Young piglets are often born with the whole body trembling and shaking to a variable extent. The consequences of this trembling will depend on its severity. The condition is known as Conditional Tremor or Myogenesis congenita.

It is widely assumed that, because pigs are born affected, it must be an inherited (genetic) condition and a common reaction is to blame and cull the boar. This is only rarely the cause.

The Disease

Five specific types of Congenital Tremor are recognised. The first two are extremely rare and are inherited conditions:

- A sex linked inherited defect occurring in pure Landrace pigs affecting only a small proportion of males (Type A III).

- An inherited recessive genetic defect in pure Saddleback pigs tending to affect only 25% of a litter (Type A IV). It is unclear whether this genetic defect is present in the Hampshire breed which was originally part of the Saddleback group (the others being the Essex and Wessex breeds).

The most common causes are infectious agents which attack the naïve gilt or sow, usually in the first half of pregnancy and can be one of two viruses:

- Classic Swine Fever (Hog Cholera). During the last 15 years of the 20th Century in some outbreaks in Germany, associated with mild strains, Congenital Tremor was the only sign seen with Classical Swine Fever infection. In the last UK outbreak of CSF in 2000, Congenital Tremor was not seen. (Type A I.)

- Congenital Tremor Virus. Despite extensive research work over many years, the specific causal agent remains unclear, although recent evidence suggests it is related to a novel Pestivirus infection during pregnancy. Previous suggestions that it might be associated with Porcine Circovirus type II or even type I but this is now doubtful. This type of
Congenital Tremor (Type A II) is by far and away the most common form occurring in the UK herd and can present as:

a) A sporadic disease affecting odd litters - usually most of the pigs within a litter are affected.
b) An outbreak over 6-8 weeks affecting a proportion of all farrowings.
c) An ongoing problem affecting only gilt litters.

In addition to these 4 types of Congenital Tremor, specific toxins to which the pregnant sow is exposed can cause a further type (Type A V). Organophosphates have been specifically implicated in some outbreaks.

Clinical Signs

Piglets are usually born shaking, trembling and nodding whilst active stimulation (eg handling, noise) will often exaggerate the shaking; conversely it will tend to stop when the piglets fall asleep (fig 1). It is common for trembling pigs to have a simultaneous splay leg of the hind legs (fig 2). In rare cases the trembling may not become apparent until 24-48 hours of age. There is no fever.

The effect on the pig will depend upon the degree of trembling and its impact on the ability of the pig to suck on a teat. Mild cases may be barely inhibited, whilst severely affected pigs can only suck if physically held onto the teat. Where a high level of stockmanship can be applied, mortality levels can be kept within manageable levels - 15-20%.

Particular attention is needed to ensure colostrum intake in the first 6-12 hours of life but thereafter assistance may be required to prevent starvation over the prolonged period on the sow. In severe outbreaks - most commonly encountered in gilt herds or where a high level of gilt intake has occurred (eg sudden herd expansion) - mortality levels can be much higher - reaching 30-40%.

In the individual pig, providing care can be given, the trembling will gradually reduce over the sucking period to the point of ceasing by the time the pigs are weaned. In rare cases, it can take up to 8 weeks of age for complete resolution.

Pathologically, 2 distinct syndromes are recognised:

1) Lack of development of the cerebellum - the part of the brain that controls co-ordination - which does not resolve. This is typical of CSF (Type A I) for which there would be compulsory slaughter of the whole herd.

2) Lack of myelin development. Myelin is effectively the insulation material around the nerve fibres, its absence leading to uncontrolled nerve stimulation. It is these cases that can resolve by delayed development of the myelin sheaths. Hypomyelination will often be associated with poor cerebellar development

(Tremor in young pigs can also be a feature of other diseases such as Aujeszky's disease - tending to occur after the first week of life - and as a sporadic sign in PMWS in weaner pigs. In such cases it does not resolve.)

Treatment

The reason that pigs are born trembling is that there is damage to the nervous control system and this takes time to heal. There is no specific treatment. However, assisted suckling and provision of an environment where chilling and overlaying can be avoided will allow more pigs to recover with time, although weaning weights may be depressed by 1kg or more.

Prevention

If purebred Landrace or Saddleback pigs are involved and the pattern of disease suggests that the cause may be one of the two inherited conditions, then clearly the breeding programme must be closely examined and culling of "carrier" animals or even families may be appropriate.

If type A V is suspected, then the key is to stop poisoning the sows! Identify the cause of the toxin and remove it.

Presence of organophosphates in diets may produce other signs in other classes of stock such as posterior paresis (complete loss of use of back legs) in sows. (Demyelination is also a feature of organophosphate toxicity in man).

No vaccine is available as the causative organism of Type A II is not definitively known.

Classic Swine Fever is a notifiable disease with a compulsory slaughter policy. Suspicion of this disease must trigger notification to the DVM of DEFRA and an investigation will be undertaken.

Control of the most common form of Congenital Tremor (Type A II) hinges on ensuring that gilts are exposed to the causative agent long before service.
The fact that Congenital Tremor type A II is uncommon and sporadic in nature suggests that gilts produced on most multiplication farms - or indeed in-house - are exposed to Congenital Tremor virus before breeding. There are however occasional herds where the virus is absent - hence unexposed gilts are produced - and in three site production systems it is possible to 'lose' the virus from the finishing stage - even where it is known to be present in the breeding herd - with the same effect.

Whilst three-site production - which is designed to produce pigs of the highest possible health - is a valuable tool in commercial pig keeping, it may not be applicable for seedstock population if it produces animals that are 'too clean' for the recipient herds of lower health status.

It is difficult to say what a reliable source of virus is for naïve gilts but experience suggests that weaners of 8-12 weeks of age can provide satisfactory exposure for gilts over a 4-week period, stopping at least two weeks prior to service. Cull sows are also widely used to acclimatise incoming gilts to a range of potential pathogens.

In the rare herds which are free of the virus - and this will only be known if a parent herd is believed to be free, or retrospectively following a breakdown - the aim must be to keep the virus out. As the cause is not known and, therefore, no test is available, this is very difficult, particularly as there is circumstantial evidence to suggest that the virus will pass through the semen.

In practice, most herds carry the virus and see little or no disease. Any change in the source of gilts must be treated with caution to avoid upsetting the equilibrium that exists with this and may other infectious agents, particularly if high numbers of gilts are introduced.

**Costs**

At the mildest level, the disease has minimal cost, other than the time to care for affected piglets. In a severe outbreak in a new herd of 400 gilts, total mortality for the first 5 months was 35% - estimated that 25% of all deaths were associated with Congenital Tremor. The loss of 1000 pigs cost more than £35,000 to that farm.

**Boehringer Ingelheim**

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