

Performance during pathogen challenge



WHETHER MAXIMUM PERFORMANCE IS ACHIEVED DEPENDS ON THE COMPOSITION OF THE FEED AND THE ENVIRONMENT IN WHICH THE PIG IS KEPT. BUT HOW IS THE FEED INTAKE AND THUS GROWTH AND PERFORMANCE INFLUENCED WHEN PIGS ARE EXPOSED TO INFECTIOUS PATHOGENS? ILIAS KYRIAZAKIS GIVES US AN INSIGHT.

When pigs are exposed to pathogens, the animals often show a (temporary) reduction in feed intake, henceforth called *anorexia*.

Sandberg *et al.* (2006) have proposed that there are at least two functional mechanisms that may lead to a reduction in feed intake during exposure to pathogens: 1) feed intake is reduced because the potential for protein retention of the challenged animals is reduced, and 2) the reduction in feed intake is a direct consequence of the exposure to pathogens.

Black *et al.* (1999) have argued that both mechanisms may be acting simultaneously. The rate of development of anorexia is greatly dependent upon the type of pathogen to which the pig is exposed and its dose. This has indeed been observed by both Houdijk *et al.* (2007) and Fraser *et al.* (2007) who have shown that low doses of bacterial pathogens such as *Escherichia coli* and *Salmonella enterica* either do not result in anorexia, or pigs need repeated doses of these pathogens for a reduction in feed intake to be observed. Similarly the extent of anorexia is greatly affected by pathogen type and its dose. Higher pathogen doses lead to very dramatic decreases in the feed intake and even complete cessation of feeding (Black *et al.*, 1999).

Duration of anorexia can be very short, as shown in the cases of exposure to *Salmonella typhimurium* (Turner *et al.*, 2002) and the porcine reproductive and respiratory syndrome (PRRS) virus (Greiner *et al.*, 2000). On the other hand, duration of anorexia can last for several days or weeks for gastrointestinal parasite challenges (Hale, 1985).

FEED COMPOSITION

There is some evidence in the literature that the composition of the feed offered to pigs exposed to pathogens may affect the characteristics of anorexia. In experiments by Williams *et al.* (1997a), pigs were offered *ad libitum* feeds of different protein (lysine) contents in environments intended to have either high or low levels of “stimulation of the immune system” (Table 1). This was achieved by exposure to a “dirty environment”, in the absence of dietary antimicrobials and through repeated vaccination. The data suggest that as the lysine content of the feed was reduced the difference in the feed intake between the “control” and “immune stimulated” pigs was reduced; the feed intake of these two groups of pigs was identical at the lowest level of lysine. This effect can be accounted for by the fact that at the lowest level of lysine the feed intake of the “control” pigs was reduced compared to the intake of the control pigs on the highest lysine level. Wellock *et al.* (2003 b) have suggested that the inability of the pigs to increase their feed intake on the low lysine feed is due to their inability to cope with the excess energy intake that

TABLE 1 – THE FOOD INTAKE AND LIVEWEIGHT GAIN OF PIGS THAT WERE KEPT EITHER IN A CLEAN ENVIRONMENT (C) OR A HIGH IMMUNE SYSTEM ACTIVATION ENVIRONMENT (IS). PIGS WERE OFFERED FEEDS WITH DIFFERENT LEVELS OF DIETARY LYSINE CONTENT FROM 6-27KG BODYWEIGHT (WILLIAMS *ET AL.*, 1997A)

	Dietary lysine (g/kg)				
	Immune activation	6	9	12	15
Feed intake (g/d) C		896	1025	1052	1002
	IS	889	954	889	911
	Ratio IS:C	0.99	0.93	0.85	0.91
Gain (g/d)	C	400	556	644	663
	IS	357	495	510	504
	Ratio IS:C	0.89	0.89	0.79	0.76

would accompany this situation. Feed intake of pigs on the lowest lysine level was thus constrained. Stimulation of the immune system would not reduce any further the already constrained actual feed intake.

Different conclusions on the effect of feed composition on the extent and duration of anorexia may be drawn when comparisons between control (uninfected) and infected pigs are made at a weight or a time basis. This can be accounted for by the different growth trajectories of the control and infected pigs, which in turn may have direct effects on their feed intake. Thus, for a fair comparison, control and infected pigs may best be compared at either the same body weight, or through scaling feed intake relative to body weight.

EFFECT ON REQUIREMENTS

In the experiment of Williams *et al.* (1997a) the associated increase in maintenance requirements for protein of challenged pigs was approximately twice as much as that of their controls. The same magnitude of increase was seen in the experiment of Williams *et al.* (1997b) on bigger pigs whose immune system was challenged through the same methodology.

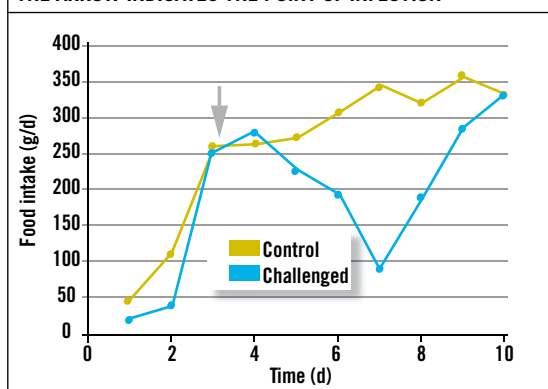
Experiments of Fagbemi *et al.* (1991) and Otesile *et al.* (1991) measured the energy retention of pigs challenged with the same dose of *Trypanosoma brucei* at two different liveweights. The 10 kg pigs had a greater increase in maintenance requirements for energy (2.2 times the healthy controls), than the 100 kg pigs, that had maintenance requirements 1.7 times the healthy controls. Pathogen challenges may also cause increased requirements for protein due to the requirements for the immune response, and the repair and replenishment of damaged tissues. Given that the stimulation of the effector mechanisms of the immune response will depend on the nature of the pathogen challenge, it is reasonable to suggest that both pathogen type and dose will affect maintenance protein requirements. Increases in energy



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requirements during a pathogen challenge may arise from the functioning of the immune system, the cost associated with damage and repair (such as additional N processing) and expression of fever. In the case of gastrointestinal parasites, the most significant energetic cost appears to be associated with damaged tissues resulting in additional amounts of N appearing in the urine (MacRae *et al.*, 1982). Such costs have not been estimated directly on pigs, but would be expected to contribute to a reduced efficiency of feed use.

FIGURE 1 – THE EFFECT OF CHALLENGE WITH 10^8 COLONY FORMING UNITS OF ENTEROTOXIGENIC ESCHERICHIA COLI ON THE FEED INTAKE OF CONTROL AND CHALLENGED PIGS OVER A 10-DAY PERIOD POST WEANING (HOUDIJK ET AL, 2007). THE ARROW INDICATES THE POINT OF INFECTION



Pathogen challenges can cause increased protein requirements due to the requirements for the immune response, and the repair and replenishment of damaged tissues.

GROWTH DURING RECOVERY

Unfortunately, there is a lack of suitable experiments that observed the growth of pigs during recovery from exposure to pathogens. Experiments with feed restricted pigs showed that a period of healthy low feed intake leads to similar body composition changes to pigs whose feed intake has been reduced due to pathogen induced anorexia (Bassaganya-Riera *et al.*, 2001; Escobar *et al.*, 2004). In such instances the pigs will be expected to attempt to restore their fatness to a level determined by their genotype; in other words they would show compensatory fattening (Stamataris *et al.*, 1991). Providing that the period of recovery is sufficiently long, this compensatory fattening should result in the same end lipid weights in the carcass (Kyriazakis and Emmans, 1992). The issue of whether pigs will also show compensatory growth in protein gain and thus reduce some of the time lost during the period of feed restriction is more contentious. Kyriazakis and Emmans (1992) have summarised an extensive body of literature on growth following re-alimentation in pigs and concluded that compensatory growth in protein of skeletal muscle does not seem to exist. In other words, time lost in the growth of muscle protein can never be regained. A compensatory gain in lipid and no compensatory gain in body protein will manifest as compensatory liveweight gain during recovery until body fatness is restored. On the other hand, if the level of fatness has been undisturbed by the exposure to pathogens, then liveweight gain will not show any compensatory gain.

FEED INTAKE AFTER PATHOGEN EXPOSURE

In order to restore any differences in its composition at the point of recovery from the uninfected controls, the pig will need to modify its feed intake. If the pig attempts to show compensatory fattening, as suggested above, then the expectation is that the pig will also increase the rate of its feed intake. The extent of the increase will depend on the level of fatness at the point of recovery (Stamataris *et al.*, 1991). It has not been possible to identify suitably designed experiments that have measured feed intake during recovery in pigs previously exposed to pathogens. This is because, in most cases, the point of recovery can not be clearly identified and the feed intake of animals is reported over a period of time that includes both anorexia and recovery. The rate of feed intake that enables restoration of the body composition will depend on the composition of the feed, the digestive capacity of the pig and the environment it is kept in (Stamataris *et al.*, 1991). An increase in feed intake will also increase the amount of heat production by the pig and for this reason the ambient temperature may define the extent of this increase (Wellock *et al.*, 2003a). In the literature of compensatory growth, the

conditions of realimentation are considered responsible for the confusion that exists over the issue of if and how quickly pigs overcome the abnormalities in their body composition (Kyriazakis and Emmans, 1992). Here it is suggested that the environmental requirements of a pig that is recovering from exposure to pathogens may be very different from an uninfected pig. A special case to consider is the feed intake of the pig that continues to be exposed to pathogens following recovery. This is a realistic scenario, as exposure to most pathogens is a constant feature of commercial practice. On the basis of the arguments developed in previous sections, this stimulation of the immune system will be associated with an increased nutrient requirement, even for a fully immune pig. This increase in requirements will be expected to be manifested as an increase in feed intake. The extent of the increase will depend on the pathogen type and its dose, as discussed above. It has not been possible to identify relevant experiments to support this suggestion. It would be useful to investigate this issue, as it may affect the requirements of a pig continuously exposed to pathogens.

CONCLUSIONS

The effects of pathogens on pig performance are common across pathogen types and doses (e.g. degree of anorexia during subclinical disease). However, to be able to better predict the effect of pathogens on feed intake, growth and performance needs to be understood even more. This can be achieved by closer communication between pig breeders and other animal scientists, such as nutritionists and veterinarians. Similarly, the characterisation of the pig's immune response has been a consistent limitation in our understanding of how animals deal with pathogens and has had implications upon our ability to enhance them. For example, under what circumstances can the immune response of a pig be enhanced through nutrition? Recent advances in the characterisation and quantification of the immune response should enable progress on this subject to be made.

In addition, consumer concerns, legislation and pathogen resistance to drugs will probably increase the interest in the understanding of the performance of animals in the presence of pathogens. A framework that accounts for the performance of pigs during exposure to pathogens may then have a value in the developments of strategies, including nutritional and breeding ones, to deal with this challenge. <-

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