worked with all food animal species.
Final Printed Issue

The Veterinary Extension unit at The Ohio State University first published the *Ohio Veterinary Newsletter* in 1974 to relay current information of interest to Ohio veterinary practitioners in food animal and general practice. We are in the process of enhancing this newsletter and its distribution by making it available on the Internet to a much larger audience; therefore, this will be the final mass mailing of print issues. We are updating our e-mail list and will send an electronic link whenever a new issue is posted to our website. Those without e-mail access who would like to continue to receive a paper copy by mail may subscribe for a fee of $10 per year. If you have any further questions, please do not hesitate to contact Mr. Jeff Workman, Extension Program Assistant; or Dr. Teresa Morishita, Extension Unit Coordinator.

*Please send your e-mail address to workman.45@osu.edu; call (614)292-9453; or fax (614)292-4142.*

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**Update on West Nile Virus Infections in the U.S.**

So far in 2004, there is West Nile Virus (WNV) activity in 24 states including Ohio. We have had dead birds confirmed in Warren, Williams, and Knox counties. There are also mosquito positives in Hamilton and Summit counties with a live bird positive in Wayne county. That means that the virus is still here. Depending on the weather, we have no way of knowing how much WNV will occur throughout the state. Therefore, precautions should be taken to reduce standing water on your property and protect yourself from biting mosquitoes using DEET products. Remember, adults should use 30% DEET and children should use DEET at a concentration of 10% or less. There have already been 14 human cases reported in the US including Arizona (10), New Mexico (1), CA (1), Wyoming (1) and South Dakota (1).

As far as horse cases are concerned, there have been 16 positive horses in Alabama, Arizona, Missouri, Oklahoma, Texas and Virginia. If you have not already done so, you should have your horse vaccinated for WNV along with the other vaccinations. If you have never vaccinated your horse for WNV, make sure the horse gets the primary vaccine and the booster. If the primary series was given in an earlier year, a booster should be all they need unless the horse is transported a great deal or it is greater than 10 years of age. Then a second booster around the first of August would be appropriate in Ohio. Just remember, approximately 1/3 of the horses with WNV encephalitis will die if not vaccinated.

—William J. A. Saville, D.V.M., Ph.D., Associate Professor, Extension Veterinarian - Epidemiology, The Ohio State University.

For more information and links to the various agencies working with the control and prevention of West Nile Virus, please visit the following website:

http://prevmed.vet.ohio-state.edu/ext_4b1.htm
List of WNV Fact Sheets Available for Veterinarians and the Public on the Website.

- West Nile Virus Fact Sheet For Veterinarians, WNV-1005-04
- What Horse Owners Should Know About West Nile Virus, WNV-1007-04
- Farmers and West Nile Virus, WNV-1001-04
- What Outdoor Enthusiasts Should Know About West Nile Virus, WNV-1002-04
- West Nile Virus and Scrap Tires, WNV-1004-04
- Frequently Asked Questions About Birds and West Nile Virus, WNV-1000-04
- People and West Nile Virus: Frequently Asked Questions, WNV-1003-04
- Frequently Asked Questions About Wildlife and West Nile Virus, WNV-1006-04
- Wetlands And West Nile Virus, WNV-1008-04
- West Nile Virus Fact Sheet for Physicians, WNV-1009-03

Cause of Jejunal Hemorrhagic Syndrome (JHS) Still Remains Enigmatic

Jejunal Hemorrhagic Syndrome (JHS), also known as intestinal hemorrhagic syndrome, “bloody gut” syndrome, and hemorrhagic bowel syndrome (HBS) is an emerging peracute clinical disease of both beef and dairy cattle (Figure 1, copied from ref 3). Affected cows may simply be found dead, others may show sudden onset of anorexia, lethargy, decreased milk production or have bloody feces or melena. Eighty-five to 100 percent of affected animals die within 24-36 hours after the onset of clinical signs. Most affected animals are not pyreptic. Distended loops of small intestine can be palpated in many, but not all cows with JHS. Differential diagnoses include salmonellosis, winter dysentery, abomasal ulceration, RDA, intestinal intussusceptions, and acute peritonitis.

Risk Factors

The disease is considered uncommon, affecting only a few animals in some herds; while other herds in the same geographic region remain unaffected. There is some concern that the disease is under-reported. Given the high mortality rate in affected animals, economic costs can be considerable for affected herds. Presently there is not enough information to determine risk factors for disease. Typically it is adult cows, up to 100 days in milk that are affected. High milk production and the increased intake of soluble carbohydrates have been linked to increased risks for JHS. Other reports suggest that large herds (> 500 cows) or those that feed TMR, or those in the western states are at higher risk for this disease. But dissecting apart these factors is difficult because the herds in the western states tend to be larger than in other areas and they also frequently feed a TMR.
Diagnosis and Etiology

A definitive morphological diagnosis is made on post-mortem examination. Invariably, there are sections of the jejunum (2-3 feet long) that are distended with large amounts of blood. Occasionally, intestinal intussusception will occur adjacent to the hemorrhagic lesions. Determining the etiological diagnosis is much more complicated. The true cause (or causes) of this syndrome remain unknown. Two prevailing theories include a role of Clostridium perfringens type A and improperly fermented feeds. C. perfringens produce a variety of toxins, and in fact, C. perfringens type C causes hemorrhagic enteritis in fast growing calves, lambs, and piglets. C. perfringens type A causes necrotic enteritis in poultry. However, C. perfringens type A is present in the intestinal contents of most healthy cattle! Nevertheless, it has been suggested that contributing factors such as feed intake, stress, available carbohydrates, etc. may alter the microenvironment of the gastrointestinal tract allowing for the proliferation of this organism to very high numbers and the increased production of toxins. The exact role of C. perfringens in this disease has yet to be proven. The second proposed mechanism of pathogenesis also remains un-tested. In this hypothesis, poor feed management is predicted to lead to the accumulation of harmful molds, bacteria (including C. perfringens) and the production of toxins in the feed that are subsequently ingested by the cow. Elucidating the cause of this syndrome is complicated by the fact that C. perfringens type A 1) may overgrow cultures masking the presence of other potential pathogens, 2) the toxin produced is highly potent and may be present in only minute quantities and 3) the toxin is rapidly destroyed and bound to cell membranes which makes it undetectable.

Treatment and Prevention

Medical or surgical interventions have been unsuccessful. In light of the hypotheses that the syndrome is somehow feed related, some researchers suggest ensuring that rations are of the highest quality and forages are well-fermented. These common sense suggestions are good for the prevention of many problems but have not been proven to impact the incidence of JHS. There is no scientific evidence that vaccination against C. perfringens provides any protection against JHS.

Outlook

Before this costly disease syndrome can be controlled a more complete understanding of its etiology must be achieved. As with many other disease syndromes it is likely that the cause of jejunal hemorrhage syndrome is multifactorial. At present there is no concerted effort to track the magnitude of this problem in Ohio dairy farms. In an effort to gather preliminary data you are encouraged to either fax (330)-263-3677 or e-mail o157@osu.edu, reports of JHS in Ohio to Dr. Jeff LeJeune at the Department of Veterinary Preventive Medicine, OSU. A downloadable report form can be e-mailed or faxed and is available at:

http://www.oardc.ohio-state.edu/fahrp/lab_pages/lejeune/extension.htm

Additional Information can be found in the following references:


-- Jeffrey T. LeJeune, D.V.M., PhD, Assistant Professor, Extension Veterinarian - Food Safety, The Ohio State University, OARDC.
Preventing Zoonotic Disease Transmission at Agricultural Fairs

Each year, millions of people across the United States visit agricultural fairs during the summer months. Fairs provide many urban and suburban residents a unique opportunity to see and interact with a variety of farm animals. In fact, many fairs have petting zoos or other animal displays that actively encourage direct contact between visitors and animals. Unfortunately, direct contact with livestock can also provide the opportunity for exposure to disease-causing organisms that may be shed by these animals. These can include *Escherichia coli* O157:H7 and *Salmonella enterica*. Exposure to these organisms can cause serious disease in some individuals, but more often cause only mild symptoms such as diarrhea or upset stomach. Although fair attendance is a safe activity, disease outbreaks among visitors to agricultural fairs have occurred. In order to better assess the potential risk that fair visitors will be exposed to these disease-causing organisms, scientists at The Ohio State University College of Veterinary Medicine and the USDA Agricultural Research Service conducted research at county and state fairs in three states during the summers of 2002 and 2003. The research was designed to estimate the proportion of animals at fairs that were shedding either *Escherichia coli* O157:H7 or *Salmonella enterica* while they were at the fair. They found that most fairs have some animals that are shedding these organisms, although fair animals appeared to be no more likely to be shedding than animals on farms. The shedding of these organisms by the animals leads to contamination of the fair environment. As a result, it may be impossible to completely prevent any exposure of fair visitors to these organisms. Fair visitors can best protect themselves from infection by washing their hands with soap after animal contact and before eating. Recommendations for fair management to reduce the risk to visitors are available and should be implemented at all fairs. These recommendations* include:

1. Signs informing the public about how diseases are transmitted and the importance of hand washing should be posted at every entrance of an animal exhibit.
2. Food and drink should never be permitted in animal contact areas.
3. Food concessions and animal exhibits should never be located in the same building.
4. Adequate hand washing facilities should be available.
5. Animal contact areas should be supervised to prevent hand-to-mouth activities. Children too young to practice adequate hand hygiene should not be allowed contact with animals.
6. Animals that are ill should not be included in exhibits.
7. The layout of the exhibit should provide hand washing stations between the animal contact area and food concession areas.
8. Animal feces should be removed from the exhibit areas as frequently as possible.
9. Fencing and rails in public areas should be cleaned and disinfected daily.
10. Buildings used for exhibiting livestock should be adequately ventilated.
11. Buildings that have been used previously to exhibit livestock should be thoroughly cleaned and disinfected before being put to any other use.


--Thomas E. Wittum, M.S., Ph.D., Associate Professor, Epidemiology, Preharvest Food Safety, Veterinary Preventive Medicine, The Ohio State University.
Survival of *E. coli* Deposited on Pasture

Researchers from the United Kingdom investigated the fate of *Escherichia coli* (*E. coli*) originating from livestock feces deposited directly onto pasture. This is important because pathogens present may result in food safety issues due to pathogen recycling. Recycling of pathogens results from livestock grazing grasses with surface contamination, consuming crops with internal contamination via the roots, and consuming water contaminated from run-off. This direct, natural application of manure is different than that from livestock housed indoors or in confinement because manure is often times stored for a period of time before being spread onto cropland or pasture. The length of time from the production of the feces to disposal reduces the number of microorganisms. This study was also unique because feces was used from animals who were naturally shedding pathogens; whereas, other studies examined feces that was inoculated with bacterial pathogens.

This study examined 3 separate high density pens consisting of 8 cattle, 12 pigs, and 20 sheep respectively. No rationale was given for the number of animals used in each pen. The livestock were kept in these pens for 14 days starting in November of 2002. During this time, animals had free choice water and were fed concentrates. An initial sample was taken from each pen after 7 days and consisted of 30 handfuls of fresh feces taken from the ground. After removal of the livestock, one week later, 20 sampling templates were created in each pen where the most manure was visibly present. The templates were not disturbed; however, they were exposed to weather conditions. Five cm deep soil core samples were collected within each of these templates periodically for up to 218 days. Samples were also collected from adjacent land without animal grazing to serve as controls.

After being occupied by livestock for 14 days, the condition of the pens varied based on species. The cattle pen was highly grazed with some intact grass and manure still visible. The sheep pen contained visible grass attached at the root and fully intact manure. The hog pen was visibly trampled with no grass and very little visible manure as it was mixed with the soil. The initial samples taken 7 days after livestock introduction showed *E. coli* levels of 7.70 log\(_{10}\) CFU g\(^{-1}\) for cattle, 7.59 log\(_{10}\) CFU g\(^{-1}\) for sheep, and 7.48 log\(_{10}\) CFU g\(^{-1}\) for hogs. These levels are similar to those previously found in the digestive system of livestock. Seven days after removal of livestock the levels averaged 5.34 log\(_{10}\) CFU g\(^{-1}\) for cattle, 4.31 log\(_{10}\) CFU g\(^{-1}\) for sheep, and 4.96 log\(_{10}\) CFU g\(^{-1}\) for hogs. The average levels of *E. coli* declined to less than 0.50 log\(_{10}\) CFU g\(^{-1}\) after 134 days which was similar to the levels found in the control samples. Recorded levels may contain error and should not be compared across species because templates were placed over intact cattle and sheep manure, but no intact hog manure remained. However, the objective of the study was to determine the survivability of *E. coli* on naturally deposited feces from each of the three species.

They found that the *E. coli* levels in hog manure declined at 1 log\(_{10}\) CFU g\(^{-1}\) over 28 days, while it took levels in cattle and sheep manure 38 and 36 days to decline that much. Remaining *E. coli* concentrations in all soils decline about 90% every 4-5 weeks. The longest measured survival time of *E. coli* was 162 days; however, an additional 14 days could possibly be added because contaminated feces may have been deposited on the first day livestock were placed in the pens. They report that the rate of decline is influenced by outside factors such as precipitation, soil type, temperature, UV radiation, animal diets, other organisms present, physiology of the organism, and the strain variability. They suggest that the shorter survival rate in pig feces is due to either the strain of *E. coli* or the chemistry of the feces. Other studies have shown that *E. coli* survives longer in cooler temperatures under laboratory conditions. The researches believe that survival times would be slightly lower if this experiment was replicated during the summer months. The researches have determined that *E. coli* pathogens do survive for at least 162 days in a natural farm environment, which could pose a food safety risk. However, they suggest that
more research is needed to determine if the length of survival contributes to the contamination of plants and water.


-- Abstracted by Jeff Workman, M.S., Extension Program Assistant, The Ohio State University.

-- Edited by Jeffrey T. LeJeune, D.V.M., PhD, Assistant Professor, Extension Veterinarian - Food Safety, The Ohio State University, OARDC.

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**Continuing Education in Fish Disease and Diagnostics**

The Ohio State University South Centers at Piketon and Veterinary Extension in collaboration with the Laboratory for Wildlife & Environmental Health in the Department of Veterinary Preventive Medicine at The Ohio State University is pleased to announce the Fish Disease and Diagnostics Workshop. The 2-day workshop is designed to provide in-depth lecture presentations and hands-on training for those individuals interested in gaining more knowledge about fish diseases and becoming more proficient at performing diagnostic testing and on-farm evaluations. The workshop will be held on October 1st & 2nd at the College of Veterinary Medicine on the campus of The Ohio State University. Since the course is designed to provide personalized training in a small group setting, registration will be limited to 30 participants on a first-come, first-serve basis. The registration fee for the course is $375 which includes course proceedings, transportation to a fish farm, 2 continental breakfasts, 2 lunches, dinner, and refreshment breaks. For more information, please contact Mr. Jeff Workman, Registration Coordinator, at (614)292-9453 or workman.45@osu.edu

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Faculty in Veterinary Preventive Medicine with Joint Appointments in Ohio State University Extension

Gary L. Bowman, D.V.M.
Extension Veterinarian, Swine (Emeritus)
bowman.7@osu.edu

William B. Epperson, D.V.M., M.S.
Extension Veterinarian, Dairy
epperson.1@osu.edu

Grant S. Frazer, B.V.Sc., M.S., M.B.A.
Extension Veterinarian, Reproduction
frazer.6@osu.edu

Kent H. Hoblet, D.V.M., M.S.
Extension Veterinarian
hoblet.1@osu.edu

William L. Ingalls, D.V.M., M.S.
Extension Veterinarian, Swine (Emeritus)
ingalls.1@osu.edu

Jeffrey T. LeJeune, D.V.M., Ph.D.
Food Safety (OARDC - Wooster, Ohio)
lejeune.3@osu.edu

Teresa Y. Morishita, D.V.M., M.P.V.M, Ph.D.
Extension Veterinarian, Poultry
morishita.1@osu.edu

Y. M. (Mo) Saif, D.V.M., M.S., Ph.D.
Poultry Health (OARDC - Wooster, Ohio)
saif.1@osu.edu

William J. A. Saville, D.V.M., Ph.D.
Extension Veterinarian, Epidemiology
saville.4@osu.edu

William P. Shulaw, D.V.M., M.S.
Extension Veterinarian, Cattle and Sheep
shulaw.1@osu.edu

Jeff Workman, M.S.
Extension Program Assistant
workman.45@osu.edu

WEBSITES:
http://prevmed.vet.ohio-state.edu/ext_int.htm
http://ohioline.osu.edu/vme-fact/index.htm

OHIO STATE UNIVERSITY EXTENSION
U.S. DEPARTMENT OF AGRICULTURE
1920 COFFEY ROAD
COLUMBUS, OHIO 43210-1092

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