

## Swine - Diseases of the Digestive System

Disease	Etiology	Epidemiology	Transmission	Pathogenesis	Signs	Diagnosis	Treatment	Prevention & Control
<b>Transmissible Gastroenteritis (TGE)</b>	<b>coronavirus</b>	epizootic (epidemic or acute): all ages, winter, naive herds; enzootic (endemic or chronic): younger pigs, 1-8 weeks, peak in winter, experienced herds	from other infected pigs, dogs & starlings carry it, fomites	ingested, infects & destroys SI epithelial cells, atrophy of jejunum, deranged digestion/absorption, undigested lactose yields osmotic diarrhea, dehydration, lactic acidosis	(epizootic) baby pigs V/D, 1Mb, 1Mt <2 weeks; diarrhea w/ milk curds, Ga, Fa & As; anorexia, V/D, poss fever, occas abortion; (enzootic) not before 6-7 d or after 2w postweaning; variable Mb&Mt. absent in adults	signs (esp epizootic form); gross (undigested milk, thin intestinal wall, chyle absent from lymphatics); histo (jejunal villus atrophy), VI, serology, fecal pH	no specific tx; fluids & electrolytes, antibact therapy in mixed infections; warm, draft free, dry env't	mgmt improvements (exposure, introduction, sanitation); immunization (IgA more protective than IgG); eradication (close herd, equalize immunity, sanitation, all in/all out, monitor)
<b>Swine Dysentery (SD) (bloody scours, black scours, vibronic dysentery)</b>	<b>Brachyspira hyodysenteriae</b>	growers & finishers: Mb up to 90%, Mt up to 30%; asymptomatic carriers common; survive up to 2 mos. in manure pit	fecal-oral route: mice (reservoir hosts), other animals (mech hosts); fomites	ingested; invades and attaches to colonic mucosa, deranges colonic reabsorptive capacity, catarrhal hemorrhagic colitis, diarrhea, dehydration	yellow/grey soft stool → mucousy (bloody) → watery bloody mucousy diarrhea with white exudate; anorexia, fever, thirst, gaunt, weak emaciated; recover in 2 wks, signs may recur; occasional peracute death	signs, gross (mucohemorrhagic colitis, pseudomembranes, hyperemia & edema of LI (no SI signs), microscopic (thick submucosa of colon, goblet cell hyperplasia, hemorrhage & necrosis, culture	Antibiotics in feed or water (water preferable) Prophylaxis with feed/water meds	prevention (closed herd, purchase from clean herd, isolate new piglets & medicate) vaccinate (reduce severity, not infection) eradication (depop, clean up, disinfect, repop from clean herd; meds & sanitation)
<b>Porcine Proliferative Enteritis (garden hose gut)</b>	<b>Lawsonia (Ileobacter) intracellularis (obligate)</b>	growers & finishers mostly, but all ages; little is known, widespread subclinical dz; dz more apparent in SPF or minimal dz herds; induced by stress; genetics	(nothing written)	poorly understood	feed intake & growth rate, intermittent diarrhea, anorexia & wt loss, variable Mb, low Mt; melena, hemorrhagic diarrhea (older pigs), some acute deaths; anemia	gross (thick intestinal mucosa [garden hose], ulcers, necrosis, hemorrhage, pseudomembranes; distal 1/3 SI, cecum, prox 1/3 spiral colon; histo (proliferation of intestinal epithelial glands, intracellular organisms in cytoplasm; fecal extracts with DNA probes	preventative better than curative tx. Prophylaxis with feed/water meds	reduce stress, medicate in stressful times, genetic selection?
<b>Salmonellosis - ZOONOTIC</b>	<b>3 serotypes in pigs: S. choleraesuis (Sc - most common), S. typhimurium (Stm) &amp; S. typhisuis (Sts - ZOONOTIC)</b>	low endemic level in most herds; asymptomatic carriers common, 2nd most common bact isolate from pneumonic swine lungs; assoc with ventilation probs, stress, poor sanitation, overcrowding & immunosuppression	fecal-oral route	invade intestinal mucosa & become septemic; most signs d/t endotoxin; may produce exotoxin → secretory diarrhea; inflammation, vascular damage & necrosis of gut d/t toxins	septicemic form: dyspnea & cough; pyrexia, anorexia, depression, purple skin (ears & ventral abdomen); GI form: acute or chronic diarrhea ± blood; pyrexia, anorexia, dehydration; occasion. Distended abdomen d/t rectal strictures; Sc	signs, septicemic form - lesions: pneumonia w/ edema & hemorrhage, pleuritis, splenomegaly, hepatomegaly, hemorrhagic inn, renal ecchymosis & liver nodules; GI form - lesions: diffuse necrotic colitis & typhilitis (& some ileum), button ulcers, enlarged he	use antibiotics for prophylaxis (amino-glycosides, ceftriaxone, trimethoprim)	mgmt (all in /all out) & sanitation; vaccination - killed bacterins; avirulent live vaccines - intranasal
<b>Rotavirus</b>	<b>RNA virus in the Reoviridae family</b>	Virus is ubiquitous, herd infection rates nearly 100%; infection more prevalent than clinical signs.	None specified.	Similar to TGE, virus is synergistic with other enteric pathogens like E. coli, C perfringens, Coccidia and TGE.	Similar to TGE but less severe, diarrhea 3-4 days post weaning, can also see neonatal diarrhea.	Hx, Clinical signs, Gross pathology: thin walled intestines with poorly digested food material within. Histopathology: villus atrophy (duodenum not spared), FA, EM	Glucose and electrolytes, antibacterial for concurrent E. coli	Weaning management: wean pigs into clean, warm, dry environment, ensure good nutrition, etc. Vaccination.
<b>Enteric Colicacilliosis</b>	<b>Enterotoxigenic Escherichia coli (ETEC)</b>	Most important primary cause of diarrhea in piglets <5 days. Can contribute to diarrhea in older nursing piglets or weaned pigs. Pure infections are rare. Can cause septicemia.	Non enterotoxigenic E coli are normal gut inhabitants. Spread through fecal contamination by other scouring piglets. Poor sanitation or continuous flow farrowing cause build up of ETEC in environment.	E coli enters orally, adheres to receptors on epithelial cells of SI via fimbria. Produces Heat labile (LT) and heat stable (ST) enterotoxins. LT stim Adenyl Cyclase, inc intracellular cAMP, inc secretion of bicarb, Na, H2O, causing diarrhea, dehydration.	Clear watery to yellowish brown pasty diarrhea, dehydration, depression, gauntness, inflamed perineum, variable death loss (higher in younger piglets). Death usually due to dehydration and electrolyte disturbances.	Clinical signs, alkaline feces (>8), culture of SI: smooth colony types on Tergitol 7, isolates characterized by fimbrial antigens and enterotoxin production, Histopath: lack of villus atrophy. Gross Path: distended, gas filled intestine, intact villi, ch	Antibiotics, oral E. coli antiserum, fluid and electrolyte therapy.	Management: warm, dry environment, all in/all out farrowing, sanitation. Vaccination of sow, efficacy depends on adequate milk flow from sow.
<b>Clostridial Enteritis</b>	<b>Clostridium perfringens type C and type A</b>	See in first 3-4 days of life, occ in weaned pigs.	Carried in feces and on skin, pig inoculated soon after birth. Can be in soil.	Organism consumed shortly after birth, attaches to and invades jejunal villi, beta toxins cause massive intestinal necrosis, death from secondary bacteremia, hypoglycemia and toxemia.	Peracute: sudden death in 1-2 day old piglets, +/- bloody diarrhea. Acute: 2-3 day course, death, bloody diarrhea with shreds of necrotic mucosa. Subacute: 5-7 day course, diarrhea +/- bloody, gradual wasting, emaciation and dehydration. Chronic: intermit	Clinical signs (hemorrhagic diarrhea), Lesions: blood fluid in jejunum, necrotic membrane in jejunum, emphysema in jejunal submucosa, see large gm pos rods in acute cases, necrotic enteritis in chronic cases. C perfringens type C beta toxin in intestinal	Usually ineffective if clinical signs are evident, administration of type C antitoxin may help in acute and subacute cases.	Sanitation, parenteral or oral type C antitoxin within minutes of birth, vaccination of sow with type C toxoid at 5 and 2 weeks pre farrowing, prophylactic antibiotics.
<b>Coccidiosis</b>	<b>Isospora suis</b>	Component of up to 27% of 5day to weaning diarrheas. Problem with continuous farrowing and poor sanitation. Mixed infections common, esp with E.coli.	Scouring baby pigs main source of infective oocysts, sows rarely shed.	Sporulated oocysts are ingested, invade epithelial cells and become trophozoites which divide into merozoites or shizonts, these give rise to merozoites which are release from and destroy host cell to invade other epithelial cells and repeat cycle or become	Diarrhea usually between 7 and 10 days of age, yellow to gray-green feces (not bloody), acidic pH, no response to antimicrobials, gauntness, dehydration.	Clinical signs (diarrhea in pigs >7days), Necropsy: mild to severe fibronecrotic enteritis in jejunum and ileum, Histopath: villus atrophy, ulceration and necrosis of intestinal mucosa, merozoites in epithelial cells, Impression smears to demonstrate mero	Amprolium solution, Trimethoprim sulfa. Limited efficacy and labor intensive.	All in/All out, disinfect with Clorox, change to raised farrowing crates.