Special Issue • Swine A magazine of **Biomin**

Science & Solutions

Special

What's Wrong With My Pigs?

Contents

The What's Wrong with My Pigs? series offers practical 1-page articles on common issues in modern pigs production. Each differential diagnosis identifies potential cause, description of problem, checklist and corrective actions to help you to maintain production performance.







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Part 1: Immune Depression

A handy checklist for diagnosing swine ailments that you can cut out and keep with you for reference.



Anum

number of biological agents are able to compromise immune integrity of animals, from mycotoxins to infections to drugs. vaccine failures. Most often the syndromes stem from socalled "conditioned diseases" such as *E. coli*, *Streptococcus*, *Salmonella*, *Pasteurella*, *Influenza*, etc..

A single instance or concomitant administration and poisoning of the above mentioned caused by these elements can undermine through suppression or disrupt regular development of body defenses and are resulting in lower performances, increased disease outbreaks and

It is well known nowadays that diseases come from multifactorial causes; immune depression can let loose many latent infections, presenting a challenge for practitioners regarding etiology and therapy.

	Potential cause	Checklist	Corrective action
MYCOTOXINS	• AFB ₁ , DON, DAS, T-2, OTA, FUM	 Positive raw materials (ELISA) or feed (HPLC) Origin of raw materials historically contaminated Symptoms pertaining to mix of infections, vaccine failure Decline of herd/phase performances 	□ Check raw materials and feed □ Hygiene of feed and water lines □ Use Mycofix [®] at suitable inclusion rate
PATHOGENS	Virus: • Circovirus, Herpes virus (Aujeszky's), Asfivirus (ASF), Orthomyxovirus (Flu), and Arterivirus (PRRS) Bacteria: • Mycoplasma, Pasteurella, APP, Salmonella	□ Epidemiology, symptomatology □ Necropsy □ Immune-histochemistry, PCR, ELISA	☐ Biosecurity ☐ Vaccination ☐ Antibiotics
ANTIBIOTICS	 Tetracyclines, penicillins, sulphametazine, streptomycin, chloramphenicol (forbidden in EU) 	 Overdosing Prolonged treatment Unwise adoption 	 Proper management and nutrition (acidifiers, phytogenics) Alternative antibiotics

References are available on request

For more information, visit www.mycotoxins.info



Part 2: Abortion

t is generally accepted that some 2% of sow pregnancies end in abortion. In these cases, an investigation must be carried out to identify causing agents that could span anything from environmental factors such as heat stress to nutritional ones such as toxins. In autumn, there may be a natural increase in the abortion rate due to daylight fading, though this ought to remain sporadic. This can be fairly well counteracted by shrewd management of lamps regarding both time and intensity. In some cases, abortions result from consumption of drinking water contaminated by yeasts or other bacteria. In other cases they result from too little water intake during early gestation.

If the abortion outbreak is dramatic and affects a wide number of sows in the herd then it is likely that an infectious disease is entering in the herd (see table) without any specific timing and the sows have often fever and there are other signs from the disease in sows, aborted fetuses and newborn piglets. Characteristic is PRRS abortion occurring mainly during last gestation period.

In large farms a rise in the abortion rate may be more difficult to witness, though evidence may come from a drop in feed intake and weight along with vulvar discharge.

Mycotoxins may also be a causing agent. Aflatoxins, deoxynivalenol, fumonisins, zearalenone (ZEN) and ergot alkaloids can all play a significant role in fertility problems and abortions. Pregnant sows' consumption of feed with high levels of ZEN, especially during early gestation, can lead to smaller litters and mummification. Between days 7 and 10 of pregnancy are the most critical ones with a high rate of embryonic death.

Moreover, if the feed contains low to medium amounts of ZEN during the whole gestation, this will result in smaller fetuses and large variations in the weight of piglets of the same litter. Furthermore, ZEN can also lead to stillbirth and neonatal mortality and in the worst case it can even lead to the death of the entire litter. There evidence that ZEN intoxication is link to splay legs.

Decrease of feed intake and sometimes feed refusal have a self-limiting effect in mycotoxicosis.

	Potential cause	Checklist	Corrective action
MYCOTOXINS	• Zearalenone, Deoxynivalenol, Aflatoxins, Ergot alkaloids, Fumonisins	 Positive raw materials (ELISA) or feed (HPLC) Origin of raw materials historically contaminated Elevated sphinganine/sphingosine ratio in serum 	 Check raw materials and feed Hygiene of feed and water line Use Mycofix[®] Plus at suitable inclusion rate
PATHOGENS	 Virus: African swine fever, Classical swine fever, Foot and mouth disease, PRRS, PCV type 2, Parvovirus, Influenza A virus Bacteria: Actinobacillus spp., Brucella suis, Erysipelothrix rhusiopathiae, Lawsonia intracellularis, Listeria monocytogenes, Leptospira spp., Salmonella spp., Streptococcus spp., Staphylococcus spp. 	 Epidemiology Symptomatology Necropsy Bacterial culture Histopathology PCR ELISA Immunohistochemistry (IHC) 	 □ Biosecurity □ Vaccination □ Antibiotics
OTHERS	 High environmental temperature Draught, especially in autumn Water deficiency 	 Check room temperature Check water flow direct after feeding time 	□ Temperature range: 10-21°C □ Water flow 1.0-1.2 liter / mins (min. 8 liter/day)

References are available on request

For more information, visit www.mycotoxins.info



Part 3: Vomiting

Vomiting should not be confused with regurgitation. Vomiting is the ejection of stomach contents through the mouth. Regurgitation is swallowed food that does not reach the stomach, and is ejected through the mouth. If there is any confusion of whether vomiting or regurgitation is occurring, it may be settled by measuring the pH of the ejected material. Vomitus has an acid pH and regurgitated material is alkaline. Pig vomiting is a symptom of several swine diseases.

Pathogenic causes of pig vomiting. Vomiting is a prominent clinical sign of Hemagglutinating encephalomyelitis (HEV), Porcine epidemic diarrhea (PED), Transmissible gastroenteritis (TGE). It is also a clinical symptom of Hog cholera (HC), African swine fever (ASF), Classical swine fever (CSF), and Aujesky's disease. Younger piglets are more susceptible to viral infection, as are older piglets. To do differential diagnosis, check major symptoms and organ systems affected by pathogens, then start disease control. Toxigenic causes of pig vomiting. Deoxynivalenol (DON), also known as vomitoxin, and in rare cases T-2 toxin can be a cause of pig vomiting. Vomitoxin is a trichothecene mycotoxin isolated and characterized in 1973 as a major emetic and feed refusal factor for swine. It can be found in corn and cereal grains contaminated in the field mainly by Fusarium graminearum.

Nutritional causes of pig vomiting. Clinical sign and subclinical deficiency symptoms for several nutrients are a wide variation in the amount of time that elapses before symptoms of nutrient deficiency begin to appear. Certain nutritional deficiencies are more common and have greater consequences than others today in swine. For example, some vitamins as niacin, riboflavin, thiamine (vitamin B1), pyridoxine (vitamin B1), vitamin D, and zinc deficiencies can be a cause of pig vomiting. On the other hand, too much vitamin D and B1 can sometimes cause vomiting.

	Potential cause	Checklist	Corrective action
MYCOTOXINS	• Deoxynivalenol, T-2 toxin	 Positive raw materials (ELISA) or feed (HPLC) Origin of raw materials historically contaminated 	 Prevent molds, purchase clean raw materials Use Mycofix[®] at suitable inclusion rate
PATHOGENS	Virus: • TGE, PED, Aujesky's disease, ASF, CSF, HEV Parasites: • Strongyloides spp., Ascaris suum	 Epidemiology Virus isolation Necropsy Histopathology PCR, RT-PCR ELISA IHC 	 Biosecurity Vaccination Good sanitation procedures Anthelmintic
OTHERS	 Foreign body Vitamin deficiency (Niacin, B₁, B6, D) Excess of vitamin D Toxicity of microelements (arsenic, fluorine, selenium, etc.) Zinc deficiency 	□ Analyze feed samples	 Remove foreign body Check nutrient requirement Adjust diet formulation

References are available on request

For more information, visit www.mycotoxins.info



Part 4: Ear necrosis

Porcine ear necrosis syndrome (PENS) in pigs has been reported as an increasing health problem in many countries with intensive pig farming. PENS exhibits as tissue damage to the tips of ears. Researchers have found it occasionally in pigs at three weeks, but it is most common in pigs 5 to 16 weeks of age.

S ometimes only a few pigs are affected and the lesions are barely noticeable, but there are outbreaks where all the pigs are affected and many with extensive lesions.

The disease does not result in mortality, but can make it difficult for a farmer to sell feeder pigs. The cause of this disease is complex and the presumed triggering factors can be divided into infectious and non-infectious agents.

Weissenbacher-Lang (2012) has published a research in which they looked for the causative factors of porcine ear necrosis syndrome (PENS) in 72 pigs, 5.5-10 weeks. Streptococci and staphylococci were isolated from most of the pinnae.

Porcine circovirus 2 (PCV2) could not be detected and porcine reproductive and respiratory syndrome (PRRS) in only 10% of the piglets. As main mycotoxins deoxynivalenol and ergot alkaloids were detected in the feed.

A positive correlation was found between deoxynivalenol and focal epidermal necrosis and bacterial growth in the superficial cell debris. Ergotamine is positively associated with vasculitis and acute phase of PENS. The findings suggest that PENS is multifactorial in origin and that although infectious agents can be involved in the development of the syndrome they are not the exclusive triggering factor. Also in some cases there is a direct link between disorder of gut micro flora and the levels of endotoxins (lipopolysaccharide in the gut and symptoms of PENS).

Specific actions to prevent ear tip necrosis have proved very difficult to formulate. Clearly, control of co-existing disease is a vital part of any herd health strategy and will help to minimise development of lesions. Minimizing trauma to ear tips by attending to pen divisions, feed hopper design and water access as well as reducing competition by increasing space provision and decreasing group size may help. Provision of fresh air may also help. Streptococci and Staphylococci require high humidity to thrive, so increasing air exchange and reducing humidity as well as attention to hygiene could be helpful. Providing mash feed instead of pellets has been shown to reduce ear tip necrosis. It is not clear whether decreased particle size during the pressing operation is the cause, or the heat treatment which destroys certain important substances in the feed. It is also important to promote the healing of wounds so that infections cannot strike quickly. The inhibition of inflammation in the capillaries of the ears could be improved through nutritional measures such as adding higher levels of B-vitamins, introducing certain phytogenics and ensuring the right balance between omega 6 and omega 3 fatty acids.

Checklist	Corrective action		
Potential cause: Trichothecenes (for example de	Potential cause: Trichothecenes (for example deoxynivalenol), ergot alkaloids		
 Positive raw materials ELISA, feed HPLC. Origin of raw materials historically contaminated Symptoms pertaining to mix of infections Decline of herd/phase performances 	 Check raw materials and feed Hygiene of feed and water lines Use Mycofix[®] at suitable inclusion rate 		
Potential cause: PATHOGENS: Streptococci and Staphylococci			
 Epidemiology, symptomatology Necropsy Immune-histochemistry, PCR, ELISA 	BiosecurityVaccinationAntibiotics		
Potential cause: dysbiose of gut micro flora			
 Increase of Gram negative bacteria / endotoxin levels Digestibility and level of protein Content of fermentable fibres 	 Check raw materials and feed formulation Use Mycofix[®] to bind the endotoxins 		
Potential cause: Nutritional factors			
 Too low digestible protein level Vitamins levels (B, K, E and anti-oxidants) Ratio omega 3/omega 6 fatty acids Mash feed instead of pellets 	 Check raw materials and feed formulation Proper management and nutrition (Fish oil, Mycofix[®], phytogenics). 		
Potential cause: Management			
 Overcrowding, mixing Ventilation/oxygen levels Design of feed hopper and water access 	Increasing spaceCheck ventilation		

DISCLAIMER:

This table contains general advice on swine-related matters which most commonly affect swine and may be related to the presence of mycotoxins in feed. Swine diseases and problems include, but are not confined to the ones present in the table. BIOMIN accepts no responsibility or liability whatsoever arising from or in any way connected with the use of this table or its content. Before acting on the basis of the contents of this table, advice should be obtained directly from your veterinarian.

References are available on request



Part 5: Rectal Prolapse

Rectal prolapse can look quite ordinary but can have a serious impact on animal health. It can prevent removal of metabolites produced during the digestive process, cause pain, encourage biting by pen-mates – thus leading to infection and even bacteraemia – and, if left alone, can cause necrosis.

The last part of the gut, the rectum, has a huge unselective absorbtion capacity, meaning that if elimination does not happen regularly, together with water, a number of toxins can enter the bloodstream causing intoxication, liver burden and discomfort.

Rectal prolapse can have many causes, the most common is constipation with hard to release stools that adhere to the gut walls, such that pushing to get rid of them culminates in projection of the last segment of the rectum outside the anus.

Similarly it can happen when swine are coughing as the sudden rise in inner pressure is able to bring protrusion. It can also happen in overcrowding situations when pen-mates step on one another's bellies.

Generic diarrhoea such as enterocolitis and some gut worms can also result in prolapse. A more specific cause can be stricture of the anal sphincter, called tenesmus. Less well known is salmonella two months after diarrhoea and local inflammations affecting the last part of the urogenital tract (vaginitis, urethritis, etc).

Then come the factors which are able to cause relaxation of keeping the rectal structure in situ, the most classic being ageing. In young animals it may be caused by mycotoxins such as zearalenone that also have swelling effects similar to those of phytoestrogens.

The most common treatment is to isolate the animal to avoid pen mates biting and then waiting until the protruded segment necrotises and falls. However, in the process lower feed intake, constipation and bacteraemia often occur, and this is quite commonly associated with high weight loss.

Surgical treatment, cutting and sewing with the tobacco bag technique, requires expert skill, is considered expensive and does not always allow for a complete recovery. It is therefore better to focus on prevention, through providing adequate water, providing fibre in the correct amount and quality according to the production phase, and ready treatment for fever. Prevention and treatment of gut and urogenital tract diseases, along with toxic agents, are reported in the table.

Checklist	Corrective action		
Potential cause: MYCOTOXINS			
 Vulvovaginitis, vaginal and/or rectal prolapse Reproductive issues; stillbirth, splay leg, low litter size Carry-over in sow milk 	Prevent mold growth • Purchase clean raw materials • Use Mycofix®		
Potential cause: MANAGEMENT			
 Water intake/constipation Overcrowding Cough Transport Seasonality Tail docking, tail biting Sudden diet changes, soft faeces, ingestion of wood shavings 	 Control water flow per minute and pressure as well as drinker efficiency Increasing space Control fibre content of feed, avoid sudden diet changes 		
Potential cause: PATHOGENS			
ELISAPCRFlotation procedure of faeces samples	 According to etiology 		
Potential cause: AGE			
 Average sow population Number of farrowings	Replacement rate		
Potential cause: GENETICS			
 Control of heterosis effect Avoid overconsumption/overfeeding/ excess of feed 	• Discuss with genetic company		
Potential cause: NUTRITION			
 Check fibre content and fibre sources Too high levels of barley (β-glucanase) Grinding fineness Control feed intake High sodium or potassium levels 	 Check feed formulation and grinding fineness 		
	References are available on request		

For more information, visit www.mycotoxins.info



Part 6: Depressed voluntary feed intake

Voluntary feed intake is one of the most important and challenging factors of swine production. The amount of feed consumed is central to determining growth performance and tissue accretion rates. Feed intake measurement and constant monitoring on commercial units is essential to formulating diets to meet nutritional requirements. This article details the main factors that impact feed intake and highlights actions where management can influence the outputs (see table below).

- Environmental factors: The major climatic factor that directly influences feed intake is the environmental temperature. Pigs exposed to extreme cold consume excessive quantities of feed in an effort to maintain normal body temperature.
- Diet composition: Dietary energy levels have a significant impact on feed intake. Pigs can use a wide variety of feed ingredients and adjust their feed intake pattern to maintain a constant energy intake. Feed intake will also be influ-

enced by the level of other nutrients in the diet, especially in case of deficiencies or imbalances.

- Health status: The immune system has a major impact on swine performance. Exposure to pathogenic agents has been shown to result in the release of cytokines that activate the immune system. Cytokines alter metabolic processes, resulting in decreased protein synthesis and increased protein degradation to maintain homeostasis during a health challenge.
- Genotype: Substantial genetic variation for lean growth rate, lean efficiency, carcase percent lean and feed intake exists between different genetic populations of pigs.
- Mycotoxins: Toxins can dramatically reduce feed intake levels. Many common feedstuffs contain natural toxins that may impair pig performance and/or voluntary feed intake, thus affecting nutrient requirements.

Guidelines / Corrective action

Environment

- Optimum barn temperature range based on age and phase of production
- Optimum barn ventilation rates
- Proper operation of misting or water drip systems to promote evaporative cooling
- Barn controller adjustment based on seasonality were performed properly and
- equipment is fully operational

Diet and feed management

- Constant revision of nutritional program
- Proper levels of dietary energy
- Proper levels of protein
- Ingredient palatability limit the inclusion of unpalatable ingredients and/or make gradual changes
- Feed form: pellets tend to give higher intake
- Particle size: ensure proper micron size
- Pigs have continuous access to feed
- Feed system supplies a continuous feed to the feeder

Feeder

- Feeder space is adequate for individual pig as well for total number of pigs in the pen
- Proper pan coverage
- Physical condition of feeders
- Consider in the potential benefits of wet dry feeders and overall feeder design with targeted operation outputs

Mycotoxins

- Check raw materials and complete feed. Establish a sampling monitoring system with analytical methods (LC-MS/MS, HPLC)
- Ensure hygiene in feed bins, feed lines and water lines
- Consider the inclusion of a feed additive with multiple counteracting strategies (adsorption, biotransformation and Bioprotection)

Water

- Drinkers are functioning properly
- Flow rate is adequate
- Drinkers are accessible to pigs (adequate height especially during the nursery phase)
- Ratio of drinkers to pigs is sufficient
- Water quality



Part 7: Conjunctivitis

Conjunctivitis can be caused by a variety of reasons. Here we describe some of the risk factors related to it and some useful tips for the differential diagnosis of this pathology. Conjunctivitis is the inflammation of conjunctiva, a thin and delicate membrane that covers the eyeball and lines the eyelid.

Conjunctivitis is an extremely common eye problem because the conjunctiva is continually exposed to micro-organisms. Also, environmental agents can cause infections or allergic reactions.

The clinical signs can be seen in one or both eyes and, if caused by infection, can be very easily transmitted to other animals due to close physical contact. Outbreaks may be associated with conjunctivitis infection Chlamydiae but there may be other manifestations of diseases such as influenza, Aujeszky's disease and Porcine Reproductive and Respiratory Syndrome (PRRS), among others. However, clinical and laboratory results are necessary to obtain the correct diagnosis.

Disinfection with most common detergents and disinfectants will inactivate Chlamydiaceae. Current infections are being treated with antibiotics. Generally, tetracyclines are the first antibiotics of choice to control it. In case Chlamydia suis is resistant to those, quinolones (enrofloxacin) or macrolides (erythromycin) could be the second choice.

Facilities with poor ventilation and poor environmental hygiene can also cause conjunctivitis. Improvement in environmental conditions is the key to solve the problem. The presence of some mycotoxins, especially trichothecenes, are sometimes related to this problem. From this group, the most frequently observed are deoxynivalenol (DON) and T-2. In addition to the signs of conjunctivitis, intoxicated animals with trichothecenes will show other symptoms like vomiting, decrease in feed intake, reduced weight gain, weight uniformity in lots and high incidence of diarrhoea. The use of strategies for inactivation of mycotoxins, especially from the trichothecenes group, is important to control the situation.

Check list	Corrective action	
Potential cause: MYCOTOXINS (Trichothecenes)		
Chronic poisoning	 Check raw materials and feed 	
Positive raw materials ELISA, feed HPLC	Use Mycofix at suitable inclusion rate	
Potential cause: ENVIRONMENT		
 Smell and lacrimation Presence of high concentrations of toxic gases (NH3 and H2S) Excess of powder and dust 	 Adjust the conditions of hygiene, ventilation and relative humidity 	
Potential cause: CHLAMYDIA SUIS		
 Enteritis, pneumonia, pleuritis, pericarditis, arthritis, lameness, orchitis, uterine, infection, late abortion, stillbirths, mummification Carrier pigs, flies, dust, bird droppings 	BiosecurityHygieneDisinfectionAntibiotics	
Potential cause: PRRS		
Periorbital oedema	Biosecurity	
RT-PCR, ELISA, Indirect Fluorescent Antibody Test (IFAT)	Vaccination	
Potential cause: AFRICAN SWINE FEVER (ASF) AND CLASSICAL SWINE FEVER (CSF)		
Vomiting Epidemiology and clinical signs	BiosecurityStamping out	
Potential cause: INFLUENZA		
 Fever, lethargy, coughing, dyspnoea, infertility, decreased litter size, abortion, stillbirths Epidemiology, clinical signs RT-PCR and/or ELISA 	VaccinationAnti-inflammatory drugs	
Potential cause: AUJESZKY'S DISEASE		
 Keratoconjunctivitis, retinitis, optic neuritis, rhinitis, laryngitis, tracheitis, necrotising tonsilitis Epidemiology, clinical signs, dog or cat dead bodies PCR, ELISA 	Biosecurity Vaccination	
Potential cause: MYCOPLASMA HYORHINIS		
 Reddening of conjunctiva, crusting of eye lid margin by exudation, tearing, polyserositis, rough hair, slight fever, depression, reduced FI, respiratory distress, abdominal cramp, lameness and swollen joints PCR 	• Antibiotics	



Part 8: Jaundice

Jaundice, or icterus, is an increase of biliary salts in the blood that takes one of three forms. Pre-hepatic jaundice, or haemolytic icterus, occurs due to massive blood destruction that overwhelms the detoxifying capacity of the liver. Hepatocellular icterus comes from direct liver injury, and post-hepatic icterus is caused by obstruction of biliary drainage.

The main symptom is yellow colouration of white connective tissue in the body, skin or eye sclera, the latter being the only sign in pigs. Several infections can directly affect the blood or the liver: Leptospira (mainly foetuses), mycoplasma, E. coli and salmonella. In all cases other signs can help to address infective causes. Ascaris suum can also cause icterus through direct parasitosis of the liver with later migration to the lungs. At the abattoir white spots are evident in the liver.

Toxicoses such as copper excess and mycotoxins that primarily target the liver can lead to jaundice, particularly when aflatoxin and fumonisin concentrations reach high levels in feed. \checkmark

Symptoms	Detection
Factor: Mycotoxins: aflatoxins and fumonisins	
Aflatoxins: Reduced protein synthesis, lower productivity and immune function; coagulopathy; depression, anorexia, anaemia, ascites, haemorrhagic diarrhoea, rough hair coat, elevated alkaline phosphatase; claycoloured liver with centrilobular haemorrhage, fatty change, subserosal petechial to ecchymotic haemorrhages, intestinal and colonic haemorrhage; hepatomegalocytosis, interlobular fibrosis, biliary hyperplasia.	
Fumonisins : Reduced feed intake, hepatosis, pulmonary oedema, liver necrosis, bile retention, characteristic increased serum AST-GGT-bilirubin-cholesterol levels.	
Factor: Leptospirosis (foetal)	
Fever, anorexia, depression, infertility, mummification, abortion, stillbirth, weak born piglets, haemoglobinuria	Epidemiology, serology (MAT-OIE 2008), PCR
Factor: Mycoplasma suis	
Pallor, fever, cyanosis of extremities (ears), anaemia, poor growth, anorexia, decreased milk production, poor maternal behaviour	PCR, ELISA
Factor: Postweaning Multisystemic Wasting Syndrome (PMWS)	
Growth retardation, dyspnoea, enlargement of inguinal lymph nodes	Multifactorial
Factor: Ascaris suum	
Major cause of icterus in swine; liver milk spots, pancreatic duct obstruction, cholangitis	Eggs in faeces (flotation), liver milk spots
Factor: Copper (Cu) excess	
Anorexia, bloody faeces, reduced weight gain, haemoglobinuria, nephropathy with haemolytic crisis	
Factor: Haemolytic anaemia	
Immune-mediated mechanism, erythrocyte parasitism (Mycoplasma suis), erythrocyte fragmentation: Haemoglobinuria	
Factor: E. coli (ETEC) septicaemia	
Petechial haemorrhages serosal membranes, splenomegaly, secretory diarrhoea, dehydration	IHC, indirect immunofluorescence, ELISA, PCR
Factor: Salmonella choleraesuis	
Cyanosis of ears-feet-tail-ventral abdominal skin, enlargement of mesenteric lymph nodes, spleen: enlarged-purple-pulpy	PCR, ELISA

For more information, visit www.mycotoxins.info



Part 9: Tail necrosis

Tail necrosis is a common swine affliction that occurs unpredictably and jeopardises performance. Tail necrosis can happen in very young piglets during the first days of life up to finishing pigs, often leading to culling and condemnation of carcases at the abattoir. The primary damage may result from abrasion, fight wounds or tail docking. Causes of tail necrosis can be attributed to many environmental, nutritional and infectious agents that often coincide. A number of environmental factors can lead to tail necrosis including air humidity, small injuries from slatted or abrasive flooring, or a failure to rinse off strong (alkaline) disinfectants after use.

Pen mates may step on or bite the tail – a behaviour more commonly observed with a lack of foraging activity or manipulating materials. High stocking density and competition for space also plays a role, potentially in response to discomfort caused by ambient temperature (too hot/cold), a lack of draught free areas, or in an effort to secure feeding space.

Cross-fostering, tail clipping and other environmental stressors may incite frustration and see vices such as tail biting develop. In terms of nutrition, an imbalanced diet, certain deficiencies, for example biotin or tryptophan, or a craving for salt, protein or some specific amino acids can influence tail necrosis. Excess energy and intestinal discomfort may also be contributing factors.

In some cases, naturally occurring toxins may be the cause, including endotoxins, mycotoxins (aflatoxin, trichothecenes, ergot alkaloids) and biogenic ammines. Bugs can also be the culprit. Skin parasites (mites), streptococcosis (beta-haemolytic), staphylococcosis or erysipelas can be at fault. Bacteria penetrate into the skin causing inflammation and then block the blood supply, leading to necrosis. Prevention can be carried out by thoroughly revising management and feeding practices in order to avoid the identified environmental, nutritional and disease factors, and by maintaining good hygiene when tail clipping.

The only treatments that are available involve isolating the affected pigs and then providing local disinfection and parenteral antibiotics. \checkmark

Factor	Symptoms	Detection
Toxins		
Mycotoxins, for example: • Aflatoxin • Trichothecenes • Ergot alkaloids Endotoxin Biogenic amines	Depressed immune competent tissue. Reduction or refusal from direct neuronal depression of hypothalamic appetite nucleus, oral/dermal irritation, digestive disorders with ulceration and vomiting and bleeding visceral Agalactia, reduced piglet birth weight, piglet starvation, gangrene ears, tail, or feet Restlessness, trembling, recumbence, tachypnoea, pale mucous membranes, blue ears, shock, death, nausea, headaches, rashes and changes in blood pressure	Positive for Afla, DON, T-2, HT-2, FUM, Ergot; ELISA raw materials, HPLC feed. Origin of raw materials historically contaminated Epidemiology, signs, RT-PCR, ELISA for Gram-negative bugs Digestibility of proteins
Environment		
	Overcrowding, ventilation, temperature, hygiene and rinsing, manipulating materials, mixing, fostering, hierarchy	Revise management practices
Disease		
Mange	Skin irritation, rubbing, ear shaking	Lesions, skin sample microscopy. Epidemiology, signs, RT-PCR, ELISA
Staphylococcus aureus H	Listlessness, skin reddening, vesicles, pustules, faver	
Streptococcus	Depression, incoordination, paddling, opisthotonos, convulsions, nystagmus,	
Erysipelothrix rhusiopathiae	death	
	Depression, fever, stiff joints, anorexia, erythema	

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