# Equine Proliferative Enteropathy (Lawsonia)

**Disease Name**: Equine Proliferative Enteropathy (EPE)

**Disease definition**: Equine proliferative enteropathy (EPE) is an enteric disease caused by the obligate, intracellular bacterium *Lawsonia intracellularis*. The disease causes intestine inflammation and thickening.

**Transmission**: Unknown, although fecal-oral exposure is suspected. While the environmental reservoir for the bacteria is unknown, it is suspected that wildlife, rodents, and feral animals may play a role.

**Frequency**: A disease that is typically restricted to weanling and yearlings in the North American fall and winter months, only a small portion (5-10%) of those horses exposed to *L. intracellularis* will develop clinical or subclinical disease.

**Incubation period**: Approximately 14 days based on experimental challenge models.

**Carrier status**: Unknown, although some evidence suggests that infected horses could be a source of infection for other horses.

**Shedding period**: Shedding of *L. intracellularis* typically ends within several days following the initiation of antimicrobial treatment.

**Severity**: Clinical signs can range from lower daily average weight gain (sub-clinical cases) to those of moderate severity in clinical EPE (see below). Rare cases of EPE associated with rapid decompensation and death have been reported.

**Clinical signs and symptoms**: Clinical signs of ventral edema, diarrhea and hypoalbuminemia combined with the time of year and age of the horse can be almost pathognomonic for EPE. North American cases are most commonly seen in August through January with young horses between two and eight months of age most commonly represented.

Clinical signs may include:

- Anorexia (partial or complete)
- Rapid weight loss
- Dependent/ventral edema (typically head, legs, and prepuce)
- Rough hair coat
- Fever
- Colic
- Diarrhea
- Depression

  

**Complications can occur in rare cases**: As noted above, rare cases of EPE associated with rapid decompensation and death have been reported. These cases often are preceded by fever and CBC changes, possibly indicating bacterial translocation across the damaged gastrointestinal mucosa.

**Diagnoses**: A presumptive diagnosis of EPE can be made based on hypoalbuminemia coupled with clinical signs, age, and season. Testing to confirm a diagnosis of EPE includes:

- Hypoalbuminemia: This is a consistent finding across almost all cases of EPE and always accompanied by hypoproteinemia.
- Abdominal ultrasonography: Used to identify areas of small intestinal wall thickening (>5mm), the absence of thickening does not rule out EPE.
- Fecal PCR testing: Utilized to demonstrate the presence of *L. intracellularis* in the gastrointestinal tract. False negative results are possible, especially once antimicrobial therapy has started.
- Serologic assays: Several different assays exist, all of which detect antibodies to *L. intracellularis*. Ideally, paired samples 7-14 days apart will be submitted, but many will initiate therapy based on a single positive result at the time of clinical signs and other testing. In certain geographic areas, exposure to the bacteria can approach 100%, so serologic diagnosis should always be paired with clinical signs and other diagnostic options.
- CBC and biochemistry analysis: These findings are typically mild in normal cases of EPE, although leukocytosis, hyperfibrinogenemia, and increased hematocrit have been reported. However, severe leukopenia, neutropenia, thrombocytopenia, and other changes have been associated with necrotizing-EPE, which often results in sudden decompensation and euthanasia/death.

**Treatment**: Treatment for EPE consists of antimicrobial treatment and supportive care. The most commonly used antibiotics include tetracyclines (oxytetracycline, doxycycline, minocycline) or chloramphenicol. The antimicrobial choice should reflect the age of the animal and the risk for gastrointestinal or renal toxicity. A standard time course of treatment, based on the antimicrobial selected, is typically sufficient for cases of EPE. Supportive care typically involves intravenous fluids, in addition to oncotic support such as plasma or colloids. Additional treatments may include anti-ulcer medications and enteral or parenteral nutrition.

**Prognosis**: Most horses diagnosed with clinical EPE will fully recover following treatment, although the clinical and physical condition of the horse, as well as normalization of albumin levels, may take significantly longer (months) to completely recover. In cases of necrotizing-EPE, the prognosis is often grave due to secondary complications from bacterial sepsis.

**Prevention**: While limited studies have described immunization of foals via rectal administration of the porcine vaccine against *Lawsonia intracellularis*, this represents an off-label use of the product and practitioners are advised to check their state rules regarding whether it is acceptable to vaccinate animals with vaccines labeled for another animal species.

  

**Biosecurity**: Isolation of affected horses for approximately 7 days and prompt removal of feces is suggested. Additionally, it is best to limit wildlife and rodent exposure as much as possible, especially in barns.