Effect of low phosphorus diets on pig health and welfare

Key external stakeholders:
Pig farmers, feed manufacturers, veterinarians, policy makers

Practical implications for stakeholders:
- The outcomes of this study provide information for pig farmers and feed manufacturers regarding the effect of low phosphorus (P) diets on pig growth, bone strength and the excretion of P by pigs. Additional knowledge was generated regarding the pros and cons associated with practices used to decrease P excretion, such as phytase supplementation and compensatory growth (early feed restriction followed by a period of realimentation). Based on this information pig diets based on growth stage can be developed to minimize P excretion without compromising pig health, welfare and growth performance.
- New knowledge is also provided with respect to the incidence and onset of joint pathology and lameness in breeding sows.

Main results:
- Reducing phosphorus levels in pig diets reduced P excretion, but also reduced bone strength and growth performance of both slaughter pigs and replacement gilts. Phytase may compensate for this, but not in cases where P restriction is too severe. After a period of being fed low P diets, pigs showed partial compensation in growth performance and bone mineralisation following realimentation, but a long period of realimentation was necessary to accomplish this. Minimum P requirements for optimum bone development are estimated at 5.8-6.7g/kg total P or 3.2-3.7 g/kg digestible P for weaners and 5.5.g/kg total P or 2.8 g/kg digestible P for finishers.
- Multiple parities in sows did not reduce bone integrity in sows, but the incidence of lameness was high (~50%). Lameness was not correlated to joint pathology, suggesting that alternative indicators should be looked at to determine joint pathology. Stiff hind limbs correlated negatively to sow longevity. Early signs of lameness appear as early as after the first service.

Opportunity / Benefit:
Pig farmers are informed of several strategies to reduce P excretion from pigs though dietary intervention. The outcomes of this study enable them to minimize P excretion without compromising pig health, welfare and growth performance. Information on the high incidence of joint pathology and lameness in breeding sows from this study will stimulate further research to identify appropriate management practices to prevent these conditions.

Collaborating Institutions:
UCD

Contact
Peadar Lawlor
Email: Peadar.Lawlor@teagasc.ie.

http://www.teagasc.ie/publications/
1. Project background:
Reductions in dietary phosphorus will reduce the level of P in manure with consequent reduced handling costs and reduced environmental risk. However, there may be consequences for bone strength due to reduced P deposition in bone when low P diets are fed. Reports of an increased incidence of bone fractures in pigs at slaughter and in breeding sows may indicate a related health/welfare problem which merits investigation.

There is concern among veterinarians that bone fractures in live pigs have become more common and that broken ribs before or during slaughter are causing a downgrading of product. This is consistent with data from poultry plants in the US processing chickens fed low P diets. Supplementing diets with phytase has become increasingly common as a method to improve the availability of P in plant ingredients containing high levels of phytate P. Animals subjected to early P restriction followed by a period of realimentation with high P diets in order to promote compensatory growth may also contribute to these problems.

In addition, little information existed on the consequences of low P diets for sow health/longevity. Bone strength is especially important for breeding stock, yet replacement gilts are usually fed diets created for grow-finisher pigs (high protein, low P). Body P stores at first mating are depleted over the following parities and it is inevitable that sows with less initial P reserves are most at risk. Lameness is a common cause of culling in sows and a significant welfare problem. Therefore this condition requires further investigation.

2. Questions addressed by the project:
- What is the effect of dietary P and phytase supplementation on bone characteristics of slaughter pigs?
- What is the effect of dietary P during rearing on bone characteristics of replacement gilts?
- Do multiple parities affect the bone strength of breeding sows?
- Which characteristics of gilts can be used as predictors of sow longevity and lameness?

3. The experimental studies:
Growing pigs were fed diets varying in P levels, crude protein and phytase addition at different growth stages (weaner, finisher or both) in a series of experiments. Effects on nutrient digestibility, P excretion, growth performance, bone mineral density at slaughter and immune response were measured. Periods of low P intake were followed by periods of high P intake to determine if pigs were capable of demonstrating compensatory growth.

In another series of experiments, replacement gilts were fed diets varying in P level at different stages of growth and effects on growth performance and bone strength were measured. Bone strength was determined with the use of a dual energy X-ray absorptiometry (DXA) scanner, which allowed the determination of bone mineralisation in live pigs. Gilts were not served, but rather were slaughtered at 100kg. Periods of low P intake were followed by periods of high P intake to determine if gilts were capable of demonstrating compensatory growth.

Two surveys on bone strength, lameness and joint pathology were carried out on culled sows from a number of commercial farms. The effects of parity number and piglets produced/sow were investigated.

Replacement gilts were monitored for gait and conformation on entry to the breeding herd. They were followed up to first serving or to slaughter.
4. Main results:
Feeding grow-finisher pigs low P diets significantly reduced P excretion, but also compromised growth performance and bone mineralisation. This was found especially during the finisher phase. Minimum P requirements for healthy bone development were determined as 6.4g/kg total P in the weaner stage (11 to 30kg) and 4.4g/kg total P or 2.0g/kg digestible P in the finisher stage (45-100kg). Pigs were capable of demonstrating compensatory growth. If pigs were fed a low P diet (4.2g/kg total P or 1.7g/kg digestible P) up to day 59 post-weaning, followed by a high P diet (5.8g/kg total P or 2.9g/kg digestible P) up to 131 days (slaughter), growth performance and bone strength were similar to that of pigs fed a high P diet throughout their life (6.0g/kg total P or 3.0g/kg digestible P) up to day 59, followed by 5.8g/kg total P or 2.9g/kg digestible P up to slaughter) Including phytase in the diet decreased faecal P excretion, and increased growth performance, compared to diets without phytase inclusion. However, in finisher pigs, phytase inclusion in low P diets (3.7g/kg total P or 1.5g/kg digestible P) resulted in pigs that had a similar carcass weight, but weaker bones than pigs offered medium (4.4g/kg total P or 2.0g/kg digestible P) or high P diets (5.3g/kg total P or 2.5g/kg digestible P) without phytase. Reducing dietary P levels (from 6.0g/kg total P or 3.6g/kg digestible P to 4.2g/kg P or 1.8g/kg digestible P) did not increase susceptibility to the Newcastle disease in pigs from 11kg to 50kgs.

In replacement gilts, the dietary P level also positively affected bone strength in both the weaner (8-26kg) and finisher stage (34-100kg). Based on this, the minimum dietary P requirements for optimum bone development in replacement gilts were determined at 6.7g/kg total P or 3.7g/kg digestible P in the first 4 weeks of the weaner stage, followed by 5.8g/kg total P or 3.2g/kg digestible P in the second weaner stage and 5.5 g/kg total P or 2.8 g/kg digestible P for finishers. In the finishing stage, decreasing the dietary total P from 4.6-7g/kg (2.2 g/kg digestible P) to 3.7-8g/kg (1.6g/kg digestible P) also decreased growth performance. Replacement gilts were not found to be capable of compensatory growth if the deficiency period (weaners: 5.6g/kg total P or 3.0g/kg digestible P for 28 days, finishers: 4.0g/kg total P or 1.6g/kg digestible P for 35 days) was followed by a realimentation period (weaners: 6.3g/kg total P or 3.5g/kg digestible P, finishers: 5.6g/kg total P or 2.3g/kg digestible P) of the same duration. However, in finishers full recovery (compared to a continuous high P diet of 5.6g/kg total P or 2.8g/kg digestible P for 35 days followed by 5.6g/kg total P or 2.3g/kg digestible P to slaughter) was possible if the deficiency period was followed by a realimentation period of twice the length (70 days) of the restriction period.

No evidence was found to suggest that bone integrity in sows reduced due to multiple parities. Stiff hind limbs were negatively correlated with longevity. About half of the surveyed sows (48%) exhibited clinical lameness. Joint pathology was not correlated with clinical lameness.

At entrance to the herd (at approximately 90-100kg), 16% of the gilts showed early signs of lameness, and this increased to 71% after first service. Heavier gilts at entry to the herd had a higher lifetime productivity than gilts that were lighter at entry (group average: 93kg).

5. Opportunity/Benefit:
The findings of this study provide new knowledge on the effect of reducing phosphorus levels in diets on the health and welfare of both grow-finisher pigs and breeding sows. Feeding low dietary levels of P (<6.4 in the weaning stage and <4.4g/kg in the finishing stage) compromises bone strength and growth performance, especially in the finisher stage. Compensatory growth may be used to reduce P levels by about 1.6-2.0g/kg (1.2-3g/kg digestible P) for a short period (up to 59 days) in the pig's life, without compromising pig performance and bone strength at slaughter. However, this is only possible if pigs are given high P levels (5.6-8g/kg P or 2.3-9g/kg digestible P for finishers) during a realimentation period of twice the length of the deficiency period. The severity of lameness and its early onset in young breeding females is evident from this study. Further studies on the prevention of lameness through improved nutrition and management are warranted.

6. Dissemination:
Main publications:


Compiled by: Dr. Lisette Leliveld and Dr. Peadar Lawlor