Premature culling or on-farm euthanasia is a commonly recorded sequel to lameness in the young breeding animal - both male and female - and lameness in general can be regarded as one of the most common ailments and welfare problems affecting breeding stock.

In broad terms, lameness in adults can be divided into three distinct groups:

- **Infectious arthritis** of which Erysipelas and Mycoplasma hyosynoviae are the most common causes.
- **Septic laminitis** - bush foot due to bacterial infection.
- **Physical lameness** associated with deformed or damaged cartilage (variably termed osteochondrosis, osteochondritis, dyschondroplasia or degenerative joint disease (DJD)) and bony pathology leading to weakness and fracture (osteomalacia). There can be overlap in both underlying causes and pathogenesis of these two conditions.

This paper will concentrate on physical lameness (see other health bulletins for the infectious conditions).

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**Fig 1:** Postmortem examination picture of epiphysiolysis - separation of the head of the femur (thighbone) has occurred through the growth plate.

**Fig 2:** Mycoplasma arthritis and mild osteochondrosis can be clinically indistinguishable.

**Fig 3:** Extreme deformation of joint cartilage does not necessarily mean there was lameness.

**Fig 4:** Erosion of cartilage to expose underlying bone is initially very painful.
**Underlying Pathology**

**Degenerative Joint Disease**

The skeleton is comprised of a mixture of bony tissue and cartilage. The latter, for the purposes of this paper, are restricted to the joint surfaces where they act as lubricated cushions and the growth plates (epiphyses), which are layers of cartilage below the ends of the bone, the expansion of which leads to elongation and growth of bones. As the growth plate expands the cartilage becomes ossified (converted to bone). In the pig these growth plates do not become fully ossified until approximately 3½ to 4 years of age.

Any damage or deficit in either articular or epiphyseal cartilage potentially leads to lameness. In extreme cases the head of the bone can separate through the growth plate causing epiphysiolysis, which presents effectively as if there is a fractured hip (fig 1).

**Osteomalacia**

Lack of calcification of bone may lead to weakness of bone that can fracture with little trauma. The defective calcification can be due to inadequate dietary provision, physiological abnormality of absorption and utilisation of calcium and phosphorous and extreme demand such that calcium and phosphorous are drained from the bones of young growing animals (especially during lactation). Rickets is seen in young growing pigs especially in Scotland and is a similar type of condition.

**Clinical signs**

**Degenerative Joint Disease**

Two distinct patterns of clinical presentation are seen:

1. Where articular cartilage is damaged animals may simply be lame on the affected leg, have difficulty rising or walk with a stilted gait or swaying hindquarters. It can be very difficult to clinically distinguish the condition from Mycoplasma arthritis. (Fig 2.)

It should be noted that even major deformity of cartilage - viewed post mortem - does not necessarily mean the animal was lame (fig 3).

Damage can progress to erosion of cartilage, which initially is intensely painful. In time, however, granulation tissue can fill the erosion and, lacking nerve ending leads to, tolerance or even a cessation of pain (fig 4).

2. Epiphysiolysis. This occurs suddenly, typically in young gilts and sows. If both hips are affected the animal will dog sit and be unable to rise. Manipulation of the joints in early cases can induce an intense pain reaction (screaming) and grinding of the bone may be felt. If only one leg is affected the animal may still walk with 100% lameness on the affected leg.

With time, some degree of healing can occur and weight will be born on the affected leg. However, it remains unstable and affected animals will almost certainly suffer further pain, discomfort and lameness.

**Osteomalacia**

This is most typically seen in weaned gilts and presents as sudden onset lameness or collapse depending on the bone or bones affected (It can include the spinal column leading to broken backs).

If back legs are affected, it can be very difficult to distinguish between epiphysiolysis and fracture.

**Causation**

**Degenerative Joint Disease**

Studies have shown evidence of cartilage defects in piglets of one day old in all breed types - included wild boar.

However, it is the rearing and growth of the young pig that will determine whether significant cartilage damage occurs and lead to pathological lesions. Contributory factors may be:

**Fast growth and development of heavy muscling** particularly of the hind quarters may be significant in increasing compression forces on cartilage. (In the 1980s experiments with growth hormone treatment increasing muscle mass led to extreme cartilage malformation and Degenerative Joint Disease.)

**Housing conditions** of rearing animals. Hard floors, slippery floors, large group sizes and high stocking densities may all contribute to compression and shearing forces on joints potentially damaging cartilage that contains underlying defects.

**Genetics.** Some breed types are more affected than others e.g. Landrace. In general nowadays hybrid sows are used commercially and the
Introduction of Duroc blood into female lines in the 1990s was largely intended to improve leg quality.

Nutrition. Incorrect balances of major minerals (calcium, phosphorous and magnesium) and inadequate intakes - possibly on finishing diets - as well as trace element inadequacies and Vitamin D shortages may all be implicated.

Physiological acidosis. Resulting from nutritional factors, a systemic acidosis has been implicated in the pathogenesis of osteochondrosis. Acidic diets potentially can influence development of the condition whilst use of alkalis of agents such as Sodium Bicarbonate may help reduce joint damage.

Hormone factors, particularly associated with puberty, pregnancy and parturition. Sudden onset lameness with underlying Degenerative Joint Disease will normally result from trauma. This can be the result of slippery floors, bullying, riding behaviour or collision with hard objects such as gateposts, pen division, feeders etc. and is particularly seen following mixing e.g. at weaning.

Osteomalacia

This condition largely results from either a primary lack of calcification of bones due to inadequate diet in the growing gilt or most likely decalcification of bone during lactation with inadequate calcium, phosphorous and Vitamin D intake. It is most likely to happen when gilts are required to rear large litters to extended weaning ages and can form part of the syndrome that manifests as second litter drop. In older sows dietary imbalance is most likely exacerbating the drain on bone minerals during repeated lactation

Treatment

When single leg lameness occurs, immediate isolation in a well-bedded hospital pen is essential. Provided some weight is borne on the leg treatment can ensue including anti-inflammatory pain killers (NSAID) and antibiotic cover. Rest in such cases will often allow some degree of recovery but it must be born in mind that the weakness will remain and early culling (e.g. following farrowing) is appropriate.

Where there is sudden onset 100% lameness affecting one or more limbs, epiphysiolysis or fracture must be suspected and if confirmed immediate humane slaughter is required.

Intermediate cases can be isolated and provided with treatment as above but if no improvement occurs within a few days, in response to treatment, or the animal continues to degenerate, euthanasia is required.

Any lame animal under treatment must be provided with any necessary assistance to eat and drink.

Prevention

Prevention of physical lameness in breeding animals begins with the selection of genetic base. Use of breeds and lines of pigs with less prevalence of "leg weakness" e.g. Duroc, Hampshire are appropriate. Breeding companies generally apply selection techniques nowadays which take account of leg and joint integrity.

Rearing of the potential breeding animal should be considered with the following points relevant:

- Rear gilts from 60kg onwards on non-slip surfaces, preferably bedded.
- Moderate growth in gilts and boars post selection.
- Avoid use of organic acids and consider efforts to raise pH of diet, e.g. sodium bicarbonate. (If active Salmonella control is required the use of organic acids will have to be carefully considered in the light of the potential increased risk of joint disease)
- Use diets for breeding animals with additional mineral content (i.e. specific gilt rearer diets).
- Avoid overcrowding of breeding animals, particularly in the growing stages and maintain small stable groups.
- Following weaning, ensure plenty of space is provided with minimal competition i.e. small groups of equal sized animals with at least 3.5m² space per animal.
- Ensure flooring is non slip by grooving concrete, increasing straw bedding or applying sharp sand to the dunging area.

Specific recommendations relating to gilts during first lactation to minimise calcium depletion and resulting physical lameness (Degenerative Joint Disease or Osteomalacia) should include:

- Limit number of piglets reared to ten per litter.
- Never use gilts as "late foster" animals - i.e. limit lactation to 28 days.

"PREVENTION IS BETTER THAN CURE"
Plan for health with your vet - get a veterinary health plan
- Maximise feed intake by ensuring water is freely available, room temperatures are maintained below 20° C and provide multiple feeds beyond ten days post farrowing.
- Ensure floors in farrowing areas allow the gilts to stand up and lie down with ease.
- Supplement litters with creep feed from ten days of age.
- Supplement gilts with 200g/day bone flour during lactation and from weaning to service (palatability can be a problem).
- Consider partial weaning of gilt litters (removing 2 or 3 of the biggest pigs early). Care of inducing lactational oestrus.

Boehringer Ingelheim

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