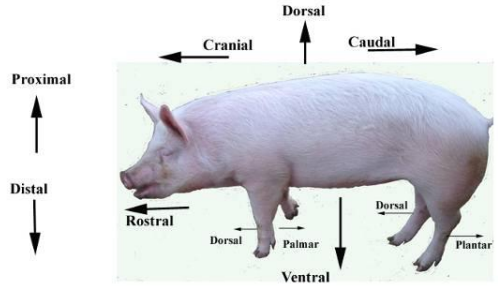
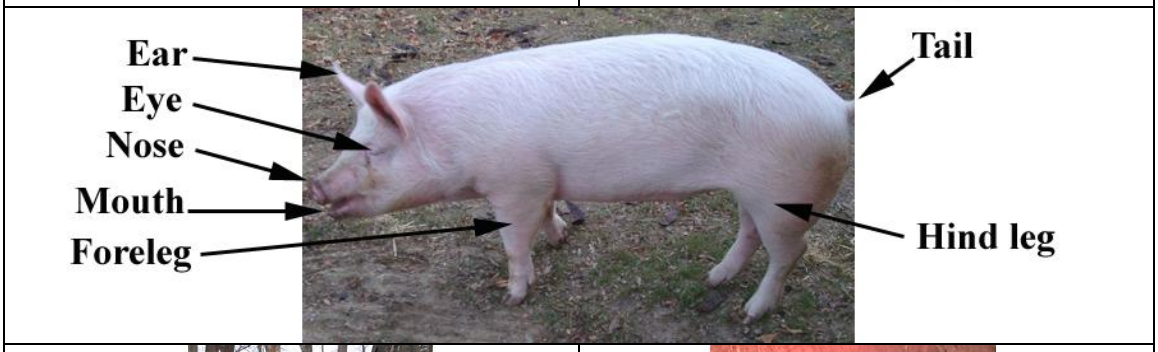
Disorders of the skin

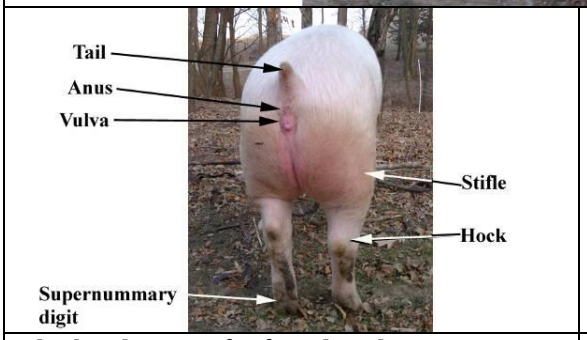
Diseases present in	Australia	Europe/Asia	North America
Anatomy of the skin			
Erysipelas	Yes	Yes	Yes
Foot and Mouth Disease	No	Yes	No
Greasy Pig disease	Yes	Yes	Yes
Herniation	Yes	Yes	Yes
Mange	Yes	Yes	Yes
Porcine Dermatitis and Nephropathy Syndrome	Yes	Yes	Yes
Swine Fever(s)	No	Yes	No
Tail biting and other vices	Yes	Yes	Yes
Other skin conditions			
Pityriasis rosea	Yes	Yes	Yes
Pig Pox	Yes	Yes	Yes
Ringworm	Yes	Yes	Yes
Epithelium imperfecta	Yes	Yes	Yes
Dermatitis parakeratosis	Yes	Yes	Yes
Abscess	Yes	Yes	Yes
Flaking skin	Yes	Yes	Yes
Areas of skin trauma	Yes	Yes	Yes

CLINICAL GROSS ANATOMY OF THE SKIN

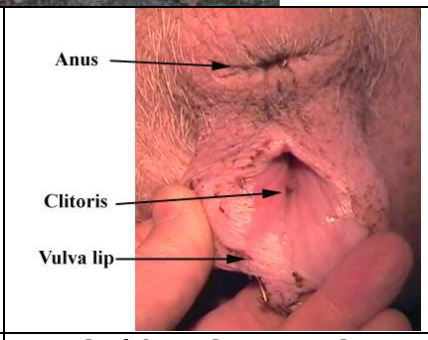
Describing the directions around the pig



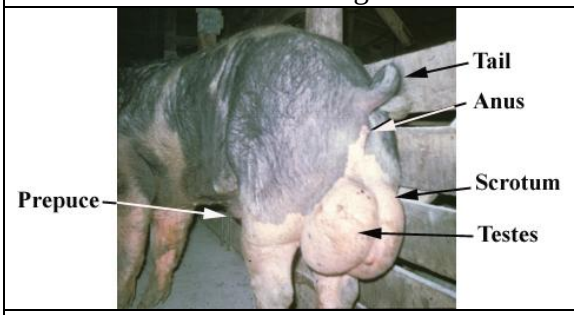




The hind view of a female gilt

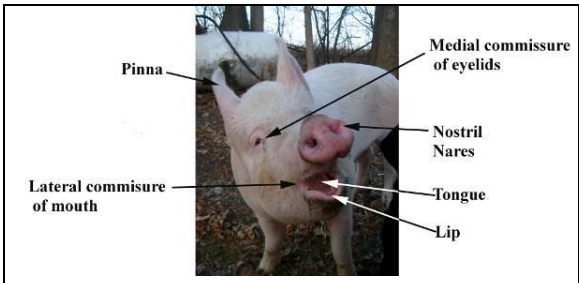


Detail of the vulva opened

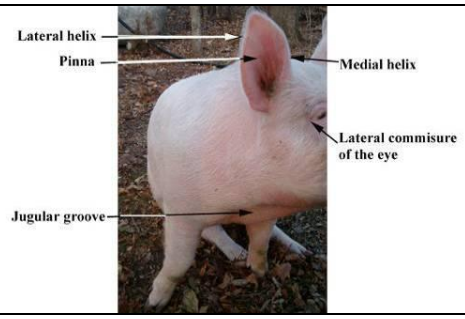


The hind view of a male entire boar

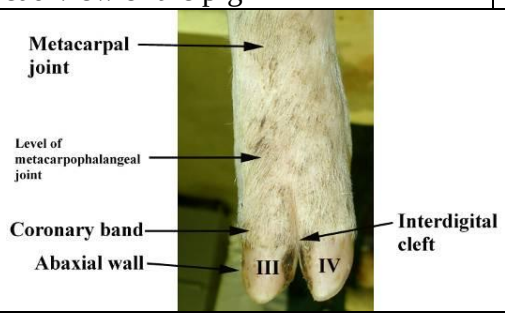
Detail of the prepuce of the boar



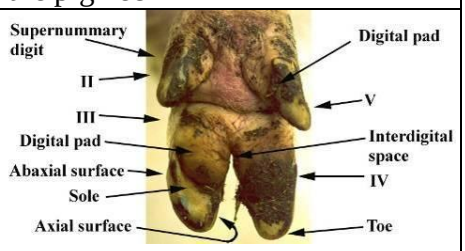
The head view of the pig



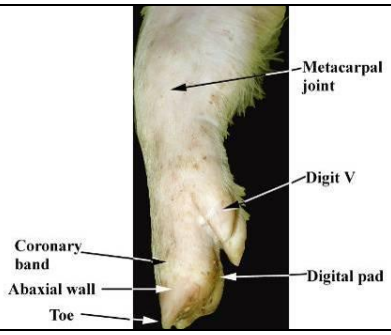
View of the pig neck



Detail of the dorsal surface of the front foot



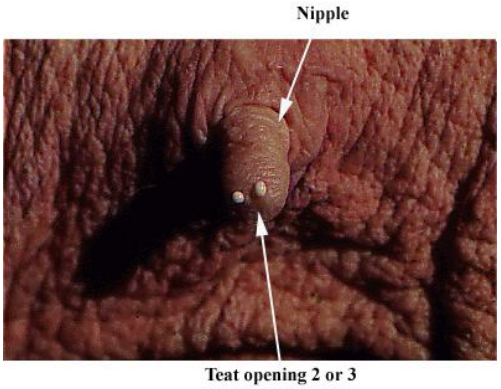
Plantar view of the foot



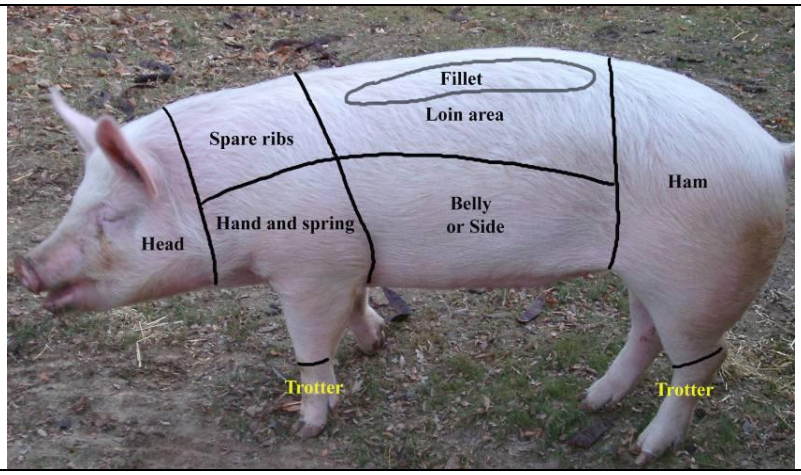
Lateral view of the front foot

Carpal glands

Lateral view of the mammary glands

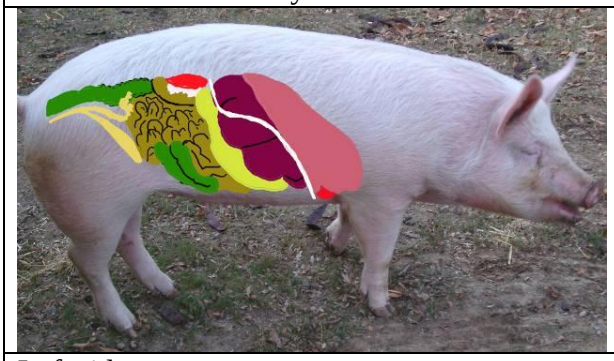


Detail of the teat with milk from the supplying mammae being expressed

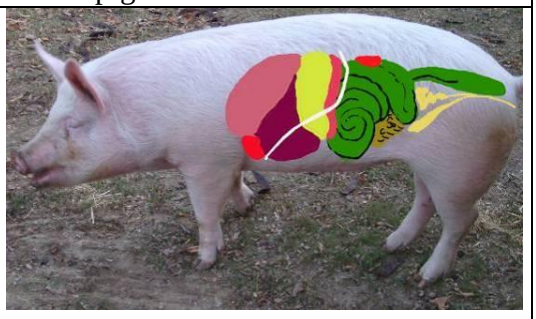


Detail of the major meat joints of the pig

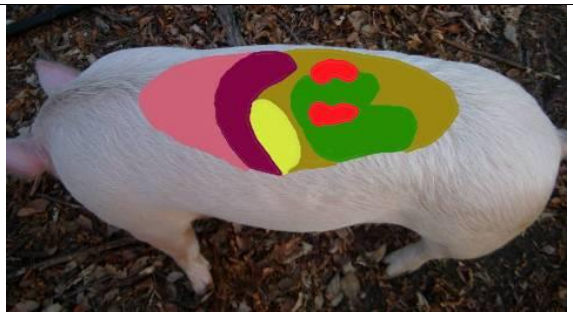
Outline of the anatomy reflected onto the surface of the pig



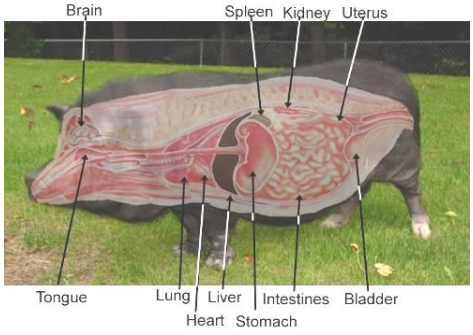
Left side



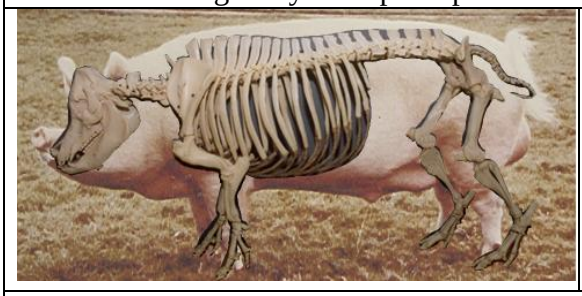
Right side

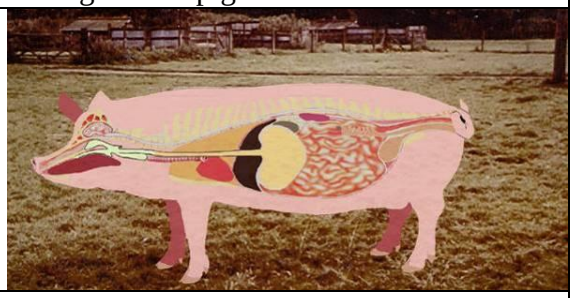


Dorsal view

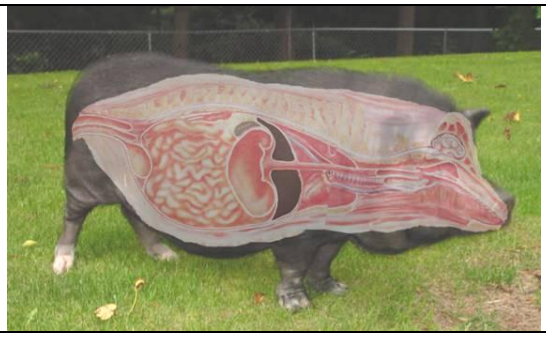


Comparison of the normal commercial pig (Large White/Yorkshire pig) with the pet Vietnamese Pot Bellied pig
Skeleton and organ layout superimposed on the image of the pig





The commercial Large White



The Vietnamese Pot Belly

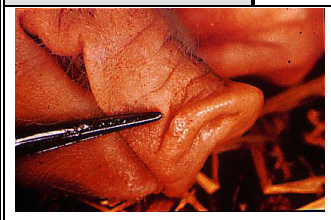
ERYSIPELAS

Other names	Diamonds, Diamond skin disease, Measles,
Causal agent	Bacteria ï *Erysipelothrix rhusiopathiae*. There are over 25 serotypes, however types 1 and 2 are the most common
Age group	The disease can affect any age group. However, most weaners below 12 weeks of age are protected from colostrum antibodies passed on from their mother
Clinical signs	
	There are five phases of the disease recognised
Peracute	Pig found dead with no clinical signs
Acute	The disease starts with a sudden onset.
 The classic diamond lesions of erysipelas	The animals present with a high temperature (40-42C). The infected pig is often separated from the rest of the group and may appear chilled and cold (typical of a high fever). The pig is generally found lying down and when encourage to rise will rapidly lie down again. The pig appears to have a sore abdomen, walk stiffly and tucked up. The pig generally appears very dejected. The pigs are often off their feed and may be constipated (although young animals may have diarrhoea). Sows may abort with the high temperature. Boars may become infertile, which may be permanent or last 6 weeks. 2 to 3 days after initially becoming infected pigs develop diamond skin lesions which can be pink to dark purple. The lesions are generally raised which may be the only way to diagnose the disease in black pigs. Pigs untreated may die or start to recover in 4 to 7 days. The skin lesions may go necrotic in the centre. The more severe diamond lesions tend to indicate a poorer prognosis.
Subacute	The pig presents with diamond lesions with few other clinical signs including no loss of appetite.
Chronic	Two forms: arthritis and endocarditis affecting the heart
Endocarditis 	The pig develops breathlessness and poor circulation especially after exertion. This can result in sudden death, especially after mating for instance. The pigôs ears and tails may go purple with the poor circulation.
Arthritis	3 weeks after infection the pig may present with a chronic lameness in one or two joints. It can affect the vertebrae and thus the pig has a sore back and difficult painful walking.
Carrier	Some 20 to 50% of pigs may carry the organism on their tonsils

Infectivity	
	Many pigs carry the organism on their tonsils without any clinical signs
	Soil, bedding, faeces and drinking water can become contaminated
	Stressed pigs are more likely to show clinical signs, cases are more likely after pigs have become stressed - sudden changes in diet, sudden changes in temperature or introduction of other disease such as Swine Influenza
	The organism can cause problems in turkeys which can then cross-infect pigs
	The organism also lives in fish and fish meal can be an infected source
	Poor vaccination may also result in unvaccinated pigs that are believed to be covered, classic reasons are vaccines inactivated by being frozen in a poorly maintained ófridge.
Post-mortem Lesions	
Peracute	May have very few lesions. Possibly an enlarge spleen
Acute	The skin lesions may be seen or felt. The spleen is generally very enlarged
Chronic-heart	The endocarditis is seen a cauliflower lesions growing on the heart valves. The heart may be enlarged associated with the poor circulation
- arthritis	Severe arthritis in one or more joints
Pathogenesis	
	The organism can gain access by many routes. Classically most infections are via the mouth from contaminated feed and water.
	In acute cases the disease enters the blood stream via the pharynx and infects the blood vessels hence the widespread clinical signs. The diamond lesions are actually an immune response. The circulation to the diamond lesions can be so compromised that the centre may die and become necrotic.
	In the chronic form arthritis can take months to develop and therefore diagnosis can be difficult as the lesions are sterile.
Diagnosis	
	Clinical signs
	Response to penicillin in suspected pigs. With penicillin the pig should response within 24 hours.
	Bacterial culture can be done on acutely infected pigs
	In chronic cases the organism can be difficult to isolate and serology may be useful.
Treatment	
Acute/ subacute	Penicillin and Tylan based medicines are very effective in the treatment of erysipelas.
Chronic	There is no practical treatment excluding treatment with pain killers in the arthritic form. Arthritis is generally permanent
Prevention	Vaccination is a cheap and effective control measure
	Vaccination lasts about 6 months, therefore the following programme is recommended: Pigs over 3 months vaccinated once and again 3 weeks later
	Sows vaccinated either pre or post farrowing or every 6 months
	Boars vaccinated every 6 months, the boar is often forgotten
Common differentials	
Acute	Swine Fever. Salmonellosis. Anthrax. Other causes of sudden death. Food allergic responses
Chronic	Arthritis ï Mycoplasma arthritis. Endocarditis - Streptococcus
Zoonotic	
	Erysipelas can infect humans and infection usually only results in a skin infection, however, the condition can be more severe.

FOOT AND MOUTH DISEASE
AND OTHER VESICULAR DISEASES

Other names	Foot and Mouth Disease - FMD Swine Vesicular Disease - SVD Vesicular stomatitis-VS Vesicular Exanthema of Swine and San Miguel Sea Lion Viruses
Causal agent	Foot and Mouth Disease - Virus Picornovirus (Aphthovirus) Swine Vesicular Disease - Virus Picornovirus (Enterovirus) Vesicular stomatitis - Virus - Rhabdovirus (Vesiculovirus)
Age group	All ages of pigs can be affected
Clinical signs	
Foot and Mouth is the definitive disease, the other diseases present with similar signs which may be confused with foot and mouth	
	Incubation period 1 to 5 days but can be 21 days
	Fever to 40.5°C
	Skin around the snout, lips, tongue, inside the mouth, around the coronary band and the soft skin on the feet becomes whiter (blanched). Vesicles may develop on the sow's teats
	Vesicles (blisters) develop
	Vesicles rupture up to 24 hours after development and if no secondary infection occurs healing is rapid
	The animals are lame. Lesions in the mouth may not occur obviously in the pig
	With the feet, the hoof may become detached, revealing the painful raw tissues underneath. The hoof can re-grow, but is often deformed. This can take several weeks
	The disease affects nearly all susceptible animals, but few animals will die specifically with the disease



One day un-ruptured vesicle on the snout

Ruptured vesicle on the snout

Horn separation

Ruptured vesicles and some hoof separation

Blanched un-ruptured vesicle

Vesicles on feet

Pig lame with FMD

Infectivity of Foot and Mouth	
	Affects all cloven-hoofed animals - pigs, cattle, sheep and goats. VS also affects horses
	Rapidly spread through the air, animal contact and vectors, such as clothing, utensils, vehicles
	Can be spread through meat and meat-by products, especially fast frozen feeds
	Spread through semen
	High humidity, cloud cover and moderate temperatures favour airborne spread (over 20 km)
	Pigs produce aerosols 3000 times more concentrated than cattle
	Carrier status occurs in cattle. FMDv can be excreted in the milk for up to 7 weeks
Post-mortem lesions	
	Vesicles, generally ruptured, in the mouth, nose and on the feet
Treatment and control	
Treatment	Notify your vet and government official if clinical signs are suspected
	None. In endemic areas a vaccine is used
Control	Strict regulation of importation of animals and animal products infected with vesicular diseases
	Euthanasia and disposal of animals- burial, composting, rendering or burning
	  
Zoonotic implications	
	Human infection does occur but is extremely rare, often without any clinical signs

GREASY PIG DISEASE

Other names	Exudative epidermitis
Causal agent	*Staphylococcus hyicus* plus fighting
Age group	3-20 kg typically. A chronic form may be seen in adults.
	At any age wounds that do not heal properly may have a localised region of greasy pig disease
Clinical signs	
Classic Picture	Newly weaned pig suddenly presents covered in patches of dirty brown greasy wet skin. The hair is matted and may become a grey colour. The condition extends rapidly, covering the whole body. The pig stops eating and drinking and becomes very dehydrated. After a week to 10 days the pig may be found dead.
	  
Chronic	The pig presents with patches 3-5 cm of the above skin condition but the disease does not spread. The condition is most common/severe affecting the upper neck and hind legs - areas where the pigs fight 
Piglet	Facial necrosis is a form of greasy pig disease associated with poor milk output resulting in excessive fighting between the piglets and damage to the face 
Adults	A chronic black spotty appearance on the back and neck of sows is often associated with *Staph. hyicus*.
Infectivity	
	Nearly all pigs carry *Staph. hyicus* on their skin.
	Infection is from the mothers shortly after birth. Infection can even occur during birth
Post-mortem Lesions	
	A severe if local exudative epidermitis.
	In severe acute cases lymphnodes may be swollen and abscessed.

Diagnosis

	Clinical examination of the animal
	Culture relatively meaningless as culture from normal skin also positive

Treatment

Individual	Isolate and place in compromised pig pen
	Inject with a staphylococcus active antimicrobials which concentrate in the skin ï lincomycin for example
	Wash pig in Savlon or other disinfectant. Ideally with Lanolin in the wash to soothe the skin
	Inject with multivitamins
	Provide ad lib water through cube drinkers and if necessary provide extra water by mouth. The animals are quite dehydrated. Note a pig may drink 1 litre per 10 kg, therefore a couple of syringe fulls will not be significant
	Provide heat from a light source and clean dry straw
Control	Greasy pig disease is the end result of fighting
	Review all causes of fighting and increased aggression
	Check feed space availability
	Check water supply number of drinkers and speed of flow
	Check for draughts and piling
	On rare occasions it has been necessary to change genetics to a more sociable pig
	Reduce mixing and moving
	Check fly control in particular look for biting flies ï *Ophyra calcitrans*
	In herd ówoutbreaksô it is possible to control by adding lincocin to the water supply together with a sweetener to encourage the pigs to drink
	Control mange on the farm
	Ensure feedback programme and colostrum management adequate in piglet cases in the farrowing house

Fighting over water

Fighting over feed space

Common differentials

	Severe Pityriasis rosea, parakeratosis associated with zinc deficiency Mineral deficiencies ï generally a milling mistake

Zoonotic

	None

Herniation

There are four common forms of herniation normally seen in the pig

1. Umbilical hernia

A congenital defect with some genetic basis. Can occur as a major problem on pig farms. Normally clearly recognised over 30 kg when the hernia can reach a gigantic size. The animal only has an economic future if the diameter stays below 30 cm and none of the skin is ulcerated. Once the hernia makes contact with the ground, euthanasia in advised. Umbilical hernias require a casualty certificate. There is no economically viable treatment.

<table>
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<tr>
<td>  </td>
<td>The umbilical hernia in the left top is too large to sell and is ulcerated. The hernia above is too large and euthanasia is to be advised. The hernia on the left is a small hernia.</td>
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</table>

2. Trauma hernia

Typically occur due to sow biting the piglet results in a hernia through the abdominal wall. Only of consequence if intestinal strangulation occurs. If the conformation is so badly disfigured that it may result in problems in the slaughterhouse, immediate euthanasia is advised.

3. Inguinal hernia

These normally occur in the male which has a very large inguinal canal. They can be very large. Rarely strangulation of a portion of the intestines can occur through the hernia. Assuming the animal is not castrated normally these animals will grow without problems to a slaughter weight. If castration has to be performed ensure that the pig does not have a scrotal hernia and then carry out a closed castration.

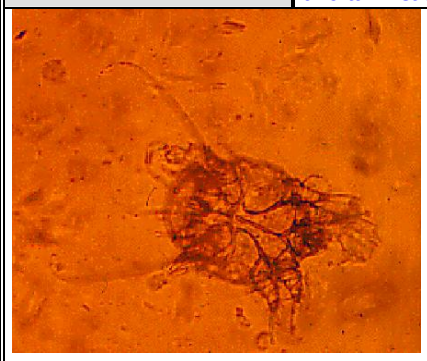
Scrotal hernias appear more common in Vietnamese Pot Bellied pigs

4. Perineal hernia

In sows the whole of the perineal region can present in collapse. The rectum and vagina may prolapse into the hernia. The hernia can be very large. There is no economic treatment. If the sow is close to farrowing keeping until farrowing may be an option, however, manual removal of piglets are likely to be required. Provide the sow with a bran diet or add liquid paraffin from time to time to help with the passage of faeces until slaughter

MANGE

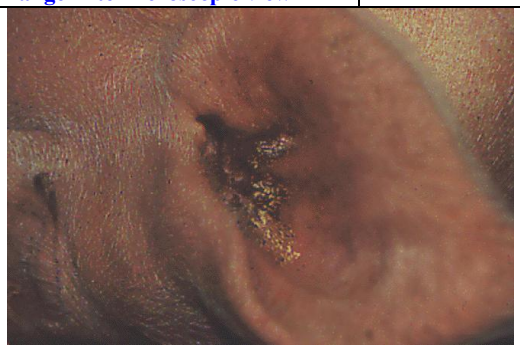
Causal agent	*Sarcoptes scabiei* var. *suis*
Age group	Affects all age groups, although sows and growing pigs most often exhibit the characteristic clinical signs
	World wide distribution with some 70% or more herds infected
Life cycle	
	Eggs are laid in the skin of the pig. The female lays about 1 to 3 eggs a day and the adult female lives for about a month; therefore some 30 to 40 eggs are laid per female. Most of the eggs are laid in the soft tissues of the inside of the ear. There may be as many as 18,000 mites per gram of ear skin.
	The eggs hatch out in about 5 days
	The larvae moult to the nymph, which moult to the adult in 10 to 15 days. The life cycle remains on the pig at all times
Clinical signs	
	Scratching
	Piglets may be uncomfortable and have intermittent body scratching
	A few weeks later the weaned and growing pig demonstrate persistent itching and rubbing
	Ear wax increases, sometimes forming plaques
	Chronic lesions may occur with thickened skin, hair loss and abrasions, especially behind the ear and tail head.



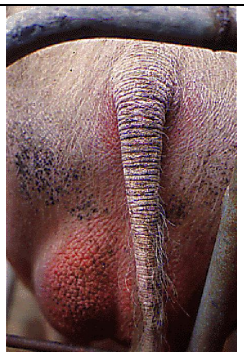
Mange mite microscopic view

Itchy pig

Examination of the ear reveals lots of wax



Chronically infested ear with yellow plaques



Chronically infested thickened skin

Infectivity

	The disease is spread through pig to pig contact and through pigs coming into contact with infested buildings
	The mite is able to survive 21 days off the host in ideal situations. The warmer the conditions the shorter the survival time.
	The pig mange mite does not live in or on other hosts

Economic Importance

	Economic importance varies depending on infestation, but a loss of 10% growth rates is not unusual in moderate to severe infestations. Mange will weaken the pig and is an added stress. Note the constant rubbing causes damage to buildings

Diagnosis

	Examination of ear wax and scrapes from the skin of the inner ear. However, individuals may be necessary to examine to find evidence of the mite to confirm the diagnosis. Absence is very difficult to ascertain
	An ELISA test is being developed
	Examination of the skin of finishing pigs in the slaughterhouse
	The problem may be more apparent in the cooler months

Skin score 1

Skin score 2

Skin score 3

Treatment

Control	Use Ivermectins via various routes. Note failure to adequately treat large boars is a common reason for failure to provide adequate control
Eradication	Where possible mange should be eradicated from units. Need to purchase animals from mange free farms

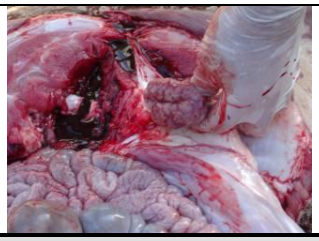
Common differentials

	Sows may scratch when exposed to cigarette smoke or perfumes including after shaves
	Forage mites in straw/bedding
	Other causes of skin hypersensitivity / allergy
	Skin may be thickened with parakeratosis or dry and scaly with deficiencies of essential fatty acids
Zoonotic	None

PORCINE DERMATITIS AND NEPHROPATHY SYNDROME

Other names	PDNS
Causal agent	Unknown, the causal agent(s) has not been recognised. The disease is suggestive of a type III hypersensitivity reaction. The role of circovirus II is as yet undefined. Association with *Pasteurella multocida* A electrophorectic type 01.
Age group	The problem classically affects pigs from 40 to 70 kg (12 to 16 weeks of age). It has been seen occasionally in adults
Distribution	Reported worldwide especially with acute PMWS.
Clinical signs	
Normal farm	The condition occurs sporadically
PMWS farm Clinical signs	Since the occurrence of PMWS the condition can reach a prevalence of 10%.
	The pigs show anorexia, depression and lie down a lot with a stiffened gait and may have problems rising.
	The most obvious clinical sign is the presence of irregular red to purple patches (macules and papules) in the skin, particularly around the hind legs and perineal area. The lesions tend to merge with time and if the pig survives scarring may occur.
	Pigs affected before 10 weeks of age (30kg) die. Pigs older than 10 weeks mortality may reach 25% and pigs generally die within a few days of showing clinical signs.
 	Two pigs with PDNS with the characteristic red blotchy lesions. Note particularly affecting the hind area
Infectivity	
	As the condition is an allergic response, treatment is generally not infective
Transmission	
	Causal agent not yet recognised

Post-mortem Lesions

Skin lesions	As described in the clinical signs	
Kidney lesions		Bilateral enlarged (2-3x normal) and pale kidneys with cortical petechiae. Histological examination reveal acute glomerulonephritis and systemic necrotising vasculitis.ï looking very like Swine Fever
		Lymphnodes around the pig may also be much enlarged with typical PMWS changes. The association with PMWS and PDNS is still unsure.

Diagnosis

	Striking skin clinical signs
	Definitive diagnosis following renal histology

Treatment

	None
	Control PMWS, which as yet there are few real strategies that are effective. Ensure that management is excellent.
	Corticosteriods may help recovery

Common differentials

	Classical Swine Fever, African Swine fever, Possibly salmonellosis

Zoonotic

	None

THE SWINE FEVERS

Other names	Swine Fever - Hog Cholera, CSF, Swine Fever African Swine Fever - ASF
Causal agent	Classical Swine Fever - Virus - a Flaviviridae, genus Pestivirus. Enveloped RNA virus African Swine Fever - Virus - Enveloped DNA virus related to Poxviruses
Age group	Any age group of pig can be infected with CSF or ASF
Clinical signs	
	It is not possible clinically to distinguish between CSF and ASF
Naive herds	Initially a few pigs appear drowsy and less active, with some anorexia and they may appear chilled
	Within days, pigs will present with a marked fever (41-42ÅC), sometimes with a reddening of the skin
	The pigs develop a conjunctivitis and constipation leading to yellowish diarrhoea
	The pigs appear chilled and will huddle together
	A few pigs may convulse before they die
	Pigs start to die with a spreading purple discoloration of the skin. Death often occurs some 10 to 20 days post-infection
	Pigs which survive will be chronically affected with severe retardation of growth and often present with arched backs
	In the adult herd, returns, abortions, and an increase in mummified and stillborn piglets
On established herds	
Congenital infection	Piglets infected from their mothers during pregnancy can result in abortion, mummification, malformations (may present with a congenital tremor with cerebral hypoplasia with Classical Swine Fever), stillbirths and weak born piglets. Piglets born from CSF infected mothers may remain healthy but continually spread the disease throughout their lives
Rest of the herd	An almost in apparent infection can also be present on chronically infected herd. These herds can be very difficult to identify
Diagnosis	
	Your vet and the government vets must be informed of any suspicious clinical signs
Treatment	
	None
Prevention	
	Prevent any pork products entering the farm and being fed to the pigs
	Prevent any infected pigs entering the farm
	All pigs from infected herds are slaughtered and destroyed and the farm intensely disinfected
	In ASF areas, control ticks and flies that may transmit the disease
	In endemic parts of the world vaccines are available
Common differentials	
	PDNS, Salmonellosis, Acute pasteurellosis, Erysipelas, Acute septicaemic streptococcal infections, PDNS, Thrombocytopaenia, Warfarin poisoning. Reproductive diseases. Other causes of congenital tremor.

Piglet with multiple haemorrhages over the skin

Infectivity

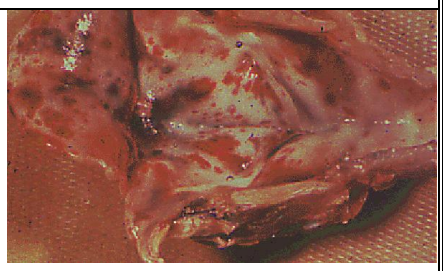
Both 	The virus is able to survive in uncooked and cured pork and pork products for months
	The introduction of new animals or pork products from infected herds, into a herd is the most likely source of infection
	The diseases can be carried on boots, vehicles, clothing, and also pets and birds
	The diseases can be carried by wild boar
Classical Swine Fever	The virus is quite resistant in the environment, surviving a couple of days
	The virus is quite readily inactivated by approved disinfectants
	Other members of the Pestivirus genus can cause disease in pigs, notably Bovine Viral Diarrhoea and Borderôs Disease.
	The virus is excreted from pigs for 10-20 days post-infection in large amounts
African Swine Fever	The virus is very resistant in the environment, surviving for months
	The disease can be spread by ticks (Ornithodoros species in Africa)
	Recovered animals remain infective for at least 6 months
	The virus is inactivated by approved disinfectants

Post-mortem Lesions

Peracute	The pigs may die so rapidly that there are few post-mortem signs
Acute	Multiple haemorrhages throughout the carcase
	Swollen, oedematous and haemorrhagic lymph nodes
	Infarction of the spleen (large areas where the blood supply has been cut off resulting in blood filled blebs on the surface of the spleen)
Chronic	In CSF ulceration (button ulcers) can be seen in the large intestine

Button ulcers in the large intestine

Haemorrhages in the bladder



Haemorrhages on the epiglottis and larynx

Splenic infarcts

Petechial haemorhages kidney

Note the feeding of waste feed, including household scraps, unless it is cooked in a plant operating under a licence is prohibited in many countries.
Several countries have now banned the feeding of any waste feed containing mammalian meat proteins.

Tail Biting and other Vices

Causal agent	None specifically
Age group	The demonstration of vices can occur in all age groups

Clinical signs

Facial necrosis

- Tail biting - grow/finishing pig, rarely in adults
- Flank biting ï grow/finish
- Vulva biting - adult females when loosed housed
- Ear sucking/ear biting ï in nursery pigs
- Penile sucking ï newly weaned pigs
- Bar biting and other stereotypyôs

Tail biting

Flank biting

Vulva biting

Ear biting

Ear sucking

Penile sucking

Causes

Check the following:	Stressed and deprived pigs
	Check stocking density. Check tail length, in particular variability. Check feed particle size (target> 500 µm). Check salt (NaCl) concentration in feed. Check water supplies. Check for evidence of a draught at pig heights (draft air speed > 0.2 m/sec). Check air quality (target - NH_3 < 20 ppm H2S < 10 ppm and CO_2 < 3000 ppm). Check humidity (target between 50 and 75%. Check light intensity. Check water supplies. Check feeder space availability. Check 24 hour temperature fluctuations. Mixing pigs. Moving pigs. Facial necrosis associated with lactation failure.

Treatment

Find offending pig ï this may be difficult

Look for the gaunt smaller middle order pig, often with chronic mild diarrhoea

Remove affected pigs to a hospital pen

Treat with sprays/wound dressings

Consider euthanasia if pig severely affected, lame or has other abscesses

Control

Review environmental factors

Increase salt (NaCl) concentration to 0.9% - ensure the water supply is excellent

Air	in particular draughts ï 90% association with tail biting Gasses ï in particular NH_3, CO_2, CO Weather changes ï high pressure Inappropriate/variable temperatures
Water	Fighting over inadequate water Urine concentrated in sows makes vulva biting more likely Water trough placement in sows
Feed	Check for mycotoxins Fighting over feed availability
Floor	Check stocking density ï both under and overstocking Inadequate sleeping area
Stock	Some genetics may be more aggressive in some environments

Provide distractions through toys ï chains for example

Improve pig flow ï remove under and over stocking

Check tail docking principles ï pigs do not like variable pig tail lengths

Chains can provide great distraction for pigs

Tires should be avoided as they contain metal parts and may block parts of the pen

Facial necrosis

Enhance lactation output ï three major areas to examine:
Overfeeding in gestation, poor water intake in lactation and too high a farrowing house room temperature.

Post-mortem findings

Injury to the skin. Sequelae to vices include ï pulmonary millary abscesses, vegetative endocarditis, bacteriaemia, spinal abscessation and single or multiple discrete abscesses throughout the body

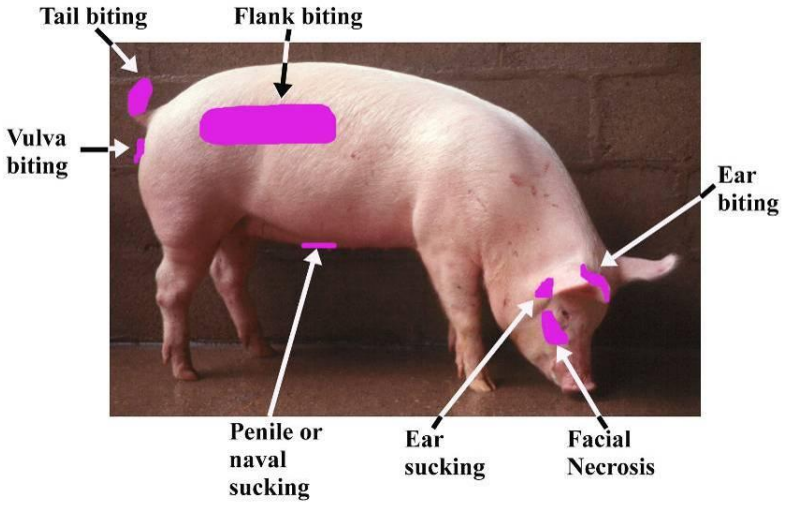
Areas of Vice

Tail biting

Flank biting

Ear biting

Vulva biting



Penile or naval sucking

Ear sucking

Facial necrosis

ADDITIONAL SKIN CONDITIONS

Pityriasis rosea

A genetic condition which suddenly appears in pigs 10-60 kg. The animal presents with scabby lesions over its body, in particular the ventral abdomen. The lesions are often in rings with a red raised edge and a blanched centre. With time the lesions may grow and coalesce. The pig is not ill and grows normally, although looks quite alarming. No treatment is necessary. Rarely does the condition present by the time of slaughter. It is wise not to breed from afflicted animals.

Pig Pox (Swine Pox)

Associated with a pox virus. This is generally seen as small circular scabs 10-20 mm in size. Occasionally small vesicles may be seen. The disease is probably widespread on most farms. Can occur as a herd epidemic problem. The pigs recover in 10 days. Provide skin disinfectant washes to control secondary infections. Improve basic pen hygiene.

Ringworm

The pigs show characteristic round light brown gradually spreading circular lesions on their bodies. Healing can take several weeks. They otherwise demonstrate no undue clinical signs. If treatments, is necessary then wash pigs with skin disinfectant or in a herd situation consider the use of antifungal antimicrobials such as griseofulvin may be used.

Epitheliogenesis imperfecta

Pigs are born with a portion of their skin is missing. Assuming the lesion is not too extensive treat with skin disinfectants and the lesion will progressively heal. Even the pig shown healed with only a scar area visible by the time of slaughter.

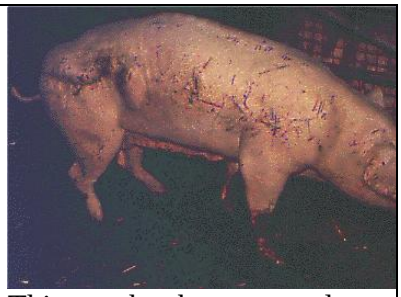
Dermatitis parakeratosis

Classically associated with zinc deficiency or calcium excess. Problems generally arise because of a feed mixing problem, particularly on home mill and mix units. The pigs present with a variety of behaviour changes from nibbling excessive vice and pica with licking of the walls and floors. The skin breaks out into a proliferative dermatitis resembling greasy pig disease. The legs can be particularly affected. The condition is generally seen in a large group of pigs all on the same feed. Treatment is through providing a properly balanced feed.

Abscesses

Pigs are prone to subcutaneous abscesses which can be very large ï containing 6 litres of purulent material for example. The abscess can be released once the contents are fluid, which is assessed by inserting a clean needle into the softest part of the lump and drawing back with a 10 ml syringe to reveal a yellow creamy liquid. If the abscess contents are fluid, release using a cross-cut at the bottom of the abscess, not at the point. It is essential that the skin wound does not heal too fast as the abscess will reappear. The cut at the bottom allows adequate drainage; no pocket of abscess should be left. Flush with running water 2-3 times daily. If necessary inject with routine antibiotics to reduce secondary infection. In the early stages of an abscess, possibly injecting with lincomycin may clear the infection. Review causes of fighting among stock to try and eliminate the cause of the abscess. However, pigs will fight when housed together and abscessation is an inevitable consequence.

Following oil based vaccination a granuloma may appear in the neck. There is no specific treatment possible. Review your injection technique and hygiene.

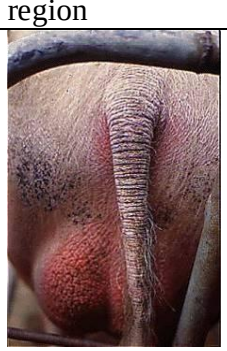


This sow has been severely beaten by her pen mates and the cuts have become infected with multiple abscesses evident. Treatment in such cases is futile and euthanasia is preferred.

A large abscess in the hip region

Flaky skin

It is not unusual for adults to present with dry flaky skin. Mange as a cause should be ruled out by treatment. If the flaky skin presents a problem, to the owner generally more than the pig, wash the pig with a skin disinfectant. Add cooking oil/olive oil to the pigôs diet to increase the fat content which will be expressed on the skin. Several pet pig diets are quite basic, to reduce calories, to reduce pigs putting on too much weight and to keep costs down and thus are short in essential oils.



Scaly skin on the tail head which was associated with chronic mange

Thrombocytopaenic purpura

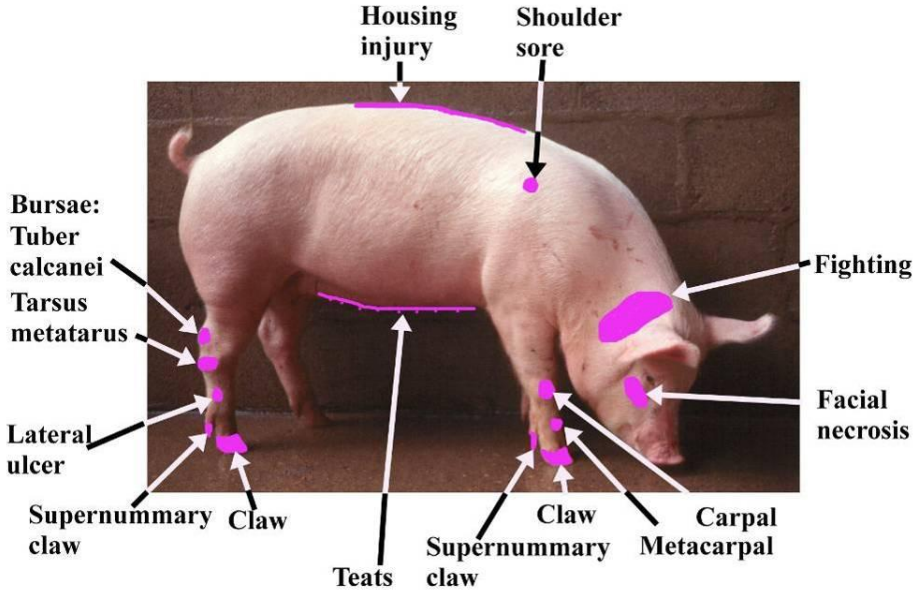
Seen only in young piglets from 3 to 10 days of age. Due to sowôs colostrum contains antibodies to the pigletôs platelets. Piglets present with death which on close examination reveal small haemorrhages on the skin. Post-mortem examination reveals small haemorrhages throughout the carcase (as shown in the photograph). Remove surviving piglets to another sow.

AREAS OF SKIN TRAUMA

Tuber ulcer

Housing injury

Shoulder sore



Lateral ulcer

Teat trauma

Facial necrosis

Bursa

Claw injury

Carpal injury