

VETS 4223

Pig Health and Production



UNIT OF STUDY HANDBOOK
2009

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Chapter 1

GENERAL COURSE INFORMATION

WELCOME

*“Dogs look up at you, cats look down at you, while pigs look you straight in the eye” –
Churchill*

We welcome students to “Pig Health and Production”. It’s been a long time since you were first introduced to pigs via “Animal Husbandry”. In this 4 credit point Unit, we will build on and apply principles and learning methodologies from previous disciplines to the study of all things porcine. The pig industry is dynamic and challenging. Veterinarians who choose to devote themselves to it have to become cognizant of factors driving its sustainability-from cost-effective production of pig meat to welfare-friendly housing of pregnant sows. Not only will you have to act as veterinarian, but also be nutritionist, economist, geneticist, environmentalist and reproductivologist! I hope that at the end of this Unit you will have the confidence to consider a career in the pig industry and an understanding of how the industry works.

UNIT OF STUDY AIMS/GOALS

Pig Health and Production takes basic principles learnt in the first three years of the veterinary course, and applies them to one particular species-the pig. Students will gain an understanding of the role that veterinarians play in the pig and pig meat industries and within the general public. This Unit has a high commercial focus, so that graduates will be empowered with the ability to assist pig producers produce low-cost, quality-controlled food made from pork. Students will also become familiarized with the major external factors driving the sustainability of the industry, particularly from the consumers’ standpoint.

LEARNING COMMITMENTS

Students are expected to participate in all scheduled classes and to engage in learning activities, independently and in groups, outside timetabled hours.

- Daytime attendance – 1-4 hours of lectures per week plus 2 practical sessions (2-3 hours duration) and a 2-hour tutorial during the semester
- On-line commitments - 6 hours of computer-based independent learning (1 USYD eLearning/formerly WebCT exercise). This is a group exercise.
- It is also anticipated that each student will spend time outside of daytime attendance in lecture preparation and/or revision (approximately 30 minutes per lecture)

LEARNER PREPARATION

All students should have basic skills in handling, restraining and performing simple management activities with pigs (covered in Animal Husbandry). They should also have completed some professional experience on farms or animal management properties.

It is assumed that students will be able to utilise and apply the concepts and information introduced in Animal Husbandry.

Students should have well developed skills in seeking and obtaining information from a variety of sources, including the University library electronic resources, the internet, textbooks and journals. They should be able to communicate effectively (written and oral) in English.

- Students should be able to use a personal computer to access the University of Sydney web sites, search electronic databases in the University library, type up an assignment and use USYD eLearning/formerly WebCT.
- Students should be able to communicate effectively in English to a variety of different audiences. They are expected to be proficient in written and oral communication.
- Students must understand and abide by the University of Sydney's policies on plagiarism.
- It is expected that students will adopt a mature, independent approach to manage their own time and ensure that they actively engage in learning during the Unit. Students who experience difficulty in time management, assessment stress, learning disabilities or personal problems should seek assistance from the University's counselling and disability services.

LEARNING OUTCOMES

- 1) Problem-solve poor biological productivity and excessively high pork production costs on pig farms.
- 2) Diagnose, treat, control and eradicate common pig diseases in Australia.
- 3) Understand factors that affect the sustainability of the pork production industry, including food safety, welfare, prudent antibiotic use and meat quality.
- 4) Explain how exotic diseases can be introduced into pig herds and be able to recognise and act appropriately to minimise their spread.
- 5) Undertake clinical interventions in pigs (blood collection, pregnancy diagnosis, intravenous catheterization).
- 6) Necropsy a pig and conduct health checks at processing for disease diagnosis.

GROUP EXERCISE ON USYD eLearning/formerly WebCT

Students will work in groups of 4-5 (you select your own group members) to undertake a USYD eLearning/formerly WebCT problem-solving exercise. You must answer the questions and submit your answers to the Unit of Study Co-ordinator (maximum of 2000 words plus references). Due date for this will be advised during the semester, but it is anticipated that you will have about 5 weeks to do this exercise. Groups who do not submit reports by the due date will lose 10% of their mark for each day they are late. Students should refer to class notes and published literature in peer reviewed journals when resourcing reference materials. Reference material from Web Sites is a less preferred option for this.

The **Harvard Flinders Referencing System** is recommended for faculty assignments. Check this style at: <http://www.lib.flinders.edu.au/services/infolit/nureference.pdf> *or*

<http://vip.library.usyd.edu.au/elibrary/citation.html#harvard>

The Library has an amended Harvard Flinders output style file that can be used with EndNote. Go to the library website: <http://www.library.usyd.edu.au/databases/endnotex/faq/stylelist.html>

and download the Harvard Flinders style from: `harvardflinders_usyd.ens` and put it in your Endnote XI/styles folder.

ASSESSMENT

Students will be assessed using the following guidelines:

Task No.	Component	Weight
1.	USYD eLearning/formerly WebCT exercise (group assessment)	15%
2.	1 hour multiple choice exam (intrasemester)	35%
3.	1 hour final written exam	50%
TOTAL		100%

REFERENCES

Core references:

- Diseases of Swine, 9th edition (2006) BE Straw *et al*
- Managing Pig Health and Treatment of Disease (1997) MR Muirhead and TJ Alexander
- Pathology of the Pig (1996) LD Sims and JRW Glastonbury

Useful references:

- Care and management of miniature pet pigs (1993) DE Reeves
- Animal-to-human transplantation research: A guide for the community (2003). Australian Govt. NHMRC (<http://www.dcita.gov.au/cca>.)
- PigStats –Australian Pork Ltd Publication
- Mating and Reproduction-Australian Pork Ltd Publication
- Farrowing-Australian Pork Ltd Publication
- Weaning to Sale-Australian Pork Ltd Publication
- The Managers Toolbox-Australian Pork Ltd Publication
- The Good Health Manual-Australian Pork Ltd Publication
- Eradicating Diseases of Pigs-Australian Pork Ltd Publication
- Plan it-Build it-Australian Pork Ltd Publication

It is not a requirement that students purchase the textbooks listed above for this course.

Web Links

<http://www.library.usyd.edu.au/VIP/>

<http://www.vetmed.iastate.edu/departments/vdpam/swine/>

Chapter 2

PRACTICAL CLASSES

General Information

The class will be divided up into eight groups (A1-D2), each consisting of approximately 15 students. Practicals will run for 2-3 hours. See the timetable for group times.

It is a requirement that by the completion of the course you will have the following basic handling skills and management techniques (A=must have undertaken, B=must have observed, C=must have knowledge of).

Practical skill	A	B	C
Identification of common breeds			X
Catching and restraining pigs	X		
Intramuscular injection	X		
Subcutaneous injection	X		
Teeth clipping		X	
Tail docking		X	
Moving pigs with a stockboard	X		
Estimate liveweight and carcass weight	X		
Ear notching		X	
Branding		X	
Handling boars		X	
Cleaning, disinfection		X	
Euthanasia		X	
Intravenous injection		X	
Intraperitoneal injection			X
Stomach tubing & bottle feeding		X	
Castration		X	
Pregnancy diagnosis (visual, RTU)	X		
Backfat measurement	X		
Blood collection	X		
Condition scoring	X		
Oestrus detection		X	
Natural mating supervision		X	
Artificial insemination		X	
Assisted farrowing		X	
Semen processing			X
Fostering piglets			X
Colostrum milking & split suckling			X
Recognising sick pigs	X		

ANIMAL HUSBANDRY: BREEDER AND GROWER FARMS

Learning outcomes of the class:

1. Be competent at catching and restraining piglets and older pigs, moving pigs with a stockboard and weighing pigs to calculate growth rate, carcass weight and profitability of pigs.
2. To collect blood from the jugular vein of pigs.
3. To conduct condition scoring and P2 backfat testing of sows.
4. To conduct pregnancy diagnosis using a real-time ultrasound machine.
5. To observe and understand the principals of inserting an IV catheter into the ear vein of pigs.
6. To have observed and have an understanding of best practice principles for castration of male pigs and stomach-tubing piglets.
7. The ability to undertake an “environmental audit” of a farm.

Site:

Camden piggery breeder site on Mayfarm Rd:

We will start by viewing a video on piglet castration. The class will then be split into 2 groups of approximately 16 students per group. One half of the group will work in the dry sow house to conduct pregnancy testing, backfat testing and condition scoring. The other half of the group will undertake blood collection and observe piglets being “processed” (teeth clipped, tails docked, iron injection).

Camden piggery grower site on Werombi Rd:

The group will come together after the initial practical at Werombi Rd where we will practice blood collection and ear vein catheterization of grower and finisher pigs. Total duration of exercise = 2-3 hours.

Bring:

Clean overalls and boots. As our piggeries are of high health status, any student with dung of any nature on their clothes or boots will not be allowed to participate in the practical class. You will need to have transport between piggery sites.

References:

Short videos and self-tests are available on-line to review these husbandry tasks.

CLASS NOTES FOR PRACTICAL 1

PIGLET HANDLING

As a veterinarian working with pigs, you will be expected to advise clients (mainly farm managers and stockpeople) on “best practice” methods for undertaking routine husbandry procedures on piglets. Stockpersons authorised to perform minor surgical, medical or intervention tasks must be trained in accordance with the Code of Practice (Welfare).

The following are guidelines that will assist you in assuring these practices are undertaken using “Best Practice”.

- Stockpersons should understand that minor surgery causes little distress if carried out efficiently and with minimal restraint. Attention should be paid to:
 - Suitability of area in which the operation is to be performed.
 - The catching facilities.
 - Type and amount of restraint.
 - Selection and maintenance of instruments.
 - Hygiene of syringes, needles and site of injection.
 - After care of animals.

Tail docking



Carry out preferably within 72 hours after birth, but at least before 7 days of age. Use sharp, clean instruments. At least 2cm of the tail from its base should be left after docking at this age. Clipping too much off may result in ascending infections into the spine. A veterinarian should perform tail docking of pigs older than 7 days.

- Where tail biting is a problem, all aspects of the environment, feeding and management should be investigated to identify the contributing factors so that remedial action can be taken.
- Tail docking is undertaken to minimize the risk of tail biting. Tail biting is considered a vice. It occurs sporadically in groups of animals, and is likely to be the result of a number of sub-optimal environmental challenges (too cold/hot/crowded/stuffy etc).
- During outbreaks of tail biting, it is important to rectify environmental, feeding and management sources of discomfort to pigs that may lead to the problem:
 - Provide good ventilation, minimise high humidity and the build up of ammonia.
 - Regulate temperature: Prevent chilling and overheating.
 - Where possible, identify the aggressors and remove them.
 - Remove the badly bitten pigs. Daub the bitten tails and ears with a repellent.
 - Provide distractions in the pen, such as paper sacks, straw or balls.
 - Make sure there is adequate feeding and drinking space.
 - Reduce the stocking rate in the pen and group pigs of similar size.
 - Change the feeding system or physical form of the feed.

Teeth clipping



Carry out within the first 2 days of birth. It is recommended that clippers are replaced every 200-300 litters to avoid cracking a tooth or leaving sharp edges.

Clip only the very tips of the teeth off

- This procedure is not routinely required. “High risk” farms include those that do excessive cross-fostering, have poor-milking sows and high ambient temperatures.
- Piglets are born with eight needle teeth. The teeth allow newborn piglets to bite sideways especially in the competition for teats, which is greatest during the first few hours after farrowing. The competition lessens as the piglets establish a stable ‘teat order’. The fighting for teats can cause severe facial wounds which can lead to infections. Sharp teeth can also cause discomfort to the sow and damage the sow’s teats.
- Piglet teeth are often clipped so as to reduce injuries within the litters and to the sow. If clipping is not undertaken correctly, this can cause gum damage that may result in lesions to the insides of the oral cavity. Polyarthritis is one possible infection arising from poor teeth clipping techniques.



*Piglet with facial lesions resulting from bite wounds.
The litter of piglets did not have their teeth clipped.*

Castration

- Castration should be avoided. If necessary for marketing purposes, non-surgical methods are preferable.
- Chemical castration using Improvac® requires two injections at least four weeks apart. The last shot should be 4-5 weeks before slaughter.



If surgical castration is necessary, do it after 2 days of age, after piglets have established their sucking order, and before 7 days of age. A veterinarian should perform castration of boars older than 21 days using local or general anaesthesia.

- Ensure the animal is adequately restrained & equipment is clean and disinfected.
- Surgical castration requires use of a sterile sharp implement such as a knife or surgical scalpel.
- Good post operational drainage is essential.

Ear notching and tattooing



Carry out before pigs are 7 days of age.

- Where it is necessary to mark pigs for permanent identification, the ear may be tattooed, tagged, notched or punched, or the body may be tattooed or micro-chip implanted.
- Ear notching is a common procedure on many farms in Australia to identify the ages of pigs (week born) and/or the litter (for replacement breeding stock). Tagging is usually used for older breeding stock for identification. Tattooing is rarely undertaken for on-farm identification in Australia.
- Use an antiseptic spray after ear notching.
- If notching/tattooing ears on older or larger pigs, it may be necessary to house them in separate pens until healed. Pen mates may be attracted to the bloody notches and start ear biting.



A litter of piglets with their ears notched for identification.

Iron injection



It is recommended that piglets be provided with a supplemental source of iron within a few days of birth (72 hours) to prevent anaemia.

- Piglets will become anaemic within 10 days of birth if not supplemented with iron.
- Iron can be administered via injection or orally. Most farmers prefer to administer iron intramuscularly (1-2mL, depending on the product) into the muscle behind the ear. It should not be injected into the “ham” muscle as it may stain the carcass.
- Always ensure needles are not blunt, dirty or burred.
- For pasture-based systems, soil can provide an adequate amount of iron for the nursing pig, reducing the need for an iron injection.

Stomach tubing

- Piglets may need supplemental colostrum or milk if they are not strong enough to suckle, or if they are unable to gain sufficient access to a sow (eg through death of the sow or insufficient teats).
- In most cases, supplemental colostrum/milk can be provided by cross-fostering to a “foster sow” in the first 24 hours after birth. Farmers may make up a “fall-back” litter to rear runt pigs. Otherwise, avoid fostering where possible – it upsets the hierarchy in the litter.
- If piglets have a sucking reflex, provide colostrum/milk via a baby’s bottle. Make sure you warm the liquid first to avoid chilling the piglet.
- If there is no sucking reflex, this means a poor prognosis. However, you may attempt to rear these little guys. To do this, you will need to administer the fluid using a stomach tube. Get a small tube (eg a drip line or model aeroplane fuel line). Measure on the outside of the piglet where the stomach is (the extended elbow is a guide). Make a mark on the tube to ensure you get the tube in the correct place.
- Hold the piglet securely so it doesn’t bite you or the tube, Insert the tube down the oesophagus to the level of the mark.
- Administer 10-30mL of warmed fluid down the tube. You should do this as frequently as possible (remember piglets drink hourly while they are on Mum!)

BREEDING HERD PROCEDURES

Semen collection

Collection of semen:

- Equipment: esky, thermos & screw-on lid, gauze & rubber band, 2 rubber gloves, diluent

Method:

- Clean up the boar
- While mounting a sow on heat or dummy sow
- Boars respond to pressure on their penis>>ejaculate
- Avoid pre-sperm fraction (no sperm, contaminated)
- Collect into pre-warmed thermos flask covered with gauze
- Discard gauze, add diluent and screw on thermos lid

Examination:

- Colour (creamy white), volume (50-450mL), motility (>70% moving forward), concentration (minimum dose rate $3-5 \times 10^9$ sperm/dose diluted to 80mL), morphology (<20%)
- Use immediately or add extender-shelf life 3-5 days
- Keep at 30-35C if used within hours; cool slowly to 18C if stored
- Invert at least BID to re-suspend (don't shake)

The quality of an insemination is determined by:

- Semen quality
- Experience and motivation of the inseminator
- Soundness of the insemination procedure

Artificial insemination

The insemination process aims to place as many live sperm as possible inside the tract of an aroused, oestrous female. The elements of a successful artificial insemination are:

- good oestrus detection to ensure the sow is in standing heat
- provision of head-to-head contact with a mature boar throughout the insemination process (N.B. the boar should be used to stimulate no more than 3 sows at a time)
- insert the catheter into the vulva at a 30° upward angle, pushing gently until the resistance of the cervical opening is felt
- if using a foam tip catheter push gently into the cervix until a lock is achieved (catheter remains fixed when it is gently tugged back)
- if using a spirette catheter gently rotate the catheter counter-clockwise (to the left) until it springs back a quarter turn
- attach the semen tube, bag or bottle to the catheter (if using a couchette bag this step could have been taken prior to catheter insertion)
- start semen flow by applying gentle pressure to the semen container

- allow free flow of semen into the sow (be patient – allow 2-6 minutes) – if semen doesn't flow re-position the catheter slightly
- throughout the insemination – including for 1-2 minutes after the semen container has emptied - continue to stimulate the sow by massaging her udder, flanks and back
- either crimp or plug the catheter and leave in place for a further 5 minutes or so

Pregnancy diagnosis using real time ultrasound (RTU)



RTU scan 22 days post-insemination.

Measurement of P2 backfat in lactating sows

Backfat is usually measured in live animals using ultrasound machines (eg. The Renco machine). Ideally, sows should have a backfat of 20-22mm at farrowing so that their lactation feed intake is high. Overly fat sows do not eat well during lactation. Backfat testing is also undertaken in gilts at selection as a guide to their body condition. Ideally, gilts should weigh more than 130kg at mating and have a P2 backfat of at least 13mm.

To measure P2 backfat, find the last rib of the pig. Then measure 6.5cm off the midline. You may need to scrape off a bit of hair in hairy animals. Apply some ultrasound gel and a place the Renco machine over the top of the area. Hold the button down until you get a steady signal.






Condition scoring sows

The body condition of sows is scored from 1 (emaciated) to 5 (overly fat). The ideal body condition of a sow at farrowing is 3 and at weaning about 2. The key landmarks to look for with condition scoring are the backbone, hip bones and tail bones. You should just be able to feel these with light pressure on the skin. If they are protruding, then is a body condition of 1-2. If they are impossible to feel then this is a CS 5.



Sow with CS 5

Appendix I: Condition Scoring of Sows

Numerical Score	Pelvic Bones, Tail Head	Loin	Vertebrae	Ribs
1	 <p>Pelvic bones very prominent. Deep cavity around the tail head.</p>	<p>Loin very narrow. Sharp edges on transverse spinal process. Flank very hollow.</p>	<p>Prominent and sharp throughout the length of the backbone</p>	<p>Individual ribs very prominent</p>
2	 <p>Pelvic bones obvious but some slight cover. Cavity around tail head.</p>	<p>Loin narrow. Only very slight cover to edge of transverse spinal process. Flank rather hollow.</p>	<p>Prominent</p>	<p>Rib cage less apparent. Difficult to see individual ribs.</p>
3	 <p>Pelvic bones covered.</p>	<p>Edge of transverse spinal processes covered and rounded.</p>	<p>Visible over the shoulder. Some cover further back.</p>	<p>Covered but can be felt.</p>
4	 <p>Pelvic bones only felt with firm pressure. No cavity around tail.</p>	<p>Edge of transverse spinal processes felt only with firm pressure.</p>	<p>Felt only with firm pressure</p>	<p>Rib cage not visible. Very difficult to feel any ribs.</p>
5	 <p>Pelvic bones impossible to feel. Root of tail set deep in surrounding fat.</p>	<p>Impossible to feel bones. Flank full and rounded.</p>	<p>Impossible to feel vertebrae.</p>	<p>Not possible to feel ribs.</p>

Appendix: A Quick Semen Checklist

1. BOAR/SEMEN:

- a. Did the boar have a temp (>39C) and/or off-feed/depressed?
- b. Were the boar's testicles normal? (appearance, consistency?)
- c. Sperm quality: Motility >70%, morphology > 75%

2. SEMEN COLLECTION TECHNIQUE:

- a. Was the boar's prepuce cleaned?
- b. Gloves not spermicidal?
- c. Avoid pre-putial fluid (spermicidal)

3. DILUENT:

- a. WATER - correct volume used?; Water equality (pH 5-7, nitrates = 0, alkalinity (0-80ppm)
- b. POWDER - within use-by date?; correct measures?
- c. Was the diluent aged? (risk of bacterial contamination if heated for >6h+)

4. GLASSWARE:

- a. no disinfectant/soap residues?

5. SEMEN PROCESSING:

- a. Is the diluent 35C (within 2C of semen?)
- b. Avoid exposure to water, temp changes, direct sunlight
- c. Was the diluent added within 15 mins of collection?
- d. Was the diluent allowed to settle at least 2-3 hours before use?
- e. Was an equivalent volume of semen added to extender in a water bath for 10mins before adding the final volume of extender? (reduces osmotic shock)

6. SEMEN STORAGE:

- a. Were the insemination bottles stored at 17-20C?

Appendix: Pig Restraint And Sampling - Bleeding

Restraint is necessary for bleeding, swabbing or anaesthetising pigs and can be a stressful operation for both pig and operator. This is especially so if the operator is not routinely working with pigs or adept at sampling procedures. It is a pity that many veterinarians are put off from accessing pig blood samples because they feel they are not proficient in this area. Sampling across different age groups on the one day (cross-sectional sampling) or following groups of cohort animals over time (longitudinal sampling) provide useful information on health status of a herd during the production cycle. If you want to brush up on this aspect of clinical work, here are some tips.

RESTRAINT

Rope nose snares are less traumatic but are unhygienic and too slow for working when numbers of animals have to be sampled. A very practical nose snare for pigs from 6 to 26 weeks can be made from stainless steel cable and galvanised pipe, plus a couple of welded bits as follows:

Handle:	11-12 cm x 16 mm ID/22 mm OD galvanised pipe
Body:	30-32 cm x 16 mm ID/22 mm OD galvanised pipe
Cable:	90 cm x 5 mm diameter stainless steel cable
Fittings:	Small nuts x 2; circular steel rod 25 mm x 4 mm dia; solid plastic bike handlebar grip to fit over one end of snare body
Other:	Welding gear and some epoxy glue; hammer and heat source to flare one end of the snare body outwards

The following pictures and instructions will help you out if you want to make a reliable pig snare.



Make the snare as follows:

- Drill a 7 mm hole in centre of handle on one side – file at an angle on one side to enable the cable to slide through easily.
- Drill a similar hole 45 mm from the pig-end of the snare body and file at an angle on one side to allow the cable to slide through easily when the cable is passed in the direction of the pig-end of the snare at an angle of 45° or less.
- Heat the pig-end of the snare body and flare out the end to a diameter of 25-26 mm. File the flared edge to prevent it cutting into the cable.
- Weld the circular steel rod across the middle of the flared end, keeping the hole 45 mm from the end lateral to the line of the steel rod.
- Attach the plastic grip to the holder's end of the snare body and glue into place.
- Feed the cable via the hole at 45 mm from the end of the snare body, loop this over the steel rod dividing the pig-end of the snare body and then feed the cable up the middle of the snare body



- Feed the cable into the hole in the centre of the snare handle and out one end.
- Weld a steel nut or cap onto each end of the cable so it cannot pass back through either hole. The nut or cap at the operator end of the cable should be small enough to fit inside the handle (e.g. 13 mm hex nut).
- Withdraw the cable at the handle end into the handle.



The object of snare restraint is to hold the pig with its head held only slightly lifted - i.e. in a normal standing position. The snare is placed over the top jaw well back from the tip of the snout, behind the level of the canine teeth if possible. The best place to restrain the pig is in an area where there is firm, slip-free flooring and in an area just away from its cohorts. A corner position in a pen is ideal.

The restrained pig can be moved easily into the preferred site by using your hand to grasp its skin in front of a hindleg, moving the pig laterally into position.

Remember to use ear muffs during this procedure.

BLEEDING PIGS

Many veterinarians and veterinary students may receive limited exposure to bleeding pigs and as a result, have limited proficiency in accessing useful sites for IV pig anaesthesia. The best site for blood sampling for most pigs is the precava. The best means of sampling pigs from 3-26 weeks is via a vacutube (vacutainer) and appropriate gauge disposable needles:

Pigs 3-8 weeks: 0.5 - 1" x 20G

Pigs 8-26 weeks: 1 – 1.5" X 18-20G

For adult pigs, 1.5" x 18G vacutainer needles (Terumo) or hypodermic needles are available from suppliers such as Clifford Hallam.

With baby pigs (up to 6-8 weeks), these can be bled most readily on their back. A V-shaped wooden cradle is recommended to provide stable positioning of the pig.

Note: For a general introduction, see the following website address:

<http://oslovet.veths.no/teaching/pig/pigbleed/>

This article and photos by Tore Framstad *et al* at the Oslo Veterinary School is also accessible via the VIP site described earlier under "Sourcing Pig Information".

The following comes from information provided to NSW Department of Primary Industries for its new edition of the publication "Animal Care - NSW Agriculture Approved Procedures for the Use of Animals in Teaching, Research and Extension".

COLLECTION OF BLOOD - PIGS

Details of the procedure:

Sites and method for bleeding depend on the age of the animal and the volume of blood required (see table below). The use of small gauge needles, especially in pigs up to 20 weeks will minimise the risk of blood leakage from the anterior vena cava to the thorax and potential respiratory distress. 20G needles are recommended for small pigs < 8 weeks old. 20G needles can be used in pigs from 8-26 weeks, however experienced operators can safely and successfully use 1.5" x 16-18G needles in larger pigs (> 16 weeks). Vacuum tubes (vacutainers) are recommended for all sites except the ear vein. The use of a 1.5" vacutainer needle with a rubber sleeve that covers the stopper-puncturing end is recommended. This will prevent splashing of blood onto the stopper when the evacuated tube is removed.

Bleeding from the anterior vena cava/caudal jugular site is suitable and recommended when a number of animals have to be bled or multiple bleeds are required. Tail bleeding, ear bleeding and cephalic vein bleeding are more problematic and not recommended where repeated samples have to be taken.

Impact of procedure on the wellbeing of animal(s):

Nil, unless excessive volumes of blood are taken or unless excessive blood leakage to the thorax occurs associated with laceration or damage to the anterior vena cava.

Reuse and repeated use:

Where small volumes of blood are removed (10-20 mL), weekly or fortnightly sampling on several occasions can be safely undertaken by an experienced operator. For repeat sampling it is important to avoid damaging the vein, using good technique and restraint. Where an animal is inadequately restrained, avoid repeated attempts at venipuncture.

Blood collection sites

Age	Site	Position/Restraint	Technique
<p>≤ 4-5 weeks.</p> <p>Can be undertaken up to 8 weeks</p>	<p>Anterior vena cava</p>	<p>Lie flat in dorsal recumbency with neck extended straight, and front legs held back along the chest by an assistant. A V-shaped cradle is recommended. The operator holds the pig's snout with his/her free hand or the pig's head can be restrained under a cross-strut on the cradle, provided access to the anterior vena cava is not impeded and head restraint is secure.</p>	<p>From the cranial point of the sternum, on a line to the base of the ear, locate a craniolateral depression in the skin, approx 2 cm cranial and 2 cm lateral to the right of the cariniform cartilage (cranial tip of the sternum). Direct the needle towards, or just lateral to, the midline and going inwards and backwards at an angle of approximately 60° to the skin surface.</p> <p>Entry on pig's right side is recommended to avoid the phrenic nerve. In some small pigs, the vein may lie only 1 cm under the skin.</p>
<p>≥ 6 weeks</p> <p>Always suitable in pigs 8 weeks or older.</p>	<p>Anterior vena cava to caudal jugular vein</p>	<p>Standing square on all four legs, restrained by a snout rope or snare, head slightly forward and slightly raised. The vein should not be occluded, and must be punctured blind. Restrain in an area of the pen apart from other pigs and on a slip-free surface, to avoid movement that will interfere with safety of the procedure.</p> <p>The operator can work from a position either parallel with the pig (facing forward, working to the left) or from in front of the pig (facing backward). The former position enables better control over pig movement, overall restraint and safety.</p>	<p>From the right side of the pig, find the same depression as indicated above, craniolateral to the point of the sternum, on a line between the sternum and the base of the ear. Find the base of this depression or a slight bulge just below (ventral to) the deepest part of the depression. The needle is directed inward, upward and backward to puncture the vein. Direct the needle toward, or just lateral to, the dorsal midline of the pig's back. The needle is typically directed at an angle to the skin of between 60° and 90°. In adults, the vein lies deep, and in large adults a 1.5" needle may not be long enough. In adult pigs, for a right handed operator, position the left hand on the top of the shoulder blade and aim the needle toward that point</p>

RECOGNISING NORMAL FROM ABNORMAL

Learning outcomes of the exercise:

The ability to diagnose disease in individual pigs by doing a necropsy and to conduct herd health surveillance by inspecting carcasses during processing.

Site:

Wet lab-Shute annex. Duration of practical - 1.5 to 2 hours.

Bring:

A lab coat or overalls, boots, pen and paper

In this exercise, students will:

1. Conduct a necropsy on a dead pig to determine the cause of death using the pictorial course notes provided.
2. Participate in a tutorial on disease diagnosis during inspection of viscera at processing.

Conducting Post Mortems

What you need for a PM

- Sharp knife
- Steel & stone
- Forceps
- Scissors
- Gloves
- Jars/bags
- Swabs

Consider:

- Has the pig been destroyed or was it found dead?
- If destroyed, record why (if obvious).
- If destroyed for illthrift, do a PM.
- If found dead, follow the flow-charts on the next pages.

Histology

- In jars or plastic bags
- Send normal & abnormal tissues
- Handle samples gently
- Take samples about 1cm thick
- Don't freeze

Culture

- Clean tissue in a jar or plastic bag
- Need at least 3-4cm²
- Swabs in transport media
- Keep in the fridge (don't freeze)
- Send to the lab within 24h with an ice-pack

MORTALITY CHECKLIST

Date:	Pig ID:	Age:	Weight:	Gender:
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External examination (Y/N):

Abrasions on feet:	Walked:	Swollen joints:
Abrasions on limbs:	Taped limbs:	Dried mucus on skin:
Hyperaemia of skin:	Pallor of skin:	Condition score 0-4:
Eye sunken:	Tongue protruding:	Squashed appearance:
Obvious bruising:	Anus patent:	Oro-facial deformities:
Other defects:	Teeth clipped:	Gums inflamed:
Wounds:	Meconium in oral cavity:	Umbilicus swollen:
Umbilical cord fleshy:	Perineal faecal staining:	

Special comments:

Subcutaneous tissue (Y/N):

Bruising:	Iron injection:	Oedema:
Fat reserves:	Dry sticky tissue:	Pallor:

Other:

Head & Neck:

Tonsils:	Nasal cavity:	Larynx/trachea:
Brain:	Lymph nodes:	

Other:

Thorax:

Lungs:
Pleural cavity:
Heart:
Thymus:

Other:

Abdomen:

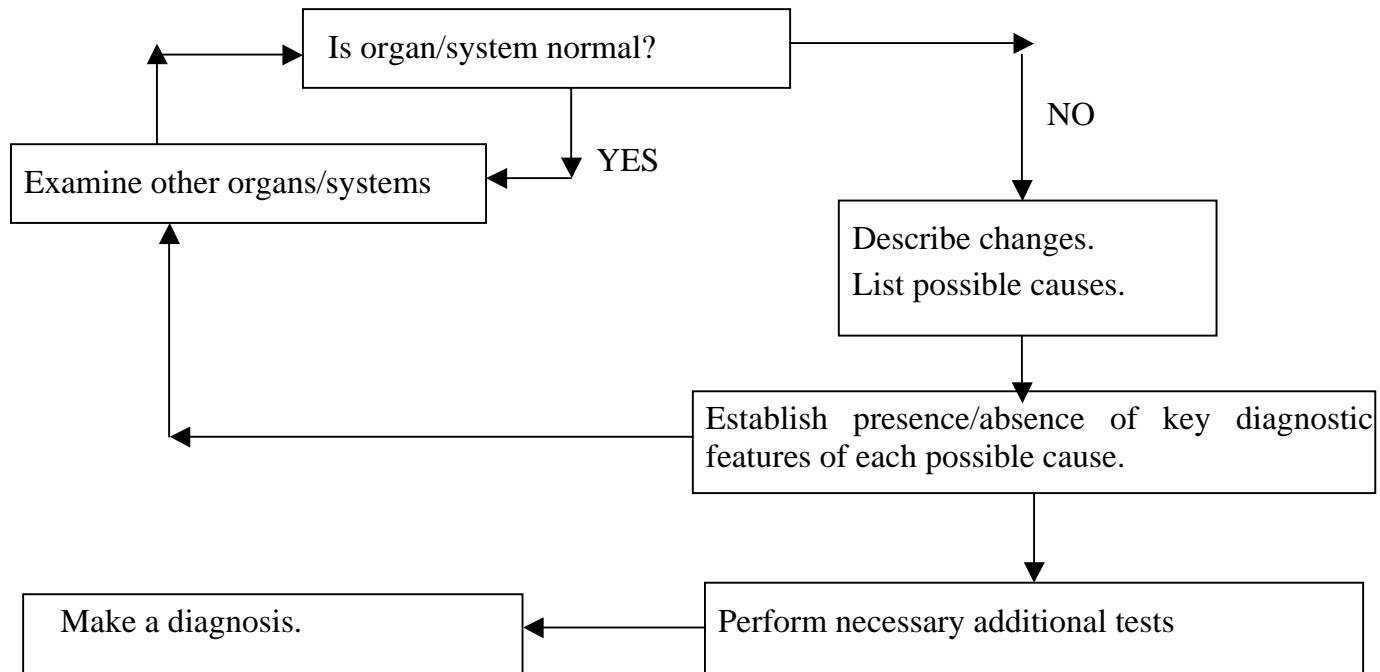
Peritoneal cavity:
Stomach & contents:
Small intestine & contents:
Large intestine & contents:

Spleen:	Liver:	Lymph nodes:
Kidney:	Adrenals:	Urinary bladder:

Notes/Specimens collected:

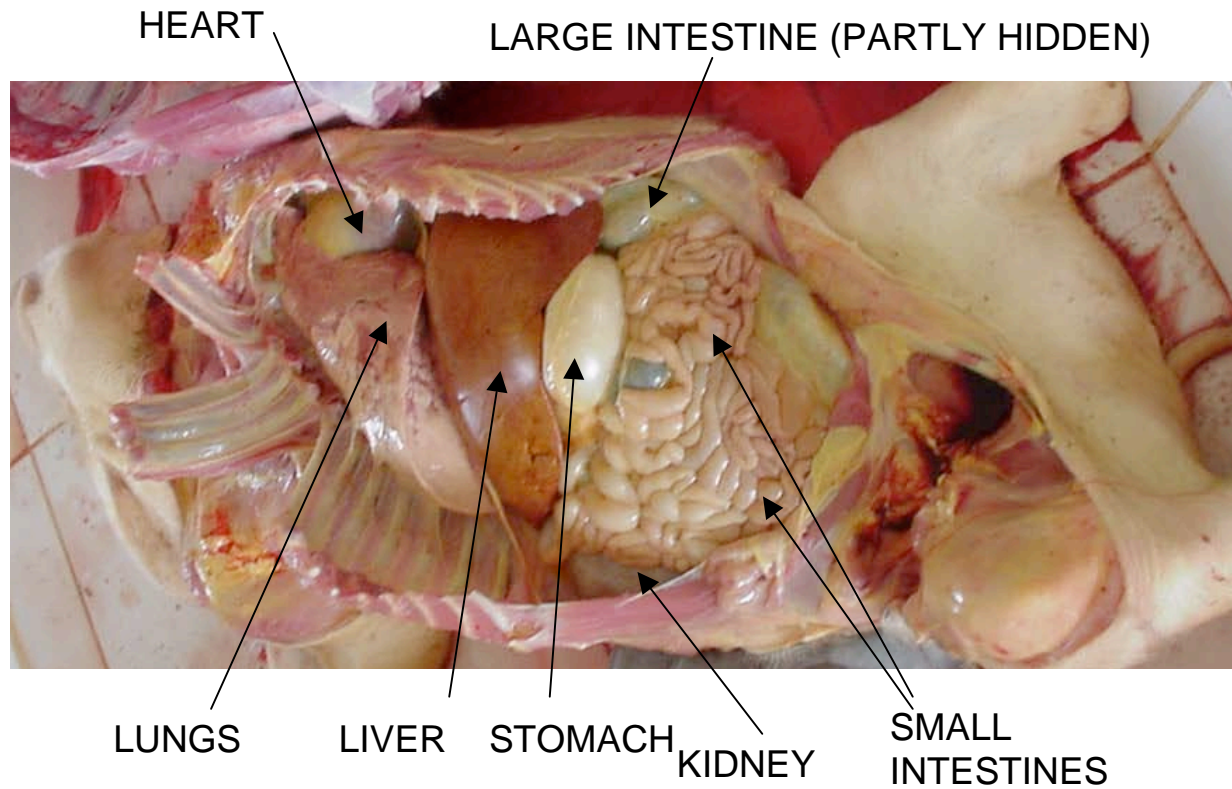
Probable diagnosis:

HOW TO START EXAMINING AN ANIMAL

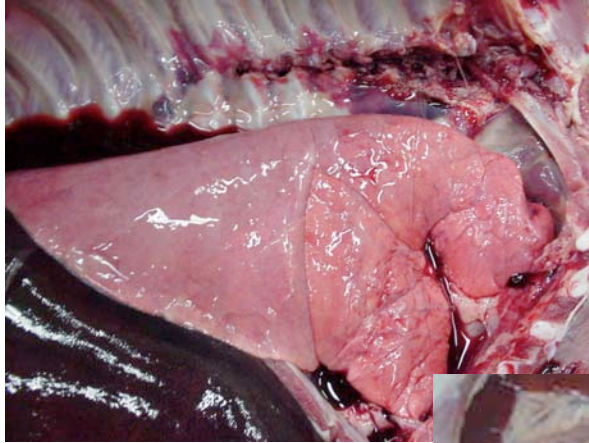


BITS AND PIECES

(note that this pig is jaundice (yellow))



What is normal?



Normal lung



Normal heart



Normal intestines

Baby pigs

We don't routinely post-mortem suckers. The external signs (below) usually tell you the cause of death.

Main external finding	Diagnosis
Piglet has not walked	Stillbirth
Piglet has sunken eyes	Dehydration/Scours
Piglet has obvious abnormality	Defect
Piglet has swollen joints	Arthritis
Piglet is in good condition	Overlay or acute disease
Piglet is less than 800g	Small/non-viable
Piglet with bite wounds	Trauma (gilt or other piglets)
Piglet is pale	Anemia
Piglet has abrasions on inner legs	Splayleg
Piglet is in poor condition	Chronic disease/starvation

TUTORIAL

“Bringing home the bacon”

Students will work in pairs to participate in a 2 hour tutorial session to identify production-limiting issues affecting financial performance on a pig farm.

Site:

General Teaching Building - Room TBA

Bring:

Pen, paper, calculator

Supervisor:

Dr Trish Holyoake

Tutorial exercise

“Bringing home the bacon”

As a practicing rural veterinarian, you have been called in by the owner of an 85-sow piggery that is currently totally non-viable. He wishes to offer you a lucrative annual consultancy contract if you can genuinely improve the viability of his unit – he intends to judge this on the advice you offer him during this tutorial.

He provides you with all the performance & financial data that he has on the herd, together with a basic plan of the piggery (enclosed).

You will have 30 minutes to read over the information provided. You need to decide if he is meeting performance targets. During this time, you will be provided with an opportunity (5-15 minutes) to question the owner. At this time you should have prepared a list of questions you need answered – N.B. The owner will not have all the answers, otherwise he would not need to pay you as a consultant!

Now you should have all the necessary information to compile a brief report outlining the piggery’s strengths and weaknesses & to suggest both immediate and longer-term strategies to enhance the overall viability of the unit.

Annual Herd Performance

Average herd size	85 sows
Sows mated	162
Gilts mated	12
Sows farrowed	153
Total pigs born	1897
Pigs born alive	1745
Pigs weaned	1431
Pigs sold	1391
Av sale weight (kg)	77
Avg sale age (days)	155
Dressing %	76
% 1 st grade carcasses	92

Annual Financial Performance

Income

1280 pigs @ \$1.85/kg carcass	\$138,522
111 pigs @ \$1.65/kg carcass	\$10,714
<i>Total income</i>	<i>\$149,236</i>

Expenditure

Salaries:

Manager	\$33,163
Assistant	\$22,919
Overtime	\$9,9936
<i>Total salaries</i>	<i>\$66,018</i>

Feed:

85T dry sow diet @ \$246/T	\$20,910
39T lac sow diet @ \$280/T	\$10,920
47T creep/weaner diet @450/T	\$21,150
121T grower diet @ \$380/T	\$45,980
201T finisher diet @ \$295/T	\$59,295
<i>Total feed</i>	<i>\$158,255</i>

Repairs/maintenance	\$4,860
Health costs	\$9,804
Power costs	\$14,909
Breeding stock leases	\$19,236
Miscellaneous costs	\$4,008

<i>Total expenditure</i>	<i>\$277,090</i>
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Balance	(\$127,854)
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Farm layout

Changing room

Stage 1 weaner room. 200 weaners

Dry sow house. Mating area (14 boars + 48 gilts/sows. Plus early gestation area

Dry sow yard – mid & late gestation area. 40 dry sows.

Farrowing house 1: 16 sows & litters

Farrowing house 2: 8 sows & litters

Stage weaner room: 2 100 weaners

Grower shed: 192 growers

Finisher shed: 304 finishers

Use of Veterinary Medicines in the Pig Industry

An extract from AVA's Prescribing and Dispensing Guidelines

Introduction

With the increased intensification in the pig industry, the role of the veterinarian is focussing more on herd health management, frequently necessitating treatment or preventive measures on a mass basis. Practices of Schedule 4 (S4) drug supply and usage in the pig industry have legal and ethical restraints which are outlined below.

Legal Obligations for Veterinarians Supplying S4 Substances

Veterinarians must fulfil the obligations imposed on them by the relevant legislation in the jurisdiction(s) in which they practise, that direct procedures to be followed in the supply of S4 restricted substances.

Any current practices which are contrary to this legislation should either be curtailed or modified to meet all requirements. Contrived arrangements between veterinarians and wholesalers that attempt to circumvent these mandatory requirements are to be avoided, since they jeopardise both the wholesaler's authority and the veterinarian's registration.

Responsibilities of the Veterinarian

Responsibilities of veterinarians supplying S4 restricted substances within the pig industry are detailed below.

Veterinary care and supervision of livestock

If a veterinarian is involved in the supply of a S4 substance, he/she must demonstrate due care and supervision of the recipient stock. This care and supervision should be real and not merely nominal.

When given responsibility for the health of the animal or herd in question by the agent or owner, the veterinarian demonstrates care and supervision by at least either:

- having seen the animal or herd for the purpose of diagnosis or prescription immediately prior to supply or;
- having visited the farm or other premises on which the animal or herd is kept, sufficiently often and recently enough to have acquired from personal knowledge and inspection an accurate picture of the current health state on the farm or premises, to enable him/her to diagnose and/or prescribe for the animal or herd in question.

Areas of responsibility

In situations where a veterinarian is called on to prescribe or supply S4 substances, responsibilities additional to the legal obligations to be taken into account are the:

- care and welfare of the pigs that are the subject of the proposed drug supply and
- professional responsibilities of the veterinarian as described by the AVA Code of Professional Conduct.

The S4 drug supply chain

Veterinarians should carefully analyse the drug supply chain in which they are involved and delineate wholesale from retail activities. They should also check the bona fides of persons to be supplied.

The S4 drug supply chain between manufacturer and end user may be described as follows.

The wholesaler may purchase S4 medications direct from a manufacturer and subsequently supply to a veterinarian, a pharmacist, another licensed, authorised or permitted wholesaler, or an authorised receiver as stipulated by the relevant poisons legislation. The latter includes government departments, universities and hospitals, overseas countries and interstate distributors.

In all states, except South Australia, a wholesaler may not supply S4 substances direct to an end user, and cannot be authorised to do so by a veterinarian. Thus, in all states except SA, a licensed or authorised wholesale dealer is not permitted to dispense a prescription under any pretext. In some states (such as Queensland) under exceptional circumstances, direct supply to an end user can be directly authorised by the Director General of Health, but no other person. In SA, registered wholesalers may supply an end user directly, but only on the authorisation of a veterinarian responsible for and with a knowledge of the end user's pig herd. Authorisation in such cases can be given by telephone but must be followed by written confirmation within three days.

An approved feed miller can supply feedstuffs containing S4 substances under specified conditions, such as for and on behalf of, and on the written order of, a veterinarian.

A pharmacist may dispense S4 drugs to an end user but only on veterinary prescription, with one exception: in WA a pharmacist may supply certain S4 drugs in limited quantity (as specified in Regulation 39 Appendix H of the WA Poisons Act) in an emergency under certain specific conditions without a prescription.

Emergency supply of S4 drugs by a pharmacist is permitted in all states on the oral order of a veterinary surgeon, who must forward written confirmation by prescription within 24 hours.

The veterinarian accepts professional responsibility for the supply and use of S4 substances in the animals under his/her care. A veterinarian can possess S4 drugs only for the lawful practise of his/her profession. The veterinarian is not permitted to merchandise them, that is he/she cannot sell them without proper professional involvement in their use and can only supply them when he/she has made a diagnosis or has planned a medication program.

Before a pig veterinarian can supply S4 drugs he/she must be practising his/her profession. To do this, the following criteria must be met:

- the pig herd must be under the care and supervision of the veterinarian;
- the treatment recommended and the drugs supplied must be recorded;
- the client must be advised of the correct usage of the drugs; and
- if the drug is intended for treatment over a period of more than three days, it must be correctly labelled by the veterinarian, or by an assistant working under the veterinarian's personal supervision.

All veterinarians involved in the supply chain of S4 substances should continually update their knowledge of those individual or corporate entities who are registered as authorised or licensed veterinary wholesalers. State departments of health maintain an updated list of those wholesale dealers authorised, licensed or permitted under the relevant legislation.

Professional intervention

Veterinarians should fulfil the definition of 'professional intervention' in the supply chain of S4 substances. 'Professional intervention' can be defined as intervention between the drug wholesaler and the end user of the substance, in such a way as to ensure that the drug is necessary, appropriate and will be used correctly.

Veterinarians must not act as 'rubber stamps' for transactions between wholesalers and end users, but should instead be fully involved in the disease treatment and/or control program requiring the use of S4 drugs.

Documentation of professional intervention:

The involvement of the veterinarian in the supply of S4 substances must be fully documented. Professional intervention should include the use of:

- The veterinarian's own stationery or his/her stamp on invoices, prescriptions, authorisations and orders and
- The veterinarian's recorded direction to supply.

When supply is made, the veterinarian must ensure that each pack or bottle of the S4 drug bears labelling as required by law, including the name and address of the veterinarian and the name of the owner or farm manager.

Instructions on drug usage should be given to the end user by the veterinarian with clear details of the method of administration, dose rate, withdrawal times and so on. These instructions can make reference to specific disease control literature originating from the veterinarian.

Records of the name and quantity of S4 drugs supplied, together with the name and address of the pig owner, must be kept for two years.

Supply of S4 drugs as part of a forward-planned medication program

There is no obligation for the veterinarian to own the drugs he/she is supplying or is responsible for supplying.

Supply of S4 substances to end users by a veterinarian is permissible according to a forward-planned medication program under the full professional control of the veterinarian. Use of medication in such a program must be a routine and not at the discretion of the end user.

In accordance with such a forward-planned medication program, use of S4 drugs from farm-held stocks supplied by the veterinarian (or a person designated by the veterinarian) can be undertaken in the veterinarian's absence but it must be done with the veterinarian's knowledge. This designated person can be defined as a piggery owner, manager or contract grower who can demonstrate that he/she has received clear instructions, in writing, on the use of these S4 substances by the responsible veterinarian. Such a program must be kept under continual review by the veterinarian, whose written instructions are valid for a period of no more than six months.

In the case of a routine preventive program, the date of supply, the drug used, the farmer's name and volume of supply must be regularly recorded as required by law. Supply by the veterinarian must be accompanied by an invoice bearing the veterinarian's name, and the drugs correctly labelled and recorded.

In a disease outbreak, administration according to a forward-planned emergency medication program may be undertaken only when the veterinarian is confident the correct drug, from stocks supplied by him/her, will be used (if diagnosis is made from a distance). When prescribing in this manner, the veterinarian has no diminished responsibility for the diagnosis.

Stocks of S4 drugs on farms

The supply of S4 drugs for animal use to an end user other than by a veterinary surgeon or by a pharmacist on a veterinary prescription, is illegal. S4 drug stocks legally dispensed by a veterinarian for use on a pig farm are commonly stored in a central drug store. The storage of non-dispensed S4 drugs on farms remote from the veterinarian, eg the storage of unlabelled or unprescribed S4 drugs in a locked area with access exclusive to the veterinarian, is considered outside the spirit of the Poisons Act and a contravention of the Veterinary Surgeons Act, in that the veterinarian would have difficulty in demonstrating the maintenance of absolute control over these stocks.

In cases of emergency where S4 drugs may be urgently required, delays may occur if drugs have to be sent to the veterinarian by the wholesaler before being dispatched to the end user. However, only in

SA is there legal provision for a wholesaler to despatch directly to the end user on oral, followed by written, authorisation from the veterinarian ordering the drugs.

Feed mills

Feed mills do not usually conform to the definition of a wholesaler but may be registered or authorised under state legislation as wholesalers, able to supply:

- Schedule 4 stock medicines to a veterinarian, pharmacist or another wholesaler; and
- feedstuffs containing therapeutic substances at exempt or S6 levels (for unrestricted sale), or at S4 level under certain conditions, as outlined below. The conditions of supply of restricted substances at S4 level to an end user specify that such supply must only be in a feedstuff, and must be on, and in accordance with, the full written instructions of a veterinary surgeon, and on his behalf, to the holder of such an order from a veterinarian.

The authorisation or registration of a feed mill under the relevant legislation does not permit that feed mill to supply S4 stock medicines for open retail sale with or without veterinary authority. Thus feed mills may not, under any circumstances, supply S4 substances to the public other than when incorporated in feed, and then only as described above.

Where a person who mixes their own feed requires S4 medication for their herd, it must be acquired from a veterinarian, a pharmacist (on a veterinary prescription), or from a feed mill as a feed concentrate (in accordance with detailed written instructions from a veterinarian). The concentrate may contain a therapeutic substance at such a level that it can be further mixed to produce medicated feed containing that drug at a specified lower therapeutic level.

There is no restriction on the supply of premixes or concentrates at levels not exceeding those set out in Schedule 6. Such premixes may be in the form of registered stock medicines or made to order by a feed mill.

The veterinarian (including those in the employ of a feed mill) must show professional intervention in the supply chain of the drug to the end user via the feed mill. In effect, the feed mill acts as an agent for the veterinarian by acting on their order or authorisation in a similar manner as a pharmacist acts in filling a prescription for a veterinarian.

Veterinarians Employed by a Company

It is recognised that companies may be directly involved with the pig industry, either by direct ownership of livestock or manufacture of S4 substances likely to be used within the industry, or both. Such companies may employ veterinarians whose responsibility may be either the health care of company-owned livestock or the provision of technical expertise in the use of S4 substances in pigs.

Veterinarians employed in such companies in whatever capacity must meet their personal obligations under the relevant State/Territory legislation regarding the use of restricted substances and their own professional activity.

Veterinarians have an obligation to point out to their employer any contravention of the legislation affecting the supply or use of S4 substances and should make every endeavour to have them rectified.

The obligations and responsibilities of a veterinarian fully employed by a company, where that company is directly involved in ownership of pigs, whether or not that company also is an authorised, licensed or permitted wholesaler of S4 substances, are the same as those of other veterinarians not so employed.

The existence of a wholesale drug purchasing group within a company does not allow that company to provide retail supply to end users (including its own stock). There should be neither direct supply nor appearance of direct supply of S4 substances by the wholesale arm of a company to outside customers, franchises (unless they also hold a wholesale authority, licence or permit), the company's own piggeries or contract growers. All are end users and can be supplied only by a veterinarian, who must

intervene in the supply chain of S4 drugs and demonstrate professional intervention as previously described.

When supply is made from the company's wholesale arm to a company veterinarian, obligation to record transactions must be taken over by the veterinarian at that point. Such S4 drugs supplied to the company veterinarian (or any other veterinarian) must be held physically separated from the company's wholesale drug supplies. These supplies should be kept in a locked cupboard or room accessible only to the veterinarian. The veterinarian is required by law to keep a record of those drugs subsequently supplied.

* This Appendix is adapted from the Code of Practice prepared by the Australian Association of Pig Veterinarians (AAPV) in June 1989.

APPENDIX 10: Order for Medicated Feed

Veterinary authorisation to supply feed containing a PAR antibiotic

1 To feed mill:

Name:

Address:.....

.....

Please provide the following feed for:

2 Farmer name:.....

Address:

Consign to:

3 Medicated feed required

Type of feed (eg broiler starter, porker):

Form of feed (eg pellets, mash):.....

Quantity of medicated feed (order only as much as required):.....

Active ingredient:

Inclusion rate (eg g/tonne) required:

Product (trade name):

Inclusion rate (eg kg/tonne) required

4 Animals to be treated

Location:.....

Species:.....Type:

Age:..... Sex..... Number:.....

5 Directions for use of medicated feed on farm

▪ Quantity of medicated feed to be given daily:.....

▪ Duration of treatment:.....

▪ Withholding period (NOTE: medicated feed must NOT be fed during the withholding period):

Do not slaughter animals for human consumption until days after their last consumption of medicated feed.

Eggs/milk must not be taken for human consumption until days after the last consumption of medicated feed.

▪ Precautions: Ensure that animals or birds other than those specified on this form do NOT have access to this medicated feed.

6 Veterinarian placing order:

Name:

Address:

.....Telephone:.....

Signature: Date: / /

[3 copies: Original - Feed mill, Copy 1 - Farmer, Copy 2 – Veterinarian]

Chapter 3

LECTURE NOTES

Definitions & Concepts

Pigs:

- **Boar:** An intact male pig
- **Barrow:** A castrated male pig
- **Sow:** A female pig that has **farrowed** (given birth to a litter) at least once. The number of times that a sow has farrowed is referred to as her **parity** (i.e. a sow that has farrowed 3 times, has produced 3 litters, and is a parity 3 sow).
- **Gilt:** The strict definition is a female pig that has not farrowed (Parity 0). However, many times the first litter of a female is referred to as her “gilt litter” even though she is truly a Parity 1 sow once she farrows.
- **Piglet:** A pig that is still nursing the sow.
- **Weaner/Nursery pig:** A pig that has been weaned and resides in the weaner/nursery accommodation.
- **Growing pig:** A pig that has exited the nursery. Growers are usually 8-16 weeks of age and weigh less than 50kg.
- **Finishing pig:** A pig that weighs between 50kg and market weight (90-120kg).
- **Stillborn pig:** A pig that is fully developed but dead at birth.
- **Mummy/Mummified fetus:** A pig that is not fully developed, is discolored, shriveled, or decomposed at birth.

Breeds:

- **Boars:** The sire of a market pig is either a purebred or more commonly a cross between colored breeds of boars. Colored breeds are generally considered meat breeds because of their leanness and carcass quality. However, some of these colored breeds have been crossed to produce a non-pigmented boar. For example, a white-Duroc boar is used in some systems. Common examples of sire breeds are Duroc, Hampshire, Pietran, Spot, Poland China, or Berkshire.
- **Females:** The dam of a market pig is usually a white breed cross because of their good milking ability and large litter sizes. Common white breeds are Yorkshire and Landrace. Some females will have a small percentage of Duroc (a traditional sire breed) to enlarge her body frame.

Buildings:

- **Farrowing house:** The building where female pigs give birth to their piglets (farrow). Piglets nurse the sow until they are weaned. Weaning ages can have a wide range among production units but will have a narrow range within a single production unit. For example, pigs worldwide are somewhere between 7 and 28 days old when weaned. However, some production units will wean pigs at a maximum of 16 days of age and some production units will wean pigs at a maximum of 28 days of age. Pigs are usually weaned once a week (typically on a Thursday) so the actual age range of weaned pigs would be 9 to 16 days of age for a herd that has a maximum wean age of 16 days, and 21 to 28 days of age for a herd that has a maximum wean age of 28 days.
- **Weaner building:** Pigs that have been weaned reside in the nursery for about 7 weeks. They usually weigh between 20-30kg when they exit the nursery.

- **Growing building:** Pigs that exit the nursery reside in a growing building until they weigh about 50kg.
- **Finishing building:** Pigs that exit the growing building reside in the finishing building until they reach market weight (90-120kg). Pigs are sold for slaughter at this weight.
- **Wean to finish building:** An alternate production system in which piglets are weaned into a single building and remain in the same building until they reach market weight. There are two main advantages to a wean to finish system: (1) The number of times that pigs are moved is reduced, thereby, reducing stress on the pig; and, (2) Only one building needs to be cleaned thereby, reducing labor needs.
- **Breeding:** The facility in which females are either bred naturally by boars or artificially inseminated (A.I.). Most production units today are use either a combination of A.I. and natural mating or all A.I. mating.
- **Gestation:** The facility which houses pregnant females. Gestation lasts approximately 115 days in the pig.
- **Isolation:** The quarantine facility for new stock is called isolation. New breeding stock should be isolated for at least 30 days before entry to the main herd.

Pig flow:

- **All-in, all-out:** Pigs are moved through production facilities in groups. Groups consist of pigs no more than 1 to 2 weeks apart in age. Pigs are moved into an empty cleaned and disinfected facility. The pigs stay in the same area until they are all moved out at the same time. The facility is then cleaned, disinfected, and allowed to dry before the next group of pigs enters. Facilities can be run all-in, all-out by room, building, or site.
- **Continuous flow-** Pigs are continually moved through rooms such that pigs of various ages and weights are in the same room. Rooms are never completely empty and cleaned. Continuous flow production is not recommended for health reasons.
- **Early weaning-** Pigs are weaned at less than 16 days of age and placed in a **weaner building** or **wean-to-finish** building in rooms with pigs no more than one to two weeks apart in age. These rooms are flowed in an **all-in, all-out** manner. The reasoning is that pigs are colonized/infected primarily by organisms from their dam. Early weaning moves pigs to a clean environment while they still have colostral immunity to protect them from organisms that their dam is carrying. Once in the clean environment with pigs of the same age and health status, a decline in colostral immunity has minimal consequences because there is no other source of infection. **Medicated early weaning (MEW)** uses antibiotic treatment of the sow and/or pig prior to early weaning. **Segregated early weaning (SEW)** is an early weaning process that uses no antibiotic treatments. Results of **MEW** and **SEW** are usually the same. Early weaning protocols will not eliminate clinical signs of most viruses. Additionally, they will not eliminate *Streptococcus suis* or *Haemophilus parasuis* from pigs.
- **Farrow to finish operation-** An operation that contains all phases of pig production from breeding to finishing.
- **Contract producer** - A producer who raises pigs that someone else owns. Any phase of production can be contracted.
- **Pure bred producer/ breeding companies-** The source of breeding stock for pork production units.

PIGS AS INDIVIDUALS

PIGS AS PETS

Pigs are kept rarely as pets in Australia, compared with in countries such as North America. This may be due to the rarity of miniature breeds here (there are some mini pig herds in Australia, but they are mainly confined to human research), or it may be that we are further from Hollywood!

Whatever the reason, there are still some people who keep 1 or 2 pigs in their backyard (and sometimes house) as pets. As a veterinarian, you may be called upon to inspect these animals in their home environment. Alternatively, the owner may bring their pig into you if it appears unwell.

Clinical examination

Pigs in general do not like to be restrained. If the animal is quiet, ask the owner to hold it (ensure you have a non-slip surface). Alternatively, use a Panepinto sling (this suspends the animal in the air). Take care with using snares on pet pigs-not good PR!

Clinical examination of a pig is much like that of any other animal. Rectal temperature should be 38.5-39.5C. Higher temperatures may be due to high ambient temperature/humidity, excitement or fever. Check the heart and respiration rates (see below).

Age	Respiration rate (breaths/min)	Heart rate (beats/min)
6-9 weeks	25-40	90-100
10-15 weeks	30-40	80-90
15-26 weeks	25-35	75-85
Pregnant females & boars	13-18	70-80

Depending on the complaint, the other areas to check include:

- their ability to see and hear
- demeanor
- skeletal soundness
- check feet for sores & fissures
- monitor the animal's walk for signs of lameness
- correct positioning of head (nystagmus = CNS or inner ear disease)
- examine skin for dermatitis due to allergies, external parasites, seborrhea, infectious agents, pityriasis rosea
- check mucous membranes
- observe character & quality of respiration

If you are asked to do a home visit, check the following:

- Does the owner have council permission to have the pig?
- Ensure the housing has an area that is dry, clean and draft-free
- Avoid sunburn if outdoors-ensure lots of shade
- Avoid slippery and/or overly rough concrete floors
- Pigs need a resting area (warm, dry, draft-free) and a toilet (1-2m from the resting area).

- Pet pigs should have bedding (pine shavings, straw, rice hulls) for rooting & nesting
- Take care with fencing-pigs will root under a fence
- Commercial pig pellets are best to feed (take care with feeding illegal swill!)

Anaesthetics & Surgical Manipulations

Venipuncture:

- anterior vena cava most common: 18-20G, 1-1.5 inch needle; go on right to avoid left vagus nerve, direct needle toward top of opposite shoulder
- auricular vein: side restraint by 2 people; pick a large vein, use finger pressure at base of ear to raise it; butterfly infusion sets good

Chemical restraint:

- atropine sulphate: 0.02-0.04 mg/kg IM, SC or IV as a pre-med to prevent bradycardia & salivation
- ACP (0.03-0.1mg/kg) or Azaperone (0.5-2mg/kg) or Ketamine 1-2mg/kg /Xylazine 0.5 mg/kg combo:facilitates IV catheterization for subsequent anaesthetic administration

Anaesthetic options:

- minimize stress & pain; small pigs-mask induction/large-IM or IV induction
- take care!; high % body fat; tendency to malignant hyperthermia (care with halothane); small heart, lung & circulatory capacities (take care with prolonged dorsal recumbency); poor thermoregulatory capacities
- withhold food 24h and water 4h prior to administration

Inhalation anaesthesia:

- isoflurane preferred-short recovery
- halothane-care with malignant hyperthermia-if temp increases + tachypnea, hyperventilation, muscle rigidity, blotchy cyanosis or tachycardia>> take off Halothane
- hard to intubate

Surgical procedures:

- Castration: on-farm 2-5 days of age; in older animals use local anaesthetic or general anaesthetic; similar to neutering dogs
- Ovariohysterectomy: best at 4-8 months; as for dogs (ventral midline abdomen approach); care! Ovarian ligaments not as strong as dogs.
- Rectal prolapse: if fresh, clean, lubricate & replace with purse-string suture; infuse antibiotic/steroid cream into rectum for 3-5 days to aid lubrication & reduce swelling; remove suture in 5-7 days; if prolapse torn or dried, amputate-beware! Prone to anal strictures-rectal ring+ elastic band-amputation in 3d under sedation
- Cutting boar's tusks: every 6-12 months under heavy sedation or GA; Gigli wire at gum level
- Hernias: inguinal & umbilical lesions occur spontaneously-genetic predisposition; repair dorsal recumbency under GA

Vaccination:

- Depends on the number of pigs kept
- Probably not necessary
- Erysipelas & Leptospirosis (+ Parvovirus if kept for breeding)

BIOMEDICAL RESEARCH/XENOTRANSPLANTATION

Pigs have similar anatomy & physiology of the cardiovascular & pulmonary systems; hence they are used in biomedical research as models for human disease. They are also a similar size, morphology & physiological characteristics to humans and may be used for models of coronary blood flow, growth of the CV system and pulmonary development.

Specific models for research

- Cardiovascular studies: myocardial infarctions, aneurisms, vascular surgery, cardiac bypass, pacemaker implantation
- Digestive system studies: intestinal bypass/anastomoses, fistulation/colostomy, rectal prolapse, gastrotomy, pyloroplasty
- Endoscopy/Laparoscopic surgery: less invasive, decreased morbidity, increased recovery, improved cosmetic effect, training for surgeons
- New areas: replace dogs & primates for musculoskeletal and CNS models

Xenotransplantation

- Xenotransplantation=transplantation of organs, tissue or cells from one species to another
- Research stems from a shortage of human organ donors + developments in genetic technology to make donor and recipient animals more similar to avoid rejection problems
- Pigs are favored: anatomy & physiology similar to humans, easy to breed, suitable for genetic modification.

Issues:

- Is it ethical?
- How well does it work?
- What are the risks?
- How can the welfare of animals be protected in both animal-to-animal studies & animal-to-human trials?
- How would animal-to-human transplantation be managed?
- What are the alternatives?
- How would resources be allocated?
- How would animal-to-human transplantation research be regulated?

SUSTAINABLE PIG PRODUCTION

THE AUSTRALIAN PIG INDUSTRY

There are a bit over 300,000 sows in Australia housed on about 2000 farms (Australian Pig Annual 2005). New South Wales is our biggest pig producer, with around 30% of the National sow herd. The number of pig producers has decreased from 49,000 in 1960. Despite this, sow numbers have increased from approximately 220,000 sows. This means that the average herd size is getting bigger. Pig farms are also becoming more vertically-integrated. In other words, the same company grows and/or buys the feed, farms the pigs and also owns the abattoir. In some cases, they may also own marketing brands (eg. Castle Bacon, Don Smallgoods).

OVERSEAS MARKETS

Until the mid 1990's the focus of the industry was almost solely on the domestic market because international competitiveness was not critical to its profitability. We slaughter about 5 million pigs each year. As the Australian pork industry is quite small, producing only 0.4% of the world pork production, it is unable to compete on volume. Therefore, Australia tends to focus on niche markets. Our main export markets are Singapore, New Zealand and Japan.

In the past few years, exports have contracted significantly. This is due primarily to the high Aussie Dollar. In 2008 we are exporting very little – mainly offal.

The advantages we have in terms of global competitiveness are proximity to Asia (capability to export chilled pork to these markets) and freedom from some diseases.

Our weaknesses are a small domestic population, unstable feed grain prices and security of supply and a lack of scale economies within the pork processing industry.

One of the problems facing the Australian pig industry is overseas imports. Imports from Canada, Denmark and North America have now become a significant component of the Australian market place. In 2002, import levels were at 20% of processed meat production. In 2008 they are approximately 80%. The bulk of this increase has come from North America, where over-supply is an issue. Meat coming in from countries with PRRS virus must be cooked at 70C for 11 minutes to destroy the virus. Imports can arrive raw and be cooked on-shore, or they can be cooked overseas before coming in. Imports coming in from countries that have PMWS must be boned-out and the major lymph nodes removed before it can be imported. So if you buy ham-on-the-bone you should be buying Aussie grown!

Exotic and zoonotic disease will play a major role in determining market accessibility and where pork will be produced globally. Combined with this, consumers globally are becoming increasingly aware of the quality of their food and how it is grown-freedom from disease, food safety and product integrity must be safeguarded. Australia remains free of the major epidemic diseases of livestock and many of the serious diseases of pigs. Hence prevention of disease entry is a key priority for the industry.

What the Australian pig industry is trying to achieve

The ultimate aim of pig production is the efficient, sustainable and consistent production of high quality pork. Our customers' requirements are different now than they were 10 years ago. Today we are competing in a global market. The wants of our importing countries are different from those of our domestic markets. For example, Asian communities are very reluctant to eat meat that is derived from male pigs, due to the potential for "boar taint". To compete against the likes of Canada, North

America and Denmark, we have to produce meat at a low price. All this means that we have to have a greater control of production events.

COST OF PRODUCTION

Piggery targets

The main factors that drive piggery sustainability are:

- Consistent throughput (kg saleable dressed meat) every week
- Cost of production (cents per kilogram carcass weight)
- Pigs weaned per mated female per year (breeding herd efficiency)
- Growing herd throughput/volume (kg sold per m² piggery space per year)

Targets for these are:

Parameter	Target
Kg saleable dressed meat every week	Minimal variation (< 5%)
Cost of production (c/kg dressed)	Less than \$2.00
Pigs weaned per mated female per year	23 pigs/sow/yr
Grower herd throughput (kg live pig meat /m ² /yr)	450-550

One of the major factors driving the profitability of a pig farm is its cost of production. Production costs are divided into the following areas:

Expense:	Feed	Labor	Overhead (R&M, cartage, contractors)	Herd (health, recording, AI, livestock purchases)	Shed (power, gas, bedding, cleaning, water, effluent)
% total	65	15	10	6	6

The vast majority (approx 95%) of pigs are sold or moved directly to an abattoir. The rest go to abattoirs via saleyards. Processing weight is normally about 100kg live (or 75kg dressed). Payment is made on a grid of weight & backfat (P2 measurement). Prices are around \$2-3/kg carcass for 'bacon' pigs and \$1/kg for cull breeders. Therefore if price received is \$2.50 and COP is \$2.20, then the margin per pig is 30cents/kg x 75kg = \$22.50.

On a 100-sow unit, the value of improving litter size is (PigStats 2002):

Average litter size born alive	A	10.5
Average cost of production/kg dressed	B	\$2.11
Sale price/kg dressed	C	\$2.63
MARGINAL COSTS & EXTRA PROFIT OF EXTRA PIGS BORN ALIVE		
Cost per extra pig (Bx60%) 60% of costs are repeated	D	\$1.27
Margin per kg dressed weight (C-D)	E	\$1.36
Extra profit per extra pig (E x carcass weight)	F	\$95.48 for 70kg pig
Increase in litter size born alive	G	0.5 pigs
Increase in litter size sold	H	0.4 pigs
Increased profit per litter (FxH)	I	\$38.19
Litters per sow per year	J	2.18
Increase in profit per 100 sow piggery	J x I x 100	\$8,325.42

VOLUME & CONSISTENCY OF VOLUME

With other livestock species (eg cattle, sheep), there is usually an annual pattern of activity set by the breeding season – the unit of time in terms of production is the year.

Pigs are quite different. The unit of time on which piggeries operate in a production sense is the week. In any week in most piggeries, all the activities associated with production occur-sows are mated, sows farrow, sows are weaned and pigs marketed.

It is this constant production flow that is critical to efficient production-ideally a piggery should have the same number of sows mated each week, farrow each week and same number of pigs marketed each week. Facilities are optimally used all the time-fluctuations in production create troughs and peaks in production.

The system should be balanced around the farrowing accommodation because this is the most expensive part of the system to establish and maintain.

EFFICIENCY

* Potential litters/sow/year = 2.6 (365/114d gestation+21 days lactation+5 days wean to re-mate).

Parameter	Target
Litters/sow/year	2.3
Farrowing rate	85%
Pigs born alive/litter	11
Pre-weaning mortality	10%
Pigs weaned/litter	10
Weaner death rate	<2%
Grower/finisher death rate	<1%
Average growth rate (birth to sale)	600g/day

Assuming target 10,000kg pig meat sold per week:

10,000/75kg per pig = 133 pigs per week

At 13% death loss post birth 133 x 0.13 = 17 pigs

133+17=150 pigs born per week

@ average born alive/litter = 10.5 >> 14 litters/week

If farrowing rate = 85% >> 14/0.85 = 17 sows to mate each week.

HOUSING SYSTEMS

Pig housing is broken up into phases:

Mating (Boar shed) >> Gestation (Dry sow shed) >> Farrowing >> Weaner >> Grower >> Finisher

Commercial pig production started in Australia in the late 1960's. This was the start of indoor or "intensive" rearing of pigs. The intensive housing of pigs provides a number of advantages: control of feeding & environmental temperature, efficient labor utilization and waste product collection.

There are six areas of basic information needed for sound design of animal housing:

- climate modification
- zoometrics and animal behavior
- structural design

- work routines and labor requirements
- handling of pigs, feed and wastes
- economics (capital and operating costs)

Site

Choose a piece of land that is well suited for piggery operations:

- isolation from neighbors
- sufficient area to allow compliance with waste management guidelines well into the future
- adequate high quality water
- suitable soil type which must cover the needs of building site, waste disposal area and holding ponds
- favorable aspect and slope since solar effects and drainage considerations will be easier to plan for if the location is carefully chosen
- proximity to feed (grain growing areas)
- proximity to processing plant
- a tree screen is useful to obscure the enterprise from public view

Management plan

A management plan for the piggery should be based on the following:

- restrictive or ad-libitum feed
- age of piglets at weaning
- stall or loose sow housing
- age of pigs at processing
- animal movement plan
- waste management techniques

Design of buildings

As building costs are high, it is important they are designed with the following considerations:

- be able to move pigs in an “all in/all out” fashion to maximize health
- capable of being expanded without disruption to the enterprise
- constructed of low maintenance materials
- well insulated
- designed to handle heat loadings through natural ventilation
- capable of discharging waste without contamination of the pigs’ living area
- laid out to facilitate pig movements and to reduce feed handling
- designed to reduce unproductive work while encouraging stock supervision and improving animal welfare

“Traditional” housing types are concrete-based. The floors are usually built from a combination of concrete (solids or slats) and/or woven wire mesh. Some floors are made of wood, but this is hard to clean, wears quickly and can be slippery when wet. Pigs are usually housed in pens of 20-30 pigs per pen. The pen dividers are usually made from concrete or metal. Ventilation can be “natural” (ie. non-automated) eg. side blinds and top “monitor” blinds or “mechanical” (ie assisted or automated) eg. fans and air inlets. Effluent is usually stored for a short time in pits under the pigs’ pens. They can be deep or shallow. They are generally cleaned out using a flushing system with water (hence they are referred to as “liquid effluent systems”). The specifics of the housing system will depend on the age and physiological state of the animal.



More recently (early 1990s), producers in Australia started rearing pigs in bedded shelters. These buildings are also referred to as “ecoshelters”, “igloos” or “hoops”. They made be specifically built or may be made by converting existing buildings. These types of buildings are attractive to build because of their apparently welfare-friendly nature, low capital costs and their use in expanding existing piggeries to improve efficiency (eg. Conversion to all in/all out production). These sheds usually have little internal structure (pigs are grown “free range”), use bedding (straw or rice hulls) to soak up effluent, are on a concrete or clay base and are naturally ventilated. Some of the key issues to consider when growing pigs in bedded systems are:

- availability & cost of bedding – this is particularly important in drought times
- growth performance-pigs tend to grow faster but have poorer feed conversion efficiency
- meat quality-pigs tend to be fatter
- health-difficult to treat individual pigs; may be a buildup of pathogens in shelters over time
- air quality-generally worse in bedded systems due to increased dust, ammonia and bacteria levels
- cooling systems-bigger pigs tend to suffer from heat extremes in these systems if overhead sprayers are not supplied
- welfare-probably improved in well-managed systems. Can be a problem if soiled bedding is not removed and replaced
- odor-probably reduced over liquid effluent systems
- use of compost



THE BREEDING HERD (MANAGEMENT)



TARGETS

PARAMETER	TARGET
Farrowing rate	>85%
Totalborn per litter	>11.5 pigs
Number of pigs born alive per litter	10.5 pigs
Percent stillborn pigs	<6%
Percent mummified fetuses	<1%
Pre-weaning mortality	<10%
Pigs weaned per litter	>9.5
Pigs weaned/sow/year	>23

RECORDING SYSTEMS

It is vital that pig producers are able to keep track of the performance of their herd using herd records. Depending on the size of the herd, the farm may do this manually or using a computer. Producers may develop their own computer-based herd recording schemes or they may purchase a software program (eg. PigChamp, PigWin, MIPs, PigTales). These systems allow the producer to track their herd's performance, generate "Action Lists" and to data analysis to undertake problem solving in the herd.

Basic records that should be kept include:

- number sows mated & farrow each week
- number of sows returning on heat and preg-test-negative (to determine conception rates & pregnancy failure rates)
- number piglets born per litter (alive & dead)
- number piglets weaned each week
- deaths (by reason) in each stage of production
- sale weight, P2 backfat and age (to determine growth rate)
- feed usage (to determine feed efficiency)

REPLACEMENT STOCK

As close to 99% of new diseases are brought on to farms via the introduction of pigs, it is important to select a genetic supplier with similar or improved health status than that of the purchaser's herd. Use a single supplier of breeding stock (if bringing in live pigs) to reduce the risk of bringing in new diseases to the piggery. This is also important from a nutritional perspective as different genotypes may require different specifications.

Some of the tools used to determine the disease status of a herd are:

- clinical observation (eg. coughing, diarrhoea, deaths, scratching)
- herd health records (does the herd meet acceptable performance targets for growth and mortality?)
- post-mortem results from pigs that have died on farm

- results of health checks undertaken at abattoir processing (by a veterinarian or trained officer)
- medication records (many medications can mask disease symptoms)
- diagnostic tests undertaken on specimens submitted from pigs and conducted by laboratories with specialist pig expertise (eg. bacterial culture on dung samples for the swine dysentery bug, blood tests for pleuropneumonia, PCRs for Enteic and respiratory pathogens)

There are some diseases that are not present in all pig herds, and which are major production-limiters. Eg Swine dysentery , Mycoplasma pneumonia.

There are also some diseases that are present in all pig herds. The table below lists examples of these:

Performance-limiting pig diseases. Those in the first column can be eliminated from pig herds. Those in the second are found in virtually all herds.

Diseases to watch out for	Diseases that you can't avoid
Swine dysentery	Glassers disease**
Mange	Ileitis
Mycoplasma pneumonia	Colibacillosis***
Pleuropneumonia	Greasy pig disease
Atrophic rhinitis	Strep. Meningitis***
Worms	Erysipelas
	Leptospirosis

By sticking with the one supplier of genetics you may be able to avoid certain serovars of these bacteria , some of which are more devastating than others.

Quarantine

The purpose of quarantine is to protect the main herd against the introduction of new infectious agents that cause economically damaging disease. The following should be considered part of a quarantine program:

- Identify the health status of incoming gilts, in relation to the health status of the purchaser.
- The gilts should be vaccinated against erysipelas, parvovirus, leptospirosis.
- The ideal quarantine housing would be at least 3km away from other pigs. It would have provision for clothing and boots used only in the quarantine area. It would be emptied of pigs, cleaned, disinfected prior to occupation by newly purchased stock. Non-medicated feed would be used.## Any feed, equipment and vehicles present on the main farm would not enter or leave the quarantine area without thorough cleaning and disinfection.
- If no separate shed is available away from the piggery, house incoming gilts in a separate pen in a well-ventilated area in the piggery, preferably with separate drainage and in an area lightly stocked. ***
- Use separate boots and protective clothing when working with animals in quarantine.
- ## If in doubt about the Swine dysentery or Spirochaetal status of the supplier herd you may elect to medicate the pigs for a 2 week period before allowing them into the herd.
- *** Bear in mind if the newly introduced pigs break with Mycoplasma the breeding company will reimburse you for the 20 replacement gilts only, not the other 10,000 pigs already there which subsequently come down with the disease.. A separate quarantine facility is part of the purchasing contract.

Acclimatisation

Acclimatisation allows new replacement gilts to adjust to the diseases present in the purchasing herd that they have no immunity to, as well as new feed, housing and management system. Major problems arise when gilts of high health status herds (eg. a herd free of *Mycoplasma pneumoniae*, pleuropneumonia or swine dysentery) are put straight into a herd where these diseases are present.

The following procedures will assist in dealing with specific diseases:

- Feed-back*-using tissue and/or excreta from animals is a way of naturally immunizing incoming animals. It may be a useful practice to prevent diseases that for which there are no vaccines currently available. These include congenital tremors, ileitis and rotavirus.
- Parvovirus-vaccinate the gilts twice, the first injection on arrival and again 2-4 weeks later. This is usually given as a combination Parvo_Lepto_Ery injection as these 3 diseases can cause pregnancy failure. Exposure to the faeces* of the sentinel growers will also boost their immunity.
- For E coli and rotavirus-expose the gilts to weaner and sucker manure* three times a week. Vaccinate gilts at 9 and 13 weeks of pregnancy with an E coli vaccine. Lots of farms give a combination booster at this stage of E.coli_Erysipelas_ Lepto_. This confers good maternal immunity to offspring as well as boosting the sows immunity.
- If the gilts are entering a *Mycoplasma*-positive herd and or a glassers problem herd from a negative herd, consider using a *Mycoplasma* vaccine and a Glassers vaccine. Vaccinate gilts on arrival and again 2-4 weeks later.
- Congenital tremors-exposing the gilts to farrowing house and weaner house manure* three times a week will assist in exposure to this. Some vets also favor wiping the vulva of recently served sows with a tissue to obtain boar semen and place these in the pens three times a week. Feeding this concoction via the water through a dosatron or medication proportioner is also a convenient way of doing this.
- Leptospirosis and Erysipelas-ideally the replacement gilts will have received two vaccinations prior to arrival. If not, vaccinate them within a week of arrival and again 2-3 weeks later.
- Ileitis-exposure to faeces* and growers may infect replacements breeders if they are naïve. Ideally they should have a period of 7-14 days with no medication and then treatment with an effective antibiotic prior to another period with no medication. This may have to be done after any initial medication coverage you give incoming breeders.

*Check with the State Regulatory Department before undertaking feedback. In some states (NSW) it is regarded as swill feeding.

During quarantine and acclimatisation, any outward signs of disease (excessive sneezing, coughing, diarrhoea, presence of blood or mucous in faeces, skin lesions, loss of appetite or lameness) should be noted. It is best to avoid masking the disease symptoms with medication. Note that you should not allow the replacement gilts out of quarantine into the main farm without written permission from the veterinarian who is in charge of the breeding company. Verbal will not stand up in court, so do it via E-mail or fax.

Routine vaccination programs for gilts/sows and boars

Vaccination is low-cost insurance against disease. A number of vaccines are available off-the-shelf for routine use in pig herds. Some vaccine manufacturers are also (GMP) licensed to produce specialist “autogenous” vaccines to protect against diseases caused by pathogens when no commercial vaccine is available. The following points provide background information to assist you to develop a routine vaccination program.

- The vaccination program starts with an injection soon after selection but full protection comes only 2 weeks after the booster shot. Combination vaccines are now available:

- Routine vaccination against erysipelas, leptospirosis, parvovirus and E coli should be considered when introducing young gilts to the herd.
- When naïve animals are introduced into a herd with endemic disease, other vaccines are recommended. These include Mycoplasma, Glassers disease, proliferative enteritis and pleuropneumonia vaccines.
- Leptospirosis, parvovirus and erysipelas boosters must be given before mating to ensure adequate protection for the developing fetuses.
- With E coli vaccinations, the objective is to produce maximum antibody levels in the gilt's colostrum at her first farrowing. The booster for E coli must be given 3 weeks before farrowing. The primary injection can be given at selection, or as late as 4 weeks before the booster shot.
- Booster vaccinations should be given for erysipelas and leptospirosis about 3 weeks before farrowing. This will boost antibody levels in the sow's colostrum, giving additional protection to the litter, as well as providing protection to the sow for her next pregnancy.
- Vaccination of older sows against E coli and parvovirus may not be necessary and will depend on the immune status of the herd.

A vaccination schedule for new unmated gilts

	Leptospirosis	Erysipelas	Optional*	E coli	Parvovirus
At selection	+	+		+	+
2-4 weeks later	+	+	+		+
2-3 weeks before farrowing (gilts)	+	+	+	+	
2-3 weeks before farrowing (sows)	+	+		+	+

*Depends on the immune status of the herd.

*Optional will depend on the health status of the herd and may include Glassers and APP vaccination

GILT MANAGEMENT



Performance targets

Gilt cycling within 3 weeks of joining herd	More than 70%
Average time taken to reach puberty	1-3 weeks
Average age at puberty	27 weeks
Gilts non-cyclic by 30-32 weeks	less than 5%
Average age at first service	30 weeks
Weight at first service	> 125kg
First litter size (born alive)	10.0

To meet these targets:

- Maintain sufficient gilts in gilt pool (rule of thumb is a gilt pool roughly 10% of the breeding herd size). If you have a 100 sow herd, you will mate about 6 females each week to get 4 farrowings each week. Then you need to have at least 7 gilts in the gilt pool each week. This allows for a 10% gilt wastage (1 gilt), 1/3 will be cycling (3 week cycle), so you will end up with 6 matings per week (4 weaned sows + 2 gilts).

- Apply suitable quarantine and acclimatisation procedures (for purchased gilts).
- Handle gilts gently at all times. Minimise the number of negative handling events.
- Vaccinate at selection and 4-6 weeks later. Vaccinate against any endemic disease in your herd (eg *Mycoplasma hyopneumoniae*, APP, Glassers, proliferative enteritis) before the sows arrive.
- Ensure gilts are housed on good quality flooring. Solids are better than slats.
- Feed a Grower/Finisher or Gilt Developer diet ad libitum up to mating.
- Provide 1.5-2.0 m²/gilt.
- Implement a puberty stimulation program for all gilts.

Use mature boars (10 months or older)

Use a boar that gets regular matings – at least one each week

Put gilts in the pen with the boar, for best results. Fenceline contact with one or more boars is much less effective, although it is still better than no boar contact, A lot of the stimulation comes from the boar pheromones which can only be achieved by direct contact, not through a fence.

Repeat boar exposure daily, and don't stop over the weekend – especially in summer and autumn if there is a problem with gilts not “coming on”.

20 minutes of daily exposure is sufficient for small groups of gilts (4-12 pigs/pen).

With larger gilt groups use boar “gangs” - approximately one boar for each 12 gilts

- Delay mating until at least 28 weeks of age and 120-130kg liveweight.
- Gilts that have not cycled by 6 weeks into the mating period should be culled. But you may like to try an injection of PG 600 before you cull stale gilts as you have a lot of money invested in them.
- Regumate (oral progestagen) can be used to synchronize gilt batches. Feed for 18 days continuously. Take care not to under-dose as this can cause cystic ovaries.

MATING MANAGEMENT



Performance targets

	Target	PigStats (2002) average
Conception rate (3 week non-return rate)	>92%	89%
Farrowing rate	>85%	85%
Litter size (born alive)	11	10.5

To get good conception/farrowing rates and large litters at birth it is essential to conduct good oestrus detection daily and to time matings or inseminations accurately. Once this has been achieved it is still possible to reduce the sow's reproductive performance if the quality of the mating or insemination is poor.

Heat detection

“Oestrus”, “on heat” and “cycling” all refer to the same phenomenon – a two to three-day period, at either puberty, the end of the 21-day reproductive cycle of a gilt or a few days after weaning for a sow, when the female shows the standing response to the boar and can be mated/inseminated.

Poor oestrus detection results in gilts and sows which are ready for mating not being mated/inseminated. This leads to fewer matings/inseminations overall and subsequently fewer pigs sold. Poor oestrus detection can be a major problem, particularly in gilts where the oestrous period is shorter and less marked.

It is not uncommon for the oestrus detection rate (% of oestrous gilts actually detected) to be as low as 50%. These rates are based on abattoir examinations of ovaries which showed that many gilts, culled because they had not been detected in oestrus, in fact had been cycling. Good housing conditions make oestrus detection easier. Any conditions which make sows more comfortable help with oestrus detection.

There are two basic methods to detect oestrus: (1) **Back Pressure Test (BPT)** and (2) **Boar Test**.

Whichever method is used, there are early indications of which gilts/sows are coming on heat: swelling and reddening of the vulva, increased restlessness, attempts to court, mounting pen mates and attraction to the boar.

Back Pressure Test

The essential requirements for doing the BPT successfully are:

- move the sows out of their own pen into the corridor and next to the boars' pen. Do not do the BPT in the sows' own pen.
- the boars used to stimulate the sows should be more than 10 months old.
- an important principle of the BPT is that it most efficient if there are a number of boars present to stimulate the sow.
- when the sow is in head-to-head contact with a boar, approach quietly and gently massage her flanks and then apply hand or sitting pressure on her back (shoulders to mid – region), and look for signs of the standing response, indicating the sow is in oestrus.

A slight response may indicate the gilt is at the beginning of her oestrous period, and will respond more positively in eight to 12 hours. If suspicious, leave the gilt near the boar for a few minutes, and then repeat the procedure. For best results, the BPT should be conducted when the sow is first introduced to the boar. It is harder to get a response to the BPT if the sow has been next to the boar for some time – even five minutes can make a difference.

The standing response signs are:

- standing rigid in response to the back pressure test. Sows that stand should allow the stockperson to quietly sit on their backs
- pricking of ears
- attentiveness to boar
- characteristic grunts
- mucus check

The BPT is less effective if gilts are continuously housed next to the boar pen, compared to over the corridor or with at least one pen separating the gilts and boars. These gilts become too familiar with the presence of the boar and the boar has less impact in encouraging the standing response. The same problem does not occur with weaned sows. Housing them next to the boar does not affect the oestrus detection rate, because they are next to the boar for a relatively short period of time before cycling commences.

The Boar Test

This test relies on running a boar with the sows to see if they show the standing response to the boar:

- take the sows to the boar's pen or to a pen familiar to the boar. The boar has fewer distractions and wastes less time exploring the surroundings.
- the boar should feel comfortable. High temperatures, an unfamiliar pen, a slippery floor or an aggressive stockperson may inhibit the boar; to the extent he may not court the sows.
- to be effective, a "keen" boar must be used. The process will not work unless the boar has good sexual motivation. It is a good idea to do the oestrus checks in the same pen the boar uses for matings, to encourage their sexual interest.

- older boars should be used where possible because boars under 10 months of age are less efficient at stimulating sows.

Housing and oestrus detection

Housing conditions which improve the females' comfort make oestrus detection easier.

- **Stocking rate:** There is an opportunity in many herds to improve oestrus detection by providing post-pubertal gilts and sows with more space. Post-pubertal gilts should have space allowance of at least 1.4m². Allow 2m² for weaned sows ready for mating.
- **Floors:** Firm, flat and dry floors make oestrus detection easier. Slippery floors will make oestrus detection more difficult. Slatted floors may be harder on gilts' feet and result in premature culling due to lameness.
- **Hot weather:** Oestrus detection should be done in the cooler parts of the day – early in the morning and in the evening. Intermittent spray cooling will help in hot weather, but be careful the spraying does not lead to the problem of slippery floors in the detection pen.

Matings/inseminations –when is the right time?

A single mating at the correct stage of the oestrous cycle will give a good conception rate and litter size. The difficulty is knowing the exact time the sow comes into oestrus. Most gilts and sows are mated two or three times during oestrus to be sure of a good conception and large litters. The timing of matings depends on how frequently oestrus checks are done, and whether the female is a gilt or a sow. A recommended mating (or insemination) schedule, using two matings, is:

- **If oestrus detection is done once daily** (i.e. oestrus may have commenced up to 20 hours ago):
Gilts–mate at first detection and 12-24 hours later; Sows–mate at first detection, or delay first mating 12 hours. Mate again 24 hours later
- **If oestrus detection is done twice daily** (i.e. oestrus may have commenced 4-10 hours ago):
Gilts–delay 12 hours then mate, followed by a mating 12-24 hours later. Sows–delay for 12-24 hours then mate. Mate again 24 hours later. If litter size is considered low, a third mating, 12 hours after the second, could be included. Sounds like witchcraft to me? This is the very issue that the fat controller went to water on!



WARNING: Only mate/inseminate females that are definitely on heat (i.e. showing a standing response to the boar). This is particularly important in late oestrus as matings/inseminations given after the sow has ovulated may cause infection and wastes time and semen. Equally don't serve animals too soon, they may be showing classic signs of proestrus in their behaviour but will not stand rock solid for the backpressure test. The problem is if you start to serve too soon you will invariably stop serving too soon.

Natural matings

- ensure a good mating pen (eg. Use a “detection mating area (DMA)
- supervise each mating
- assist where required
- 3 minute ejaculation
- ensure the boar and sow are compatible sizes

Artificial insemination

Why use AI?

- introduce new genes without risking health status
- use boars with superior genetics over more sows
- reduce the number of boars required at the piggery
- reduced labor requirement

Collection of semen:

Equipment: esky, thermos & screw-on lid, gauze & rubber band, 2 rubber gloves, diluent

Method:

- Clean up the boar
- While mounting a sow on heat or dummy sow
- Boars respond to pressure on their penis>>ejaculate
- Avoid pre-sperm fraction (no sperm, contaminated)
- Collect into pre-warmed thermos flask covered with gauze
- Discard gauze, add diluent and screw on thermos lid

Examination:

- Colour (creamy white), volume (50-450mL), motility (>70% moving forward), concentration (minimum dose rate $3-5 \times 10^9$ sperm/dose diluted to 80mL), morphology (<20% abnormal)
- Use immediately or add extender-shelf life 3-5 days
- Keep at 30-35C if used within hours; cool slowly to 18C if stored
- Invert at least BID to re-suspend (don't shake) This is to ensure adequate mixing of the extender which becomes depleted in the area closest to the settled sperm.

The quality of an insemination is determined by:

- Semen quality
- Experience and motivation of the inseminator
- Soundness of the insemination procedure

Semen quality should not be a problem in most situations assuming that it is maintained at 17-180C, rotated gently twice daily while stored and is used within 72 hours of collection.

Oestrus detection and the timing of inseminations should be the same as described for natural mating. Insemination staff need to be well trained to understand the full procedure required to maximise the success of an insemination & should not conduct more than 20-30 consecutive inseminations before taking a break – otherwise farrowing rates and litter size will start to drop for the later inseminations.

The insemination process aims to place as many live sperm as possible inside the tract of an aroused, oestrous female. The elements of a successful artificial insemination are:

- good oestrus detection to ensure the sow is in standing heat
- provision of head-to-head contact with a mature boar throughout the insemination process (N.B. the boar should be used to stimulate no more than 3 sows at a time)
- insert the catheter into the vulva at a 300 upward angle, pushing gently until the resistance of the cervical opening is felt
- if using a foam tip catheter push gently into the cervix until a lock is achieved (catheter remains fixed when it is gently tugged back)
- if using a spirette catheter gently rotate the catheter counter-clockwise (to the left) until it springs back a quarter turn

- attach the semen tube, bag or bottle to the catheter (if using a couchette bag this step could have been taken prior to catheter insertion)
- start semen flow by applying gentle pressure to the semen container
- allow free flow of semen into the sow (be patient – allow 2-6 minutes) – if semen doesn't flow re-position the catheter slightly
- throughout the insemination – including for 1-2 minutes after the semen container has emptied - continue to stimulate the sow by massaging her udder, flanks and back
- either crimp or plug the catheter and leave in place for a further 5 minutes or so

Trouble-shooting AI problems:

- Poor oestrus detection
- Incorrect timing of mating
- Poor overall herd performance
- Operator fatigue
- Poor technique: equipment handling, hygiene & storage; sow stimulation; catheter insertion; insemination hygiene
- Semen quality: storage & transport; boar fertility

Gestation Management

After mating, sows may be housed in groups or stalls. They are generally fed a restricted ration of 2.2-2.4kg of a dry sow ration (12.5MJ energy) per day. Overfeeding sows in gestation results in overly fat sows at farrowing, which in turn results in poor lactational feed intake, poor weaning weights and excess condition loss. It is also bad management in terms of feed wastage.

The good and bad points of any housing system can be exaggerated by how the system is managed on a day-to-day basis.

Group housing:

The advantages of group housing include:

- Cost (it is probably the least expensive design);
- Pens may be used for a variety of purposes;
- Because grouped sows can lie together, the temperature requirements in the shed are lower than required for individually stalled sows.
- More opportunity for exercise;
- Fewer injuries than stalled sows;
- Sows are in better physical condition at farrowing, and therefore have a shorter farrowing time and fewer stillborn piglets.

The disadvantages of group housing include:

- Increased early pregnancy loss
- Lack of control over amounts of feed given to individual sows;
- Increased opportunity for fighting and bullying among sows;
- More difficulties managing sows (stock movements, pregnancy testing, vaccinations).

Grouping of sows in pens is prone to over-stocking. The welfare code currently states that at least 1.4m² per sow should be the absolute minimum. Overstocking will lead to an increase in the potential problem areas associated with group housing (anoestrus in unmated sows, fighting, bullying, vulval biting, disparity of feed consumed by each sow etc).

Grouped sows are likely to be in better condition than stalled sows because they exercise more. The extent of lameness and injury will depend to a large extent on the condition of the floor and slats. Muirhead (1981) reported on a case history where the incidence of lameness in sows was reduced from 14% to 1% when faulty slats were repaired.

Sows penned in pairs at the recommended minimum space allowance have been shown to be chronically stressed (Barnett et al, 1984), possibly because of the limited space or because they prefer more company. Include at least three sows in each group.

Provide sufficient floor space or trough space at feeding time to ensure sub-ordinate sows get their share.

Stall housing

The use of sow stalls attempts to overcome disadvantages of penning sows. There is no doubt that sows housed in stalls during the first 6 weeks after mating have better reproductive performance (less early embryonic loss) than group-housed sows. However, stalled sows may suffer from a lack of physical exercise, inability to huddle and an increase in specific health problems. There are a number of factors that may predispose sows to increased bacterial infection of the genitourinary tracts. These include a tendency to lie for long periods and to empty the bladder only when standing, and soiling of the vulva and perineal area.

Shed temperature is particularly important when sows are stalled. As sows are generally fed less than growers relative to their size and maintenance requirements, they require warmer shed temperatures. The lower critical temperature (that below which an animal must eat more feed just to keep warm) is 40C higher for sows in stalls than for grouped sows. The sow's temperature requirements will be increased if there is a breeze. Outbreaks of abortion in winter months may be associated with low sow house temperatures and failure to compensate for increased energy requirement. The insulation of buildings and particularly the control of drafts under doors, through ridge vents and windows are all important.

The Model Code of Practice for Welfare (pigs) was reviewed in 2007. This code states that from 2017, no sow is to be housed in a stall for more than 6 weeks of her gestation period.

Bedded systems

There is increasing interest in the use of semi-intensive bedded housing systems for group-housed sows. These systems are relatively new, and the ideal housing parameters are yet to be determined. However, in general:

- Ensure adequate bedding to ensure sows have access to a clean, dry place to lie;
- Replace overly-soiled bedding;
- Ensure adequate space allowance (more than 2m² per sow);
- Provide enough feeding space and/or use stalls to ensure individual animals have ready access to feed;
- Install spray cooling and/or wallows to protect against heat stress;
- Minimise bullying by avoiding the introduction of single animals into a defined group;
- Ideally, pen animals of similar gestational stage together.

REPRODUCTIVE FAILURE

The profitability of a piggery is determined to a very large degree by the reproductive performance-number of piglets born alive/sow/year. This is governed by two factors-litter size and number of litter each sow produces each year:

Gestation length	114 days
Lactation length	21 days
Weaning-to-oestrus	6 days
TOTAL	141 days
Potential = 2.6 litters/sow/year (actual 2.2)	
Average pigs born alive per litter	10.4
Therefore, number of piglets/sow/year = 23	

Infectious diseases can play a role in reproductive failure, but there is a tendency to blame them for everything. Many micro-organisms affect the pig and be isolated from cases of infertility. However, it is often hard to prove they are involved with the reproductive failure.

REPRODUCTIVE EVENTS

- Fertilization in the ampulla
- 48 hours in the oviduct
- 4 cell stage to the uterus
- In the first 5 inches of the uterus in 4 days+
- Blastocysts hatch on day 8
- Migration begins day 6 (passive for the embryo)
- Migration ceases day 12 (maternal recognition of pregnancy now)
- Implantation, days 13 to 18
- Organogenesis, day 21
- Calcification, days 30 to 35
- Fetal membrane growth stops, day 65-70
- Fetus immunocompetent, day 70-75
- Farrowing, day 112-116

Reproductive failure occurs when the bred sow does not conceive or fails to farrow a normal healthy litter. It may appear as:

- normal return to oestrus (18-24 days)
- delayed return to oestrus (beyond 25 days)
- abortion
- pseudo-pregnancy
- failure to farrow

- mummified foetuses
- stillbirths
- small litter size
- abnormal piglets
- decreased neonatal survival.

TROUBLE-SHOOTING REPRODUCTIVE PROBLEMS

FAILURE TO MATE

Characterised by returns to oestrus after 18-24 days and/or small litter size. The fault may be with the boar or the sow.

■ ***Boar factors:***

Management of mating:

(see previous section)

Boar infertility:

- Incapable: Immature, lack of experience; Defects in penis (persistent frenulum) or prepuce (constriction of preputial orifice); Mismatched for size of sow
- Unwilling: Immaturity; Overuse (5 is a good number of matings/week); Hot weather; Any generalized disease; Arthritis, OCD or foot problems;
- Disease that causes high fever: Erysipelas is most common. Reduced fertility likely for up to 6 weeks; Cull
- Sperm production defects: hypoplasia; high environmental temperature/fever; toxins; infection of testes or accessory sex glands (eg Brucellosis); over-use

□ ***Diagnosis:***

- Examination with an oestrus sow: behaviour; allow the boar to mount and settle on the sow then remove the penis from the vagina & hold by the coiled tip; examine penis; can collect some semen from the anterior vagina/ cervix with an AI catheter post-mating

■ ***Sow factors:***

Anoestrus:

- True anoestrus-no ovarian activity
- Behavioural anoestrus-ovulation occurs but sow fails to show normal behaviour associated with oestrus-not mated.
- Failure to detect oestrus-in situations where the boar is not run with the sows

□ ***Gilt anoestrus a significant problem.***

→ Prevention:

Maximise correct boar exposure-house across aisle & not next to the boar; boar > 10months; run boar in with groups of gilts 15 mins BID; novel boar exposure better than continual exposure to 1 boar

>1.4m²/gilt; maximum gilt number 6 per pen

Acute stress eg. change pens of gilts around; change sheds; transport

Seasonal problem

Hormonal treatment (desperation): PG600 (400IU PMSG+ 200IU HCG) induces oestrus in a proportion of anoestrus gilts 3-5 days post-injection. Results often disappointing

Synchronisation of oestrus: altrenogenst (Regumate-an orally active progestagen) in feed 15-20mg in feed per day for 12-16 days-oestrus 3-6 days after removal

□ **Post-weaning anoestrus:**

- Normally sows come into oestrus 3-10 days after weaning (most 4-6 days)
 - Investigate weaning to oestrus intervals, pattern, parity
 - Most commonly a problem in sows weaning their first litter because: (1) the sow is still growing, (2) excessive body condition loss in lactation due to small appetite, (3) physiological stress of weaning may be greater
- Prevention:
- Ensure sow is not overfat at farrowing
 - Maximise feed intake in lactation
 - Feed a high protein, high lysine (1%), high energy (14.5MJ DE) diet in lactation
 - Minimise heat stress in lactation (reduced appetite)
 - Ad-lib feed post-weaning
 - Ensure good boar exposure post-weaning, commencing no later than 48 hrs post weaning
- Skip a heat & mate on 2nd oestrus-must get an increase in litter size of at least 1 pig to make it worthwhile

RETURNS TO OESTRUS

Important to determine when sows are returning to oestrus as this gives an indication as to what the reproductive problem may be:

- (1) < 18 days-unlikely –may indicate XS prostaglandins from uterus (endometritis) or a systemic reaction (eg endotoxemia). Remember CLs in pigs are resistant to the effects of PGs until after Day 12.
- (2) 18-24 days normal oestrus cycle length indicates: (1) failure of fertilization, (2) early embryonic death before Day 10 caused by a hostile uterine environment. The embryos signal their presence to the sow between days 10-24 in the form of oestrogens which prevent the effects of PGs from the uterus which normally cause involution of CLs and return to oestrus. Must have at least 4 embryos present at this time to provide a significantly strong signal.
- (3) > 24 days (delayed return to oestrus): (1) failure to detect return at 18-24 days, (2) embryonic signal had occurred but pregnancy not maintained because of: embryonic signal inadequate, failure of pituitary support (once ovulation has occurred CLs develop normally without pituitary support for first 16 days of pregnancy); involution of CLs caused by PGs (uterus endometritis, systemic reaction endotoxemia); injection of prostaglandins

DISRUPTION AT THE TIME OF MATING

>>Normal returns 18-24 days + low litter size

- (1) Failure of ovulation/fertilization
- (2) Hostile uterine environment & death of embryos before day 10:
 - a. hormonal (failure of normal ovulation & cystic follicle development)
 - b. infection during breeding or obstetrical procedures after farrowing, or from an infected bladder or kidneys. Local uterine defence mechanisms under the influence of oestrogen usually prevent infection but the uterus is more susceptible to infection

under the influence of progesterone. Results in endometritis-ubiquitous organisms such as E coli, Streps, Staphs etc but there may be a particular pathogen involved eg. Brucellosis. Sows with discharges around breeding time have a much lower farrowing rate than normal sows. Treatment is usually unsatisfactory & it may be best to cull.

- c. olaquinox (>100g/t)
- d. Mycotoxins (from moldy grains) exert hormonal effects on the sows and may reduce reproductive performance.
- e. "Stress" hormones (eg. cortisone) may depress an animal's resistance to infections, such as Salmonella and Erysipelas, and cause infertility.
- f. Parvovirus and Leptospirosis.
- g. Mixing in groups at 10-14 days has been shown a tendency to reduced farrowing rate (Kirkwood, 2007)

DELAYED RETURNS TO SERVICE (oestrus?) (> 24 days)

- (1) **Management-failure** to detect a normal return 18-24 days post mating
- (2) **Death of all the embryos between 12 and 24 days.** Eg. Infections in the first few days of pregnancy

About 48 hours after fertilisation, the embryo enters the uterus. The embryo has few nutritional reserves and relies on the uterine fluids for survival. By day 10-11, the embryos become extremely long due to rapid growth of the placenta. They migrate through the uterus until about day 13, when they are evenly spaced and attach to the uterus. The signal for the continuation of pregnancy is given around days 12-13. At least four foetuses are required to initiate the signal. It is thought that the placenta secretes a hormone (oestrogen) from about day 12, which is recognised in parts of the brain (pituitary gland) and the ovary.

Up to this stage of foetal development, infectious agents either inhibit the growth of the placenta, or affect the uterine lining and the nature of the uterine fluids. For the pregnancy to fail completely by day 12-13, not more than 3 embryos can survive the infection. With porcine parvovirus infection, embryonic death is progressive as the virus spreads within the uterus. If the pregnancy fails by day 12, the sow can be expected to return to oestrus within the normal time. Beyond day 12, up to about day 35, (the time hard bones start to form) death of the whole litter will cause a sow to return beyond the expected 18-24 day interval, depending upon when and if the embryo remnants are completely resorbed.

Infectious agents causing severe maternal or foetal reactions can cause birth defects. Others (parvovirus) kill the foetuses straight away.

- (3) **Abortions > 12 days** once the embryonic signal had been registered by the sow pregnant state will be maintained unless terminated by abortion. This abortion may be readily recognized by the presence of her fetuses later in pregnancy but early in pregnancy usually passes unrecognized because of the small amount of material passed (< 30 days).

Caused by failure of pituitary support for pregnancy. Once ovulation has occurred the CL and embryos develop normally up to about Day 16 without any pituitary involvement. After this, CLs are dependant on gonadotrophin support from the pituitary gland.

Foetal death may not alter the course of pregnancy if it occurs after Day 35. Even when the whole litter has died, the sow may farrow normally (often late), or may never farrow.(depends on the level of insult/inflammation/ prostaglandin release. See below

Beyond Day 50 of pregnancy, infection with a range of micro-organisms often results in abortion. Abortion may occur earlier, but may go unnoticed due to the small size of the foetuses.

Abortions may follow placental infections (eg Leptospirosis), or may occur when micro-organisms enter the blood stream, resulting in blood poisoning and fever (eg erysipelas).

Abortion occurs as a result of prostaglandin production by the inflamed uterus acting on the placenta or ovary. Some disease agents (parvovirus, enterovirus) cross the placenta without causing a reaction. Remember parturition is initiated by the effect of fetal cortisol on the production of Estrogen and prostaglandin by the placenta. Severe infections will cause a similar release of cortisol and prostaglandin synthesis leading to premature partition.

Seasonal infertility is the most important example of this:-what exactly is This???

occurs mid-summer to early autumn (weeks 1-16)

sows conceive and embryos are present up to about 20 days

Sows return to oestrus about 28-30 days after mating but frequently do not express normal oestrus behaviour

Litter size is normal so an all-or-nothing effect

The problem may affect up to 50% of a group of sows mated in a particular week but fluctuates dramatically from week to week and unit to unit

Some piggeries do not have a problem at all

Aetiology

Not fully understood:

- (1) As day length starts to decrease past Dec 21.
- (2) Other factors further suppress pituitary activity result in variation in severity eg. Inadequate nutrition, stresses

Control:

- (3) Individual stalls
- (4) Feed liberally for the first 4 weeks of gestation
- (5) Minimise stresses to sows: individual stalls, cooling systems
- (6) Stimulate pituitary activity by exposure to boars, particularly first few weeks of pregnancy
- (7) Manipulation of day length, melatonin etc

Other Causes of abortion:

- (1) prostaglandins-injection at any time after Day 12;natural prostaglandin release following illness? endometritis placentitis (eg brucellosis, leptospirosis); systemic reactions (eg septicemia)
- (2) Autumn abortion syndrome-inadequate nutrition to compensate for colder weather?

NIPs (not in pig) sows

- (3) Failure to detect oestrus (management, failure of oestrus expression)
- (4) Anoestrus following mating
- (5) Death of all embryos after Day 24. Foetuses die without causing sufficient inflammation (PG release) to cause abortion-common cause viral infections

Embryos dying before Day 35 are resorbed, embryos > 35 days after calcification of skeleton etc mummified-absorption of fluid leaving a dry leathery mass. Such fetuses are carried to term with normal litter mates. Fetuses dying in late pregnancy > large mass with little opportunity for resorption of fluids-sunken eyes and autolysis.

The age of the fetuses at the time of death can be estimated by measuring the crown-rump length. If all fetuses die then sow will have a prolonged gestation-usually farrow 120-130 days-some back up mechanism to initiate parturition.

At around Day 70 of pregnancy, the foetus develops its immune system, and this immune response increases with gestational age. After day 70, foetal infection is less likely to cause foetal death. However, if the placenta is infected, the pregnancy may still be lost, due to the effects of prostaglandins on the CL.

Pseudopregnancy or “false pregnancy” occurs when sows demonstrate outward signs of pregnancy, even though foetal death and resorption have occurred. If all embryos are killed at around day 30, sufficient foetal tissue mass may remain in the uterus to maintain pregnancy. Affected sows and gilts appear pregnant and develop an udder, but late in pregnancy they “lose” their swollen bellies and fail to farrow. Pseudopregnancy has also been reported in pigs following ingestion of feed containing the mycotoxin, zearalenone.

NIP treatment-(1) PGs & collect fetuses for further investigation, (2) maximize boar exposure, (3) cull

NIP control-effective early pregnancy diagnosis is important in recognition of these sows.

Normal embryonic mortality

This “normal” embryonic loss is not well understood. May be due to uterine capacity re physical space & nutrient supply. This is probably an evolutionary fail-safe mechanism to ensure maximum number born, by building in a 30% margin for error.

In normal pregnancy, 25% of embryos are lost by Day 35. Do determine this, count CLs, compare to embryos present. These embryos are resorbed and not identifiable.

Approximately 5% are lost from Day 35 to parturition, These are mummified and are identifiable at birth. Particularly common in large litters.

PREGNANCY DIAGNOSIS

Failure to detect non-pregnant pigs so they can be treated appropriately (culled) can be a source of appreciable economic loss. Non-productive days are increased (\$4/day). Sows eat 16kg feed/week @28c/kg=\$4.50/week. However, greater loss associated with her occupying space that could otherwise be occupied by a productive sow. It is estimated that every sow which fails to farrow below your target level costs you \$900.

Methods of pregnancy diagnosis

- (1) Non-return to oestrus. Usually sows closely observed when expected to return at 3 weeks. Especially if use a boar for detection.
- (2) Ultrasound. Usually used in the 5th week (28 days+)
 - a. RTU-most common & accurate now; portable machines on wrist
 - b. Depth-amplitude-detects fluid-filled uterus-inaccurate if pigs close to oestrus (fluid accumulation) or later than 60 days gestation (less fluid)
 - c. Doppler-detects pulsation in uterine arteries of sow & foetal vessels-difficult to use
- (3) Rectal palpation-rare
- (4) Oestrone sulphate in blood or urine-18-36 days post-mating
- (5) Progesterone levels-18-22 days post-mating

STILLBIRTHS

Infectious agents are not common causes of stillbirths in pigs.

Risk factors for stillbirths include: large litters, old sows, hot weather, and a history of stillbirths within individual sows. However, if stillbirth rates exceed 10% of pigs born, it may be pathogen-related. Any agent capable of crossing the placenta may cause stillbirths (eg Leptospirosis). While Parvovirus may cause stillbirths, it is more likely to be associated with foetal mummification.

If stillbirth rates >6%, check:

- (1) Parity, (2) cooling mechanisms in the farrowing shed, (3) supervision of farrowing adequate (no more than 30-45min between piglets), (4) over-fat sows

Diagnosis of stillbirths-(1) toe-caps intact, (2) meconium staining on skin, (3) lungs will not float in water.

NEONATAL DEATHS

Piglets of low viability & die within 1-2 days of birth:

- (1) prematurity (<110 days of birth)
- (2) anoxia during parturition causing brain damage
- (3) nutritional deficiencies eg Vitamin A
- (4) Infection-Lepto, Porcine Reproductive Respiratory syndrome (PRRS), congenital tremor, porcine myocarditis (PMC), Encephalomyocarditis virus (EMC)
- (5) Congenital malformations
- (6) Environment, too cold
- (7) Too many piglets/ starvation/hypoglycaemia

CONGENITAL MALFORMATIONS

Account for about 4% of pre-weaning deaths. Include:

- hernia-umbilical or inguinal-common-complex inheritance-grow well, best to cull at porker weight before they get too large
- atresia ani-absence of anus or rectum-death due to intestinal obstruction. Obstruction may be at different levels
- Cleft palate, hare lip-cannot suckle
- Epitheliogenesis imperfecta-round or elliptical areas devoid of epidermis. May be mistaken for a heater burn. May heal up with time.
- Inverted nipples-problem in selected gilts
- Splayleg-muscle weakness resulting in splaying of legs (usually hind). Worse on slippery floors; myofibrillar hypoplasia-complex inheritance-Landrace; Treatment-hobble legs together below hock & knees; supportive treatment such as heated crib and colostrum care.

CONTROLLED FARROWING

Allows supervision of farrowing to reduce stillbirths.

Prostaglandins IM or intra-vulva (half dose). Do at greater than 112 days gestation. Give at 7am Day 1...if not farrowed, give 0.5mL oxytocin 7am next day...will farrow in that work shift

Will not affect viability if done at correct gestation age.

SPECIFIC DISEASE ENTITIES CAUSING REPRODUCTIVE FAILURE

Porcine Parvovirus

It is likely that nearly all pig herds in Australia are seropositive for PPV. Therefore, all pigs receive passive immunity from their mothers. Passive immunity wanes at or near time gilts become of breeding age. Therefore, gilts in positive herds and breeding animals in negative herds are at risk. Quiz: why does the maternal antibody last out to 21 weeks when it is gone at 8 weeks for most pathogens?

□ **Transmission & Pathogenesis:**

The mode of transmission is faecal- oral. After infection, viremia lasts 1-6 days and infected pigs shed virus for up to 2 weeks. Therefore, one cannot use old sow's feces to infect gilts through feedback systems. Adults show no clinical evidence of disease.

Transplacental infection requires at least 14 days. Virus replicates in vascular endothelium. All fetal pigs are not infected at the same time.



□ **Clinical signs:**

1. Non-pregnant gilt - inapparent infection with rise in parvo titers
2. Infections that occur after conception but before implantation cause delayed return to estrus
3. Infections that occur after implantation but before 35 days
 - a) Some embryos die and are resorbed - small litters? Dodgy for pre days 35
 - b) All embryos die - pseudo-pregnancy
4. Infections from 35-65 days - mummification of fetuses at different sizes
5. Infections that occur after 65 days - fetus develops immunity and is born with + titer and is normal
6. Fetuses are immunocompetent at 70 days of age and are 7 inches or 17 cm long (crown - rump length)

In infected herds, this is reflected in reproductive problems in gilts only:

- | | | |
|---|--|---|
| <ol style="list-style-type: none">a) Abnormal returns P-0b) NIP gilts P-0c) Mummies in gilt littersd) Increased % of small litterse) Farrowing rate reduced | | not all infected at same stage of pregnancy |
|---|--|---|

□ **Diagnosis:**

- Submit mummified fetus-foetal fluids
 - Less than 14cm >>>Ag detection>>>haemagglutination or Ag ELISA
 - More than 17cm >>>Ab detection>>>haemagglutination inhibition or Ab ELISA
 - Avoid foetuses 14-16cm dt Ab:Ag complexes
- Virus isolation not efficient
- Non-suppurative inflammation in brain, lung, liver, kidney in foetuses > 16cm (non-specific)

□ **Prevention/Control:**

- Vaccinating gilts twice is sufficient in positive herds.
- Mix animals- remember that old sows shed very little - if any virus. Gilt pool mixes are best.
- Feedback - stools of older gilts and/or mummies are a good source of virus (take care with legislation!)
- If the herd is negative, keep it closed with little or no animal mixture. Don't forget young boars are a source of infection.
- The naturally infected animal has long lasting immunity and should be kept in the herd.

Leptospirosis

(*Leptospira pomona*, *tarrasovi* and *bratislava*)

Transmission

- Source - urine-contaminated water - from feral animals or swine
- Route -oral intake then 2-7 days for infection, 5-10 days for antibody production, abortion 1-4 weeks later
- Kidney is reservoir for 1-2 years - Asymptomatic

Clinical signs:

1. Infection in adult swine is inapparent
2. Spread may be confined or appear to affect all animals at once, dependent on housing and serotype of organism
3. Trans-placental infection occurs in last trimester when small amounts of blood leak across placenta
4. Signs- abortion, birth of weak pigs, stillbirths and mummified fetuses dependent on time of infection

Diagnosis:

- Petechial cutaneous haemorrhages, oedema, placentitis, bloody body fluids, jaundice
- Detection of antibody in blood or body fluids using serovar-specific microscopic agglutination test (MAT)
- PCR on foetal kidney or urine
- Rising Ab titre in the dam 1:100 >>> 1:800 in 2-3 weeks. Titers of 1:100 are common in vaccinated animals

Treatment/Control:

1. Carrier state hard to remove
2. Tetracycline – high doses (25mg/kg)
3. Tetracycline - 800ppm
4. Prevention by use of vaccination given twice before breeding (P-0) and then pre-farrowing of all other parities. Boars should be vaccinated 2x per year.

Congenital Tremors

Characterised by muscle tremor varying from a slight tremor to severe involuntary dancing movements. Death due to inability to suckle. May be due to any factor which interferes with normal myelination and/or cerebellar development.

- (1) Hereditary-Landrace pigs, saddlebacks (overseas)
- (2) Organophosphate anthelmintics in midpregnancy-Trichlorfon causes cerebellar hypoplasia & hypomyelination
- (3) Viral infections
 - Swine fever/Hog Cholera-infection of the sow in pregnancy>>>cerebellar hypoplasia & hypomyelination. Exotic to Australia
 - Infectious congenital tremor-most common in Australia; Cause? An unidentified virus. Adult animals show no evidence of infection. Outbreaks of disease usually follow the introduction of a pig carrying the virus. All sows farrowing over a period of 1-2 months afterwards produce affected piglets. Problem then disappears as sows become infected at other stages of the reproductive cycle and replacement gilts become infected before being first mated-endemic.

Can become cyclical in endemic herds with outbreaks in gilt litters occurring every 10-12 months as a new population of susceptible gilts comes into breeding herd. Feedback of weaner first stage grower faeces through the water to selected gilts may be the best way to expose gilts before pregnancy.

Clinical signs

The severity of the tremor is usually worse in those litters born early in the outbreak. The mortality is usually low and dependent on the piglet's ability to suck. The tremor regresses and is usually hardly noticeable after 3-4 weeks.

Diagnosis

History of introduction, clinical signs, histopathology (hypomyelination of the spinal cord & special stains for myelin)

Control

By the time a diagnosis is made-too late to do anything. Can try to deliberately expose all non-pregnant animals to affected sows & litters and segregate pregnant sows in the middle stages of pregnancy.

Prevention

Strict quarantine-introduce only animals of known health status.

Encephalomyocarditis (EMC)

Enterovirus of rodents but can infect pig, man & elephants. Rats are principle reservoir of infection-subclinical-excretion of virus in faeces.

Mice develop both encephalitis & myocarditis when infected & suffer a high mortality. Mice as amplifiers of infection for pigs. Infection after mouse plagues after ingestion of viremic mice or faeces (not from pig to pig).

EMC can cause a fatal myocarditis in young growing pigs and foetal death in pregnant sows. A continuing problem is North Coast of NSW as deaths in growing pigs. In the last mouse plague there was a serious reproductive problem associated with EMC in the absence of any grower deaths.

EMC in pregnant animals can result in death of all or some members of the litter-sow is NIP or farrows a mix of normal & affected piglets-stillbirths & mummies. Much more lethal for fetuses than parvo.

Diagnosis

Rat & mouse problems. Isolation of virus from foetal heart. Serological evidence of infection in the sow.

Control

Control rodents

Menangle Paramyxovirus

New disease of pigs causing stillbirths, mummies, deaths, infertility. Stillborn pigs have malformations. Spread by flying foxes to pigs and can also infect humans.

No clinical signs unless sows are infected for the first time during pregnancy.

Only recognized in one piggery.

When first introduced into the pig herd there was a major reproductive problem. Now endemic with immunity.

Diagnosis

Association with flying foxes; SMEDI and congenital malformations; serum virus neutralization test; isolation of virus

Brucellosis - *Brucella suis*

Brucellosis is a zoonosis and is notifiable in Australia. Has been reported in feral pigs and can spread to domestics. Causes orchitis and epididymitis in boars and metritis in sows resulting in infertility, abortions and stillbirths. Can infect man.

It is transmitted among pigs via oral or at mating. Post exposure, there is a latent period of 1-7 weeks when bacteremia occurs. Pregnant sows either abort or absorb fetuses about 7 weeks after exposure. Recovery is spontaneous but chronic carrier animals do result from infection.

Clinical Signs

Clinical signs are similar to PPV and Lepto - abortion, repeat breeders, occasional arthritis and vaginal discharge.

Post mortem (millet-seed) abscesses in endometrium or catarrhal endometritis; in boars-orchitis, epididymitis, seminal vesiculitis-occasional posterior paralysis due to spinal cord abscessation.

Diagnosis:

Serum agglutination test. Herd test (considerable individual variation) +ve indicates presence of infection & necessitates action; Isolation of organism.

Control:

Slaughter & re-stock; Segregation of weaners & market all adult pigs-breed up replacements from young stock.

Toxoplasma gondii

- Organism is ubiquitous.
- Zoonotic disease.
- Domestic cats are the definitive host and pass oocysts in feces.
- All other animals ingest these oocysts from the environment and they encysted in the meat. Improperly cooked meat can be a source of human or domestic animal infections.
- In Australia up to 10% of culled sows are seropositive and 1% of finishers (1997/8 data).
- Major concern is that a woman would be exposed for the first time during pregnancy or an AIDs patient would be exposed. The newborn child could incur serious brain damage from this infection.

The disease is generally, sub clinical in swine. Infected pregnant sows can give birth to premature pigs, dead or weak pigs or pigs that die soon after birth.

Diagnosis:

Diagnosis is based on serology.

Vulval discharge

Discharges from the vulva may originate from the reproductive tract (primarily the uterus) and/or the urinary tract (bladder, kidneys etc.)

It is important to realise that not all vulval discharges are abnormal. Sows will normally have some degree of discharge during heat and after farrowing. These discharges are usually relatively low volume, do not smell, are clear and colorless and the sow will show no signs of illness.

Abnormal vulval discharges (vulval discharge syndrome or VDS) may appear at other times. These discharges may be voluminous, smelly and be yellow/green. The sow may also appear ill (have a fever (>39.5C), be lethargic, off her feed etc. Sows with kidney/bladder infections will generally have urine stained with blood, and will pass pus at the end of urination.

Often the cause of the discharge does not interfere with pregnancy and the sows farrow normally. If your farrowing rates remaining high, don't be overly disturbed by discharging sows. If farrowing rates have dropped, VDS is a possible cause.

□ **General principals for control of VDS include:**

- Adopt a 1 boar:1 sow mating policy-ideally culling any boars that are associated with consistent VDS following mating.
- Improve hygiene, especially around mating areas and dry sow stalls and pens. Wash & disinfect boar pens, sow service pens and stalls used to hold mated sows for the first 21 days post mating.
- Review the quality of your housing. VDS is more likely to occur where sows are housed in non-sloping, poor-draining floors, where urine and faeces have a chance to pool under the animal.
- Animals housed in stalls may be more likely to be affected than those in pens. This is because they get less exercise, and may urinate less frequently (urinating tends to flush any bugs out of the tract).
- Old, lazy, fat sows(this is a biased comment coming from a young, slim, hyperactive parity3) are more likely offenders.
- Antibiotics may have variable success in treating/preventing VDS. Some vets report success by using high levels of in-feed medication (tetracyclines, trimethoprim-sulpha) at around the time of mating. As the antibiotic dose rates and duration of use required to treat VDS are usually higher than directed on the product label.
- Cull any discharging sows that return to service 18-24 days post-mating. These animals have about a 1 in 3 chance of conceiving subsequently.

Porcine Circovirus Type 2

- PCV2 endemic in all pig populations
- Part of PCV2-associated disease complex
- Associated with late-term mummies, abortions, stillbirths, premature & weak foetuses
- Experimental reproduction
- Diagnosis
 - histology and IHC (spleen & LNs), myocarditis
 - Amount of PCV2 related to severity of infection

Porcine Myocarditis Virus (PMC)

- New pesti-virus – not classical swine fever
- Increased stillbirths & pre-weaning deaths
- Occurred in NSW on 2 linked farms in 2003

Nutritional cause of reproductive failure

- Iron deficiency-anaemia in sows & increased stillbirths
- Vitamin A deficiency-no pre-mix, no access to green grass; increased embryonic mortality, increased stillbirths, congenital malformations, anasarca
- Mycotoxins - Fusarium roseum (pink mold)--Zearalenone (estrogen like toxin)- Clinical signs of toxicity depend on the stage of gestation. Early (10 to 15 days) pregnancy ingestion (5ppm or greater) - decreased litter size, pseudopregnancy, weak pigs at birth. Diagnosis is based on history, clinical picture (including swollen vulvas in finishing pigs), negative results from other laboratory testing (blood, mummies and stillborn pigs to lab) and testing of corn if a sample eaten prior to signs is available. Treatment relies on removing the contaminated feed. Prevention relies on milling and storing feed at correct moisture concentrations. New products are on the horizon to neutralize the toxins i.e. Nutrazone

SOW DEATHS

The proportion of sow deaths in a herd has been found to be highly correlated with herd size. It has been suggested that for herds with 150 sows or less, target death rate should be 3% or less. For herds with 200+ sows, this target increases to 5%. It is suggested that the combined euthanasia/death rate for herds be 9% (5% natural deaths/4% euthanasia). Sows appear to be most at risk during the lactation period, with about 40% of deaths occurring post-farrowing.

Variation in death rate can be due to:

- Herd size;
- Housing systems (bloat and urinary tract infections tend to be more frequent in stalls);
- Management systems (mortality rates increase as lactation lengths decrease);
- Nutrition;
- Season (deaths are usually higher at times of thermal stress);
- Culling policies (the rate at which sick sows are euthanased).
- Parity (bladder and kidney infections occur more frequently in older sows/leg problems occur more commonly in younger stock)

Most deaths can be avoided by:

- Minimising excitement and noise around feeding time;
- Maintaining the frequency of feeding (ie do not skip feeds on weekends);
- Intermittent spray cooling in summer during pregnancy/drip cooling during lactation;
- Do not allow sows to get overly fat;
- Proper ventilation;
- Ensure adequate water availability and flow rates (greater than 1.5L/min);
- Maintain floors (correct drainage problems, re-surface abrasive floors);
- Maintain hygiene (do not allow stalled animals to lie in their own faeces and urine);
- Attention to pen and shed maintenance

Target mortality rate less than 5% over 1 year

The major causes of sow death include bladder and kidney infections, gastric accidents (twists, bloat, stomach ulcers), heart failure/heat stroke, blood poisoning (septicemia) and uterine prolapses.

Pyelonephritis

Relatively common in sows. Outbreaks caused by *Eubacterium suis*. This bacterium is found in the prepuce of normal boars and infection of the sow occurs at the time of mating. It is thought that trauma of the urethral orifice area of the sow is necessary for the development of ascending infection. Occasionally get endometritis also.

Clinical signs:

Illthrift & wasting associated with purulent material in urine. May get deaths or seen at slaughter of cull sows. Smear G+ Coryneform. Strict anaerobe.

Treatment:

Penicillin-early

Control:

Treat any sows with are obviously injured at mating. Monitor boar involvement.

Cystitis

- *E coli, Eubacterium suis*
 - Poor hygiene
 - older parities
 - “dog-sitting”
 - low water intake/availability
 - Stall housing with poor drainage
- Profuse urinary discharge &/or blood in urine
- Often septicaemia-poor prognosis
- Penicillin, amoxycillin, trimethoprim-sulpha, tetracyclines

FARROWING HERD ISSUES

Sow Factors Responsible for Prewaning Problems

Failure of milk supply (Teat defects)

Insufficient teats

- hereditary

- teat necrosis within 24 hrs of birth (esp anterior teats)-due to floor abrasion

Inverted teats-glands develop but not functional-heritable

Infantile mammary glands

Failure of milk production (Agalactia)

Management

Poor water supply – quality/amount. Poor piglet viability or low litter size causes sows to eventually “dry up”.

Hormonal

Failure of milk ejection-eg. In first litter sows due to blocking of letdown by adrenaline. Try treatment with 10-30units oxytocin IM

Traumatic agalactia

Due to damage by piglets’ teeth during suckling. Reduced by trimming canine teeth tips.

Infection - mastitis

Usually due to ascending infections up the teat canal. Opportunists-E coli, Strep, Staph. Endotoxins may be produced & absorbed by the sow-pyrexia, inappetance, depression

Clinical signs of mastitis

Sow becomes dull & inappetant. Lies on her udder. Piglets become restless & hungry. Swelling in part or whole of udder (hot, hard, painful, and oedematous).

Treatment

Antibiotics (Trimethoprim sulphar best) + Antiinflammatories (Finadyne, Dexamethasone); supportive for piglets

Prevention

Good hygiene; reduce floor abrasion

Udder oedema

Most common in parity 1 sows. May be due to overfeeding before farrowing. Results in pitting oedema which interferes with suckling & milk production. Treatment with diuretic (Frusamide-50mg/ml. Give 5mls 2x/day).

Generalised conditions

Infections - retained placenta +/- piglets. Results in rapid toxemia. Always treat with antibiotics - Penicillin, trimethoprim-Sulpha after manual exam of the sow

Cannibalism (Savaging)

Usually seen in gilt litters-associated with pain of farrowing. Usually attack the head of the piglet & crush the skull.

Prevention

Sedation (Stresnil), Beer, Provide bedding, Remove piglets until sow has finished farrowing. The placement of rabbits in with the gilts for 3-4 days prior to farrowing has proved successful anecdotally. The theory is that the animals get used to little things darting past them so that is one less stress at farrowing.

Overlaying

Largest cause of death before weaning. Most occur in first 3 days post-birth.

Can be primary (pig in wrong place at wrong time) or secondary (piglet debilitated for some reason).

Diagnosis

Piglet found dead with no obvious other signs of ill-health. Evidence of trauma, suffocation & bruising.

Prevention

Ensure piglets have a warm creep area to attract them away from the sow. Sow roll-bars, specially designed crates.

Piglet Factors Responsible for Prewaning Problems

Hypoglycemia/Starvation

Caused by restricted feed intake at < 7 days of age. Exacerbated by low environmental temperatures. Piglets are born with virtually no fat reserves. If food intake is limited, liver glycogen is rapidly depleted and the piglet can become hypoglycemic. This is exacerbated in low environmental temperatures (<30-34C). This can predispose pigs to infections etc.

Clinical signs:

Hungry, squealing, hairy, hollow-sided, gaunt piglets. Uncertainty of gait>difficulty in maintaining balance>recumbency>depression. Shivering dullness. Can progress to convulsions and rapid death.

On PM see no visible lesions. Empty stomach & evidence of dehydrations. DDX: Other causes of nervous signs

Treat hungry piglets:

- bottle feed as often as possible-make sure its warm!
- Stomach tubing (if they can't suckle)-10-15ml at a time
- Hold up to the udder of the sow (but very labor intensive)
- Place in a heated crib during this time to prevent overlays
- If cold, and no suckling reflex when place finger in mouth, warm them by immersion in water @ around 40°C (not too hot) can now purchase flotation jackets to float them in.
- Give 20mls of colostrum or substitute/hr by stomach tube.
- Place them in heated crib 37°C till vigorous enough to drink from bottle

Hypothermia

Thermal neutrality for a newborn is 35°C. The temperature under the creep for newborns should be 30-35°C with less than 2°C fluctuation. The creep area of the floor should be solid (not mesh), and preferably bedded (straw, rice hulls, shredded paper) with no drafts. Using a light source (as opposed to a heater globe which gives out no light) (175W, no larger or smaller) also may attract the piglets away from the sow.

It is difficult to simultaneously provide the correct temperature for the sow (16-22°C) and her piglets. Covered creep boxes are good, but can be labor intensive. Best solution for this is to rig them up with sliding lid, which can be pushed away to get access to the pigs.

Piglets born at less than 800g are unlikely to survive. These require TLC (eg, supplemental feed, crib-housing).

Cross fostering may be used to increase piglet survivability. The general rules are:

1. Only do it if you have to, ie too many pigs on sow or agalactia!
2. Do it to even up numbers (not size) at less than 24 hours. Need to ensure that pigs you are transferring have had or will get colostrum. This may be difficult in small herds with few farrowings.
3. Do it later to form "fall-back" litters.
4. Every foster movement you do is going to disrupt someone!

Haemagglutinating encephalomyocarditis virus infection (vomiting & wasting disease)

Two syndromes:

- (1) < 1 week of age show clinical signs of encephalitis-convulsions, coma, death. Vomiting sometimes seen.
- (2) 1-2 weeks of age-persistently vomit and waste away. May succumb to secondary infection
- (3) Older pigs show no clinical signs, except pigs affected just prior to weaning will fade away in the weaner house

Due to a corona virus infection of the upper respiratory tract which spreads to the CNS causing encephalitis (+ vomiting centre in brain and or affects the ganglion cells in stomach wall which leads to the vomiting and wasting.

Usually seen in outbreak form in a piggery following introduction of new stock-affecting all litters born over a period of 2-3 weeks OR heavily pregnant sows introduced to a piggery where the infection is endemic. The virus, when introduced into a susceptible piggery spreads very rapidly, infecting pigs of all ages but it is only pigs < 14 days old that show clinical signs. Pigs born to immune sows are protected by colostral immunity. Prevent spread of virus from URT to brain. Once all sows have been infected and have had the opportunity to develop immunity no further problems. As colostral immunity wanes at 12-14 weeks of age pigs become infected and so are then immune and their piglets protected. Recent evidence that can occur cyclically over a number of years in endemic herds. Suspect it when get wasting of litters with no obvious cause.

Diagnosis - History (introduction of pigs); clinical signs;

PM-no gross signs (vomiting, dehydration and emaciation > 7 days); histo-encephalitis; serology-rising Ab titre to coronavirus

Treatment, control, prevention:

No treatment. Ensure infection spreads as quickly as possible to other pigs (eg. Mix sows with problem litters with other sows, mix with affected pigs).

Streptococcal infections (see later under "Nervous diseases")

THE GROWING HERD



TARGETS

PARAMETER	TARGET
Growth rate (birth to sale)	>600 g/day
Weaner mortality	<2% (or 0.5% population)
Grower mortality	<1-2%
Grower feed conversion efficiency (carcass)	<3.5

WEANER MANAGEMENT



TARGETS

Age (weeks)	birth	Weaning	3	6	10
Weight (kg)	1.5	4.5-8	6	13	25+
Temp (C)***	34	28-30	26-28	22-24	20-22
Stocking rate (m ² /pig)			0.11	0.25	0.32
Stocking density (m ³ /pig)			1	1	1.5

**** These are thermoneutral zones, (for a newly weaned pig eating very little it will rise to 30-32°C for the first 7 days after weaning)

A weaner is generally defined as a pig from the time it is removed from the sow until it weighs around 20kg (at 8-10 weeks of age).

Weaning is nature happens over a 10-12 week period, with a reduction in milk intake accompanied by an increase in other food. Gradual decline in the protection by the sow's milk and circulating serum antibodies allows the piglet time to respond and provides its own protection. Other stresses placed on the piglet at weaning include:

- change from a liquid to a solid diet
- Loss of milk antibacterial and viral factors
- meeting new pigs (resulting in fighting to establish hierarchy and cross infection)
- change from group feeding (ie as a litter on Mum) to individual feeding
- environmental changes-reduced temperature

To overcome these changes:

- use all-in/all-out weaner accommodation (improves air quality and decreases transfer of pathogens from older to younger pigs)
- clean, warm, dry environment. Hard to provide 28C all the time. Giving pigs a choice of micro-environments best (eg keep the ambient room temperature cooler (eg 22-25C) and provide heater lamps in each pen. These are called zone heaters and should be wide enough to accommodate all pigs)
- minimise temperature fluctuations to less than 2C in a 24hr period

- group size probably doesn't matter as much as people once thought-as long as there is adequate floor space, feeder space and drinker space
- ready access to fresh feed and water
- pigs eat approx 4%-6% body weight
- Protein 23-21%, lysine 0.95-0.78 g/MJ DE, energy 14-15MJ DE/kg
- Food needs to be highly digestible so base it on animal protein
- feed on a solid part of the floor a few times a day post-weaning. This will keep the piglets eating as a litter (what they are used to) and provide the stockperson with an idea of any poor-doing pigs. These can be treated with TLC (eg. Liquid feed, "stuffing")
- ensure there is fresh air flushed through the room at least 2 times daily to maintain ammonia < 11ppm, carbon dioxide < 1500ppm
- Zone heaters (see above) allow adequate entilation
- adhere to recommended stocking rates (provide more room if possible)
- Stocking rate is a problem at end of weaning period not in first few weeks
- grouping pigs by sizes probably a waste of time, unless you intend to feed them seperately

GROWER MANAGEMENT

How a pig grows depends on a number of interacting factors: nutrition, genetics, housing, environment and disease.

In the past, grower pigs were traditionally housed in pens of 20-30 pigs/pen on concrete solid +/- slatted floors. They were usually classed as "growers" from 10-16 weeks of age, and then moved again and classed as "finishers" at 16-24 weeks of age. These days, growers may be housed in pens of 200-1000; on concrete or on deep litter bedding (rice hulls or straw); traditionally or in ecoshelters.

However they are housed, there are a few general rules that should be adhered to ensure optimal performance:

- provide ad-libitum fresh feed of correct diet specifications
- This will vary for males (anabolic testosterone) and females and is called split or gender feeding
- As a general rule the following specs are minimal for mixed genders

Liveweight (kg)	Lysine (g/MJ)	Energy (MJDE/kg)	Protein (%)	Fibre (%)	Ca (%)	P (%)
20-50	0.65	>14.5	16	5	1	0.6
50-90	0.55	>13.5	14	8	1	0.6

- provide adequate, fresh drinking water (rule of thumb-1 drinker:10 pigs; 0.5-1L/minute flow rate)
- provide adequate space (see later)
- provide fresh air (<11ppm ammonia, <1500ppm carbon dioxide, <2.4 mg/m³ dust)
- provide correct temperature (18-22C)
- provide for overhead sprayers if temperature exceeds 22C
- Manage to prevent over-fatness (eg. PST)

GROWTH REGULATORS

Essentially these raise the genetic goalposts of the pigs so that the fat deposition plateau is reached at a later age or weight.

Porcine somatotropin (PST aka Growth Hormone)

- Increases growth rate (10-15%), leanness (5-10%) and FCE (15-25%)
- Acts by increasing protein deposition and decreases fat deposition, through either increased efficiency of dietary protein and/or increased requirement of dietary protein to support increased protein deposition
- They cause a decreased feed intake, therefore must increase the energy concentration of the diet in treated animals
- Boars-already utilizing dietary protein optimally (testosterone), therefore must increased P for a response
- Gilts-increases protein deposition is via increased efficiency of dietary protein(oestrogen) and additional dietary protein
- Recombinant protein-must be administered via injection (3-5mg/kg/day) for 35 days. Labour intensive and always a chance of a broken needle.
- Is expensive so must be done correctly
- Best response in finishers as they are the ones most likely to have reached their genetic growing potential
- Allows for increased carcass weight without XS fat
- Biggest marketing penalty is falling outside the weigh/fat matrix
- Excess may cause lameness & gastric ulceration

Immunocastration (“Improvac”)

- GnRF linked to a protein carrier-reduced GnRH activity and testosterone production
- An alternative to surgical castration to reduce boar taint problems
- Developed in Australia by CSL. Now marketed through Pfizer.
- Need 2 injections. Need at least 4 weeks between shots. Last shot given 4-5 weeks before slaughter as this is the time of puberty induction and duration of antibody
- May reduce aggressiveness in male pigs, this will probably be its stongest selling point in the future.
- Problem with lack of anabolic testosterone and hence increased backfat; marketing penalty
- Take care when administering as the antigen common to all mammals?

Ractopamine (Paylean)

- Beta agonist
- Feed additive 5-10 mg/kg 4-6 weeks
- Increased ADG, FE, lean yield, muscle depth
- Increased protein (lysine) requirements
- Not registered for use in all countries

- May increase stress sensitivity

ENVIRONMENTAL AUDITS

The key environmental factors that should be considered are:

- (1) Air quality
- (2) Hygiene
- (3) Pig flow
- (4) Stocking rate & density
- (5) Temperature
- (6) Humidity

Air quality

Influenced by many things: ventilation, hygiene, stocking density, feed quality (dustiness).



Targets

Parameter	Target
Ammonia	<11ppm
Carbon dioxide	<1500ppm
Total dust	<2.4mg/m ³
Respirable dust	<0.23mg/m ³
Airborne bacteria	<100,000cfu/m ³

Rules of thumb for natural ventilation

- Orientate building with long axis East-West
- Wide eaves on North side to protect from summer sun
- Insulate roof and walls
- Roof pitch 15° and ridge vent for sheds > 10m wide allows a stack effect for natural ventilation
- Roof pitch 5° and no ridge vent for sheds < 10m wide
- Ventilation openings: ideally automatic blinds

Each side wall, minimum 10% floor area

Ridge vent width, minimum 10% floor area

Ridge vent height 5% of floor area

(note, for buildings with full length shutters 10% of the building floor area equates to 10% of the building width)

- Ventilation openings for hot climates can be from floor to eaves
- Drop side shutters for weaner and farrowing sheds
- Automatically control ventilation openings
- Separate sheds by 5 times the height of the nearest shed or tall object as this allows correct air flow between sheds

Temperature

Age/Class of stock	Recommended temperature range
At birth	30-35C
Pre-weaning(decreases weekly)	32-24C
At weaning (first week)	LCT 30-32 C
At weaning 4-8wks	28 to 22C (decreasing 2C each week)
Growers (8-16 weeks)	20 to 22C
Finishers (16-24wks)	18-22C
Sows and boars	16-22C

	Effect of ambient temperature on growth rate	
	Degrees below LCT	
	1° C	5° C
Growth Gm/day birth to bacon (normal 600g/day)	588	540
Days to processing (normal 120 days)	122	133
Extra feed eaten	6 kg	31 kg
Cost/ pig at a feed price of \$300/tonne	\$1.80	\$9.30

Stocking rates

System	Minimum space allowance (m ² /pig)	Comments
Growing pigs up to 10kg in groups	0.11	
11-20kg	0.18	
21-40kg	0.32	Approx. 20-30% of space allowance for dunging area
41-60kg	0.44	
61-80kg	0.56	
81-100kg	0.65	
Adult pigs in groups	1.4	
Adult pigs in individual stalls	0.6x1.8	Minimum length of shortest side 2m
Boars in pens used for mating	6.25	
Lactating sows in stalls	3.2	With piglets up to 4 weeks of age
Lactating sows in pens	5.6	

DISEASE (GENERAL ISSUES)

NECROPSIES

Production medicine veterinarians are responsible for the health of large populations of animals. In case of a problem, you will be asked to make a presumptive diagnosis during the herd visit and prescribe an appropriate treatment or management change. Additionally, you should perform post-mortem examinations and collect samples to confirm your presumptive diagnosis and recommendations. Failure to collect samples for a definitive diagnosis can be costly if your presumptive diagnosis happens to be incorrect. In such cases, you will need to return to the farm (if they ask you back) to collect appropriate samples. Any preliminary treatments might interfere with diagnostics on this subsequent visit. Moreover, the delay to appropriate treatment could result in many additional pig illnesses or deaths.

Autopsies should be conducted in an area away from the piggery to lower the risk of infectious agents from opened carcasses being transmitted to live pigs.

Always wear gloves to prevent bacteria from entering skin abrasions. Some diseases (eg. Strep, Salmonella, Erysipelas and Leptospirosis) can infect humans. Therefore, all equipment used during the autopsy should be cleaned and disinfected at the conclusion of the procedure. Disposable material (eg gloves) shall be disposed of into rubbish bins once disinfected. Ensure the equipment for cutting is sharp and always cut away from yourself.

Where unusual symptoms of disease are seen, do not autopsy the pig. For example, anthrax, a rapidly fatal disease of humans, can present itself as an unusually large swelling of the neck in pigs. If in doubt, do not open the pig but rather notify your State Veterinary Officer.

A basis autopsy kit should contain:

- knife, steel/sharpening stone
- scissors and forceps
- swabs and plastic bags/bottles to collect specimens
- disposable gloves
- bucket, scrubbing brush and disinfectant

Always wash hands after conducting a post mortem. The person responsible for disposal of dead pigs should always wear rubber gloves when handling carcasses. Rubber is preferred as they can be washed with disinfectant at the end of the run.

The pigs chosen for postmortem examination and sample collection should be: (1) Acutely affected with clinical signs representative of the problem; (2) Alive; and (3) Untreated

Recently dead pigs can also be examined, but take care if they have been dead for more than a couple of hours. These may be of little diagnostic value for laboratory testing.

EUTHANASIA OPTIONS:

As a veterinarian, you can perform euthanasia by injection of euthanasia solution. Other options available on the farm are outlined below. The most appropriate method of humane euthanasia should only be determined after considering the following: human safety, pig welfare, skill requirements, practicality, cost, aesthetics, and limitations.

Euthanasia Methods for Swine. Adapted from: “On Farm Euthanasia of Swine-Options for the Producer” American Association of Swine Veterinarians/ National Pork Board

	Nursing Piglet (≤ 8 kg)	Nursery Pig (8-20kg)	Growers/Finishers	Sows/Boars
Carbon dioxide	Yes	Yes	Not practical	Not practical
Gunshot	No	No	Yes	Yes
Captive Bolt	No	Yes	Yes	Yes
Anesthetic overdose	Yes	Yes	Yes	Yes
Blunt trauma	Yes	No	No	No

LOGICAL APPROACHES TO DIAGNOSTIC WORKUPS

1. Get a complete history.
2. Perform an on-farm clinical evaluation of the problem.
 - What are the clinical signs?
 - What age pigs are affected?
 - Is the nutritional program meeting the pigs' needs?
 - What other disease problems are on the farm?
 - What is the medication/vaccination program on the farm?
3. Analyze the production system (continuous flow, All-In- All-Out, multiple site, age segregation, early weaning)
4. Study the environment
 - Is there enough space per pig?
 - Do pigs have access to feed and water?
 - Is the ventilation system adequate? It is working?
5. Does the pattern of the disease on-farm reflect an infectious disease process?
 - i. Is the spread within litters/pens/rooms/age groups?
 - ii. Does it appear to respond to antibiotic treatment? (most pig diseases in Australia are of bacterial origin)
 - iii. Can you recognize the disease from clinical signs?
 - iv. Does it co-occur with a change in management/environment/entry of new animals?

On-farm necropsy & submit animals or samples to a diagnostic laboratory. Include culture and sensitivity.

 - a. Are there recently (less than a couple of hours old) dead cases to necropsy?
 - b. Are there representative live cases to necropsy? (choose non-medicated, acute cases).
 - c. Are there changes in body organs that appear abnormal?
 - d. If so, collect representative samples of each. Also collect "routine" samples likely to harbour bacteria (liver, lung, spleen, kidney, lymph node). Target the organ you suspect to be affected (eg small intestine for scouring).
 - e. Consider bacteriology first-it's relatively inexpensive.
 - f. Take samples also for histo-you can store them in formalyn and submit them later if your bacto is inconclusive.
 - g. If bacto and histo are inconclusive, talk with a pig specialist and/or your diagnostic lab. It may be the samples you took were inappropriate or that you have a new/emerging disease!
6. Slaughter checks
7. Optimize management, environment, production system.

ENTERIC DISEASE

DIARRHOEA (def.) = increased frequency and excess water content of faeces

Diarrhoea mechanisms:

1) Maldigestion

Villi destroyed by TGE and/or Rotavirus⇒maldigestion⇒incomplete absorption of sugars and amino acids⇒water secretion to gut (osmotic effect)⇒large intestinal bacteria ferment sugars to volatile fatty acids (VFAs)⇒water secretion to gut (osmotic effect)⇒decreased pH of intestinal lumen⇒decreased absorption of ions (osmotic effect)

2) Active secretion of ions (Hypersecretion)

E. coli⇒heat-labile toxin and/or heat stable toxin⇒cAMP and/or cGMP⇒Cl excreted to lumen⇒HCO₃ excreted to lumen⇒Na absorption inhibited

Binding of bacterial enterotoxin to enterocytes luminal epithelium - Natural resolution requires replacement of affected cells by cell turnover

glucose transport mport unaffected by enterotoxins, so will carry a Na ion across with it; hence the inclusion of Glucose in electrolyte solutions

Prostaglandin synthesis due to inflammation⇒increased cAMP⇒Cl excreted to lumen⇒HCO₃ excreted to lumen⇒Na absorption inhibited

3) Increased permeability

Inflammation precipitated by toxins decreases integrity of tight junctions⇒loss of water, electrolytes, protein, blood

Protein (protein-losing enteropathy)

Blood (hemorrhagic enteritis)

4) Altered motility

Not considered a primary mechanism for the cause of diarrhoea

Hypomotility can allow pathogens to proliferate and cause diarrhoea

Hypermotility can result from increased exudation of fluid into the intestine. Results in reduced contact time for digestion. Nutrient digestion and absorption impaired⇒increased osmotic load

Remember- Extracellular fluid losses; 15% loss = clinical signs, 30% loss = death.

Total body water is 70% of weight; will perish if lose greater than 10% of Total body water.

Establishing a diagnosis of diarrhoea:

- Collection of a thorough history of the problem (age affected, mortality, character of feces, color of feces)
- Evaluate animals, premises and management practices
- Necropsy examination (choose live, recently and typically affected, untreated pigs)
- Utilize laboratory support (Bacterial culture, virus isolation, electron microscopy, histopathology, PCR)
- pH of scours (acidic = malabsorptive, basic = hypersecretory)

Common enteric diseases in Australia-differentials by age

Neonates	Post-weaning	Grow/finish
<i>Neonatal colibacillosis</i> (d 1-4 for Non hemolytic E.coli)	<i>Post-weaning colibacillosis</i> and oedema disease(1-2wks post-weaning)	<i>Proliferative enteritis</i> (6-20 wks)
<i>Coccidiosis</i> (d 5-15)		<i>Swine dysentery</i> (6-20 wks)
Clostridial enterotoxemia (d 1-21)		Intestinal spirochetosis (8-20 wks)
Rotavirus (7+ to wean)-rare Can occur at day1 in a naïve herd	Rotavirus (post-weaning)-rare	Hemorrhagic bowel syndrome (4 months to adult)
Salmonellosis 5+ days through to post weaning	Cross infection at weaning	Salmonellosis (6-20 weeks)
Hemolytic E.coli 10+ days through to 2 weeks post weaning		Whipworms, ascarid and nodule worm (8-24 weeks) Gastric ulcers 3-6 months

*Common diseases in *italics*.

PRE-WEANING ENTERIC DISEASES

Neo- natal Enteric colibacillosis (day 0- 4 of life)

Aetiology:

- **Non Haemolytic** Enterotoxigenic *Escherichia coli* (Gram-negative rod)
- Somatic antigens - : O:8, O:9, O:20, O64,O:101;
- Fimbrial types –K88 (F4), K99 (F5), 987P (F6), F41??;
- Enterotoxins – STa, LT
- STa=heat stable, stimulates cGMP
- LT=heat labile, stimulates cAMP
- Serotyping of isolate is used to determine if it is a recognized pathogen

Epidemiology/Transmission:

- NHEC used to be the most common cause of neonatal diarrhea in pigs in Australia. Now controlled by vaccination of sows in the majority of cases.
- Age affected: Day 1 to 4
- Route of infection: Fecal-oral transmission from mum
- Most often gilt litters, associated with a lack of maternal antibodies.

- E coli adhere to receptors on the epithelial cells of the small intestine via fimbriae (K88, K99, 987P, F41) & colonise. They then produce toxins that act directly on the intestinal epithelium causing active secretion of Cl⁻ ions. This takes Na⁺, K⁺ with it, resulting in osmotic diarrhea. There is no cell damage. Antibodies to fimbriae can prevent colonization and infection.
- Vaccination of mum, sanitation, environmental temperature and colostrum intake important factors

Clinical signs:



Yellow, watery diarrhea, dehydration, depression, inflamed anus and perineum, alkaline pH of feces

Gross lesions:K88 is reputed to colonise the whole of the small intestine Whereas K99 and 987P apparently are confined to the distal SI lower jejunum and ileum; dilated stomach with undigested milk curd; gas and fluid in small intestine. Can still see villi as velvet appearance due to intact vs. villous atrophy seen as flat white surface (eg. Rotavirus)

Histopathology:

No obvious changes early. Later, changes are variable - large concentrations of Gram-negative rods adjacent to microvilli +/- villous atrophy, depending on secondary invasion.

Diagnosis:

- Clinical signs & history
- Culture & sensitivity (distal SI, caecal fluid)
- PCR to detect toxins and fimbrial genes
- Serotyping for O and fimbrial antigens

Treatment:

- Clean, warm, dry environment
- Antibiotics (injectable, oral) –Neomycin, Trimethoprim-Sulphar, Tylosin, Apramycin, Spectinomycin
- Supportive measures (electrolytes containing glucose) eg Vytrate
- Transfer litter to another sow

Prevention:

- Thorough cleaning and disinfection of farrowing house
- Wash sows prior to placement in crates
- Maintain a warm, dry, draft-free environment for pigs
- Vaccinate sows to stimulate colostrum antibodies (commercial pili bacterins). Vaccinate at 6 and 3 weeks before farrowing IM. May be able to just vaccinate gilts. These vaccines stimulate IgG only, so are only useful when these antibodies are in the gut lumen at high concentrations (ie only in the first few days of life). After this, you need IgA.

Sucker colibacillosis caused by Haemolytic E.coli

- Occurs normally from day 10 of life probably associated with decline in of anti K88 IgG antibodies in sows milk at this time.
- This E.coli (usually K88:O149 - sometimes K88:O8) has genes for StA , StB and LT (cf NHEC which has StA and LT, or normal postweaning HEC which has StB and LT)

Clinical signs

Sudden death from endotoxic shock, or severe scour with dehydration.

Necropsy

Venous infarction of stomach. Severe mucosal congestion probably due to STb=heat stable, which is associated with prostaglandin hypersecretion?

Histology/smears

Villous epithelial necrosis and microvascular thrombosis along with many gram negative bacilli on the villi of the small intestine.

Diagnosis

Can invariably isolate heavy to pure cultures of K88 positive haemolytic *E.coli* from the ileum and colon of dead pigs.

Treatment and prevention:

Highly resistant to commonly used antibiotics, shock usually prevents absorption from injection site anyway

Vaccination

- Specific IgA can be stimulated and increased in sows' milk by feeding live avirulent K88 hemolytic E coli. Culture the E coli strains at 37^oC for 18 hrs, feed 200ml top dressed on sow fed for 4 days around 11 weeks of gestation.
- This approach has never failed to control the problem. Some pedants would regard this as contrary to the swill feeding act in NSW. (Other states seem more enlightened. Better to seek forgiveness than permission, so don't ask the DPI if you can do it)
- Starter cultures can be obtained from the Pig health and Research Unit of the DPI in Bendigo

Coccidiosis

Aetiology

Isospora suis

Epidemiology/Transmission:

- Common cause of diarrhea in older neonatal pigs in Australia where Baycox is not used.*****
- Age affected: Day 5-15 (sometimes seen in early weaned pigs due to cross infection at weaning)
- Route of infection: Fecal-oral transmission (feces of infected piglets, from previous litters)

Clinical signs

Yellow to yellow-brown diarrhea, dehydration, weight loss, high morbidity, death uncommon , neutral to acid pH of feces (malabsorption diarrhoea)

Necropsy

Ranges from no visible change to necrotic enteritis affecting distal ileum. Probably no chyle present in mesenteric lymphatics.

Diagnosis

Oocytes and/or faecal fat evident in pooled faecal samples from 4- 5 pigs/litter.

Gross lesions:

Jejunum and ileum commonly affected, villous atrophy and fusion, fibrinonecrotic membrane on mucosa, empty lacteals

Histology/smears

Necrosis and blunting of villi, sexual and asexual stages visible on histology and smears

Treatment

Potentiated sulphonamide injection or oral toltrazuril. Have to wait for lesion to heal after treatment therefore need mainly supportive-electrolytes, warmth

Prevention:

- Toltrazuril (Baycox) administered at 3-5 days of age
- Thorough cleaning and disinfection of farrowing crates
- Wash sows prior to placement in farrowing crates
- Open wire or metal flooring, concrete floors are a risk management disaster from a coccidiosis perspective

Clostridial enterotoxemia (Types A & C)

Aetiology

Clostridium perfringens, type C, Gram-positive rod, produces hemolyzing alpha toxin and necrotizing beta toxin

Epidemiology/Transmission

Not Significant in Australia unknown; Age affected: Day 1-14; Route of infection: Fecal-oral transmission

Clinical sign

Peracute: bloody diarrhea, sudden death

Acute-subacute: +/- bloody diarrhea, wasting, death in 3-7 days

Chronic: yellow-gray diarrhea, stunted growth; Neutral to acidic pH of feces

Gross lesions

Jejunum and ileum commonly affected, Hyperemic intestinal loops (red to black), Emphysema of bowel wall, Bloody, necrotic contents in the lumen

Histopathology

Ghosts of villi left with large numbers of gram positive rods adhering. Necrosis of mucosa and lamina propria, Large, gram-positive rods

Diagnosis

Bacterial culture, Intestinal smears, ELISA (to detect beta toxin)

Treatment

Amoxicillin, penicillin

Prevention:

- Rigid sanitation (wash sows)
- Sow vaccination with type C toxoid (twice during gestation) not available in Australia
- BMD in feed (250 g/ton for 2 weeks pre-farrowing & 250 g/ton for 3 weeks post-farrowing)
- Prophylactic antibiotics in herds where previously diagnosed

Clostridial perfringens type A

Never been positively diagnosed as a common cause of scour in Australia; although is the flavour of the year overseas.

Age of occurrence

1-21 days.

Clinical signs

Mild to moderate scour.

Necropsy

Fluid filled intestines. Neutral to alkaline faeces.

Histology/smears

Necrosis of villi due to enterotoxins. Large numbers of gram positive rods.

Diagnosis

Culture of organisms in large numbers from faeces. Demonstration of enterotoxin in faeces or intestinal contents.

Clostridium difficile colitis

Aetiology

Spore-forming, Gram +tive rod; produces A and B toxins

Epidemiology

Age affected: Day 1-7; Variable morbidity (10-90%) and mortality (20-50%); Significance in Australia?????

Transmission

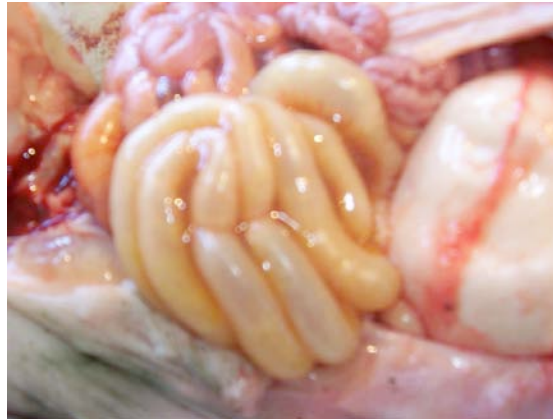
Fecal-oral transmission; Disruption of the normal flora considered a contributing factor

Clinical signs

Yellow to dark yellow diarrhea; Feces vary from watery to pasty; Rapid loss of body condition; Stunted growth of affected pigs

Gross lesions

Moderate to severe mesocolonic edema; Yellow, watery to pasty colonic contents; Undigested milk curd in stomach



Histopathology

Intact colonic mucosa; Neutrophil and fibrin exudation into colonic lumen (volcano lesions); Gram-positive rods (+/- spores) in lumen and mucosa of colon; Neutrophilic infiltrate in mesocolon; Suppurative foci in colonic lamina propria

Diagnosis

Clinical signs, rule out other causative agents, gross & histopathology; bacterial culture (anaerobic); Toxin testing (confirmative)

Treatment

Penicillin-based antibiotics, supportive fluid therapy

Prevention

Rigid sanitation practices, Autogenous vaccine?

POST-WEANING ENTERIC DISEASES

Post-weaning Colibacillosis

- Occurs in the first 1-2 weeks post-weaning. Most common cause of scours in pigs in the immediate post-weaning period in Australia.*****
- F18;0:141 and K88;0:149 most common antigen types.
- Associated with loss of lactogenic antibodies post-weaning, in the face of no active immunity and fluctuating, cold weaner houses.

Diagnosis:

Clinical signs, gross pathology, histo, culture & sensitivity & serotyping as for pre-weaning Colibacillosis

Treatment:

As for pre-weaning E coli but rely more on group than individual treatment:

- Electrolytes + Glucose in drinkers can lose 10% of their total body water a day
- Antibiotics (parenterally and in electrolyte solution)

Prevention:

- Refer to “Weaner management” notes
- Oral vaccination of sow pre-farrowing may help
- Oral vaccination of piglets pre-weaning is effective
- IM vaccination of piglets is available commercially (Weanavac), theoretically should not work as is stimulating IgG and will be neutralised by maternal antibodies. Is a good first approach, will give you time to think, and mentally prepare the farmer for oral vaccination of suckers one week preweaning

Oedema disease

(This is no different from normal post weaning colibacillosis except it is caused by strains that produce an additional toxin (called a shiga like toxin or ST 11e) This toxin is absorbed from the gut and causes disease by injuring small arteries and arterioles. Microscopically this is seen as a degenerative angiopathy. The lesions may occur in many organs and tissue and cause oedema

Aetiology:

- Hemolytic *E. coli* (serotypes O138, O139 and O141)
- Associated with F18 and F4 fimbriae
- Produces a heat labile vascular toxin (verotoxin or Shiga like toxin – ST 11e)
- Not very common in Australia any more, but the wheel will turn, usually every 10 years or so.

Epidemiology/Transmission

Same as for post weaning colibacillosis

Clinical signs:

- In addition to scouring and death you may see
- Neurological signs (staggering, paralysis, convulsions)
- Head and palpebral edema
- Sudden death

Gross lesions:

Oedema (stomach wall, gall bladder, mesentery, mesenteric lymph nodes); subcutaneous edema; mesocolonic edema; ascites with fibrin tags (serogelatinous)

Histopathology:

Oedema (stomach wall, brain); fibrinoid necrosis of arteries and arterioles; perivascular edema and hemorrhage, Areas of malacia in the brain stem

Diagnosis:

- Tied off jejunum, liver, mesenteric lymph node for culture
- PCR (to detect fimbriae, verotoxin)

Treatment:

- Too late if neurologic (death within 24 hours)
- Provide warm, draft-free environment, Corticosteroids
- Antibiotics (injected, water, feed)- based on culture & sensitivity (Apramycin, Amoxycillin)- rapid development of antibiotic resistance a problem

Prevention/Control:

Oral or injectable vaccination of pigs one week preweaning. Once again the same argument applies as for post weaning colibacillosis as to whether or not the injectable vaccine (induces IgG) will work. Essentially we are trying to stop the E.coli adhering to the enterocytes by producing F18 or F4 antibodies – IgA most likely to be effective here.

- Zinc oxide in the first stage weaner diet (1-2 weeks post-weaning only). Zinc Oxide is considered a feed additive and not a medication by the APVMA.
- provide stable environment, minimize management stresses

Rotavirus

Aetiology:

- Rotavirus (Reoviridae)- a ubiquitous pathogen (seroprevalence approaching 80%). Virus causes villous atrophy resulting in malabsorption diarrhea.
- Uncommon cause of scour in Australia

Epidemiology/Transmission

Age affected: Day 5+ to postweaning; Fecal-oral route of infection from mum or other weaners

Clinical signs

Transient diarrhea (Yellow - milk ingestion/Black-gray – feed ingestion); dehydration; vomiting; high morbidity, variable mortality; subclinical infections

Gross lesions

Villous atrophy, thin intestinal wall, fluid filled intestine

Histopathology

Villous atrophy (tips of villi)

- **Histology/smears-** Blunting of villi in jejunum and ileum. Identification of virus using fluorescent antibody.
- **Diagnosis-** Absence of cocci and HEC. Malabsorption diarrhoea .Demonstration of virus in neutral to acidic faeces.

Diagnosis

Histopathology, electron microscopy, immunofluorescence & ELISA tests (FA). Rule out other causes of diarrhoea at these ages

Treatment

Provide electrolyte solutions containing glucose and glutamine

Prevention/Control

Rigid sanitation and all-in/all-out management of farrowing facility and nursery

Porcine proliferative enteritis

Aetiology

Lawsonia intracellularis

Epidemiology/Transmission:

- Two syndromes-non-haemorrhagic and haemorrhagic
- Age affected : 6-20 weeks (non-haem); 16 wks + (haemorrhagic)
- Infection via fecal-oral route
- Very common (ubiquitous?) in Australian piggeries*****
- Incubation period 7-14 days. Peak disease occurs 21 days post-infection.

Clinical signs:

Non-haemorrhagic proliferative enteritis: Wasting, loss of condition (“slab-sided”), variable mortality, recovery possible in uncomplicated cases

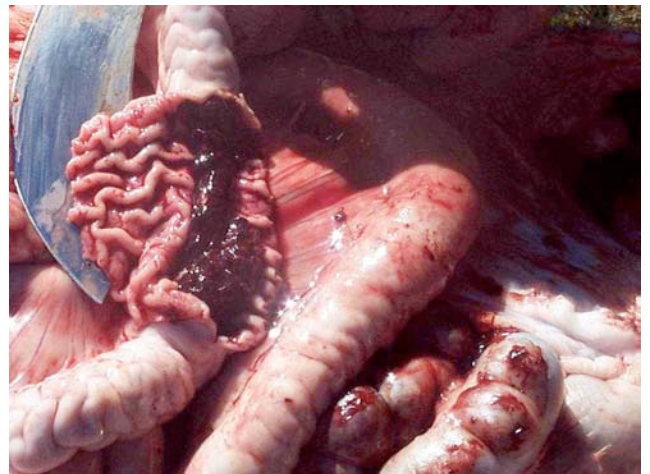
Haemorrhagic proliferative enteritis: Intestinal hemorrhage, dark, tarry stool; sudden death; Must differentiate from hemorrhagic bowel syndrome

Gross lesions:



Non-haemorrhagic: “Hose pipe” ileum, fibrinonecrotic membrane

Haemorrhagic: Terminal ileum and upper spiral colon affected, clotted blood in intestinal lumen, thickened mucosa



Histopathology

Marked proliferation of crypt epithelial cells, Mucosal hemorrhage and destruction, Demonstration of *Lawsonia* organisms (Warthin-Starry stain)

Diagnosis:

- Gross necropsy-thickening of ileum. Necrosis in chronic cases. Blood in acute cases with pigs >16 weeks.
- Histopath: Microscopic lesions, Demonstration of organisms (Warthin-Starry stain)

- PCR on faeces (not conclusive – use to rule out other ddx eg.Brachyspira)
- Culture of faeces – to rule out other ddx
- Culture of mLNs – to rule out Salmonella
- Serology (IFA) – herd diagnosis – not for individual pigs
- Main DDX is swine dysentery/gastric ulcer/intestinal spirochaetosis; salmonella

Treatment:

Parenteral antibiotics: lincomycin, tetracyclines, tylosin

Oral antibiotics: (in conjunction with parenteral treatment)

Tylosin 100 g/ton of feed for 14-21 days or 3mg/kg for 3 days in water

Chlortetracycline @ 10mg/kg for 14 days in-feed or 3 days in-water

Lincomycin – 3-6mg/kg in water

Prevention:

Aim for prevention of exposure/infection for the whole of the pigs' life or for “controlled” exposure. Antibiotics such as 50ppm olaquinox and 300ppm tetracycline have shown to prevent infection under experimental conditions. Controlled exposure can be via “pulsed” water medication (eg 12 days off/2 days on) or low doses of in-feed medication (<300ppm tetracycline, 100ppm or less of Tylosin; <50ppm olaquinox).

Enterisol ileitis (Boehringer Ingelheim) is an avirulent live vaccine delivered via water. It is registered for use in weaned pigs greater than 3 weeks of age. It is recommended for use at least 6 weeks before a herd serconverts to Lawsonia (3 weeks for vaccine to “take” + 3 weeks for antibody production). Recently introduced into Australia (2006). Can be given via drench to pigs or in water (via proportioner systems). If given in water, best to give after 6 weeks of age, when water intake stabilizes. Take care with administration. This is a live vaccine, so don't give antibiotics 3 days before or 3 days after vaccination (total of 7 day medication-free period). Use within 4 hours of reconstitution.

Swine dysentery

Aetiology

Brachyspira hyodysenteriae

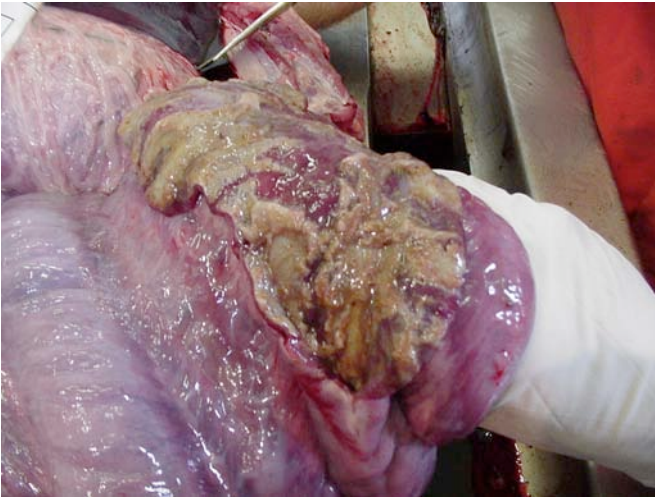
Epidemiology/Transmission:

- Age affected: 6-22 weeks (10-16 weeks most common)
- Route of infection: Fecal-oral transmission
- Organism survives for long periods in the environment – especially in moist dung.
- Carrier pigs most important in transmission and survival in piggeries
- Relatively common in Australia. Probably the most expensive enteric disease.*****

Clinical signs:

- Variable colored (yellow, brown, gray) diarrhea that progresses to bloody diarrhea (dysentery)
- Undigested feed, mucous casts and necrotic material in stool
- Wasting, emaciation
- Feed refusal

Gross lesions:



Large intestine primarily affected- mainly colitis, catarrhal to fibrinonecrotic typhocolitis, serosal edema, swollen colic lymph nodes, spiral colon contains mucous, blood and necrotic material, ascites

Histopathology:

Goblet cell hyperplasia and dilation of crypts, hemorrhage, fibrinonecrotic debris and inflammatory infiltrates, demonstration of *Brachyspira* organisms (Warthin-Starry stain)

Diagnosis

Clinical signs, demonstration of organisms (Warthin-Starry stain), culture, PCR

Treatment

Antibiotics (tiamulin, lincomycin). Must be given IM to be effective. Water medication as an adjunct to treatment.

Prevention

- Rigid biosecurity, restriction of traffic and control of rodents
- Obtain pigs from *B. hyodysenteriae*-free sources
- Multi-site production with medicated early weaning (MEW)
- Depopulation, cleaning, disinfection and repopulation of infected units
- Whole herd treatment and aggressive rodent control

Eradication.

This can be achieved without depopulation by feeding high levels (6-9mg/kg) of an effective antibiotic for a period of 2 weeks; cleaning the environment of all visible manure which was there prior to medication and placing progeny pigs on low dose (50ppm Lincomycin or Tiamulin, 200ppm DMZ) until all progeny which were on the farm at commencement of eradication have been sold ie a total of 22-24 weeks (ref Eradication of Pig diseases. APL publication, Editor Ross Cutler)

Porcine intestinal spirochaetosis

(this condition is very similar to SD in all respects ,but the clinical signs are not as severe)

Aetiology

Brachyspira pilosicoli

Epidemiology/Transmission:

- Use of PCR probes would suggest that it is more common than we thought, and we may have confused this with mild SD in the past.
- Age affected: most commonly 8-16 weeks, but any age in susceptible animals ie replacement breeders introduced into affected herds
- Fecal-oral transmission
- Associated with commingling of multiple source pigs and feed changes
- Carrier pigs important in transmission and maintenance in herd

Clinical signs:

Variable colored (green to gray) diarrhea, mucus and occasional flecks of blood in stool, loss in condition, feed refusal

Gross lesions:

Spiral colon and cecum affected, contents are watery and contain mucus, reddened, edematous mucosa, feed particles adherent to the mucosa +/- Fibrin tags.

Histopathology:

Variable superficial erosive colitis, goblet cell hyperplasia and dilation of crypts, mucosal edema, fibrinonecrotic debris and inflammatory infiltrates, end-on attachment of *Brachyspira* organisms on luminal epithelial cells (hence the name pilosi (hair like) coli in the colon originally the little serpent of the hairy colon)

Diagnosis

Clinical signs, culture, PCR most important diagnostic tool to distinguish it from SD

Treatment

Treatment and control procedures are largely modelled on the procedures developed for SD. Antibiotics (Lincomycin, Tiamulin, DMZ, Tylosin, Monensin, Tetracyclines)

Prevention:

- Strict biosecurity and control of birds, rodents and canine contact with pigs. Has a wider host range than *B. hyodysenteria*
- All-in/all-out movement of grow-finish facilities
- Careful selection of animal sources & Isolation of incoming pigs. If in doubt treat with 6mg/kg of Lincomycin or Tiamulin in the ration for 2 weeks

Salmonellosis

Aetiology

Salmonella choleraesuis (septicemic and gastrointestinal); *Salmonella typhimurium* (gastrointestinal)

Epidemiology/Transmission:

- *Salmonella choleraesuis* has not been reported in Australia since 1969. *Salmonella typhimurium* occurs but is Uncommon in Australia
- Age affected: 6-20 weeks most common, but any age may be affected
- Fecal-oral and aerosol transmission

- Associated with management stresses

Clinical signs:

S. choleraesuis: Yellow diarrhea, cyanosis of extremities, pneumonia, lethargy, death

S. typhimurium: Dark green to dark brown diarrhea, +/- blood, feed refusal, severe loss of condition, rectal stricture

Gross lesions:

- Jejunum, ileum, cecum and spiral colon affected (for *S. typhimurium* mainly distal ileum and caecum)
- Fibrinonecrotic membranes
- Multifocal hemorrhages
- Button ulceration of the colonic mucosa (*S. typhimurium*)
- Enlarged, edematous organs
- Congested, edematous lungs (around 50% of pigs with intestinal lesions *S. typhimurium* will have a bronchopneumonia affecting apical and/or cardiac lobes)

Histopathology:

- Severe fibrinonecrotic enterocolitis
- Macrophage infiltration of lamina propria and submucosa
- Thrombi in vessels of lamina propria and mucosa

Diagnosis:

- Clinical signs, histopathology, culture

Treatment/Prevention

Antibiotics on C&S results.

If occurring in suckers and weaners consider autogenous vaccination of sows against *S. typhimurium*.

Whipworms

Aetiology

Trichuris suis

Epidemiology/Transmission:

- Uncommon in piggeries in Australia but bear it in mind as a differential diagnosis for poor growth associated with colitis
- Age affected: 8-24 weeks
- Infection via ingestion of infective eggs

Clinical signs

Anorexia, dehydration, mucoid to bloody diarrhea, death

Gross lesions:

Cecum and spiral colon affected, catarrhal, fibrinonecrotic and hemorrhagic typhocolitis, mucus and necrotic debris in the lumen, whipworms in cecum and colon

Histopathology:

Nematode larvae on the mucosa, hemorrhage and necrosis, inflammatory infiltrates (eosinophils, plasma cells), fibrin exudation

Diagnosis

Gross pathology, Whipworm eggs on fecal exam & histopathology

Treatment

Fenbendazole is highly effective against all stages of the life cycle. However, it is off label use in Australia where it has never been registered. Give in feed at a total of 5mg/kg over a 7-14 day period. Morantel and Levamisole, which are currently registered for pig use in Australia, are only about 60% effective.

Control

Like most pig intestinal parasites the prepatent period is around 40 days so treatment of breeders very other month or one week in 6 will break the life cycle.

Nodule worm

Aetiology

Oesophagostomum dentatum.

Similar to whip worm in terms of life cycle and control

Porcine gastric ulcers

Aetiology

Unknown- concurrent Disease such as enzootic pneumonia, feed management and infectious etiologies proposed, concurrent respiratory diseases, feed deprivation or poor appetite due to disease, diet particle size (<700 microns), *Helicobacter spp*

Epidemiology/Transmission

Age affected: 3 to 6 months

Clinical signs

Skin pallor, vomiting (due to sticture of healed ulcer at oesophageal inlet), poor doing pigs, sudden death, black, tarry stools

Gross lesions



Hyperkeratosis of pars oesophagea , bile staining of pars, erosive lesions, frank blood (or coffee grounds) in stomach

Diagnosis

Clinical signs, necropsy

Treatment/Prevention:

- Prevent disruptions in feed consumption
- Insure diet particle size of 700 microns or greater

Roundworms (*Ascaris suum*)

Very common-found in small intestine. Adults 15-40cm long.

Eggs thick-shelled, sticky coated and resistant to disinfectants

Pathogenesis:

Damage caused by migrating larvae - damage to liver tissue, fibrosis; multifocal pulmonary haemorrhage and inflammation

Damage caused by adults – enteritis; migration causes blockages, perforations. Economic loss in abattoir due to loss of intestines as sausage casings

Clinical signs:

Usually in younger pigs-coughing, dyspnoea, secondary pneumonia, afebrile diarrhea, weight loss, illthrift

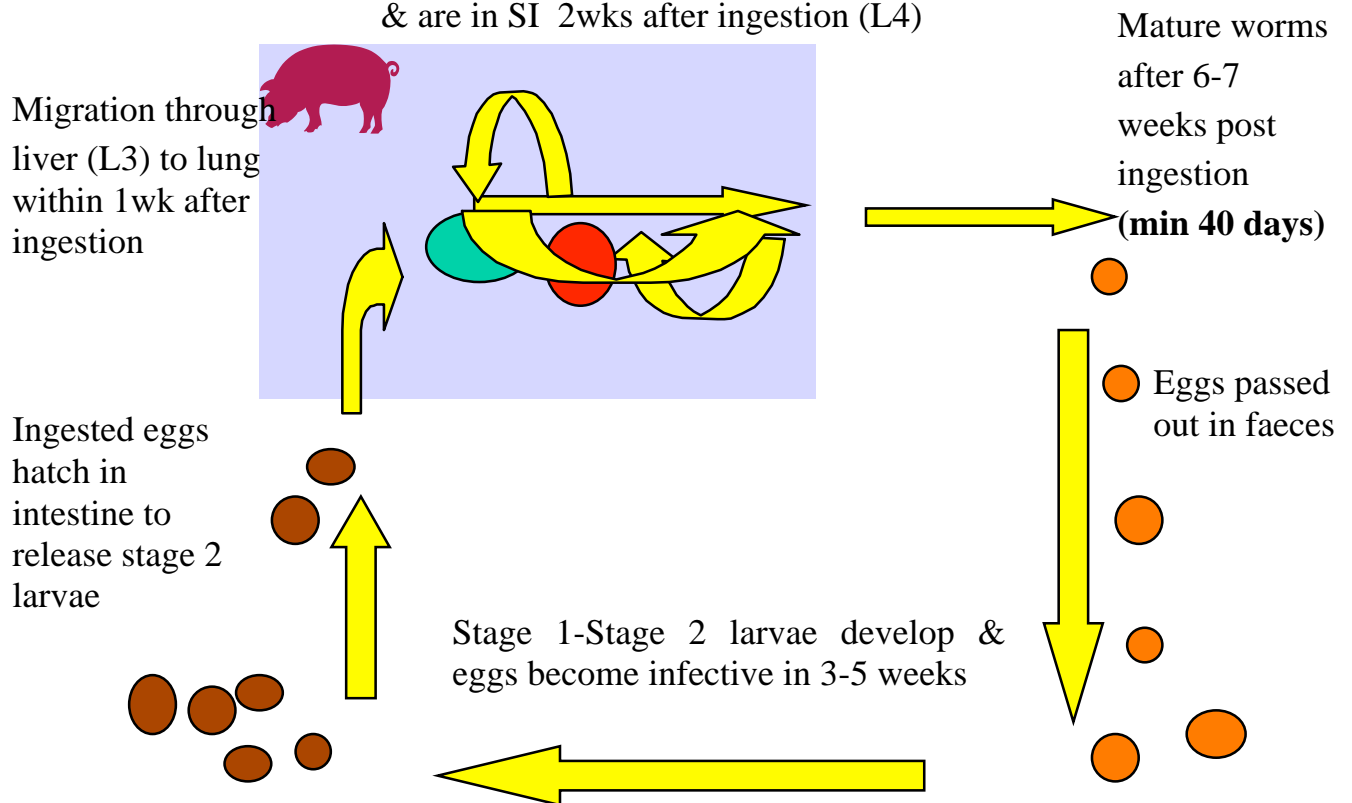
Diagnosis:

Slaughter checks, post mortem, eggs in faecal flotations

□ **Life cycle:**

Ascaris suum life cycle

Stage 3 larvae are coughed up & swallowed,
& are in SI 2wks after ingestion (L4)



□ **Treatment/control:**

In-feed easiest-Wormtec 30 (1kg/tonne), fenbendazole, Ivermectin.

Treat sows for at least once within the prepatent period of 40 days, include anthelmintic in weaner diet for at least one week before 42 days of age.

Balantidium coli

A large ciliate normally a commensal found in the large intestine of healthy pigs. An alteration in the microbial flora in the gut allows it to become a secondary invader and exacerbate existing lesions of say SD, Spirochaetal colitis, Whipworm etc. invasion of the mucosa of colon and caecum>>multiple small mucosal abscesses>>persistent watery diarrhea, normal colour, abnormal consistency. It is capable of infecting humans and some other species and may cause an explosive bloody diarrhoea

Intestinal catastrophes

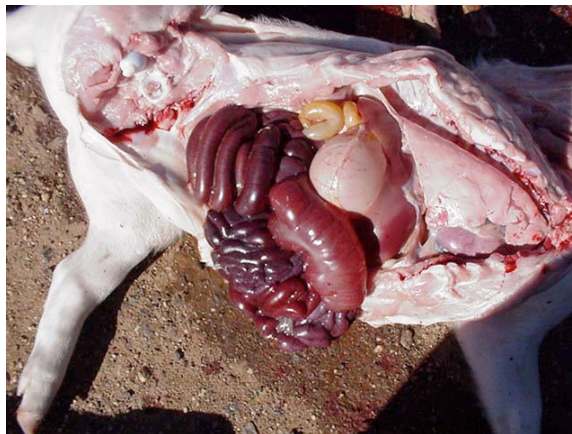
Associated with abnormal distension or torsion of different parts of the alimentary tract. Cause largely a mystery, best to blame it on the nutritionist

Stomach-distension associated with feeding whey.

SI-torsion of the mesentery

LI-distension and/or torsion often associated with whey feeding

Death-due hypovolemic shock, respiratory distress, or rupture



RESPIRATORY DISEASE

Respiratory Immunity

Non-specific clearance mechanisms:

Mechanical barriers: The tracheobronchial duct traps and eliminates airborne particles.

- Curving of the tracheobronchial duct
- Decreasing the velocity of air at the point where the tube enters the lung
- Impaction of foreign particles on the mucous layer
- Ciliary clearance of inhaled particles

Biochemical barriers: Bactericidal and antiviral substances in the nasal and tracheobronchial mucous inactivate the foreign microorganisms (IgA, lysozyme, Ferritin, Beta lysine, Interferon) note this is a mucosal surface

Cellular mechanisms: In the alveoli,(no longer a mucosal surface) where there is no mucous, alveolar macrophages phagocytize invading microorganisms. The macrophage response can be inhibited by PRRS virus, Mycoplasma hyopneumoniae, or the toxins of Actinobacillus pleuropneumoniae.

Note that the alveoli contains small amounts of IgG

Specific immune response:

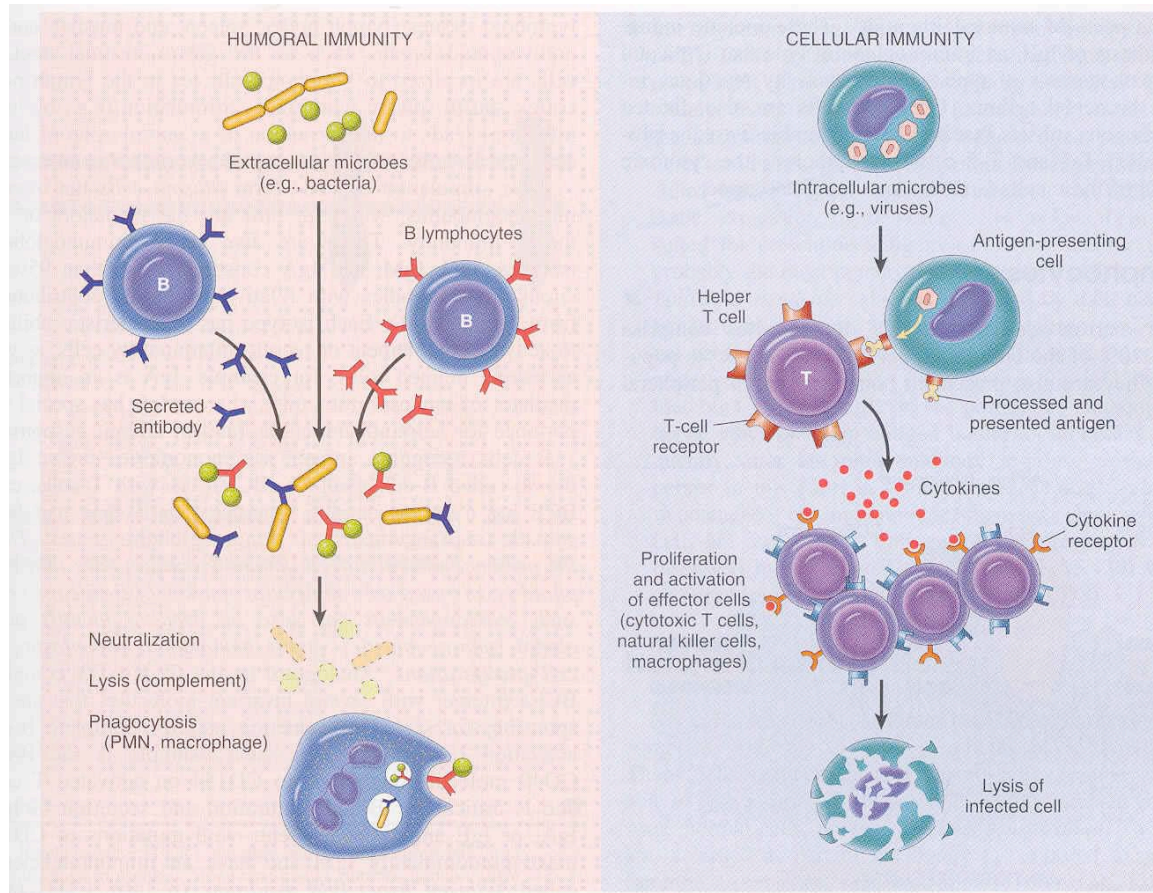
IgG: Antibacterial protection, fixes complement, opsonizing activity, located in deep respiratory sites ie below the terminal bronchioles commencing at alveolar duct

IgA: Primarily secreted in upper respiratory tract, protects against virus and bacteria.

Cell enhanced immunity as opposed to Cell-mediated immunity: Macrophages and lymphocytes are the predominant cells. Neutrophils, eosinophils, basophils, and epithelial cells are also present; Long term memory; Activated T-lymphocytes attract additional lymphocytes and macrophages to the lungs by secreting lymphokines. This enhances the destruction of microbes.

Immunosuppression:

Primary infection, stress of management, stress of environment, combination



Immunology for decision making

- First exposure to vaccine antigen or disease agent ~2 weeks for antibody production
- Second exposure to vaccine antigen or disease agent ~ 4 days for antibody production (memory response)

Humoral immune response:

- Evolved to fight extracellular pathogens
 - B cells produce antibody (IgG in blood; IgA at mucosal surfaces)
 - Good for neutralization of extracellular phases of bacterial and viral infections & toxin neutralization
 - Both live vaccines and killed bacterins can stimulate a humoral immune response (the antigen is gobbled up by a macrophage which presents it to a T helper cell which then presents it to a B cell which produces antibodies ie Plasma cells)
 - Give bacterins intramuscularly stimulates IgG; Give vaccines orally, intranasally- IgA ie horses for courses

Cell-mediated immunity:

- Evolved to fight intracellular pathogens which are protected from the humoral immune response by the cell wall; therefore we must destroy the whole cell in order to save the host. Probably evolved to combat cancer cells

- T cells search for infected host cells and destroy these infected cells
- Good for fighting intracellular bacteria and the intracellular stages of viral infections
- Only Live vaccines can stimulate CMI, as they have to invade the host cell in order to evoke the production of Killer T cells.

Mycoplasma pneumonia

Mycoplasmal pneumonia of swine is a syndrome that results from interactions among various pathogens. This syndrome is considered to be the most common swine respiratory disease in the world, and likely affects 99% of swine herds in Australia. Management, genetic, and environmental variation make quantification of the economic effects of mycoplasmal pneumonia difficult; however, estimations of an additional 11 days to market weight and 0.15 increase of feed/ gain have been reported.

Transmission:

Mycoplasma hyopneumoniae is primarily spread by direct contact with respiratory secretions from infected pigs but can be spread by wind between farms. Infected sows usually transmit *M. hyopneumoniae* to their offspring by 3 weeks of age. Infections are maintained in herds when the organism is transmitted from older infected pigs to younger naïve pigs; thus, continuous flow operations tend to maintain their disease status. Distances of less than 3.2 Km from mycoplasma-free herds to herds with endemic mycoplasmal pneumonia have been shown to be a major risk factor of transmission from herd to herd.³

Pathogenesis:

The incubation period for mycoplasmal pneumonia is approximately 10- 16 days. After inhalation, *M. hyopneumoniae* adheres to the ciliated epithelium of the trachea, bronchi, and bronchioles. The mucociliary apparatus is disrupted followed by reduced ciliary activity, ciliary loss, and epithelial cell destruction. Consequently, bacteria, epithelial debris, and secretions that cannot be cleared accumulate in the airways. Lymphocytes aggregate around blood vessels and bronchioles. Bronchioles become obstructed resulting in alveolar collapse. Damage caused by *M. hyopneumoniae* can compromise the lung sufficiently to allow secondary infections by a variety of organisms including other bacteria, viruses, and nematodes; however non-toxicogenic *Pasteurella multocida* Type A is the most common secondary agent to cause a purulent bronchopneumonia.

Clinical signs:

Most pigs do not exhibit clinical signs of mycoplasmal pneumonia until 3 to 6 months of age. High morbidity and low mortality characterize mycoplasmal pneumonia. A dry, nonproductive cough is the most prominent clinical sign. Coughing begins 2 weeks after experimental infection, peaks at 5 weeks, and resolves by 12 weeks. Recovered pigs appear to be resistant to reinfection.

Clinical signs of *Mycoplasma pneumonia* will vary according to (1) the immune status of the herd, (2) secondary infections, and (3) concurrent infections. Uncomplicated *Mycoplasma hyopneumoniae* infection in a typical commercial herd results in mild and transient clinical signs because most pigs have some degree of natural immunity to the organism. Coughing is rarely observed before 6 weeks of age, and is most common in 14-16 week old pigs (about 1 month after pigs enter the finishing facility). The cough lasts 4-8 weeks in individual pigs, but may persist in the population throughout the finishing stage as disease spreads through the group.

Early weaned pigs that have escaped colonization by *M. hyopneumoniae* or have been colonized in such low numbers that an effective immune response is not generated may be highly susceptible to mycoplasmal pneumonia and other diseases to which they have no natural immunity. Reintroduction of these "naïve" pigs into commercial herds may result in severe clinical disease or death. Thus, vaccination (as selected gilts or earlier) of segregated early

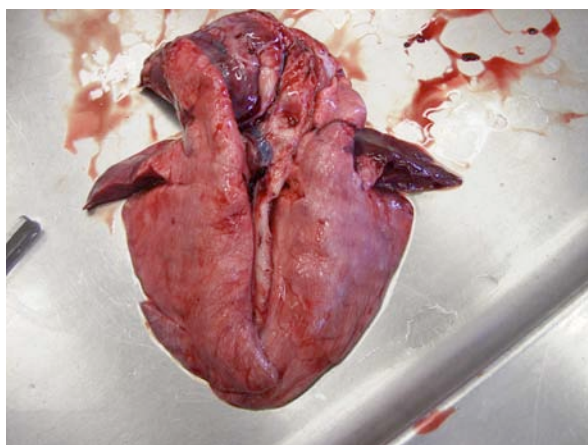
weaned pigs that have had minimal exposure to *M. hyopneumoniae* is recommended before these animals are entered into the breeding herd as replacement seedstock or commingled in nursery or finishing facilities.

A productive cough, "thumping", usually signifies a secondary infection with nontoxigenic *Pasteurella multocida* Type A. This complex has been named enzootic pneumonia. Other clinical signs associated with enzootic pneumonia include severe respiratory depression, fever, anorexia, weight loss. Pleuritis, abscessation, and death may result.

Mycoplasma hyopneumoniae infection in conjunction with concurrent respiratory pathogens (particularly APP) in a herd has been labeled porcine respiratory disease complex (PRDC). Clinical signs of PRDC usually appear when pigs are 18-20 weeks old and can be acute or chronic. This complex can result in 4-6% mortality in finishing pigs, poor daily gains, poor feed conversion, and sort loss. Control of *M. hyopneumoniae* appears to be central to minimizing clinical expression of PRDC even when APP is involved.

Lesions:

Gross lesions of pure mycoplasmal pneumonia consist of well-demarcated, purple to gray consolidation of the antero-ventral portions of the cranial, middle, and caudal lung lobes and in various portions of the accessory lobe. A purulent bronchopneumonia with fibrinous pleuritis and pericarditis can be observed with secondary infections and is characteristic of enzootic pneumonia. Caution must be taken when interpreting lung lesions at slaughter because lesions will have resolved if the pig was clinically infected at an early age.⁸



Immunity:

Natural immunity to *Mycoplasma* appears to be of long duration as recovered pigs are resistant to reinfection. Cell mediated immunity and secretory IgA provide primary protection against *M. hyopneumoniae*. Cells producing IgM, IgA, and IgG specific to *M. hyopneumoniae* have been identified from infected lung sections. Increases in IgG appear to correlate with resolution of pneumonia. The half-life of maternal antibodies to mycoplasma is approximately 15.8 days. However many factors affect the uptake of colostrum.

Diagnosis:

Presumptive diagnosis of Mycoplasmal pneumonia is based on clinical signs and post-mortem examination (bronchopneumonia in the cranio-ventral lung lobes). Most likely differential diagnosis for the gross lesion is *H. parasuis*. Culture may take up to 4 weeks so is not undertaken routinely. PCR can be used to confirm the presence of mycoplasmal DNA in lungs and is a good tool to use when herds are supposedly "Mycoplasma-free". Exposure of herds to *M. hyopneumoniae* can be concluded by serological monitoring using an ELISA, but will not confirm a diagnosis on an individual pig basis.

□ **Treatment:**

Although many antibiotics demonstrate effectiveness against *M. hyopneumoniae* in vitro, the location of the Mycoplasma on the cilia of the bronchial tree generally prevents antibiotics from reaching the organism in high enough concentrations to eliminate the infection or resolve the lesions. Antibiotic administration in feed or by injection may decrease clinical signs and mortality by controlling but not eliminating Mycoplasma and secondary infections. Lincomycin hydrochloride @ 6-8mg/kg, Tiamulin at 6-8 mg/kg or Chlortetracycline @ 25mg/kg used strategically at the time of colonization may help.

□ **Prevention/Control:**

Optimization of management and environment are essential to the control of Mycoplasmal pneumonia. All-in/ all-out management controls Mycoplasma by preventing transmission from older infected pigs to young naïve pigs. Pigs with no more than a 1-2 week age difference are moved into a clean room. All pigs are moved out to the next stage or marketed as a group. The empty room is cleaned, disinfected, and the disinfectant allowed to dry before the next group of pigs enters. All-in/all-out management has been one of the most successful procedures used to control clinical Mycoplasmal pneumonia (subclinical infection is the general result). A complete herd program encompassing optimization of air quality, room temperature, sanitation, stress reduction, and parasite control is needed to minimize the effects of mycoplasmal pneumonia in growing pigs.

Vaccination is an important tool for the control of Mycoplasmal pneumonia. Both single dose and two-dose killed bacterins are commercially available. You can expect lung lesion scores (at abattoir) to reduce to about 3% in twice vaccinated pigs (vs 15% in non vaccinated herds). Vaccination is recommended for control of Mycoplasma in (1) continuous flow units, (2) pigs commingled from different source herds, (3) early weaned replacement stock or stock from minimal disease herds, and (4) herds with severe PRDC. Vaccination has been shown to increase carcass weight by about 3.5kg, depending on herd performance before vaccination. Segregated early weaning units can usually tolerate higher increases in mortality before vaccination is economical. The jury is still out on whether maternal immunity reduces the effectiveness of vaccination. Some manufacturers may recommend that pigs younger than 3 weeks of age should not be vaccinated. This is likely to be based more on marketing than science. Certainly herds vaccinated late ie after maternal antibody has disappeared around 8 weeks of age and again 4 weeks later perform no better in terms of lung lesions or average daily gain than those vaccinated at 4 days of age (when picked up to be treated for cocci) and given a booster dose at weaning. This is probably a good example of herd immunity where if 70-80% of animals respond we get herd immunity. This of course would be an absolute disaster for a disease which causes death such as APP. If the 20% which did not respond then subsequently died, then this would be very costly.

Mycoplasma hyopneumoniae can be eliminated or made subclinical using early weaning and age segregation procedures in some herds. Early weaning prevents or minimizes transmission of *M. hyopneumoniae* from dams to offspring, and age segregation minimizes transmission among weaned pigs. Weaning ages for *M. hyopneumoniae* eradication are herd specific and dependent on a variety of sow and pig factors. Strict biosecurity protocols concerning pigs, people, vehicle, and equipment flow must be followed to prevent reinfection of the weaned pigs with *M. hyopneumoniae*.

Actinobacillus pleuropneumoniae

New technologies such as early weaning and age segregation of pigs have produced groups of high health status pigs with increased susceptibility to disease.

Aetiology:

Actinobacillus pleuropneumoniae is a gram negative coccobacillus. Colonies appear beta hemolytic on blood agar and are urease positive. Twelve serovars have been described within 2 biovars. Biovar 1 isolates are dependent on nicotinamide adenine dinucleotide (NAD) for growth and biovar 2 isolates do not require NAD for growth. Serovars 1, 7 and 15 are the most common in Australia, and about a dozen farms have serovar 5. Generally, serovars 1 and 15 are most virulent. Serovar 7 is generally of low virulence.

Epidemiology:

Pigs are the only known species susceptible to APP because the organisms can only obtain iron from porcine transferrin. Transmission occurs primarily by aerosol over short distances (less than 500 metres) and by direct contact between pigs. Most herds become infected following introduction of symptomatic carrier swine. All ages of pigs are susceptible; however, APP is usually observed in pigs older than 3 months of age, probably due to the decline of maternal antibody by 8-10 weeks of age. The incubation period can vary from hours to days; however, mortality can occur within 4-12 hours after exposure. Morbidity (50%) and mortality (1-10%) are generally high, but are dependent on the immune status of the pig and the virulence and number of organisms to which the pig is exposed. Recovered pigs can become asymptomatic carriers. Pigs exposed to low doses of organisms can become subclinically infected. Economic losses are due to mortality, decreased growth performance, and medication costs.

Pathogenesis:

Virulence factors for APP include capsule, lipopolysaccharide (LPS), repeats-in-toxin (RTX) cytotoxins, and outer-membrane proteins. *Actinobacillus pleuropneumoniae* can colonize the tonsil and alveolar epithelium. Organisms multiply in the lung after inhalation. The bacteria release outer membrane blebs containing LPS and possibly some toxins. Neutrophils are attracted to the area by the LPS and an inflammatory response begins. The neutrophils are destroyed by the cytotoxins once they reach the site of infection. The extensive tissue damage caused during APP infections is thought to be the result of neutrophil destruction. The cytotoxins can also destroy alveolar macrophages, endothelial cells, and alveolar epithelium. Organisms appear to be resistant to complement and digestion by phagocytes.

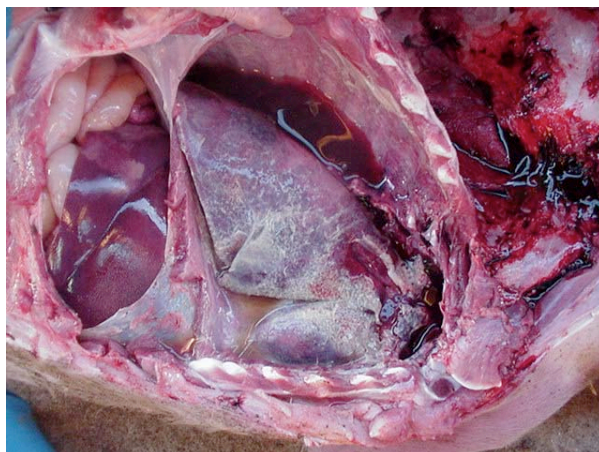
Clinical signs:

The severity of clinical signs varies with the serovar involved. The course of the disease varies from peracute to chronic. Secondary infections may exacerbate clinical signs. Stress factors such as poor hygiene, temperature fluxes, humidity, dust, noxious gases, mixing, moving, and purchasing animals from multiple sources will also impact on the severity of the disease. Single or multiple pigs in the same or different pens can become ill during an outbreak. Sudden death without observation of any clinical signs is typically reported. Pigs are febrile (40C), anorexic, and dyspneic. Cyanosis of the extremities can be observed. Pigs dog sit and exhibit open-mouthed breathing. Sometimes a foamy, blood-tinged discharge from nares and mouth will be observed (pigs literally drown in their own serosanguineous exudate) Chronically affected pigs have poor growth performance due to diminished appetite. Intermittent coughing can develop.

Lesions:

Gross lesions consist of a fibrinohemorrhagic and necrotizing pleuropneumonia having a hilar, unilateral or lobar distribution - most commonly on the caudal or diaphragmatic lobes on the lung. Distribution can be bilateral and diffuse. A serosanguineous thoracic exudate can be present

and in peracute cases the trachea and bronchi are filled with a foamy, blood-tinged exudate. Fibrinous or fibrous pleurisy can develop. Abscesses or walled off lesions are observed in chronic cases (most often detected at the abattoir). Differential diagnoses would be acute *Pasteurella* infection, outsiders may include *Actinobacillus suis*, *Haemophilus parasuis* and *Salmonella choleraesuis*.



Immunity:

Antibodies can be detected as early as 10 days post-infection, peak within 4 weeks, and remain at a low level for months following infection. Colostral antibodies can be detected for up to 9 weeks

Diagnosis:

Presumptive diagnosis is based on history, clinical signs and gross lesions.

Definitive diagnosis is based on cultural examination of acutely affected lung and serotyping. Organisms grow well on 5% blood agar with a *Staphylococcus epidermis* nurse streak or liquid NAD. Colonies will satellite along the nurse streak. Serotyping can be performed using slide agglutination or coagglutination techniques.

Treatment:

Administration of injectable antibiotics based on antibiotic sensitivity. Draxxin (tulathromycin) is an extremely effective antibiotic against respiratory pathogens so go with this first. Other options include Penicillin or a potentiated sulphonamide. Affected pigs have reduced feed and water intake; thus, treatment by oral medication is not recommended. Pigs in contact with sick pigs (pigs in the same pen or adjacent pens) may be treated by water medication or individual injection, depending on the extent of the outbreak. It is useful to also inject acutely ill animals with an anti-inflammatory (eg Flunixin).

Prevention/Control:

Preventing introduction of asymptomatic, chronically infected pigs into naïve herds is the best way to prevent an APP epizootic. Health matching, serological profiling, and strict isolation and medication of incoming stock are important preventive tools. Optimization of environment (Limit temperature fluxes, minimize dust level, optimize humidity, and provide adequate fresh air) and management (minimize moving, mixing, and crowding) are important in minimizing disease incidence.

The serological status of a herd can be determined by submitting a minimum of 30 serum samples from adult pigs using ELISA. Commercial ELISAs that are not serovar-specific (test for the APx toxin) are available.

Feed or water medications can be effective in controlling APP; however, clinical disease can recur after medication ceases. Water medication using amoxicillin (at 30mg/kg) each day for 3 consecutive days may be used as an adjunct to IM treatment as very ill pigs will not drink. Tilmicosin (Pulmotil) fed at 200-400 g/t continuously for 21 days beginning approximately 1 week before an expected outbreak of APP is labeled for the control of swine respiratory disease associated with APP.

Predictable outbreaks.

Many farms can predict when they are likely to have an outbreak. Most notably when immunologically naïve pigs are moved from a weaner shed to a contaminated grower shed or site. In these situations the use of in feed medication for the first 2 weeks, combined with diligent observation and injection of sick pigs will minimise the damage

Actinobacillus pleuropneumoniae bacterins are commercially available; however, their efficacy is unproven in pen or field trials, there is much anecdotal opinion that their efficacy is questionable beyond a possible reduction in mortality. Vaccination at 10 weeks of age and again 2 weeks before pigs are likely to be challenged is recommended. The reasoning for this timing is that maternal antibody may neutralise the first dose if given before 10 weeks of age, and because APP is such a fulminating disease, there is no time for an anamnestic response to occur. Unless the serosanguineous exudate which floods into the alveoli is rich in specific IgG the disease process will not be halted. Sows may also be vaccinated 3 weeks before farrowing in an attempt to boost maternal antibody, this will level out the playing field during cross exposure of weaners.

Segregated early weaning has been successfully used to prevent transmission of APP in some herds. Effective weaning ages will vary from farm to farm. Pigs early weaned at 10 days of age or younger and age segregated have had the most success. Approximately 50% of herds weaning at 16 days of age are successful, and herds weaning at 18- 20 days of age have approximately a 5% success rate.

Haemophilus parasuis

H parasuis is endemic to pig herds. The bacteria is transmitted from the oro-nasopharynx of the sow to her piglets and from carrier pigs to naïve younger pigs at weaning. This disease is most common in weaners, but can occur in 14 day old suckers.

Outbreaks of disease are usually precipitated by some kind of stress (temperature fluctuations, overcrowding, poor hygiene etc). In outbreaks of disease there is usually high morbidity (50-75%) with mortality up to 10%. Respiratory infection occurs, followed by septicemia.

Clinical signs in affected pigs will include some or all of the following: fever (39C), lameness, meningitis, poor growth (“fading” pigs). Lesions include polyserositis (arthritis, pneumonia, pleuritis, fibrino-purulent meningitis (rare).

In endemic situations the stress of weaning and co-mingling of naïve weaners with carrier pigs can produce a syndrome where there is reduced growth rate in the weaning house (<400gms /day with 5-7% mortality usually from destruction of animals for failure to thrive)

Diagnosis:

- Clinical signs
- Necropsy - polyserositis (fibrinous to fibrino-purulent), pseudo membranes, pneumonia(congestion/haemorrhage), fibrino-purulent meningitis, arthritis (fibrino-purulent, periarticular edema and suppuration). For isolation of the organism untreated, acutely affected or recently dead animals used.
- Culture organism (X & V factors required). Most success if less than 4 hours between when the pig has died and the sample taken. Samples are best taken in a sterile manner

from pericardial, pleural or peritoneal fluid. There are also now PCRs available for *H. parasuis*.



Prevention:

- Reduce stress at weaning
- Vaccination – commercial vaccines eg. Fort Dodge HPS vaccine with serovars 4 and 5. Claim cross-protection with serovar 13. Otherwise need to get an autogenous vaccine made up if the serovar on-farm is not in the vaccine.
- Can vaccinate sows with a booster dose 13 weeks of pregnancy (must be strain-specific). This will level the playing field so that when cross infection occurs at weaning all pigs will be protected with maternal antibody while they mount their own active immunity. Can vaccinate piglets (2 shots, 3-4 weeks apart) but may not be effective if disease occurs very early.

Treatment:

- Affected pigs respond poorly to antibiotic treatment. Try Amoxycillin and Flunixin IM. Once affected they usually continue to fade away and are eventually destroyed.

Porcine Respiratory Disease Complex

Porcine Respiratory Disease Complex (PRDC) is a name used mainly in the United States to describe respiratory disease in growing pigs. It does not implicate a specific etiological agent or agents-only that more than one pathogen is involved.

Porcine Respiratory Disease Complex usually describes acute or chronic respiratory disease in pigs 18 -20 weeks of age. It is sometimes referred to as "The Wall" because the pig's growth is curtailed. Poor daily gains, poor feed conversion, 4-6% or greater mortality, sort loss, and increased treatment costs are common consequences of PRDC.

The primary agents involved are *Mycoplasma hyopneumoniae* and *Actinobacillus pleuropneumoniae*. Opportunistic agents involved are *Pasteurella multocida*, *Streptococcus suis*, *Haemophilus parasuis* and *Mycoplasma hyorhinis*.

Preliminary diagnosis and treatment is based on clinical signs, epidemiology and necropsies. To get a definitive diagnosis, send appropriate samples from acutely affected, live, untreated pigs.

Develop a strategic, cost-effective control program based on strategic vaccination, medication, management optimization (avoid mixing, moving, crowding), environment optimization (ventilation, temperature, pig density, feeder and waterer access), biosecurity (pig, people, vehicle & equipment flow) and utilization of new technologies (AIAO, SEW).

Serological profiling may be used to refine vaccination/ medication protocols. This can be undertaken using cross-sectional sampling (1 visit) or longitudinal sampling (over time).

Atrophic Rhinitis

Infectious progressive atrophic rhinitis is a disease complex rarely seen in Australia today. As in the other respiratory diseases, the susceptibility of the pig is affected by the age of the pig, the number and pathogenicity of the bacteria, ventilation, dust, noxious gases, humidity, nutritional deficiencies, and the presence of secondary pathogens.

Aetiology:

Two syndromes are recognized. Non progressive atrophic rhinitis is caused by toxigenic *Bordetella bronchiseptica* alone. Progressive atrophic rhinitis is caused by *Pasteurella multocida* type D toxigenic alone or in combination with *Bordetella bronchiseptica*.

Bordetella bronchiseptica: thermolabile AR toxin

Pasteurella multocida type D toxigenic

Both *Pasteurella multocida* type A nontoxigenic and toxigenic strains can also be isolated

Transmission:

Aerosol droplets, dam to pig, pig to pig, purchased breeding stock which are infected

Pathogenesis:

Bordetella bronchiseptica is nearly ubiquitous in the swine population. Thus, *Bordetella bronchiseptica* can be isolated from pigs without clinical atrophic rhinitis. *Bordetella bronchiseptica* organisms adhere to nasal mucosa, attach to ciliated epithelial cells, multiply on the mucosal surface, and produce toxins. This leads to inflammatory, proliferative, and degenerative changes (loss of cilia) in the nasal epithelium. Once the *Bordetella bronchiseptica* has damaged the mucosal surface, *Pasteurella multocida* can colonize the nasal cavity. The heat-labile dermonecrotic toxins produced by *Pasteurella multocida* cause the bony changes by stimulating osteoclasts.

Clinical signs:

Sneezing, conjunctivitis with staining of skin and hair at medial canthus of eye, serous to mucopurulent nasal exudates, distortion of nasal bones, epistaxis

Necropsy & lesions:

- Transverse Cut of snout at the level of the first/second upper premolar
- Atrophy of ventral &/or dorsal turbinates
- Increased space between the ventral nasal cavity and the ventral turbinates
- Deviation of nasal septum
- Hyperemia and edema of mucous membranes
- Nasal exudate (+/- blood)

Diagnosis:

- Culture (usually from weaners) coupled with necropsy or abattoir findings. Need to demonstrate presence of dermonecrotic toxin.

Treatment:

- Optimized management, environment, and nutrition, coupled with antimicrobial therapy are needed to combat atrophic rhinitis. Autogenous vaccines may help.
- Pigs less than 4 weeks old-injection of long acting oxytetracycline-20-80 mg/kg once or twice. New antibiotic (“Draxxin”) also likely to be very effective.
- Weaners and growers- Oxytetracycline in feed (400g/ton), Tylosin in feed (100 g/ton)

Prevention:

- Autogenous vaccination (killed bacterin);
- LA 200 or Draxxin: IM of baby pigs at days 1,7,14, and 21, with 0.5,1.0,1.0, and 1.0 mls, respectively.
- Purchase all breeding stock only from a known Atrophic Rhinitis free herd
- New Segregated Early Weaning technologies can eliminate clinical atrophic rhinitis in some herds
- Monitor prevalence on slaughter check

DDx:

Porcine cytomegalovirus, Paranasal abscess (Bullnose)

Porcine Cytomegalovirus

Porcine cytomegalovirus, a Herpesvirus, causes Inclusion body rhinitis, and should be on your differential diagnosis for atrophic rhinitis.

Cytomegalovirus can isolated from: nasal/ ocular secretions, urine, semen, cervical fluids, transplacental. Transmission is most common by the nasal route or through contact with a urine-contaminated environment. The lung macrophage is a reservoir of infection.

Lesions:

No gross epithelial lesions are observed. Generalized lesions include petechiae in the kidneys and small intestine, pulmonary edema, pericardial and pleural effusions, and subcutaneous edema. Microscopic lesions include basophilic intranuclear inclusion bodies and cytomegaly in nasal mucous glands, lachrymal glands, and renal tubular epithelium

Clinical signs:

- Pigs < 3 weeks old: Mortality may reach 25% (stillborn or die shortly after birth) anemic, stunted, edema of tarsal joints and around jaw, shivering, sneezing, respiratory distress, mild rhinitis
- Pigs > 3 weeks old: Asymptomatic or mild rhinitis
- Sows: anorexia, lethargy, not pyrexia

Diagnosis:

- Virus isolation in neonates and fetuses from nasal mucosa, lung, kidney, and pulmonary macrophages.
- Immunohistochemistry on frozen tissue sections.

Treatment/Control/Prevention

* The disease is self limiting. Antimicrobials can be used for concurrent bacterial infections (tetracyclines). No vaccine is available. Optimize environment, management

Salmonella

Clinical disease due to *Salmonella* is seen rarely in pigs in Australia. Overseas, *Salmonella* septicemia is usually seen in pigs aged 6 weeks to 5 months old. Occasionally sucking pigs will become septicemic or pregnant sows will abort due to infection with *Salmonella choleraesuis*.

Clinical signs include a shallow wet cough, acute death, cyanosis of extremities and abdomen and fever.

Lesions include: lungs are firm and diffusely congested with interlobular edema and hemorrhage, splenomegaly, hepatomegaly, icterus, mesenteric lymphadenopathy, focal hepatic necrosis, renal petechiation, typhocolitis, ulcerative colitis, rectal strictures

Diagnosis is made on primary culture (Brilliant Green agar).

Treatment/Prevention: Cull affected animals if only a small number are affected; Reduce stress; Antimicrobials based on susceptibility results.

DISEASES/CONDITIONS OF THE CENTRAL NERVOUS SYSTEM

Hypoglycemia

Hypoglycemia is common in neonates who fail to suckle. Weak pigs at birth or pigs that become hypothermic due to inadequate environmental temperature do not nurse regularly. Early signs include nervousness, tremors, irritability or vocalization which progress to lethargy and convulsions.

Streptococcus suis

Streptococcus suis is common worldwide and has gained recent popularity as the bacterial pathogen able to evade medicated and segregated early weaning systems. Streptococcosis is a problem that many producers and veterinarians encounter despite advances in swine management and production systems.

It is a relatively common disease in pig herds in Australia. It may occur sporadically, and affect 1-2 pigs in a group, or it may occur as epidemics (eg. In early weaned herds of pigs).

Streptococcus suis has been reported to be an opportunistic pathogen in many animals and is zoonotic.

Epidemiology/Transmission

Streptococcus suis are currently classified according to capsular type. There are 35 serotypes+ of *S. suis*. Serotype 2 is considered the most common serotype of diseased pigs worldwide; however, many serotypes are capable of causing disease. Heterogeneous subtypes within these serotypes have been identified using genomic fingerprinting.

The epidemiology of streptococcosis is unclear at best. Both pathogenic and nonpathogenic strains of *S. suis* are subclinically carried in the tonsils and nasal passages of healthy pigs. Pigs become colonized with *S. suis* during or shortly after birth. During birth, pigs can be colonized when they contact or swallow *S. suis* infected vaginal secretions of their dam. Sows continually shed *S. suis* in their saliva, nasal secretions, vaginal secretions, and feces. Additionally, *S. suis* can be isolated from the underline of the sow. These sources provide ample opportunity for pigs to become colonized with the multiple types of *S. suis* carried by the sow. It is thought that up to 75% of all pigs are carriers of *S. suis* type 2; carrier rates approach 100% when all types of *S. suis* are included in estimates. Once in the nursery, *S. suis* appears to spread horizontally among pigs. Direct contact between infected nursery pigs appears to be the primary mechanism of post-weaning transmission; however, rodents and flies can also be a source of infection.

Although *S. suis* type 2 has been cultured from stillborn fetuses to adult swine, and clinical streptococcosis has been reported in all ages of pigs, most cases of septicemia and meningitis caused by *S. suis* occur in these recently weaned nursery pigs. Presumably, the pig succumbs to clinical disease as passive immunity declines and the pig becomes susceptible to those types of *S. suis* that are not its "normal flora. Concurrent infections, management and environmental stressors, can all predispose pigs to streptococcosis.

Pathogenesis:

Little is known about the pathogenesis of streptococcosis. There is strong evidence that the organisms are engulfed by and carried to the CSF compartment within or on monocytes. The ensuing inflammatory response results in increased pressure in the CSF compartment leading to

clinical meningitis and forcing the organism into the inner ear, nose and eyes. However, there is additional evidence that most *S. suis* are not engulfed by macrophages, and the role that these extracellular *S. suis* play, if any, in disease is unknown.

Potential virulence factors for *S. suis* such as hemolysins, hemagglutination factors, and proteins have been described although their roles remain unclear. The extent of pathogenicity is dependent on a variety of characteristics of both the bacterium and the host. Pathogenicity likely results from a combination of the virulence factors as no single factor has been a consistent predictor of virulence to date. The presence of a polysaccharide capsule appears to be necessary for pathogenicity, but is not sufficient to cause disease in and of itself. A practical method to identify meningitic strains of *S. suis* relies on isolation of *S. suis* in pure culture from the CSF of pigs demonstrating CNS signs.

Clinical signs:

Many capsular types of *S. suis* cause outbreaks of septicemia in pigs of all ages resulting in meningitis, polyarthritis, endocarditis, polyserositis, and/or pneumonia. Additionally, *S. suis* is a common opportunistic pathogen of the lung secondary to infections with *Mycoplasma hyopneumoniae* in high health status pigs. Many of these pigs appear as the typical post weaning "poor doers" or "starveouts".



Diagnosis:

Presumptive diagnosis of streptococcosis is based on clinical signs. Gross and microscopic lesions of meningitis and septicemia caused by *S. suis* are not pathognomonic. Diagnosis is based on cultural and biochemical features and confirmed with serotyping. Because *S. suis* is a commensal of the nasal mucosa, tonsil, lung, vaginal canal, etc., swabs of these areas are not good samples. Sample collection targeting affected organs is critical for a diagnosis. Isolation of the virulent strain is important. Cerebrospinal fluid is the sample of choice for cases of meningitis. Two methods have been used to aseptically collect CSF samples:

1. A vacutainer needle is inserted into the exposed C1-2 intervertebral space to collect the CSF sample while the euthanized pig is positioned in sternal recumbancy.
2. The pig is positioned in dorsal recumbancy. The head is extended and the pluck is removed. A needle is inserted ventrally between the occipital condyles to collect CSF.

Treatment:

Prompt recognition of the early clinical signs of streptococcal meningitis followed by immediate parenteral treatment of affected pigs with an appropriate antibiotic. Most outbreaks of *S. suis* meningitis occur in the first 14 days post-weaning, therefore, careful monitoring of pigs during

this critical time period is recommended. Pigs in the early stages of meningitis may be difficult to detect. Groups of pigs should be checked 2-3 times daily to maximize identification of affected pigs. The nursery area should be entered quietly to minimize pig disturbance. Affected pigs may hold their ears back, squint their eyes, or exhibit dog sitting.

The first line of treatment should be Amoxicillin + an anti-inflammatory (Dexamethasone/Flunixin) IM. However, if the animals fail to respond, antimicrobial susceptibility testing of *S. suis* isolated from CSF should be undertaken.

Although widely implemented, mass antimicrobial treatment of groups of pigs via feed or water in many cases is not efficacious for prevention of *S. suis* meningitis. Bioavailability (feed may interfere with absorption), route of administration (feed, water), competition for vehicle of administration (overcrowded pens), and serum concentration needed to kill *S. suis* should all be considered prior to prophylactic antimicrobial treatment for *S. suis*. Mass treatment should be performed with caution as it may select for strains resistant to available antibiotics lessening the chance for successful treatment in subsequent outbreaks.

Prevention:

As with other pathogens, optimization of management and environment are essential to control clinical disease. Temperature fluctuation, high humidity, crowding, high levels of pit gases, mixing pigs greater than 2 weeks apart in age, and commingling pigs from different sources can all increase the risk of streptococcosis. Cleaning and disinfection of the environment will reduce the numbers of *S. suis* to which the pigs are exposed. *Streptococcus suis* is easily killed by common disinfectants such as phenol, quaternary ammonium, chlorhexidine, and bleach. Streptococcosis may be minimized by controlling primary pathogens such as *Mycoplasma*.

All medicated early wean (MEW) and segregated early wean (SEW) protocols have failed to eliminate *S. suis*. Failure of these protocols to eliminate *S. suis* is most likely due to colonization of pigs during birth or the first few hours of life, and the inability of therapy using antibiotics available in the United States to eliminate *S. suis* colonization of tonsils.

The effectiveness of vaccination in controlling *S. suis* infection is equivocal. Commercial and autogenous *S. suis* bacterins are available overseas and appear not to be crossprotective against heterologous serotypes. It appears that they temporarily control the capsular type(s) contained in the bacterin, and as the prevalence of these types decreases, other types carried by the pig increase in prevalence. For this reason it is important to:

- differentiate the strain(s) of *S. suis* causing meningitis in the herd from commensal strains of *S. suis* carried by the pig; and,
- periodically culture cerebrospinal fluid from pigs with meningitis to ensure that the strain(s) of *S. suis* causing meningitis in the herd have not changed.

Live attenuated *S. suis* vaccines remain in the experimental phase. Extreme caution must be taken when handling live virulent cultures of *S. suis* as the organism is zoonotic agent.

Haemophilus parasuis

(see under “Respiratory Diseases”)

Oedema disease

Oedema disease is caused by an F18 fimbrial variant of *Escherichia coli* that produces SLT-I ie, a Shiga like toxin

Pathogenesis:

Escherichia coli colonize the small intestine and release the SLT-IIe toxin systemically which results in degenerative angiopathy.

Clinical signs:

Postweaning disease - usually within 10 days of weaning; Sudden deaths of good looking pigs; Dull, blind, head pressing, incoordination and loss of balance, lateral recumbency, paddling, coma, death; Edema of eyelids, nose and ears; Squeaky voice

Lesions:

Gross lesions can include edema of face and eyelids (usually observed first), submucosa of stomach, gall bladder, spiral colon, lymph nodes, around kidneys, in the larynx, and lungs. Microscopic lesions observed in the brain can include fibrinoid necrosis of arteries and arterioles, edema, perivascular cuffing, occasional thromboses. Encephalomalacia and cyst formation can be observed.

Diagnosis:

Isolation and typing of *E. coli* and toxin

Prevention:

Phase-feeding; Autogenous bacterins

Salt Poisoning/ Water deprivation

Salt Poisoning/ Water deprivation is caused by excess dietary salt or sudden water deprivation. The condition is commonly caused overseas by salt poisoning in pot-bellied pigs fed dog food, pretzels or other salty foods. The condition is usually the result of water deprivation in pork production units when a water line is turned off and forgotten or when a waterer becomes blocked.

Pathogenesis:

Wrongly formulated diet or loss of water supply/Salted fish, brine, buttermilk fed with limited access to clean water (OS)>> Considered that brain tissues dehydrate and sodium content increases - inhibits anaerobic glycolysis>> Water moves out of intracellular spaces due to the hyperosmolarity of the extracellular fluid. Neurons are more resistant to cell shrinkage because they can increase Na⁺, Cl⁻, and K⁺ ions. Neurons also generate "idiogenic osmoles" to minimize cell shrinkage. Sudden rehydration may worsen condition because sodium will exit the cells quickly, but idiogenic molecules remain causing hyperosmotic intracellular fluid. This causes cellular volume to expand, resulting in cerebral edema.

Clinical signs:

These are observed after water has been suddenly restored.

Peracute: prostration, running movements, coma and death

Acute (following restricted water intake): twitching, pruritis, thirst and constipation, blindness, circling, head pressing and inappetance follow 1-5 days later

Lesions:

Congested meninges (grossly), Microscopic - meningoencephalitis with edema and perivascular cuffing by eosinophils in cerebral cortex. Later encephalomalacia - cysts

Diagnosis:

History and clinical findings, "Pathognomonic" lesion in brain - eosinophils around blood vessels, Salt levels in brain (0.18 to 0.19% DM)

Treatment:

Re-introduce water supply gradually (Use a sprinkler), Supportive treatment of convulsions using sedation (eg SC Phenobarb); Restrict feed intake, Dexamethasone injections can be helpful if only a few pigs are affected, but are not practical if large numbers of pigs are affected.

Prevention:

Ensure adequate water supply; ensure pigs can use nipples before weaning into nursery with nipple drinkers; Provide free access clean water, especially if rations have a high content of salt.

LOCOMOTORY DISEASES OF SWINE

Diagnostic methods:

- History (records)
- Evaluation of pigs on farm (by group/location or by age)
- Walk through observing all pigs (buildings, rooms, pens)
- Evaluation of individual pigs (observe, hands-on)
- Consider environment (especially floors)
- Consider nutrition (quality control)
- Laboratory diagnosis (post-mortem)
 - Adequate numbers of pigs should be provided
 - Untreated, clinically affected pigs, (i.e. soon after onset of signs)
 - History and preliminary findings should also be provided

Abrasions/ trauma

Superficial injury or secondary infection in deeper tissues, resulting in cellulitis and/or osteomyelitis.

Clinical signs: Lameness involving one or more limbs; Piglets unwilling to stand; If pelvic limbs affected, pigs may try to balance and walk on thoracic limbs.

Diagnosis is based on observing wounds on wall, sole or heel of hoof, swollen coronary band.

Prevention is based on examining, repairing (sharp edges on galvanized woven wire, sharp slat edges, wide grooves in expanded metal, rough concrete, damp floors) and improving (epoxy paint, paper, carpet, rubber mats, straw) the floors.

Treatment relies on topical and/or parenteral antibiotic - must be used early in pathogenesis of lesions. Also treat the animal with an anti-inflammatory.

Porcine Stress Syndrome (PSS)

PSS is an autosomal dominant trait with variable penetrance and expressivity. It results in stress susceptibility.

Pathogenesis:

Stress → excess response with epinephrine → rapid muscle glycogenolysis, ATP breakdown and excess formation of muscle lactate → high metabolic rate → increased muscle temperature → Lactate acidosis + elevated temperature → death

Clinical signs:

- Muscle and tail tremor at outset
- Acute lameness and collapse
- Muscle rigidity
- Reddened skin
- Hyperthermia Temp. 107-113 degrees F.
- Shock → death

Lesions:

Rapid development of rigor mortis. Pale, soft, "watery" musculature – due to denaturation of muscle protein.

Diagnosis:

Based on clinical signs and a blood test for presence of stress gene.

Prevention & Treatment:

- Rest; Reduce temperature rapidly; use hose with cold water; Tranquilizer, hydrocortisone and bicarbonate
- Avoid mixing stress-susceptible animals
- Market animals on dry, cool days and withdraw feed for 12-24 hours beforehand
- Genetic selection: Tests incoming animals for stress gene

Splayleg in piglets

The aetiology of this condition is unclear. Possible causes include:

- Hereditary - European Landrace, Large White
- Choline deficiency
- Slippery floors – trauma
- Zearalenone in sow food

The pathogenesis is basically myofibrillar hypoplasia and muscle immaturity.

The condition is seen in newborn piglets - at or within hours of birth. Affected pigs show weakness of hind limbs and occasionally forelimbs; some are unable to stand; sit with limbs extended, although flexion is possible. This can result in traumatic skin lesions -> arthritis; traumatized muscle may exacerbate problem. Affected pigs may recover within days or up to 1 week if able to feed

Diagnosis is based on clinical signs - 1 -> 4 piglets affected/litter - sometimes whole litter

Myofibrillar hypoplasia on histologic preparations

Prevention:

Breeding selection?; Tape/hobble affected limbs - remember to remove after a few days; Improve floor to reduce slipping eg. straw, epoxy paint with filler, carpet

Neonatal polyarthritis

Neonatal polyarthritis occurs when "environmental" organisms gain entry through damaged tissue: Omphalitis (navel-ill), Knee or other limb abrasions, Tail docking, Ear-notch or castration wounds, Necrotic stomatitis /gingivitis following teeth clipping, Facial lacerations, Exudative epidemitis

As a result, a mix of environmental bacteria (Streptococci, Staphylococci, Escherichia coli and Haemophilus) enter the wound.

Clinical signs include: lameness affecting one or more limbs, swollen, painful joints, hard, swollen umbilicus, fever and lethargy



Diagnosis is based on history and clinical signs, necropsy and culture. It is important to consider reasons for entry of organisms (environment & hygiene).

Prevention requires: Determining the cause, improve flooring if needed, improving hygiene (e.g. environment, during processing, antiseptic/antibacterial on umbilicus at birth)

Treatment: Antibiotics are rarely effective if more than one joint is affected. Use antibacterials based on sensitivity profiles + Dexamethasone to reduce inflammation.

Mycoplasma hyorhinis

M. hyorhinis is an uncommon cause of polyarthritis in Australia. Where it occurs, it causes low to moderate morbidity and very low mortality.

The bacteria is transmitted from the oro-nasopharynx of the sow to piglet, and from carrier pigs to naïve younger pigs.

Disease occurs after local damage to respiratory epithelium -> septicaemia -> polyserositis/polyarthritis.

Clinical signs in affected pigs include: fever (39C+), lameness and abdominal discomfort with polyserositis.

Diagnosis:

Necropsy - polyserositis, fibrinous exudate with mononuclear leukocytes and some neutrophils & Culture organism

Treatment:

Tylosin/Lincomycin/Tiamulin + anti-inflammatory

Prevention:

Reduce stress at weaning, improve general hygiene if necessary, herds free of *M. hyorhinis*

Vitamin E/ Selenium Deficiency

Selenium and Vitamin E reduce levels of peroxide in tissues to prevent excessive lipid peroxidation of cell membranes, etc. Deficiencies can occur in conditions where: (1) there is an absolute Se/Vit E deficiency, (2) where there is Vitamin oxidation in rancid food and/or (3) rapidly growing pigs exceed average requirements in the diet and become deficient. This disease is most common in weaner pigs.

Affected pigs may succumb to a number of conditions including Mulberry heart disease (most common) and Hepatosis dietetica. Pigs with Mulberry heart disease die suddenly (usually "good pigs found dead"). This can occur quite commonly in pig herds in Australia.

Lesions include Myodegeneration of skeletal and cardiac muscle. This may not be visible grossly, hence use histologic evaluation may be required.

Diagnosis:

- History, clinical signs, and characteristic lesions
- Unexpected deaths in best pigs
- Heparinized blood for glutathione peroxidase levels
- Histology - coagulative necrosis of muscle fibers

Prevention:

- Se/Vitamin E in unthrifty animals with marginal deficiency probably at cellular level. Schering Bo-Se, 1 ml/40 lbs SQ or IM (sodium selenite, 0.55 mg = 0.25 mg Se; Vit E, 50 mg = 68 I.U.)
- Selenium supplement in diet - 0.3ppm Se (sodium selenite)

Mycoplasma hyosynoviae arthritis

This disease is most commonly seen in weaner & grower pigs, but may affect gilts. It causes low to moderate morbidity and very low mortality.

Pigs acquire the bacteria from the "Carrier" sow (tonsil). The organism is found in the snouts from 60-70% of pigs at slaughter. Six-8 week-old weaner pigs become infected with waning colostral antibody. Early mild infection -> immunity. Colonizes tonsil -> septicaemia -> localizes in lymphoid tissue and synovial membranes

Clinical signs include:

- Painful, swollen joints, but usually afebrile
- Acute or subacute lameness lasting up to 10 days in 10-20 week old animals
- Excess synovial fluid may distend joint

Lesions:

- Change limited to synovium, most often stifle, yellow and velvety
- Excess yellow joint fluid - may have fibrin flakes
- Articular surfaces and periarticular tissues normal

Diagnosis:

- Age of onset
- Afebrile and lacking polyserositis
- Lesions restricted to synovium at necropsy
- Culture organism (only likely for up to 3-4 days after onset)
- Response to treatment with antibiotics

Treatment:

Tylosin / Lincomycin/ Tiamulin + Corticosteroid /Flunixin IM

Osteochondrosis and Osteoarthritis (Degenerative Joint Disease)

DJD is a generalized condition with predilection sites, e.g. AECC of medial humeral and femoral condyles, and growth plates of costochondral junctions, distal parts of ulnae, and ischial tuberosities.

The true aetiology of DJD is unknown, however it is thought to be multifactorial: genetics (muscular, heavy pigs), nutrition- quantity vs. quality, environment and management. It is associated with fast growth, and clinical signs occur from 4 months of age. Alternatively, it may be related to absolute body weight (it affects pigs > 100kg). DJD can result in high cull rates (34-100%) in breeding pigs.

Pathogenesis

Osteochondrosis is a dyschondroplasia that results in disturbed endochondral ossification in the physal component of growth cartilages. Hypertrophied chondrocytes persist in the metaphysis. Osteochondrosis is a generalized condition so that many sites may be affected and a single growth cartilage may have one or more focal lesions. Cartilage flaps, or areas with loss of cartilage result in exposure of subchondral bone to the joint cavity and, at this time, pain and lameness are considered to develop.

Clinical signs:

- Moderate/severe lameness in 1 or more legs affected-“kneeling” walk or on tip toes
- Age of onset can be as early as 4 months. Onset may be insidious or sudden following trauma
- Chronic, progressive lameness - probably when osteoarthritis develops
- Involvement of synovial joints of vertebrae -> kyphosis

Lesions:

Radiolucency and sclerosis, Joint cartilage had folds and flaps, craters expose bone, Excess synovial fluid, Villous proliferation, Joint “mice” ossified, Osteophytes

Diagnosis:

Clinical signs; Post-mortem lesions

Prevention:

None since cause unknown; Good floor surfaces - bedding or dirt lots

Treatment:

Rest & anti-inflammatories

Rickets and Osteomalacia

Aetiology:

Rare today on commercial farms due to well-formulated diets. May be due to absolute Ca, P deficiencies, imbalanced ratio of Ca:P, feed mixing issues and/or cost cutting.

Clinical signs:

Rickets: Anorexia and unthriftiness, stunted growth by 10 weeks of age, pigs unwilling or unable to stand-will sit, enlarged joints, skull may seem disproportionately large, bowed, truncated limbs, possibly fractured long bones, ribs or vertebrae (paresis or paralysis likely)

Osteomalacia: lameness or inability to stand late in pregnancy, during lactation or at weaning - often seen when sows are in crates or when they are removed from crates at weaning

Lesions:

Rachitic lesions: Enlarged costochondral junctions (Rachitic rosary) and thickened, hemorrhagic growth cartilages at ends of long bones (seen on slabs of bone), pliable ribs –some cut with a knife, bones poorly mineralized, fractures and/or calluses

Osteomalacic lesions: Classically, poorly mineralized bone (Osteoporosis is a decreased amount of bone), fractures frequently seen - femoral neck & shaft, humerus or vertebrae, secondary osteomyelitis at fracture site

Prevention:

- Provide adequate diet - Ca, P and Vit D in adequate amounts and suitable proportions during the grower/finisher phase for adequate skeletal development and during lactation
- Monitor home-mixed rations - difficult if batches are mixed frequently, but the problem may develop only if there is repeated error in mixing
- ? Adequate exercise

Treatment:

Provide diet as above; Parenteral Vit. D

SKIN DISEASES

Erysipelas

Aetiology:

Erysipelothrix rhusiopathiae – a Gram positive rod, 28 serotypes

Epidemiology/Transmission:

- Occurs widely in pig herds in Australia.
- Worldwide distribution-Sheep, birds, reptiles, fish, man
- Sources of infection are primarily healthy carrier pigs but can come from contaminated water, contaminated feed, feces, rodents, feral and domestic animals, and organisms in soil (survives up to 35 days depending on temperature, pH, moisture).
- Most common in pigs 3 months to 3 years old
- 30-50% of healthy swine are carriers

Pathogenesis:

Route of infection - oral, wound, flybite -> systemic

Clinical signs:

- Acute form
 - Pigs are found dead
 - Fever (40C+), septicemic, lethargy, inappetant, painful joints, chilling
 - Cyanosis associated with capillary congestion, especially ears, abdomen, legs
 - "Diamonds" develop on skin over next 2-3 days
 - Abortion
 - Sloughing of skin on the extremities
 - Chronic lameness
- Subacute form
 - Less severe signs, animals not as sick, fevers not as high, still eating, mild skin lesions- can be overlooked, may go unnoticed
- Chronic form
 - Endemic or may follow acute form
 - Chronic arthritis frequently involves carpus and hock, associated with swollen joints - periarticular fibrosis and exostoses - intermittent lameness multifocal areas of skin necrosis
 - Cardiac insufficiency
 - Swollen, stiff joints 3 weeks after infection

Lesions:

- Acute form
 - Arthritis with increased fluid – early, Diamond shaped hyperemic skin lesions - vascular degeneration, Multiple petechiae and ecchymoses - vascular degeneration, Swollen hyperemic lymph nodes, Renal hemorrhages-tubular degeneration, Muscle degeneration,

Hemorrhagic gastritis, Congested and enlarged spleen, Petechiae in renal cortex, Enlarged congested lymph nodes

→ Chronic form

Arthritis - synovitis with hyperemia, villous hyperplasia, Fibrous proliferation around joint, Diamond skin lesions - arteritis -> focal necrosis and slough, Sloughed ears and tail, Vegetative endocarditis, Valvular endocarditis



Diagnosis:

- Culture organism (blood, spleen, liver of untreated animals, can be found in some chronically affected joints; can culture skin if animal was recently treated)
- Response to penicillin therapy (24 hours if treated in acute phase)
- Morbidity and mortality variable - few deaths, some febrile, some lame, depressed, inappetent
- "Diamond" skin lesions indicate septicemia
- Differential diagnosis should include any bacterial septicemia
- **Treatment:**
- Penicillin (to affected pigs and those in-contact)
- Chronic cases cannot be treated satisfactorily as lesions are "irreversible" in and around joints.

Prevention:

Vaccination in Australia with killed organism bacterins. Attenuated live organism vaccines are available overseas. Vaccination provides 2-6months of immunity

Greasy pig disease (*Exudative epidermitis*)

Aetiology:

Staphylococcus hyicus, usually infection is observed in gilt litters- prevalence of clinical disease decreases as the average parity of the breeding herd increases. Can see lesions after pigs are moved into the nursery and have fight wounds.

Pathogenesis:

Staphylococcus hyicus is a normal inhabitant of skin and enters through puncture wounds such as bites or injection sites. Once in the puncture wound it releases a toxin

Clinical signs:

Peracute form: skin lesions-extensive cutaneous vesicles --> ooze --> crust; coronary band lesions --> loss of hoof wall; death in 3 to 5 days

Acute form: scab formation, depression, anorexia, dehydration, emaciation, death in 4 to 8 days

Subacute/chronic form: slower development of lesions, lesions resolve, reduced growth rate

Pruritis is not typical of this disease

Diagnosis:

Age of the pig. Clinical signs. Culture *Staph hyicus* from lesions in heavy growth. Rule out other conditions.

Lesions:

Renal damage, bacteria, mucus and epithelial cells block ureters



Prevention:

Proper hygiene during processing, improved farrowing house hygiene

Treatment:

Individual pigs

- Inject with penicillin, amoxycillin or trimethoprim-sulphar as soon as the first reddened lesions appear (the size of mosquito bites) + Inject with an antiinflammatory such as dexamethasone to control spread of toxin
- Do not use needle to inject clinically healthy pigs as you can spread the disease by injection with a contaminated needle

- Topical application of moisturizer (eg udder cream) to prevent dehydration. Topical application of disinfectant (eg. Chlorhexidine) to reduce infection.

Sarcoptic mange

Aetiology:

Sarcoptes scabiei var suis

Pathogenesis:

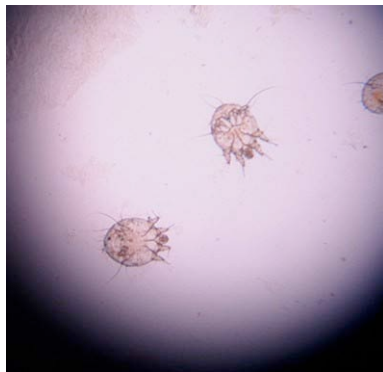
Mites acquired from sow by piglet; pig to pig transmission; mites invade epidermis and cause papules/vesicles

Clinical signs:

Pruritis (head shaking, body scratching); chronic, pruritic dermatitis in older pigs (thickened skin, dark brown/gray encrustations in ear canal); unthriftiness, considered to reduce growth rate

Diagnosis:

Collect ear wax from pigs using a melon baler or equivalent. Examine for mites under a microscope using mineral oil as a carrier or use NaOH/KOH to corrode debris. Observation of mites is diagnostic, but you cannot rule out mange if you do not see mites. Skin scrapings are useless as mites live in the ears. Skin biopsy if no mites for histo. Rule out insect bites/bedding allergies.



Treatment:

- Injections: Dormectin (1-2 doses) or Ivermectin (2 doses, 18-21 days apart)
- In-feed medication with ivermectin
- Topical: Amitraz, Phosmet etc sprays and pour-ons

Prevention:

- Mange eradication program throughout herd
- Dormectin (1-2 doses)
- Ivermectin (2 doses, 18-21 days apart) or can add to feed.
- Treat all incoming stock in isolation

Pediculosis - louse infestation

Aetiology:

Hematopinus suis (4 to 6 mm in length); host specific; only survives 2-3 days off host

Pathogenesis:

Spreads by pig to pig contact. Louse damages the skin and sucks blood causing irritation and self-inflicted trauma

Clinical signs:

Pruritis (DDx second hand cigarette smoke can cause pruritis in pigs); Nits (eggs) are attached to hair and adults can be visualized directly

Lesions:

Skin folds of neck, jowls, inside leg, flank, and base of ears

Treatment:

Dormectin (1-2 doses) or Ivermectin (2 doses, 18-21 days apart); Combination of above with in-feed medication with ivermectin

Prevention:

As for mange

*Note - *Hematopinus suis* transmits swine pox virus and *Eperythrozoon suis*

Swine Pox

Aetiology:

Swine pox virus; rare in Australia

Pathogenesis:

- pox virus -->direct contact -->skin injury; virus survives up to 1 year in skin debris
- spread by lice
- infection--> lesions--> immunity in 3 wk
- high morbidity, low mortality

Clinical signs:

Secondary infections may cause problems; no pruritis

Lesions:

Papules --> pustules --> scabs

Diagnosis:

Immunofluorescence, Electron microscopy, Differential diagnosis is insect bites

Prevention:

Self limiting, avoid introducing infected animals, eradicate lice

Pityriasis Rosea

Aetiology:

Unknown, relatively common in Australia

Pathogenesis:

Red spots at 5-12 weeks - spontaneous recovery in 2-3 weeks

Lesions:

- small red spots --> coalesce --> nodules
- depressed center with brown scab
- centrifugal spread of lesion --> irregular reddened edge (like a coastline on a map) - may involve much of the ventrum and lower parts of sides of body
- early lesions may resemble ringworm

This syndrome is benign and self-limiting



Ringworm (Microsporum nanum)

Differential diagnosis for pityriasis rosea

Diagnosis based on demonstration of fungal spores on hairs and scrapings from lesions.

Usually self-limiting. Can try iodine sprays.



TOXICITIES

Mycotoxins

Due to moldy grain (>16% moisture).

- Aflatoxins: *Aspergillus flavus*- Causes hepatitis...gross haemorrhage...liver swollen, friable, yellow/tan. Clinically-inappetance & poor growth. Treatment-remove feed.
- Tricothecenes: *Fusarium* spp (deoxynivalenol/DON or vomitoxin). Results in inappetance & vomiting.
- Zearalenone: Is luteotropic & results in oestrogenic activity. Can cause reproductive disorders (see under “Reproduction”)

Prevent mold by cleaning silos before filling, removing leftover feed from walls, store feed at moisture content < 16%, keep moisture low via agitation/aeration, use preservatives or air-free

Olaquinox

Used as a growth promotant and for control of proliferative enteritis and SD. At inclusion rates of >100ppm can result in reproductive failure.

Procaine Penicillin

Toxicity occurs when the product is stored in the heat. The Procaine dissociates from the Penicillin. Pregnant animals can abort if treated with the dissociated product.

Skin scald

Scalding of the skin can occur as a result of using high levels of Lincomycin and some other antibiotics in feed. This results in “scalding” around the perineum. Can also extend to other parts of the body if pigs roll in their faeces. Treatment is to remove the antibiotic, remove the faeces and hose down the animal.



Salinomycin/Monensin

Toxicity can occur when these products are administered to pigs in combination with Tiamulin and the combined dose exceeds 6mg/kg. Poisoned pigs show the following signs within 24 hours:

Mild toxicity: inappetence, often accompanied by vomiting. Severe Toxicity: open-mouthed breathing, frothing from the mouth, muscle weakness and shaking, inco-ordination or hind limb paralysis, death

Arsenic

Organic arsenicals are used as growth promotants and in the past to control SD. Toxic levels @ 250ppm or when there is water deprivation can result in blindness/ataxia/progressive paresis.

Diagnosis

History (feed assay), clinical signs, histo-peripheral nerve pathology.

Treatment

Remove arsenicals from feed & ensure adequate water

Selenium

Toxic levels @ 5ppm+...anorexia, alopecia, separation of hooves at the coronary band, liver/kidney degeneration...paralysis

Organophosphates

Used to treat ectoparasites. Toxicity results in parasympathetic NS stimulation....salivation, defecation, urination, vomiting, stiff gait, uneasiness...gastrointestinal hypermotility, colic dyspnea, cyanosis, muscle tremors.

Diagnosis:

Clinical signs, history, OPs in stomach contents, chilled blood/brain for cholinesterase activity.

Treatment

Atropine sulphate 0.5mg/kg, wash pig

Hygromycin

Anthelmintic-forms cataracts in sows if feed for extended periods. Recommended to feed for 8 weeks only.

PROCESSING & MARKETING

Pig producers are basically paid on a grid of carcass weight and P2 backfat (measured at the last rib, about 6.5cm from the midline). An average carcass weighs about 75kg (75% of 100kg liveweight) and has a P2 of 13mm. In the future changing consumer attitudes may mean more focus on food safety and quality assurance. This may create opportunities for the pork industry to clearly define and segment the market to achieve price differentiation with the sale of free range, organic, vegetable fed, antibiotic residue-free pork.

CONSISTENCY OF SUPPLY

It is important that the type of pig meat produced consistently meets the requirements of its customers. The general requirements of meat is that it looks good, tastes good and is tender and juicy. Inconsistency occurs mainly due to the effects of Pale, Soft, Exudative (PSE) meat, Dry, Firm, Dark (DFD) meat, variations in weight and variations in fat content. It is important that the type of pig meat produced is targeted at the right market. For example, meat exported to Asian countries must not be from entire males due to the risk of boar taint. Asian countries also do not permit the importation of meat from chemically-castrated pigs. Hence, our market for export to these countries is restricted to female pigs or surgically castrated male pigs. In addition, the weight at which we grow our pigs and the backfat specifications will depend on what part of the pig we are sending where. For example, once a month, local supermarkets tend to have a week where one type of meat is discounted (“Woolies specials”). Pig producers supplying these supermarkets may take advantage of this and sell their lightweight pigs during this week.

QUALITY CONSIDERATIONS include:

To optimize pig meat quality and to provide our consumers with some guarantee of pork quality, the Australian pig industry has developed its own on-farm Quality Assurance Program (APIQ). This program is developed by the producer, with help from Australia Pork Ltd (APL). Farms undergo a series of internal audits before an external audit to be APIQ accredited. The program focuses on the following areas:

1. Foreign bodies
2. Residues
3. Eating quality (taste, tenderness, juiciness)
4. Meat safety
5. Welfare
6. Biosecurity

1. FOREIGN BODIES

These usually arise as a result of broken needles, but there have been other FB types (eg iron solder). Although the risk of these occurring is relatively low, the impact that it can have on the consumer would be high (imagine the ramifications if a 5-year-old got a needle through his cheek while eating a Big Mac!)

To minimize the risk of foreign bodies occurring in meat it is important to:

- Use best practice with needles (eg replace them when they are bent/burred/blunt)
- Report and record any broken needles

- Identify the affected animal
- Notify the abattoir
- The needle's location and pig ID are also reported
- If you cannot ID the animal in the pen or room, then the whole batch of pigs must be considered suspect and the abattoir notified

2. RESIDUES

Residues can be from antibiotics, analgesics, hormone treatments, heavy metals etc. They can arise from any form of treatment (parenteral, in-water, in-feed). The most common reasons for residues include:

- wrong withhold
- wrong drug (eg long-acting penicillin instead of short)
- wrong dose
- cross-contamination (especially from medicated feed to non-medicated feed)
- not identifying treated pigs
- not labeling silos

Prudent use guidelines are discussed in a later section of this booklet.

3. EATING QUALITY

Pale, soft exudative meat (PSE) develops as a result of the rapid drop of pH caused by rapid breakdown of glycogen post-slaughter while the carcass temperature is still high. The high lactic acid concentration in the meat while the muscle temperature is high changes the physical properties of the muscle proteins causing decreased water holding capacity and increased light scatter. PSE meat is visually less attractive than normal meat and loses considerable mass as exudates (drip loss) and cooking of fresh pork cuts. PSE meat also yields less processed meat during the manufacturing of hams and bacon.

The ultimate pH of meat 24 hours post-mortem is a good indicator of meat quality. Normal meat will have an ultimate pH of between 5.6 and 6. Such meat will have an acceptable reddish-brown colour, be firm to touch and lose comparatively less exudates than PSE meat. This is the ideal description of pig meat. Meat with an ultimate pH of less than 5.6 is PSE. It dries out more when cooked, lacks juiciness and absorbs less cure in the manufacturing process. PSE meat, either as fresh pork or manufactured product is less appealing to consumers than normal meat.

If the ultimate pH is above 6, the meat is described as DFD (dry, firm, dark). This is normally a result of depletion of muscle glycogen pre-slaughter. DFD meat will have a dark, less visually acceptable colour. It will be firm to touch and have less surface exudates than normal meat. The texture of DFD has been described as "rubbery" and less palatable than normal or PSE meat. The high ultimate pH of this meat leaves it more vulnerable to bacterial spoilage as a fresh product however resulting in lower shelf life expectancy than normal or PSE meat. DFD meat will yield more manufactured product from processing as a result of its drier nature and subsequent ability to absorb nor cure in the manufacturing process.

To maximize pork meat quality by reducing the incidence of PSE/DFD:

- Transport & slaughter times re arranged so that pigs are removed from feed at least 6 hours and nor more than 24 hours before slaughter
- Loading or transporting in extreme temperatures are avoided
- Shade is provided on the truck and during periods of high temperature
- Pigs that are significantly different in weight are separated from each other
- Stocking rates for transport meet industry standards
- The trip to the abattoir is by the most direct route

- Dogs, electric prodders are not used for loading or moving animals
- The loading facility is designed to minimize stress.
- Avoid the Halothane gene

Another component of taste is boar taint. This arises due to the presence of two components-androstenone (produced in the testes of mature boars) and skatole (a by-product of hind-gut fermentation). Boar taint may be largely eliminated by castration (surgical or chemical) or by selling female pigs.

4. FOOD SAFETY

The major pathogens of concern are Salmonella, Toxoplasma, Campylobacter and verotoxigenic E coli. Of these, most focus is on Salmonella control. Denmark was perhaps the first country to implement a National Salmonella surveillance and control program, based on ELISA testing of carcass juice. Farms are ranked according to their Salmonella levels and if considered a problem, must take on-farm quality control steps.

In Australia, the APIQ program seeks to minimize the risks of food poisoning by:

- Ensuring there is a program to control rodents (Salmonella minimization)
- Control of cats and their access to feed stores & bedding (Toxoplasmosis)
- Provide sick pens for pigs (minimize the risk of transfer of pathogens from sick to healthy pigs)
- Clean pens regularly between batches/groups to reduce faecal buildup.
- Clean trucks before pigs are collected.
- Remove pigs from feed at least 6 hours before slaughter to allow for gut emptying and minimize the risk of ingesta spillage and contamination of the carcass
- Slaughter within 24 hours of being off-feed.

5. WELFARE

There are a number of issues relating to pig rearing that are increasingly concerning consumers. The main ones include:

- individual housing (eg in stalls and farrowing crates)
- surgical interventions (teeth clipping, tail docking, ear notching, surgical castration)
- intensive housing (on concrete with no bedding)

A number of lobby groups (eg. Animals Australia) have a stance to out-law certain husbandry practices. The majority of pig producers would like to retain the status quo. Somewhere in the middle is the answer, but we should be aware that despite the lobbying, any change to current practice should be based on science and not opinion.

It is of interest that although consumers may demand more “welfare-friendly” pig rearing, they are seldom willing to pay more for the end product.

6. BIOSECURITY

The principal areas under APIQ regarding biosecurity are: compliance with swill feeding regulations, awareness of exotic diseases among piggery staff, recording of people, animal and transport movements, a controlled entrance with hand-washing facilities in place and farm boots/clothing provided for visitors.

TREATMENT OPTIONS

The Australian pork industry has access to a wide range of antibiotics to treat and prevent disease, and to enhance the growth of pigs. At present the use of antibiotics to promote growth is still legal. It is essential for the industry to be aware of the issues relating to the development of antibiotic resistance in the bacterial flora of pigs treated with antibiotics. The introduction of Hazard Analysis Critical Control Point (HACCP)-based quality assurance programs has highlighted the importance of establishing clear and open communication between producers, their suppliers of feed and veterinary chemicals, veterinarians, abattoirs, and HACCP facilitators and auditors.

Swine can be medicated via feed, water or parenterally depending on the medication/vaccination used.

INJECTION TECHNIQUES

Medication is injected into the muscle (intramuscular) of the neck or under the skin (subcutaneous), preferably behind the ear. The most effective and accurate treatment method is to inject sick pigs with the appropriate drug at the correct dose rate. When using this method of treatment care must be taken to avoid breaking needles. If this occurs and the needle remains in the animal then it **MUST** be identified and kept separate from other animals at the time of slaughter. In general the use of injections should be kept to a minimum.

Select proper size and length of needle:

Intramuscular Injection	Gauge	Length
Piglets & Weaners	18 – 22	1/2"
Growers & Finishers	16 – 18	1"
Adult animals	16 –	1" or 1 1/2"

Injections can also be given into the side of the vulva in female pigs to reduce the injected dose rate of the drug. Drugs injected in this manner are those targeting the uterus and ovaries (eg Lutalyse and Oxytocin). For intra-vulval injections, the injection is given externally in the crease of the vulva (muco-cutaneous junction) where it meets the buttocks and at about 30 degrees. Preferred needle gauge is 22G, with 1/2" length.

Oral:

Medication is placed into the mouth. This method is effective for suckers and weaners.

In-feed:

Medication is mixed into the feed. This is the easiest and most common but least effective method for treating sick pigs. It is used for disease prevention.

In-water:



Quiz. How much water does a pig drink as a ratio of what it eats?

Medication is added to the drinking water. It is a more effective method for treating large numbers of sick pigs than in-feed medication. Sick pigs drink about 20% less than healthy pigs so it is necessary to adjust the water medication rate to account for this. In fact by monitoring water intake closely it

might be possible to detect an outbreak of scours in advance. Individual sow water intakes vary from <2 litres per day to >40 litres per day. With such individual variation water medication may result in treatment failure simply as a result of the animal not receiving enough drug.

Pour-Ons:

Medication is placed on the skin, usually along the back-line (and in the ears for Sarcoptic mange). Use the site recommended by the product manufacturer or your veterinarian.

PRUDENT ANTIBIOTIC USE

Veterinarians, in consultation with their pork producer clients, must ensure that producers:

- use only approved (registered) agricultural and veterinary chemicals
- obtain medicines only from a veterinarian, pharmacy, or a licensed reseller/distributor
- store agricultural and veterinary chemicals in accordance with label instructions
- keep written records of the use of medicines prescribed by the veterinarian and follow label instructions and restrictions
- read the label carefully before administering the medicine
- Must observe WHP's and ESI's
- dispose of unused medicines in an environmentally responsible and safe manner according to label directions when treatment is finished or the expiry date has passed.

Veterinarians working in the pig industry need to comply with:

- the Code of Practice for the use of Schedule 4 substances (Prescription Animal Remedies) in the industry that has been prepared by the AAPV
- the Australian Pig Industry Quality Program (APIQ) standards
- the AAPV guidelines for the care of the sick and injured pigs

■ ***Remember:***

- ▶ Bacteria have the ability to adapt in multiple ways to decrease the efficacy of antibiotics.
- ▶ The driving force behind the emergence and spread of antibiotic resistance in bacterial populations is from selection pressure due to antibiotic overuse in human and veterinary medicine.
- ▶ Some antibiotics used in food animals are also used for human therapy.
- ▶ Antibiotics are important for animal health, welfare, and food safety and the environment. However, they need to be used judiciously to avoid the development of resistance in pathogenic bacteria of the pig. This resistance may be transferable to bacteria that infect humans. For this reason this practice (which practice???)while currently legal, is actively discouraged by APL.
- ▶ Therapeutic antibiotics should be used for as long as needed, for as short a time as possible, at the appropriate dosage and with the appropriate withholding period before marketing treated animals. This will help maintain their effectiveness on the farm.

Establish a herd health plan that includes:

- periodic herd health monitoring
- a review of genetic sources, their associated traits and the potential utilisation in your herd
- a professional review of your herd's nutritional program
- a review of space, temperature and other environmental considerations appropriate for the phase(s) of production in your operation

- a review of management protocols to ensure an appropriate level of biosecurity for your herd.
- An economic review which covers average daily gain and cost of production all of which are underpinned by the health and environment of the herd?

Maintain good general practices:

- ▶ Records should be kept regarding the administration of all antibiotics
- ▶ Veterinary supervision and coordination is essential for the appropriate use of antibiotics in animals
 - a valid veterinarian-client-patient relationship is essential
 - this means that the veterinarian prescribing the antibiotic has an active and on going professional relationship with the owner of the pigs.

Recording requirements:

Home-mixers:

- List of Approved Agricultural and Veterinary Chemicals supplied by the veterinarian.
- Home Mix Feed Manufacture records that include descriptions (records) of in-feed medications and bins used.
- In-Feed Medication Program. This form allows both veterinarian and producer to monitor use of in-feed medications.

Purchased feed users:

- List of Approved Agricultural and Veterinary Chemicals supplied by a veterinarian.
- A medicated feed order form.(this is incorrectly referred to as a script, only a pharmacist can fillout a prescription. What the feedmills have is an authority to supply S4 medicated feed to a farm. Non S4 medications do not require veterinary authority to be added by a feed mill.
- The use of veterinary authorisations applies only to prescription animal remedies (S4 products). (*This form is intended to aid the collection of information on all in-feed medications.*) Actual usage of in-feed medications will be recorded on the Feed Manufacture Form or other form as designated by the producer's veterinarian. (*Home mixers do not require a Medicated Feed Order (veterinary prescription) if the prescription animal remedy is purchased directly from the veterinarian.*)
- Delivery Docket with similar information, provided by the feed-miller at delivery
- Under the Health Act (which may vary between States) commercial feed manufacturers are required to keep full records for use of S4 medications. Feed manufactures record batch number, date of purchase and in-feed medication details as shown on the Feed Order Form for each medication used.
- **Biosecurity** - diseases can be spread between pig farms by pigs, people, rodents, feral animals, birds, domestic pets, vehicles and wind, depending on the pathogen involved.
- control measures can include perimeter fencing to prevent the entry of stray animals, isolation facilities for holding new breeding stock prior to entry into the breeding herd, and the provision of clothing for the use by visitors to the farm.

ALTERNATIVES TO ANTIBIOTICS

Vaccines

A major difference between antibiotics and vaccines is that:

- antibiotics are used to treat animals affected by bacterial diseases and as additives to the ration for growth promotion effects.
- vaccines are used to prevent bacterial and viral diseases and consequently play a large role in reducing disease.

Improved vaccines, increased usage of current vaccines and developing new vaccines for existing and emerging diseases will decrease the reliance on antibiotics.

Probiotics

A probiotic is a live microbial (bacteria or yeast) feed supplement that benefits the host animal by improving its intestinal microbial balance. The concept of probiotics is not new, but their use has been reduced by the availability of antibiotics to effectively control disease, and the total lack of any data to show that probiotics are efficacious or cost effective.

With the emergence of antibiotic resistant bacteria in pigs, probiotics are being considered again as alternatives for improving health and performance. While there are many conflicting statements made about the efficacy of probiotics in pigs, it has been demonstrated that with careful attention to the criteria for usage, they can improve piglet health and performance, particularly during stressful periods such as immediately after weaning.

When selecting a probiotic, the target organism must be identified and a probiotic strain selected which is biologically active against that target. The probiotic must also be able to colonise the gut tissues as well as remain metabolically active in situ. For example, certain strains of Lactobacillus are known to be and can prevent inhibit growth and adhesion of enteropathogenic E. coli K88, post-weaning piglet diarrhoea caused by this pathogen.(Really????) The aim of this approach would be to utilise a mixture of strains that provide broad-spectrum protection against digestive tract diseases.

Other antibiotic substitutes

- ▶ **Minerals** – Copper and Zinc have been used to stimulate growth.They are currently being used illegally by veterinarians at high levels (2000-3000ppm) to control E.coli in weaner pigs. Remember it is illegal to use, or prescribe for use, any chemical which is not registered in at least one food producing species. Quiz: What species of food producing animal has a curently registerd product for it with inclusion rates of 2000ppm.
- ▶ **Acidifiers are used in weaner feeds to prevent scours by preventing colonization by pathogen bacteria. They work by increasing the level of acid in the stomach and this prevents some bacteria from replicating within the stomach and subsequently passing into the lower digestive tract.**

Monitoring and residue testing

Residue testing regimes

The National Residue Survey (NRS) run by Agriculture, Fisheries and Forestry, Australia (AFFA) monitors food commodities for residues of antibiotics, pesticides, anti-parasitic and other therapeutic agents. Five hundred randomly chosen pig carcasses are tested each year.(this number is sufficient for

them to be sure at the 99% level that they will pick up any residues. This program is funded by a producer levy, and is a requirement of countries who purchase meat from us.

Once a specific residue problem has been detected that may have trade access implications, a targeting testing program can be implemented to better define the nature of the problem and minimise the risk of residues entering the food chain. In this situation it is likely that producers, abattoirs, processors, State agencies and the NRS would be involved.

? Quiz: what is the difference between an export slaughter interval (ESI) and a withhold period (WHP)? Why have both?

Duty of care

People handling and selling antibiotics and other agricultural and veterinary chemicals, have a *duty of care*: a responsibility to carry out their tasks in a safe and efficient manner so that they do not cause harm or injury to themselves, others, other property or the environment.

Adverse drug reactions

The National Registration Authority for Agricultural and Veterinary Chemicals (NRA) Now known as the Agricultural Pesticides and Veterinary Medicines Authority (APVMA) is the national agency responsible for regulating agricultural veterinary chemicals including antibiotics up to and including, the point of sale in Australia. It conducts an Adverse Experience Reporting Program (AERP) to provide a national mechanism for reporting, recording and analysing adverse experiences with veterinary chemical products, with a view to develop better use practices and prevent avoidable side-effects.

? Quiz: You visit a farm and notice that about 20% of sows have lumps on their neck that vary from grape to oranges in size. When you enquire of the farmer it turns out that he uses a particular brand of vaccine which we know causes this reaction. Do you have an obligation to report this to the AERP

VACCINES

There are a number of pig vaccines commercially available in Australia. Most have been mentioned elsewhere in this booklet in conjunction with the relevant disease. In general, most are killed bacterins (with the exception of Autovac and Enterisol Ileitis). Hence, they generally require 2 shots (usually 3-4 weeks apart to be effective. Peak antibody levels are not acquired until at least 2 weeks after the second shot. Having said this, there is currently one 1-shot vaccine (Respisure 1-shot, Pfizer) available to prevent *Mycoplasma pneumoniae*.

Quiz: How would a 1-shot vaccine work, assuming it is a killed vaccine?

Veterinarians also have the option of having autogenous vaccines made up that are specific to strains of pathogens present on their farm provided there is no commercial vaccine available to control the disease. This is particularly useful for diseases such as Glasser's disease due to *H. parasuis*.

The vaccines commonly used on pig farms in Australia are:

Breeders: Erysipelas, Leptospirosis, Parvovirus (gilts only), E coli

Progeny: (depending on herd health status): *Mycoplasma*, Pleuropneumonia, Erysipelas, *H parasuis*

ANTI-INFLAMMATORIES

The most common anti-inflammatories used in pigs in Australia are dexamethasone (eg Dexapent) Flunixin (eg Finadyne), Tolfenamic acid (Tolfedine CS) and Metacam. These are all administered IM. Common reasons for their use include anti-inflammatories for symptomatic treatment of greasy pig disease, meningitis, lameness (particularly breeding stock) and mastitis. Flunixin may be used as an anti-shock treatment to assist in pigs with pleuropneumonia.

DISEASE ERADICATION

Disease impacts significantly on herd performance by:

- reducing feed intake
- reducing growth rate
- reducing feed efficiency by directing protein toward tissue repair or the immune response
- increasing mortality rate and, in acute cases where herds are exposed to the disease for the first time, reducing reproductive performance and increasing mortality rates before weaning
- increased medication costs (typically around 10c/kg dressed)
- impact on staff morale

The impact of a disease will depend on the farm and the pressure that its environment puts on the pigs. In buildings that are well-ventilated and where hygiene and air quality standards are met, disease may have minimal impact. However, on other farms, disease may impact profit to the point where only eradication will allow sustainability.

It is important to remember that probably 99% of pig diseases enter a herd through the entry of an infected animal. The other 1% is made up of aerosol spread (eg. *Mycoplasma* can travel 2-3km), fomites (dirty boots and overalls) and vectors (eg mosquitoes can carry PRRS). Probably the best biosecurity is to ensure that there is a perimeter fence around the farm and that all visitors are required to wear clean boots and overalls.

The targeted pathogens for biosecurity and/or disease eradication programs in Australia are *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, *Brachyspira hyodysenteriae* and *Sarcoptes scabiei*. Pathogens that cannot be eradicated (yet!) include *Lawsonia intracellularis*, *Erysipelothrix*, *Haemophilus*, *E coli* and *Streptococcus suis*.

DEPOPULATION-REPOPULATION

This involves the complete removal of infected animals from the site, followed by repopulation with healthy animals. Between the depopulation and repopulation phase the site has to be empty of pigs for a period appropriate for the diseases being eradicated.

Considerations for a depop-repop:

- The proximity to other piggeries-if poor health status herds are close by then the risk of re-infection post-eradication is high.
- Ensure the health status of incoming pigs is good.
- The cash flow budgets for depopulation and repopulation
- The timing of depopulation and subsequent repopulation needs to be carefully managed to try and minimize the period where no pigs are being sold at the same time as maximizing the income from dispersal and sale of first pigs
- The cleaning program is performed to maximize the likelihood that all trace of the disease is removed.

SWISS DEPOPULATION

The principles of “Swiss” depopulation are:

- all young animals (piglets, weaners, growers, finishers, young replacements) are removed from the herd to a facility at least 2-3km away, leaving behind only adult breeding animals older than 10 months of age
- for a period of 14 days following removal of young stock no farrowings take place and the remaining adult breeding herd are medicated. Adult stock should also be fully vaccinated with a Mycoplasma vaccine prior to removal of young stock
- all sheds, pens and equipment are thoroughly cleaned and disinfected
- piglets born after the medicated 14-day no farrowing interval remain disease free
- the program may be used to eradicate M hyopneumoniae, mange and swine dysentery

The removal of young stock is based on research and field observations that infected replacement stock tend to recover from infection with M hyo and cease to shed the organism after about 10 months of age. Medication and vaccination of the adult herd is designed to ensure that shedding of mycoplasma by any remaining stock is eliminated or reduced to a level which infection does not persist.

SEGREGATED EARLY WEANING

This is also referred to as “Medicated early weaning” or “MEW”. It is a technique which takes advantage of the antibody protection piglets acquire from the sow while suckling and involves the transfer of piglets from the sow to a new site before they can become infected with diseases that the sows carry and usually spread to the young pigs. This naturally immune advantage is assisted by the strategic use of medications, vaccine and hygiene rules to reduce the chance of infection occurring in young pigs.

Most farms undertaking SEW wean pigs at less than around 17 days. The jury is still out whether it is the “early” part or the “segregated” part of SEW that is most effective in preventing disease transfer. It is the author’s personal view that pigs should be weaned at around 21 days and have a minimum weight of 4.5 kg for maximum performance. Weaned progeny should be segregated from the breeding herd at a distance of at least 2-3km.

SNATCH FARROWING

Snatching piglets at farrowing-farrow in non-contaminated environment. Piglets are manually delivered, dunked in disinfectant and not allowed to breathe the same air as the sow. They are transported to a foster sow on a clean site via a humidicrib. This is a costly way of cleaning up health status and piglets may suffer from colibacillosis.

PROLONGED MEDICATION

This method of disease elimination is most often used to eradicate swine dysentery. The object of the eradication program is to eliminate the infection in the animals that are seeding the herd with disease; therefore it is important to identify these animals. In the vast majority of breeding herds it is the adult sow that carries the infection in a subclinical form. The sows then infect their piglets and the infection is maintained in the population across generations. Cross infection will occur from continuous-flow housing, pen-to-pen contamination, communal dunging channels and rodents are the main source maintaining the infection in the herd will be the carrier sow at farrowing time.

Medicated eradication is based on:

- the faeces of the carrier pigs are the only source of contamination (ie, rodents are eliminated and the environment cleaned)
- over a period of 3-6 weeks, medication will be delivered to all animals at a high level that will eliminate the SD organisms from the carrier pigs

- lower levels of medication (that prevent reinfection with the SD organism) will be continued for a further 8-18 weeks. Should the cleaning or rodent elimination be sub-optimal this allows for the organisms to die out in the environment and in the rodents
- clean and disinfect the piggery to rid of all residual contamination. Without cleaning and disinfection the SD organism may survive in manure for up to 10 weeks. Eradication in summer minimizes the survival time of the organism
- when medication is withdrawn at 6 months, no medication that will mask the SD in the growers should be used for a further 6 months.

EXOTIC & EMERGING DISEASES

General indications of an exotic disease include:

- Unusually high number of sick animals
- Unusually high number of deaths
- Blisters or vesicles on animals' snout, or feet
- Unusually high number of lame animals
- Unusually high number of animals with fevers
- Unusually high number of animals not eating
- Unusually high number of animals that do not want to get up
- Discoloration of the ears, belly, rump, legs, or tail

Call your State Veterinary Officer at your local Department of Primary Industries if you suspect a reportable disease.

Aujesky's disease / Pseudorabies

Pseudorabies has only recently been eradicated from the United States. The pig is the only natural host for pseudorabies virus. Infection can result in subclinical, clinical, or latent disease in the pig. Pseudorabies virus can also infect ruminants, dogs, cats, raccoons, rodents, etc. The disease is lethal in these animals and is called "mad itch" disease due to the classical severe pruritis which is observed following infection in these dead-end hosts. Pseudorabies infection has not been definitively diagnosed in man.¹

Aetiology

Pseudorabies or Aujesky's Disease virus is a herpesvirus. The virus contains many proteins. Glycoproteins gI, gIII, gp 63 and thymidine kinase are important virulence determinants. Induction of immunity relies on gII, gIII, and gp 50. Live attenuated vaccine strains of pseudorabies virus have been engineered by deleting gI, gIII, gX, and thymidine kinase. These are very useful for eradication programs.

Epidemiology

Except for higher primates, all warm-blooded animals are susceptible. Latency and recrudescence very important (virus hides in the trigeminal nerve). Transmission occurs primarily by direct contact between infected pigs. Direct transmission can also occur transplacentally or through infected semen. Indirect transmission can occur by aerosol for distances up to 2 miles under proper atmospheric conditions. The virus can also survive in water and manure for short periods of time. The virus does not survive well in the environment, especially in direct sunlight. Direct contact or consumption of infected dead-end hosts can contribute to disease spread in individual herds. Temperature stress and improved virus survival at cold temperatures predispose pigs to outbreaks of Pseudorabies during winter months.

Pathogenesis

From upper respiratory tract through the cranial nerves to the brain. Spreads through the upper respiratory tract. A difficult-to-detect viremia also occurs.

Clinical signs

Except for the pig most warm-blooded animals are dead end hosts. Clinical signs vary with the viral strain, infectious dose, and the age and immune status of the pig. Infected neonatal pigs usually die within 24-48 hours after birth. Fever, anorexia, and central nervous system (CNS) signs are commonly observed. Nursery pigs can also exhibit CNS signs but to a lesser extent. Mortality can reach 50% in recently weaned pigs. Pigs exhibit fever and anorexia 3-6 days after exposure. Sneezing, coughing, nasal discharge, and dyspnea persisting for 5-10 days is common in older nursery pigs. Mortality can reach 10% in these pigs; however, most production losses are a result of poor performance and stunted growth. Respiratory disease with high morbidity and low mortality is most common in growing/finishing pigs. However, infected adult pigs may not show clinical signs. Recovery from clinical signs usually occurs within 10 days; however, secondary infections with *Pasteurella multocida* can prolong the disease period. Sows and boars can develop respiratory disease. Reproductive consequences are dependent on the stage of gestation during which infection occurs and include returns to estrus, abortions, mummies, stillbirths, and weak born pigs.

Lesions

Gross lesions include fibrinonecrotic rhinitis, necrotic tonsillitis, conjunctivitis, focal hepatic necrosis, focal splenic necrosis, laryngitis, and tracheitis. Lung lesions have multifocal firm, red areas with hemorrhage. Gross lesions may not be apparent older pigs.^{1,2} Microscopic lesions include nonsuppurative meningoencephalitis and ganglioneuritis.¹

Diagnosis

Call the State Veterinary Officer at the Department of Primary Industries immediately if you suspect pseudorabies based on clinical signs and gross lesions.

Diagnosis is based on disease history, clinical signs and fluorescent antibody tests on tonsil sections for virus detection.

Vesicular Diseases of Swine

Vesicular diseases of swine include foot-and-mouth disease, swine vesicular disease, vesicular exanthema of swine, San Miguel sea lion virus, and vesicular stomatitis.

Clinical signs

Clinical signs include fever and vesicles. Excessive salivation and lameness can be observed. Blanched epithelium will become fluid filled vesicles. Vesicles rupture and hemorrhagic erosions can be observed. Vesicles can form on the snout, nares, inside the mouth, on the tongue, at the coronary band, interdigital cleft and heel bulb. Vesicles can also be observed at mechanical pressure points such as teats, shoulders, carpi.

Diagnosis

Call the State Veterinary Officer of the Department of Primary Industries immediately if you suspect a vesicular disease based on clinical signs and gross lesions. A foreign animal disease diagnostician will investigate and collect samples.

Classical Swine Fever (Formerly known as Hog cholera)

Classical swine fever is caused by a Pestivirus of the family Flaviviridae. The virus is a single stranded RNA virus related to BVD. Strains vary in virulence

Epidemiology

The virus can be detected in all secretions, excretions, and body tissues. The virus is primarily spread by pig-to-pig contact, but can also be ingested or inhaled or transmitted in utero. Infected pigs can shed virus up to 20 days. Mechanical vectors include flies, arthropods, birds, feral animals including feral swine, vehicles, people, and equipment. The virus can survive in uncooked pork products for 85 days and frozen pork products for 5 years.

Clinical signs

Clinical signs usually appear 5 to 10 days after infection. In peracute cases young pigs can die without signs. Reproductive problems are described and pigs usually die after 1 to 2 weeks. Acute cases can present as lethargy, conjunctivitis, arched backs, drooping heads/tails, anorexia, constipation followed by diarrhea, fever, cyanosis of extremities, and CNS signs- staggering gait.

Lesions

Lesions may not be observed in peracute cases. Septicemia, hemorrhages in the kidney, ileocecal valve, lymph nodes, bladder, and larynx can be observed. Splenic infarction and colonic ulcers can also be present.

Diagnosis

Call the State Veterinary Officer at the Department of Primary Industries immediately if you suspect a classical swine fever based on clinical signs and gross lesions. A foreign animal disease diagnostician will investigate and collect samples.

Porcine Reproductive and Respiratory Syndrome Virus

Porcine Reproductive and Respiratory Syndrome (PRRS) was first seen in the United States in 1987 and was later given the name "Mystery Swine Disease". In 1990, a similar syndrome was reported in Europe. The European strain caused cyanosis of the extremities, and was named "Blue Ear Disease". The causative agent has been named the Lelystad virus. It is an RNA virus (Classification Arteriviridae) that grows in alveolar macrophages. A major problem is that it mutates readily.

Epidemiology:

Transmission of PRRSV occurs primarily through direct contact between pigs. Waterfowl may also spread the virus. The virus can be shed in the saliva, feces, and urine of infected pigs for at least 99 days, and can persist in the tonsil of infected pigs for at least 157 days. Moreover, seronegative pigs can still harbor PRRSV in their tonsil. New outbreaks result from the purchase of infected pigs and via semen from infected boars. There is rapid spread within a herd and between herds. Aerosol transmission up to 3km. Expt aerosol transmission < 1m.

Transplacental infection occurs after 50 days of gestation. Pigs can be born viremic and have circulating antibody against PRRSV. Such pigs either die from clinical PRRS or survive and become asymptomatic carrier pigs. Aerosol transmission has not been confirmed in controlled studies but may occur over a distance of 120-140 meters. The virus does not readily persist in the environment.

The virus appears to be immuno-modulating with major co-infection problems in weaners. Recurrence of the reproductive problem within a herd is unusual. Continuous flow nurseries tend to maintain virus infection. There may be subpopulations of infected sows in large herds, within which the PRRS virus cycles

Pathogenesis:

The virus enters following inhalation, ingestion, coitus, and possibly wounds. PRRSV then replicates in regional, mucosal, or pulmonary alveolar macrophages and dendritic cells in tonsils.

The virus is in regional lymph nodes and viremia is detectable by 6-24 hours post-infection. Further replication can occur in lungs, lymph nodes and other tissues. Systemic distribution occurs to monocytes and tissue macrophages. Impaired function and destruction of macrophages occurs within the first week of infection and probably facilitates secondary infections. Viremia may persist for up to 42 days. Antibodies are present within 7 days and persist for up to 1 year. Virus can infect fetus after 50 days gestation and produce clinical signs. Some herd infections are subclinical and are detected by sero-conversion only. Clinical signs in sows and boar are predominately reproductive. After infection animals either completely recover or become shedders

Clinical signs:

In the first weeks after infection, clinical signs may be seen in pigs of all ages. Signs in the breeding herd include: animals off feed, fever (39-41C), listlessness and abortion. In the piglets and nursery pigs, signs may include respiratory distress ("thumping", mouth-breathing), off-feed and listlessness. Prewaning mortality may increase to 50 to 60% from starvation/diarrhea/fading. In the growing/finishing pigs, animals may be off feed, have increased respiration rate, listlessness, fever (39-41C) and there may be some hyperexcitability when stimulated. These signs gradually abate after 6-8 weeks, with a gradual recovery in respiratory symptoms but an underlying poor reproductive performance.

Lesions:

Gross lung lesions can be inapparent or appear as a multifocal to diffuse gray or tan mottling of non collapsing lungs. Microscopic lesions of interstitial pneumonia are characteristic of PRRSV infection.

Diagnosis:

Diagnosis is based on history, clinical signs and serology (immunofluorescent antibody test (IFA), ELISA, serum neutralization (SN) and virus isolation from sera.

Prevention/Control:

*There are commercial and autogenous vaccines available. Use of vaccines in sows to attempt to serologically stabilize an unstable sow herd is used by some. Modified live vaccination of gestating sows **not** now being recommended by most (too many strains of virus possible cause of disease—vaccine usually has one strain). Use of killed vaccines still questionable

*Negative herds try and stay negative by closing herd and purchasing negative breeding stock and use semen from negative sources (boar studs)

*Positive but serologically stable herds add gilts by:

Purchase negative gilts and isolate for 60 days

Blood test at entry to assure gilts are seronegative

Vaccinate 2 times 3 weeks apart

Enter herd at least 3 weeks later

***Use AIAO production systems (by site best) to prevent endemic infections**

*Because acutely infected sows generally quit shedding in about 4 months, many veterinarians now trying and assure all breeding animals are infected ASAP after diagnosis

*If all sows are effectively exposed and infected (stabilized, then negative progeny can be maintained in off-site nursery and finishers after 4 months.

*Repopulation - dependent on herd

Swine Influenza Virus

Swine influenza was first described in 1918 when it was responsible for an epizootic in swine and a pandemic in humans. Today swine influenza is endemic in many pork production units overseas. It has not been recorded in pigs in Australia.

Aetiology:

Swine influenza is an influenza A virus in the family Orthomyxoviridae. Influenza viruses are described by the major surface antigens, hemagglutinin (H) and neuraminidase (N). Hemagglutinins aid in the attachment of the virus to cells and neuraminidase is responsible for the release of virus from infected cells. Swine influenza in the United States is caused by both H1N1 and H3N2 strains. In other countries, H3N2, H1N2, and H1N7 strains can cause disease in pigs.

Epidemiology:

All ages of pig can become infected. Acute seasonal influenza outbreaks were commonplace in the past. Most outbreaks occurred in the Fall or early winter. Today, endemic swine flu causes continuous low levels of disease year round in many herds. The virus is primarily spread by direct contact between pigs resulting in the transmission of virus infected nasal secretions. Pigs of all ages can be affected. Interspecies transmission occurs among avian, human, and swine influenza viruses. Although human cases of swine influenza are rare today, swine influenza viruses may cause acute respiratory distress and death in humans and should be considered an occupational hazard.

Pathogenesis:

The incubation period for swine influenza ranges from 1 to 3 days. The virus attaches to the cilia and replicates in nasal and tracheal epithelium. Damage to the mucociliary apparatus allows infection to spread the lung and predisposes pigs to secondary bacterial infections.

Diagnosis:

Presumptive diagnosis is based on clinical signs. High morbidity, low mortality are characteristic of uncomplicated influenza outbreaks. Pigs on the same site can be affected simultaneously. The onset of disease is sudden. Pigs are anorexic and lethargic. They lie down and are reluctant to move. Pigs may pile due to the fever. Nasal and ocular discharges are observed. A characteristic dry, hacking, "goose-honk" cough is associated with movement. Open-mouthed, labored, abdominal breathing can be observed. Recovery usually occurs in 5-7 days, but can be longer if secondary infections are involved.

Gross lesions are typical of viral pneumonia. Red, wet, heavy lungs are observed. Lesions may be multifocal or diffuse. Lesions are well-demarcated, purple, and firm and interlobular edema can be present. Blood tinged, fibrinous exudate can be observed in airways. Pigs can have enlarged bronchial and mediastinal lymph nodes.

Microscopic lesions are typical of interstitial pneumonia and include degeneration and necrosis of bronchial and bronchiolar epithelium. Bronchial and bronchiolar lumens are filled with neutrophils and monocytes. Alveolar septae are infiltrated with lymphocytes, macrophages, and plasma cells. Peribronchial and perivascular cellular infiltration is observed.

Definitive diagnosis is based on virus isolation from nasal mucous or lung. The sample of choice is a [nasal swab](#) using a Dacron swab (cotton can kill the virus.)

Immunohistochemistry and fluorescent antibody testing of lung sections can be used in the early stages of disease.

Serological profiling using HI, ELISA, or IFA tests can be used to establish the prevalence of exposure to swine influenza. Samples should be collected from the breeding herd, nursing pigs, nursery pigs, and growing finishing pigs. A positive titer indicates natural exposure, vaccination,

maternal antibodies, or infection. A negative titer indicates that the pigs were never exposed, or that they were exposed but antibody was not detectable.

Prevention/Control:

Optimization of the management and environment to reduce stressors is important to control influenza. Water should be available to pigs at all times. Antibiotics may be added to water (or feed once pigs begin to eat) to control secondary bacterial infections.

An inactivated swine influenza vaccine (MaxiVac-Flu) is commercially available in the United States. The withdrawal period is 21 days. All ages of swine may be vaccinated and duration of immunity about 3 months. Colostral immunity can interfere with pig vaccination at < 10 weeks of age.

Control of influenza in nursing pigs can be accomplished by vaccinating sows 5 and 2 weeks pre-farrowing to raise maternal antibody levels. Control of influenza in growing/ finishing pigs can be accomplished by vaccinating pigs at 10 weeks of age and 2 weeks later.

Contact with avian species and infected humans should be prevented to eliminate interspecies transmission.

Transmissible gastroenteritis (TGE)

Aetiology:

Transmissible Gastroenteritis Virus of Swine (TGEV)- a Coronavirus

Epidemiology/Transmission:

All ages affected; Fecal-oral and aerosol transmission; Starlings implicated in spread of disease; High mortality in neonates; Virus is hardy in cold temperatures (winter outbreaks)

Clinical signs:

Epizootic (Acute): Severe illness in young pigs, Explosive pattern of infection, Vomiting and yellow diarrhea, Dehydration, Death, High mortality of pigs under 3 weeks of age, Sick sows, Diarrhea, vomiting, anorexia, agalactia, Acidic pH of feces

Enzootic: Dilated stomach with undigested milk curd, Usually less severe than acute TGE, Variable mortality, Sows not sick

Gross lesions:

Villous atrophy; thin intestinal wall, empty lacteals, extensive dehydration

Histopathology:

Villous atrophy, variable mitotic activity

Diagnosis:

Virology-Virus isolation, Immunofluorescence testing (IFA, IHC)

Serology- Acute and convalescent samples, Blocking ELISA test (Differentiate between TGE and PRCV)

Electron microscopy

PCR Feces

□ **Treatment:**

- Exposure of pregnant sows to the virus will stimulate colostral immunity for unborn litters in the case of an outbreak
- If sows > 3 weeks away from farrow date - intentionally expose to virus (*feed ground intestines from infected neonates to sows*)
- If sows < 2 weeks away from farrow date - farrow sows off site and prevent exposure of litters to the virus
- Supportive measures (add electrolytes to water containing glucose), increase temperature in farrowing facility

□ **Prevention/Control:**

- Rigid sanitation and all-in/all-out management of farrowing facility and nursery
- Expose all susceptible sows during outbreaks
- Vaccinate recovered sows to maintain herd immunity
- Control starlings
- Obtain animals from TGE negative sources

Isolate incoming replacements and use feed-back procedure

Post-weaning multi-systemic wasting disorder (PMWS)

First observed in Canada in 1991-in animals from a farm with excellent sanitary conditions, resulting in progressive weight loss, generalized lymphadenopathy and high mortality. First described in 1996 at a Canadian conference as PMWS. It is a disease reported mainly in parts of Europe and the US. It was diagnosed in New Zealand in late 2003. It has not been diagnosed in Australia.

The “necessary” agent is thought to be Porcine CircoVirus Type 2 + some other agents (parvovirus has been implicated). Although we have PCV 2 in Australia, we do not seem to have PMWS.

PMWS affects animals in the weaning and early growing period. The disease is subacute or chronic; it affects all types and sizes of farming operations. Clinical signs include:

- Progressive and chronic weight loss of animals
- Anaemia and jaundice
- Dyspnea
- Generalised lymphadenopathy (the inguinal superficial ganglion is increased in size)
- Unspecified respiratory and/or digestive signs
- Occasional fever
- Morbidity and mortality varies



□ **Diagnosis relies on:**

- An array of somewhat non-specific clinical signs (e.g. wasting, diarrhoea, respiratory diseases) associated with increased mortality amongst pigs 8-12 weeks
 - 1a. Gross pathology (e.g. non-collapsing lungs, enlarged lymph nodes, polyserositis, gastric ulceration)
 - 2. Histopathology
 - Lymphoid depletion (non-specific)
 - Giant cells (more specific)
 - Inclusion bodies (specific – for PCV2-associated disease)
 - 3. Demonstration of **abundant** PCV2 in lesions

Clinical criteria is (case definition):

- “Current or historical evidence of a herd syndrome characterised by elevated morbidity and mortality (twice the baseline for the herd), associated with non responsive, otherwise unexplained obvious wasting, in pigs between weaning and 12 weeks of age. Other signs may be present including dyspnoea or tachypnoea, enlarged lymph nodes, and, less frequently, diarrhoea, pallor or jaundice”.
- *Wasting means loss of body condition, typically over a 7-10 day period. Older pigs, to about 18 weeks, may be involved. Wasting in pigs due to PMWS can occur at any time during the growing phase, but should be distinguished from poor growth during the immediate post-weaning period.*

□ **Treatment & Control :**

The Europeans seem to be on top of PMWS and control it using good management practices (small group sizes, late weaning, AIAO etc). The Americans seem to have less success.

PCV2 vaccines have been developed and introduced into the American and European markets. These have been hugely successful to the extent that vaccine suppliers are pushed to meet with demand.

Porcine dermatitis-nephropathy syndrome (PDNS)

Has been diagnosed in Australia. Occurs sporadically. Thought to be associated with an “overload” of the immune system-seen OS in association with PRRS, PMWS. Seen in Australia associated with high level of mosquitoes and biting flies.

Pigs appear lethargic or may be normal. Extremities (ears, belly, tail, nose) are purple. Also blotches on the skin. Pigs usually recover.



Porcine myocarditis virus (PMC)

Occurred in a large piggery in NSW in mid 2003. Characterised by a decrease in the number of piglets born alive per litter, increased stillbirths and an increase in pre-weaning deaths (sudden death in apparently good pigs). Pigs older than 5 weeks were not affected.

At post-mortem-dilated hearts and evidence of congestive heart failure. Histo-multifocal, non-suppurative, interstitial myocarditis.

A “new” pesti-virus has been isolated and is thought to be the aetiological agent

VETERINARIANS IN THE PIG INDUSTRY

Pig-specialist swine veterinarian: consultant or large company employee

- Health
- Nutrition
- Economics
- Welfare
- Reproduction
- Quality Assurance

Do pigs as part of general practice

- “Fire engine” calls

Research

- Pig industry
- Pigs as models for human research

Pharmaceutical industry

- R&D
- Technical support

Chapter 4

PAST EXAM QUESTIONS

Past exam questions

What is the differential diagnosis of diarrhea in 8-14 day old pigs and how do you differentiate the conditions? Select one of your diagnoses and propose how you would treat affected pigs. For the selected disease describe how you would prevent its recurrence.

You have been called in as a consultant to a pig farm that is not achieving its production target of 3000kg of dressed meat each week.

How would you approach this problem, in terms of identifying the production-limiting factors? In your answer, consider factors driving both volume & efficiency.

The growth rate of weaner pigs (4-10 weeks of age) on a farm is below 350g/day.

What management factors could be responsible for this?

What diseases may impact on the growth of weaners of this age?

How would you rule out poor feed intake as a reason for slow growth?

Describe the clinical signs of infection with *Actinobacillus pleuropneumoniae*. Discuss the treatment and control measures available to Australian pig producers.

Breeding sows in Australian herds are generally vaccinated against leptospirosis, erysipelas, parvovirus and E coli. Describe the nature of and response to these vaccines and provide details of an appropriate vaccination program.

Most producers in Australia wean their pigs at 28 days of age. Producers in the United States are weaning their pigs as early as 10-14 days of age. What are the advantages and disadvantages of early weaning? Consider both sow and piglet factors and the efficiency of facility utilization in your answer.

A pig farmer requests you to help solve a problem of poor growth rate in piglets before weaning. He is currently weaning piglets at 28 days of age with an average weaning weight of 5.5kg.

List the major sow and piglet factors which contribute to poor weaning weights of piglets.

For each factor listed, outline the management strategies which would help to organize pre-weaning growth.

Consumers demand consistent high quality in the meat they purchase.

Antibiotic residue testing of kidneys from one of your client's herds indicates chlortetracycline residues in 3% of finisher pigs at slaughter. What antibiotic treatment "errors" may have resulted in the chlortetracycline residues detected at slaughter?

What variations in appearance, taste and nutritional value can occur in pork and bacon and how can these variations be minimized on-farm and in the pre-slaughter handling of pigs?

The manager of a 200-sow piggery is concerned about low numbers of pigs weaned (8.5 pigs weaned/litter). How would you improve this? Include in your answer how you would overcome low total born, low numbers of pigs born alive and high pre-weaning death rates.

THE UNIVERSITY OF SYDNEY



FACULTY OF VETERINARY SCIENCE

PIG HEALTH AND PRODUCTION

VETS 4223

Semester 2, 2004

 **Time allowed: 1.5 hours**

- There are three (3) questions in this examination
- ALL three (3) questions are to be attempted
- Write your name, student number and the section on each examination book you use.
- **The marks allocated for, and the time you should spend on, each question are indicated next to the question.**
- The total for all questions is 60 marks.

Question 1

20 marks/ 30 minutes

Reproductive performance

- a) The average farrowing rate on a pig farm in 2003 was 75%. What other information would you need to know to define the cause of the low farrowing rate on this farm? List 5 things and justify why you need to know these. (5 marks)
- b) On another farm, the average wean-to-oestrus interval for sows is 7 days. What are three major determinants of wean-to-oestrus interval in sows? How would you manage sows to minimise the interval between weaning and return-to-oestrus. (5 marks)
- c) What three (3) diseases do pig producers routinely vaccinate replacement stock against to prevent reproductive failure? How would these diseases be expressed in non-vaccinated stock? (5 marks)
- d) What is a target stillbirth rate for pig farms? What are five (5) major contributing factors to stillbirths? Address each of these five factors and demonstrate how you might overcome a stillbirth problem on a farm. (5 marks)

Question 2

20 marks/ 30 minutes

Processing and meat quality

- a) At a processing plant, Pig Farmer A receives \$2.00 per kilogram of carcass. Pig Farmer B receives \$2.20 per kilogram of carcass. What are three (3) main factors that will determine the price a farmer receives for pig meat at the processing plant? Explain your reasoning for each factor listed. (5 marks)
- b) Pigs occasionally die during transportation to the processor or while in lairage. What three (3) syndromes may cause sudden death in 24-week old pigs in this manner? How would you differentiate between them on gross post mortem and using confirmatory diagnostic tests? (5 marks)
- c) Farmers are penalised if the pigs they send to the processing plant require extensive skinning. What three (3) syndromes that originate on-farm commonly cause skin blemishes in **slaughter-aged pigs** and how would you minimise these occurring? (5 marks)
- d) Inspection of the lungs at the processing plant is often used to monitor herd health. What three (3) diseases are you likely to detect when inspecting lungs at slaughter? What pathogens cause these diseases? What further diagnostic tests would you undertake on lung tissue to make a definitive diagnosis? (5 marks)

 **Question 3**

20 marks/ 30 minutes

Housing and environment

- a) The Code of Practice for the Welfare of Pigs is currently under review. Acting on behalf of the pig industry, develop an argument for retaining individual sow housing for sows for at least part of gestation and during farrowing/lactation. (5 marks)
- b) What three (3) pathogens commonly cause scouring in piglets prior to weaning in Australia? How would you differentiate them on clinical signs alone? (5 marks)
- c) Neonatal scours usually result from a combination of: (1) poor immunity, (2) sub-optimal temperature and (3) poor hygiene. Develop a “best practice” approach incorporating the three (3) factors above for housing and managing sows and their piglets in the period around the time of farrowing and lactation to minimise the risk of pre-weaning scours. (5 marks)
- d) Over-crowding weaner pigs can result in sub-optimal performance. Give three (3) reasons why this is the case and discuss the mechanisms for the reduced performance. (5 marks)

THIS IS THE END OF THE PAPER



THE UNIVERSITY OF SYDNEY



FACULTY OF VETERINARY SCIENCE

PIG HEALTH AND PRODUCTION

VETS 4223

Semester 2, 2005

 **Time allowed: 1 hour**

- There are four (4) questions in this examination
- ALL four (4) questions are to be attempted
- Write your name, student number and the section on each examination book you use.
- **The marks allocated for, and the time you should spend on, each question are indicated next to the question.**
- Each question is worth equal marks.

Question 1**15 minutes**

A 100-sow farm that you consult to suffers from a number of endemic diseases. These include *Mycoplasma hyopneumoniae*, mange and roundworm. The farm is currently on one site (ie. farrow-to-finish) but there is a farmer nearby who has empty pig accommodation.

- a) Is it possible to eradicate each of the pathogens present currently on this farm? Explain your answer.
- b) Outline briefly two (2) options for disease eradication on this farm and the advantages and disadvantages of each option.

Question 2**15 minutes**

The farmer in the scenario above is currently losing about \$15 per pig (75kg dressed carcass weight) as his cost of production (\$2.50 per kilogram carcass weight) is higher than the price he receives (\$2.30 per kilogram carcass weight). If you undertake a disease eradication program on this farm, you will expect to improve his profitability.

- a) List five (5) production parameters that are likely to change as herd health improves.
- b) Next to each parameter, explain why the change will occur.

Question 3**15 minutes**

You have just spoken on the topic “Abortion in sows” to the Grenfell Pig Producers Group. At supper, a farmer approaches you and says “I don’t have a problem of abortions, but my gilts won’t get pregnant. What could be the cause of that?” You reply that it could be any number of causes, and to find the cause, it will be necessary to obtain further information.

- a) List 5 questions you would like to ask this farmer about his problem that are likely to give you the most information to help you solve this problem.
- b) Next to each question, justify why you are asking it.

Question 4**15 minutes**

A farmer rings to say that 50 out of 150, 10 week old pigs housed in a straw based deep litter “ecoshelter” were found to be affected by a skin condition. You ask him to send you some photos of affected pigs. The farm is 4 hrs drive from your office and over the phone the condition does not sound life-threatening. The photographs arrive the next day (see attached).

- a) What would you advise the farmer to do at the time he rang you?
- b) What are your 3 most likely differential diagnoses, given the appearance of the lesions, housing type and age of the pigs?
- c) What additional action would you take to confirm your diagnosis from (b) above?

Figures below are relevant to Question 4.



THIS IS THE END OF THE PAPER



THE UNIVERSITY OF SYDNEY



FACULTY OF VETERINARY SCIENCE

PIG HEALTH AND PRODUCTION

VETS 4223

Semester 2, 2006

 **Time allowed: 1 hour**

- There are four (4) questions in this examination
- ALL four (4) questions are to be attempted
- Write your name, student number and the section on each examination book you use.
- **The marks allocated for, and the time you should spend on, each question are indicated next to the question.**
- Each question is worth equal marks.

Question 1**15 minutes**

You are a veterinarian working for a genetics company selling high-health status pigs that are free of the major costly endemic pathogens.

- c) List 4 “costly” pig pathogens that can be eradicated from pig farms and 4 “costly” pathogens that cannot be eradicated. (4 marks)
- d) Choose 1 pathogen from each list and discuss why it can or cannot be eradicated from a pig herd. You will need to consider in your answer the ability of the pathogen to survive on and off the pig and how this survival can be manipulated. (6 marks)

Question 2**15 minutes**

As part of your disease surveillance strategy for the above herd you conduct “health checks” at the abattoir every 3 months. During your last check you notice that 5% of lungs inspected have cranio-ventral consolidation (see attached photo). When you cut a section of this consolidated lung you can squeeze pus out of the airways. You have not noticed this before in this herd.

- c) What are the two most likely differentials for this lung condition? (2 marks)
- d) What test(s) would you request at the veterinary laboratory to confirm the diagnosis? (4 marks)
- e) What else might you do to confirm the diagnosis/determine the extent of the problem? (4 marks)

Question 3**15 minutes**

You have just spoken on the topic “Abortion in sows” to the Grenfell Pig Producers Group. At supper, a farmer approaches you and says “I don’t have a problem of abortions, but I have a problem with my sows. You see, about 10% of my sows come into the farrowing house assumed pregnant, and then I find they don’t farrow!. What could be the cause of that?” You reply that it could be any number of causes, and to find the cause, it will be necessary to obtain further information.

- c) List 5 questions you would like to ask this farmer about his problem that are likely to give you the most information to help you solve this problem. (5 marks)
- d) Next to each question, justify why you are asking it. (5 marks)

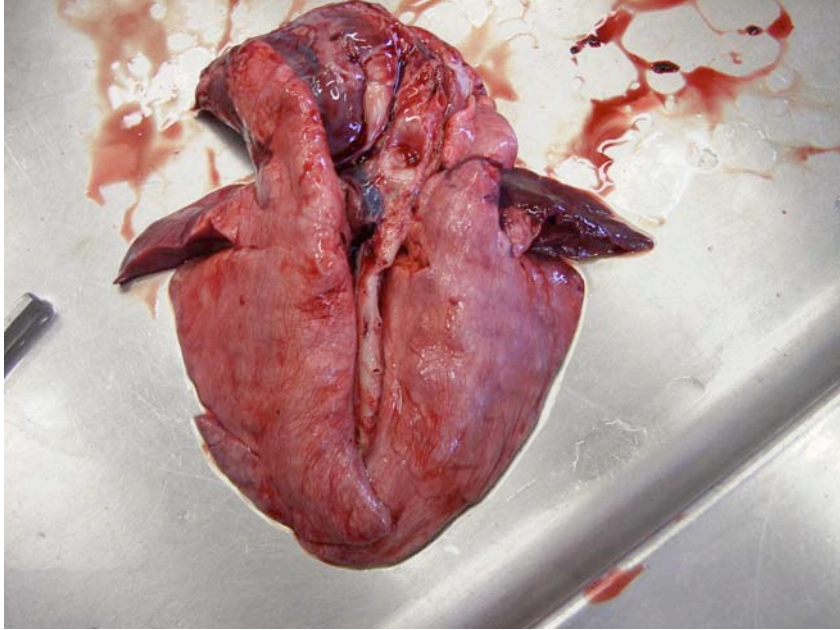
Question 4**15 minutes**

In Australia, about 60% of pregnant sows are housed individually for at least for part of gestation. The “Animals Australia” group would prefer that individual housing (ie “sow stalls”) is banned completely in Australia.

- a) List three (3) reasons why farmers may house sows individually during pregnancy. (3 marks)
- b) Next to each reason, list a management and/or housing strategy that would over-come these problems-without the need for individual housing. (3 marks)

c) Replacement rates for breeding stock can reach as high as 60%. What are two main causes of death in sows? (2 marks) What management/housing strategies might you implement to prevent each cause of death? (2 marks)

Figure below is relevant to Question 2.



THIS IS THE END OF THE PAPER



THE UNIVERSITY OF SYDNEY



FACULTY OF VETERINARY SCIENCE

PIG HEALTH AND PRODUCTION

VETS 4223

Semester 2, 2007

 **Time allowed: 1 hour**

- There are three (3) questions in this examination
- ALL three (3) questions are to be attempted
- Write your name & student number on each page of the exam.
- **The marks allocated for, and the time you should spend on, each question are indicated next to the question.**

Name: _____

Student ID: _____

Question 1

15 minutes

- a) What 7 parts of the pig's carcass are routinely examined during a health check for endemic disease monitoring at an abattoir? (7 marks)
- b) For each part, state the disease conditions that may be detected. (7 marks)

Carcass body "organ"	Differential "disease" conditions detected

Question 2

30 minutes

Complete the table (below) which refers to Photos A-E (below). Do not write outside of the table. (25 marks)

Photo	Describe the pathology	Most likely pathogen(s)	What laboratory tests to confirm the diagnosis?	Are vaccines available for these disease(s)? (Yes/No)	What is the 1 st choice antibiotic to treat
A					
B					
C					
D					
E					

Name: _____

Student ID: _____

Photo A



Photo B



Photo C (small intestine)

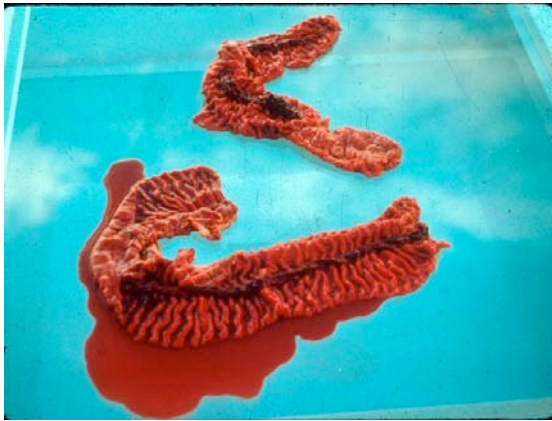


Photo D (colon)

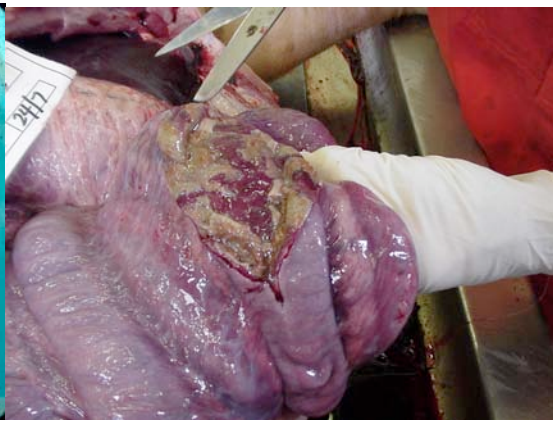
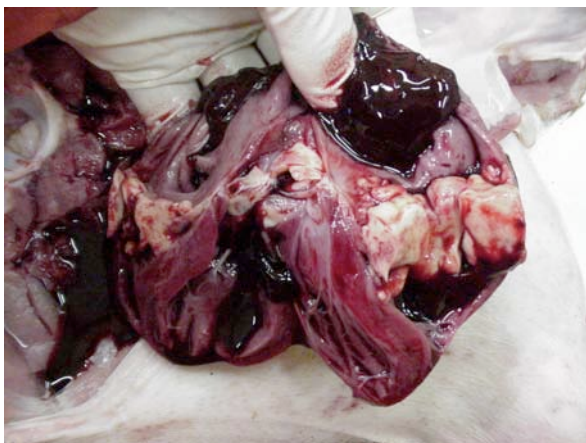


Photo E (heart)



Question 3**15 minutes**

In the table below are listed 5 points relating to pig health and medication usage. Consider each of these points, state whether you agree or disagree with the statement made, and give a justification for your answer (10 marks).

Statement	Agree/Disagree	Justification
The major public health risk with over-use of antibiotics is detection of residues at slaughter.		
The most effective way to administer antibiotics to pigs is in their feed.		
Hormones are added to pigs' feed to make them grow faster		
Feed conversion efficiency (kg feed:kg gain) is the most important growth performance parameter to measure		
Pigs that have a high disease challenge during their lifetime are likely to be fatter at slaughter than "high health" pigs		

THIS IS THE END OF THE PAPER





NSW Department of Agriculture: Veterinary Laboratory Service

The New South Wales Department of Agriculture (now called Agriculture NSW) has provided veterinary research and laboratory diagnostic services for the livestock industries of the State for many years. This article gives a brief history of these areas of activity.

Physical facilities

In 1923 the Department built its first veterinary laboratory, which was located at Glenfield about 30 km south west from Sydney. It was called the Glenfield Veterinary Research Station and it consisted of two laboratories, a post-mortem room, offices and out buildings. Over the years it grew into a large complex with laboratories covering many scientific disciplines. The limitations of having only one central laboratory to service the entire State was recognised and this led to the establishment of regional veterinary laboratories. The first was opened at Armidale in 1965, followed by Wollongbar (near Lismore) in 1968, Wagga Wagga in 1974 and Orange in 1982. Because Glenfield was becoming urbanised and the older buildings on the Research Station needed replacing it was decided to build a new complex at Camden. This was occupied in 1990. It is called the Elizabeth Macarthur Agricultural Institute because it is on part of the original Camden Park Estate where the Macarthurs established Australia's commercial wool industry in the early 1800s.

Diagnostic services

From the beginning Glenfield provided a diagnostic service on specimens and cadavers and this service was extended when the regional veterinary laboratories were opened. The laboratories serviced the livestock industries and worked on cattle, sheep, pigs and poultry and occasionally horses. Most diagnostic material dealt with endemic diseases with some coming from outbreaks of the exotic diseases swine fever and Newcastle disease. The laboratories played an essential role in the National Bovine Tuberculosis and Brucellosis Eradication Campaign.

Research

Research was carried out on many diseases concurrently with the diagnostic work. Over the years specialist discipline laboratories developed. These included pathology, microbiology, virology, immunology, serology, parasitology, biochemistry, toxicology and molecular biology. Again work was carried out on production livestock. Diseases studied changed with the passing years but overall there are few livestock diseases in New South Wales that have not come under study.

Conclusions

For over 80 years the veterinary laboratories have made valuable contributions in safe guarding the health of production animals and birds in New South Wales through their laboratory diagnostic and research work.

References

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