Chapter 12

ANOREXIA-LIKE WASTING SYNDROMES IN PIGS

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ABSTRACT In the last decades demand for high quality pork meat has led to the development of modern intensive pig production methods. As a result of the modification in the breeding, feeding, housing and management, modern intensively raised pigs have become more sensitive to social stress. The grouping imposed in many industrial pig units has consequences for feeding behaviour, feed intake, growth and the health status of pigs. Pigs, in particular those that have been bred for the purpose of extreme leanness, can develop irreversible self-starvation and emaciation. Anorexia in pigs develops mainly post-weaning as the wasting pig syndrome (WPS) or after farrowing as the thin sow syndrome (TSS). The clinical features of these syndromes show an uncanny resemblance to those of anorexia nervosa of humans. The aim of this chapter is to present WPS and TSS as possible animal models of disorders of eating and body composition in humans. WPS and TSS are related mainly to social and environmental stressors that occur during the very critical periods of lactation and weaning, and are widespread within some modern intensive pig husbandry systems

INTRODUCTION

During the last decades the demand for the production of high quality pork meat has risen worldwide, a result of the increase in the world population and greater consumer demands. This led to the development of modern intensive pig production methods, where the science and the practice of production has changed dramatically (Whittemore, 1998). Approximately one billion pigs are raised worldwide and pork is the dominant meat source, representing 40% of the total quantity of the meat consumed (Rothschild and Ruvinsky, 1997). As a result of the improvement and modification in the breeding, feeding, housing and

management of pigs, modern intensively raised pigs are becoming more and more sensitive to social stress (Andersson, 1988).

The pig is a social animal by nature, but the grouping imposed in many industrial pig units are not those occurring in the wild life. It has been established that grouping has consequences on feeding behaviour, feed intake, growth and health status of the individual animal within the group (Morgan et. al., 1999). Pigs, in particular those that have been bred for the purpose of extreme leanness, can develop irreversible self starvation and emaciation (Treasure and Owen, 1997). Anorexia in pigs develops mainly post-weaning as the wasting pig syndrome or after farrowing as the thin sow syndrome (Kyriakis and Andersson, 1989, Kyriakis, 1991). The clinical features of these syndromes show an uncanny resemblance to those of anorexia nervosa (Treasure and Owen, 1997), a condition in which people, mostly women, would voluntary starve themselves (Gull, 1874).

In addition, the pig, because of its physiological and genetic similarities to man, serves as an excellent animal model for medical research (Rothschild and Ruvinsky, 1997). Pigs may be a useful model for studying the habits of eating in humans and / or digestive disorders because the species are roughly comparable in body weight and have a similar digestive process, being omnivorous and simple-stomached (Andersson, 1996). The aim of this chapter is to present the wasting pig syndrome (WPS) and the thin sow syndrome (TSS), both of which occur in modern intensively raised pigs, as possible animal models of disorders of eating and body composition in humans. WPS and TSS are related mainly to social and environmental stressors that occur during the very critical periods of lactation and weaning, and are widespread within some modern intensive pig husbandry systems (Treasure and Owen, 1997).

Before the actual presentation of WPS and, it is useful to consider some aspects of the genetics of behaviour, in particular feeding behaviour and the role of stress on growth and reproduction. These are essential to the understanding of underlying mechanisms involved in the appearance of both syndromes.

GENETICS OF BEHAVIOUR

The wild pigs of Asia and Europe were selected for domestication mainly because of their behaviour. While there was considerable variation in the behaviour of the pig within the species, the behavioural traits that favoured domestication included their omnivorous dietary needs, their relatively weak maternal-neonatal bonds, their precocial nature and their general adaptability (Ratner and Boice, 1976, Rothschild and Ruvinsky, 1997). It is known that the oldest breeds of domesticated European pigs and the present commercial pigs share many behavioural characteristics (Mitichashvili et. al. 1991). Over the years, looking at the species in terms of possible eating disorders, we can see a gradation from: (a) the primitive wild pig, which is omnivorous and with an apparently well controlled

appetite mechanism, allied to a relatively lean body, to (b) the domesticated pig that was obese until the middle of the 20th century and to (c) thereafter, a progressively leaner animal bred to satisfy the modern consumer in developed economies (Owen, 1998).

The pig as a species, is known for its relatively copious feed intake. Pigs can reach feed intakes of 5% of their body weight per day, which exceeds the level of feed intake of most farm animal species (McGlone et. al., 1997, NRC 1998). If feed intake could be increased in young animals, it would improve the rate of weight gain and the feed efficiency. Thus, clearly there is a great economic reason to increase feed intake in the present domesticated lean lines of pigs. It is noticeable that the genetic basis for the control of the average daily feed intake (ADFI), has been less well studied than the closely correlated trait, average daily gain (ADG) (McGlone et. al., 1997). Furthermore, as a result of the intensive selection, which is applied among some modern lines of pigs for meat production, multiple selection goals may not result in increased feed intake, especially if ADG is emphasized. For example, with intensive selection for both less body fat and increased weight gain, feed conversion tends to improve, and interestingly, feed intake deteriorates (Vogeli, 1978, McGlone, 1997,).

The different problems of feed intake on commercial farms may imply genetic variation and thus, are amenable to change. One example is the adult sow whose feed intake is too high, which means wasting of feed and, if left unchecked, excessive body fat and body size. Limiting feed intake is the only solution to overcome this condition, but on the other hand it requires resources and alters the sow's behaviour (McGlone, 1997). This unusually high weight loss, especially when it takes place during lactation, is often followed by sow's failure to regain weight (Kyriakis, 1991). This condition is known worldwide as the thin sow syndrome (Kyriakis et. al, 1990, Kyriakis, 1991).

Another problem of feed intake occurs in the postweaning feeding period in young pigs. Pigs usually are not fond of dry feed in the hours shortly after weaning. Gradually, over the first few days and weeks after weaning, feed intake increases to reach the species' typical level (McGlone, 1997). Unfortunately, some pigs do not adapt in the new situation and develop a chronic stress syndrome. These pigs are known as wasting pigs or unthrifty pigs, and the pathological situation is known as the wasting pig syndrome (Kyriakis and Andersson, 1989).

Besides, changes in the feeding behaviour due to selection for modern lean pigs have possibly not only revived the 'primitive' leanness alleles, but also dredged up side effects known as the halothane gene (halothane-induced malignant hyperthermia). The halothane gene is associated with leanness, sensitivity to stress, and porcine stress syndrome. This condition can be observed in both pigs and humans. This gene, or the genes nearby, sensitizes animals to stress, sometimes affecting appetite and reproduction. The halothane mutation is due to a mutation in the calcium release channel (CRC) gene on chromosomal 6 of the pig, leading to a defect in calcium regulation (Fujji et. al., 1991). The symbol CRC is used for the locus encoding the skeletal muscle sarcoplasmatic reticulum,

calcium release channel, also known as the ryanodine receptor (Andersson et. al 1994).

Body composition shows a high heritability based on the segregation of a single gene with major effects as happens with the halothane gene. On the other hand there is also a significant, though somewhat lower, level of heritability for the feed intake. Body composition is the primary controlled trait and feed intake variation is a dependent secondary effect (Owen, 1990). Finally, body composition is reviewed as a composite of several traits, each with their distinctive genetic basis, including major effects of genes at single loci. Studies involving twins, adopted offspring and other family relatives have demonstrated the high heritability (0.4-0.7) of body composition in many of the studies involved. Genotype-environment interactions with diet and activity occur in domesticated animals and humans and associations with voluntary choice of diet and level of activity are unfavourable. Body composition is the main reference for a normal homeostatic mechanism involving appetite and energy expenditure control (Owen, 1999).

FEEDING BEHAVIOUR

Growing pigs in modern production systems are kept in groups of similarly aged animals, sometimes of the same sex, in contrast to the "natural" groups, which included pigs of both sexes, young and old (Morgan et al, 1999). In particular, this stage of production takes place 3-4 weeks postpartum, when piglets are weaned (Whittemore, 1998). It has been recognized that such grouping can affect the production performance of individuals within the group (Morgan et. al, 1999). It is important to provide some information regarding the feeding behaviour of pigs housed within groups, because the wasting pig syndrome examined in this chapter is a condition observed in group-housed pigs. Moreover, the feeding behaviour forms the link between other social behaviours as well as between feed intake and growth.

The feeding behaviour of pigs housed within groups has been found to differ significantly from that of individually penned pigs. Within the social environment, growing pigs eat substantially fewer, but much larger meals than individually housed animals (De Haer and Merks, 1992). Besides, pigs within groups grow less quickly than we would expect, because our estimations are based on data from individually housed pigs (Morgan et. al., 1999). One possible explanation for the reduced growth rate is based on the proposition that pigs are more active in a group than when kept individually, because they interact with each other (Black, 1995) and the energy normally used for growth is therefore diverted to meet the demands of activity. Another explanation is that housing pigs in close proximity to each other within groups affects the thermal environment of the individual which may prevent effective loss of body heat. This results inreduction in feed intake. It is obvious that there are differences in the behavioural constraints related to the social

environment, and in some ways social living affects the feed intake and growth.

THE ROLE OF STRESS ON GROWTH AND REPRODUCTION

Social stress is common on commercial pig units, mainly at the time of weaning of pigs and sows and during the first period of growth. Therefore, several stress induced diseases and stress related syndromes are now recognized in the pig. Before presenting the wasting pig syndrome and the thin sow syndrome, which are stress related, it is important to examine the role of stress on growth and reproduction (Kyriakis, 1989). In the terms of behaviour and welfare, stress occurs as the consequence of the restraint which is applied at intensive pig production units, and in particular, as it is seen in stall-housed pregnant and lactating sows and in common stocking densities among weaning and growing pigs (Whittemore, 1998).

Stress is an adaptive phenomenon, which represents a psychophysiological reaction to the external stimuli or stressors (Kyriakis, 1989). The major stressors are disease, injury and subjection to aggression. Stress may also occur as a result of other environmental problems, for example at weaning, when piglets are removed from the sow to another building and they are mixed with other piglets (Whittemore, 1998). A stimulus becomes a stressor by affecting directly the individual through a sensory or metabolic process, which is stressful itself. Such stressors elicit a response via lower brain sensory mechanisms without involving higher interpretive brain centers. A stimulus can also become a stressor by virtue of the cognitive interpretation of meaning that the individual assigns to the stimulus. Thus, if an individual interprets or perceives some stimulus as being aversive or challenging, stress response will be elicited (Everby and Rosenfeld, 1981). In either case, the stress response is one of pituitary adrenal activation resulting in release of the pituitary and medullary tropic hormones ACTH, cortisol, epinephrine, norepinephrine and dopamine. Short bursts of adrenal cortical and medullary output are beneficially adaptive, but chronic hyperactivity of the pituitary-adrenal axis is regarded as maladaptive response. Stress has been linked to adrenocortical function and resultant acceleration of vascular aging in several species including the pig (Brambvell, 1965, Henny and Stephens, 1987).

In particular, one possible underlying mechanism that is responsible for the observed effects is the involvement of stress in growth regulation (Chapple, 1993). Reduced protein deposition, which results in growth retardation is due to the stress of the pig resulting from the need to maintain its social position within the group. This stress is acting via biochemical factors directly on tissue growth through growth hormone, cortisol insulin-like growth factor and cytokines (Morgan et. al., 1999). A reduction of circulating growth hormone could be expected to reduce protein deposition (MacRae and Lobley, 1991, Morgan et. al., 1999)

although the mechanism by which stress should cause such a reduction of the growth hormone secretion is not clear. Cortisol increases proteolysis to supply amino acids for gluconeogenesis (Oliverio, 1987). Cytokines have also this effect and they additionally reduce the feed intake (Grimble, 1993). Both cortisol and cytokines also stimulate lipolysis. and chronic treatment of growing pigs with pharmacological doses of corticosteroid resulted in suppression of the growth rate (Oliverio, 1987, Grimble, 1993).

Another possible underlying route, through which socially induced stress might influence nutrient demand and growth rate, is through its influence on the animal's immune status. McGlone et. al. (1993), showed that low ranking pigs suffered a decrease in natural killer cell cytotoxicity following transport, in comparison to dominant pigs. Therefore, stressed pigs of low social status might be more susceptible to infections which in turn, could lead to decreased growth via clinical or subclinical pathogenic routes (Johnson and von Borell, 1994). In addition, pigs with a lesser activity of the immune system, due to medicated early weaning, have a lower ratio of T helper/T suppressor cytotoxic cells, a greater capacity for protein growth and, consequently higher amino acid requirements, than conventionally reared pigs. Furthermore, the administration of an inactivated Parapoxvirus immunomodulator (Baypamun), wasting pig syndrome, which is stress induced (Kyriakis et. al., 1998). Baypamun triggers some non-antigen mediated mechanisms of the immune system such as interferon production, and cytotoxic activity of natural killer cells. Additionally it improves the reproductive performance of gilts (young breeding females), mainly when they are exposed to transportation stress (Kyriakis et. al., 1996).

Finally, one outcome of high stress is the reduction of reproductive efficiency. Some manifestations of stress are reduced litter size, failure of oestrus, poor conception rates and sporadic attainment of puberty. In stressed young growing animals, the endocrine changes outlined augment their ability to cope with stress, but in a reproducing female there is a cascade of other hormonal events which follow. These secondary events can lead to ovarian dysfunction, complete infertility and embryonic death. Ovarian function depends upon a sequence of finely orchestrated endocrine phenomena and their disruption results in endocrine chaos. Adrenal hyperactivity is associated with asynchrony of the principal reproductive events. One mechanism by which stress influences reproduction is via ACTH, which directly suppresses pituitary response to gonadotrophin releasing hormone (GnRH) or decreases its secretion from the hypothalamus and this action is mediated by the adrenal gland (Varley and Stedman, 1994)

WASTING PIG SYNDROME (WPS)

Social and environmental stressors during the critical period of weaning frequently not only affect the performance but also the health status of the piglets. Pigs unable to cope with the changes associated with

weaning, develop a condition characterized by retardation of growth and a listless appearance, known as the wasting pig syndrome (WPS) (Kyriakis and Andersson 1989). There are many possible negative factors (stressors) to which a pig can be exposed in connection with weaning, and they could be divided into different categories: a) the fact that weaning demands a nutritional change. Prior to weaning, piglets have access to hourly meals of high quality, readily digestive ingredients consisting of simple carbohydrates, proteins and fats provided by the sow's milk (Pond and Maner, 1984). Although piglets voluntarily consume solid feed in appreciable quantities they still rely on milk as an important food source for a long time (Algers et al., 1988). On the other hand piglets are forced to adapt to a different diet which leads to dramatic changes in their enzyme activity (Hefner, 1987). b) The need to utilize the feeders more may increase the frequency of agonistic interactions between littermates. particularly if feeding space is limited, and, c) There is also an effect because of the change of the environment, if the pigs are moved after weaning and d) the removal of the mother itself will probably cause a broad spectrum of various psychological strains (Algers et. al., 1988).

One of the most important negative factors that a pig is exposed to is the mixing of pigs after weaning. In modern pig practice, disturbances in the social hierarchy are purposely introduced. When pigs from different litters are put together for the first time they start fighting, and over the next 24 to 48 hours a new dominance hierarchy is established (Andersson, 1988). The consequences of fighting among pigs are severe. Aggression among newly weaned pigs mixed together into broken family groups can reduce the growth of all the pigs in the pen for a period of up to 2 weeks while the new hierarchy is established. If pigs of equal strength and dominance meet repeatedly, individual animals can be severely affected. Pigs demoted in the social order by fighting may subsequently fare less well, may be denied full access to feeders, and fail to thrive. These pigs are not only inefficient in their growth, but they are also susceptible to diseases and are therefore, a threat to the whole group (Whittemore, 1998).

During the period of rank order establishment, there is a strong activation of the pituitary-adrenal axis. As already mentioned short bursts of adrenal cortical and medullar output are beneficially adaptive but chronic hyperactivity of the pituitary-adrenal axis is regarded as a maladaptive response, which leads to elevated levels of circulating corticosteroids, depression of the immune system and secretion of catecholamine (Andersson et. al., 1986, Fabiansson, 1986). Because pens in modern pig housing do not offer hiding places or refuges during aggression, the pigs may stay in a state of constant stress during the period of rank order establishment. Clinical signs of a stress response following immediately after mixing of pigs are a reduction in the growth rate, feed intake and feed conversion efficiency. Within a few weeks, most of the pigs adapt to the new situation and social order, so that their feed utilization and body-weight increases are normalized. However, some pigs do not recover from the initial growth restraint and the inability of these

pigs to cope with the new situation causes severe stress responses with serious health consequences. Those animals become wasting piglets, a chronic form of the stress impaired syndrome known as WPS (Fabiansson, 1986, Andersson, 1988)

WPS may have economic importance in many large commercial pig farms. Usually, it occurs 2 weeks post-weaning, and up to 7% of all weaners may exhibit a decrease in weight gain of more than 50% when compared with normal piglets of the same origin and age while maintained in the same environment. The typical clinical signs of pigs suffering from the WPS are a much reduced weight gain, prominent dorsal spine and increased hair coat. Haematological studies have shown a lowered plasma activity of alkaline phosphatase and a decreased plasma concentration of zinc, frequently combined with a non-specific eczema of the back of the wasting pig (Martinsson et. al., 1976). The hyperactivity of the pituitary-adrenocortical axis was indicated by the observation of hypertrophied adrenal glands (Martinsson et. al., 1978). The increased plasma concentration of non-bound cortisol in wasting pigs (Albinsson and Andersson, 1988) indicates that an anti-anabolic state may be present in these pigs, i.e. amino acids are directed into the gluconeogenetic pathways at the expense of normal protein metabolism. This means that ingested feed is preferably used for energy production instead of protein synthesis, thus making feed less available for body growth (Kyriakis and Andersson, 1989). Furthermore, the finding of thymus gland atrophy (Martinsson et. al., 1978) may be indicative of a stress-induced deterioration in immune function (Hara et. al., 1981). Therefore, resistance to diseases is diminished, mortality is high and poor growth/feed efficiency follows the pig up to the age of slaughtering (Fabiansson, 1986).

Before analyzing aspects of the prevention and treatment of the WPS, it is important to mention two other stress induced diseases of the early weaned piglet: Non Infectious Diarrhoea and the Post Weaning Diarrhoea Syndrome. Weaning of piglets at the age of 3-4 weeks, often leads to the development of non infectious diarrhoea, followed by the malabsorption syndrome (English et. al., 1978, Kyriakis, 1984). The malabsorption syndrome is caused by the stress of weaning and the simultaneous change of diet. Normally, this results in the following digestive alterations, to a greater or a lesser degree: 1) an increased excretion of fatty acids in the faeces, 2) an increased output of carbohydrates in the faeces, 3) watery faeces. 4) degenerative changes in the villus structure of the small intestine such as reduced number of enterocytes and decline of brush border enzymes (English et. al., 1978, Kyriakis, 1981). In most cases, opportunistic pathogens take advantage of the presence of non-infectious diarrhoea and the malabsorption syndrome. The most common pathogens which cause post weaning diarrhoea syndrome are enterotoxigenic strains of E. coli and Rotavirus, and as result of this syndrome there is a high fluid /electrolyte secretion from the small intestinal mucosa into the lumen of the intestine, severe diarrhoea, biochemical abnormalities and dehydration.

As already mentioned, a number of different social and environmental stressors may contribute to the development of WPS in early weaned pigs. In order to reduce these stressors to the minimum, efforts must be made regarding the areas of housing, nutrition and hygiene (Kyriakis, 1989). The housing requirements for the early weaned pig are already well known and established (Brent, 1985). The final composition of the diet after weaning must be based upon current knowledge of physiopathology of the digestive system of the piglet in addition to nutritional requirements during the same period (English et. al., 1978, Whittemore, 1999). The health status of the weaned piglet is also related to the hygiene standards which are set in the weaning house starting from the 'all in all out' husbandry system, proper disinfection techniques, moving and regrouping piglets in the correct way, using low densities of animals to reduce the rate of diseases and controlling the number of visits to the minimum (Taylor. 1999). Furthermore, other prevention and therapeutic measures includes (1) vaccination against specific infectious diseases which help the early weaned piglets to overcome health problems, (2) the use of antimicrobial prophylactically agents applied, either therapeutically, metaphylactically and (3) fluid electrolyte supporting therapy based upon the use of glucose, sodium bicarbonate and vitamin C added to the drinking water (Kyriakis, 1983, 1984, Taylor, 1999).

One interesting feature regarding the treatment and the prevention of WPS is that the syndrome is frequently successfully treated with amperozide (Kyriakis and Andersson, 1989). Amperozide (chemical name: 4-[4,4-bis(p-fluorophenyl)butyl]-N-ethyl-1-piperazinecarboxamide) is a typical psychotropic drug with specific effects on nerve transmission in the limbic part of the brain (Pettersson et. al., 1987). Neurochemical studies have shown that amperozide has very weak affinity for the postsynaptic dopamine receptors in striatum but is a potent antagonist on 5HT2 receptors in the limbic part of the brain (Svartengren and Christensson, 1985). Behavioural studies have shown effects on dopaminergic and serotonergic nerve transmission in the limbic system. Furthermore, amperozide has potent anxiolytic properties as evident from its effects on emotion behaviour in a number of animal conflict models. The most prominent effect of amperozide however, indicating the limbic profile of action, is its effect in various experimental models of aggression. The total lack of interference with motoric skills and the absence of ataxia, underline the selective effect of amperozide on aggressive behaviour. The effect of amperozide markedly differs from that of azaperone and acepromazine whose effects on aggression are due to unspecific sedation and motoric impairment (Christensson Gustafsson, 1985, Andersson et. al., 1987, Björk et. al., 1988). Finally, amperozide seems to interact with central neuroendocrine processes involved in the regulation of stress reactions (Andersson and Albinsson 1985).

Kyriakis and Andersson (1989) studied the effect of amperozide in an animal model exhibiting hyperactivity in the pituitary-adrenocortical axis, as is the case of WPS. The study was carried out at the largest commercial

pig farm in Greece with around 3000 sows in production. A total of 100 homebred hybrid piglets at the age of 25-30 days, suffering from typical WPS were used in the study. The pigs were weaned at 17-20 days by moving them from the farrowing pens and mixing them in the flat decks on the farm. At the time of weaning all piglets in the trial were clinically healthy. Half of the pigs were treated orally with amperozide (2,5 mg/Kg) body weight) and the control group was treated with long-acting oxytetracycline and vitamins (A+D3+E). There were significant improvements in average daily weight gain (P < 0.05) and feed conversion ratio (P < 0.01) in amperozide treated pigs compared to controls. Furthermore, there was a significant reduction (P < 0.05) in mortality in amperozide treated groups. Within 2 weeks following amperozide treatment the clinical signs of the WPS had disappeared and the pigs grew at the same rate as the healthy ones (Kyriakis and Andersson, 1989).

THIN SOW SYNDROME (TSS)

The thin sow syndrome (TSS), first reported over 30 years ago (MacLean, 1968) occurs particularly in stalled sows of lean breeds, in which weight loss occurs in lactation and early pregnancy, followed by failure to regain weight. This may become progressive and result in poor fertility and eventually in emaciation and death. The syndrome is also not uncommon in loose housed sows where individual feeding is not practised (Taylor, 1999). In Denmark, it was found that about 30% of sows in one abattoir were 'thin sows' (Kristensen, 1982).

The aetiology may be related to a combination of causes such as parasitism, low environmental changes, inadequate and/or inappropriate feed intake during lactation (Taylor, 1999). Nevertheless, in many field cases when all of the above factors are not present, the thin sow syndrome still occurs causing economic problems in farms using intensive methods of production. Thus, the syndrome may have an aetiology related mainly to social and environmental stressors during the very critical period of lactation, especially in the cases of large litters (Kyriakis et. al., 1990).

In the terms of behaviour and welfare, stress is often perceived as the consequence of the restraint of animals which occurs in intensive production units. Particular restraints are seen with tethered and stall-housed pregnant sows which cannot turn around, and have limited (less than 1m) forward and backward movement, and also with lactating sows similarly restrained in farrowing crates. On the other hand, increased movement freedom levels may themselves bring stress, particularly when adult sows are placed into groups. Adult sows may fight vigorously when placed into novel social groups, as is often the case after weaning. As such fighting may also occur around the time of oestrus, and during the first 3 weeks of the pregnancy, losses of embryos can result in the reduction of litter size. Sow groups are often formed with one dominant sow, which eats greedily and becomes large and fat. There is also likely to be one or two subservient sows which actively avoid competitive feeding situations

and, in addition to being wounded and bruised, may become emaciated (Whittemore, 1998). The underling mechanisms of stress at the individual animal level are similar to those previously described (wasting pig syndrome).

All phases of the reproductive cycle of a sow are interrelated and therefore the feeding programme in one phase can have significant effects on the sow's performance and appetite in another phase. It has been shown that feeding levels during gestation that promote backfat levels at parturition of 20mm P2 (P2 backfat is measured at the level of the last rib 65mm from the mid-line of the sow) or more, may result in reduced sow feed intake during lactation, especially in early lactation. High level of feed intake during gestation will either decrease insulin secretion during lactation or increase insulin resistance. This may reduce peripheral glucose utilization and reduce mobilization of fat stores, which in turn will reduce feed intake. The resulting reduced feed intake during lactation will lead to an increased weight and condition loss of the sow (Ahern, 1999).

The clinical signs of the syndrome include emaciation in 30-90% of sows and boars in a herd, associated with hypothermia (35.5-38 °C), deprayed appetite, restlessness, apathy and later on difficult in rising. The skin may be dirty and greasy and there may be surface abrasions. As the condition progresses, failure to return to oestrus and permanent infertility may occur (Taylor, 1999). This condition also includes features such as overactivity, and consumption of non-nutritive substances, for example straw (Owen, 1995). Analysis of blood and faeces from 'thin sows' did not show any significant changes, compared to normal sows. It was concluded that the clinical and the haematological changes had similarities to the situation of hyperfunction of the thyroid gland (Kristensen, 1982). Also, in many clinical cases of this syndrome, renal vascular lesions, unrelated to parasitism, have been identified and this is believed to be a stress induced situation (MacLean, 1968). There is much present interest in the possibilities for systems of production which increase potential movement and space whilst avoiding some of the new stressors which may be associated with it. Such stressors will usually include: (1) avoidance of injury and disease, (2) generous provision of feed and water, (3) small group size, (4) solid flooring for at least a part of the pen, (4) use of bedding (straw), (5) low density stocking, (6) individual feeding for breeding sows and stability of animal groups by obtaining constancy of individuals in the group (Whittemore, 1998). Furthermore, environmental temperatures should be restored to normal and anthelminthic treatment should be considered (Taylor, 1999).

It is of great importance to ensure that adequate quantities of feed are given to all affected animals (Taylor, 1999). It has been suggested that gilts and sows should be bred with 16 to 17 mm P2 backfat and should remain with roughly that level of P2 backfat at successive weanings. This could be achieved by having sows of all parities with about 19mm backfat at farrowing. These sows could then lose 3mm backfat during lactation and still have 16mm backfat at weaning. The nutrient and energy requirements of a gestating sow will depend on her weight, backfat level

and the amount of weight gain she will need during gestation to meet a target level of 19mm P2 at farrowing. From the data available (NRC, 1998, Whittemore, 1998) reasonable estimates of the energy, protein and lysine requirements of gestating sows can be calculated (Ahern, 1999). Finally, lactation feed requires to be provided at adequate levels. Lactating sows need to eat as much as they can, but in many cases even that is not enough (Whittemore, 1999).

As we have already mentioned TSS has an aetiology which is related mainly to social and environmental stressors during the very critical period of lactation, especially in cases of large litters. Therefore, the syndrome may be related to the wasting pig syndrome (WPS) another stress induced disease (Kyriakis et. al., 1990). The observation that in the case of WPS amperozide was used with very good results (Kyriakis and Andersson, 1989) lead Kyriakis et. al. (1990) to investigate the effect of amperozide on the weight gain and health status of sows with TSS. The field study was conducted in a commercial pig farm in Greece, with around 3000 sows in production. The prevalence of the thin sow syndrome is approximately 6% of the sows at post-lactation. The farm follows the genetic programme of a very well known, European hybrid brand name of lean breed. Sixty sows, suffering from typical postweaning thin sow syndrome, were divided into three groups: (1) 20 as negative controls (NC), (2) 20 as positive controls (PC) treated with vitamins, trade elements and antibiotics and (3) 20 injected with amperozide (2mg/kg body weight). Amperozide treated sows fully recovered (P< 0.05) and 80% became pregnant, while figures for the PC and NC groups were only 15% and 10% respectively. Mortality was up to 50% in the NC, 40% in the PC and only 15% in the amperozide treated group (P<0.05) (Kyriakis et. al. 1990).

Additionally, Kyriakis (1991) conducted another trial to determine the effect of amperozide on sow-litter productivity while it was used in sows at farrowing and, or, at weaning. The study was conducted in a commercial pig farm in Greece with 400 sows under production. The breed of sows was F1: Landrace cross Large White and the sows were served by Belgian Landrace boars. A total of 64 sows divided into four groups: (1) 16 as untreated controls, (2) 16 treated with a single dose of amperozide at farrowing, (3) 16 treated with a single dose of amperozide at weaning and (4) 16 treated with two single doses of amperozide at both farrowing and weaning. No clinical outbreak of mastitis, metritis, agalactiae complex was recorded in the amperozide groups dosed at farrowing. The number of "empty" days was decreased (about three days) in sows treated with amperozide at weaning. Furthermore, pre-weaning mortality decreased (8 %) and piglet growth rate improved when sows were treated with amperozide at farrowing (Kyriakis, 1991).

The results from both studies suggest that amperozide treats TSS and improves the health status and productivity of sows by reducing their emotional responses to novel or threatening situations (Kyriakis et. al., 1990, Kyriakis, 1991).

ANIMAL BEHAVIOUR AND ANOREXIA NERVOSA – CONCLUSIONS

Eating disorders are currently increasing alarmingly in the Western developed nations (Goodrick et. al., 1996). It is commonplace to refer to the epidemic of eating disorders in Western women but few are aware of the parallels on the farm and especially at the modern pig intensive production (Treasure and Owen, 1997). As we have already described, pigs, in particular those which have been bred for extreme leanness, can develop irreversible self starvation and emaciation (MacLean, 1968, Kyriakis and Andersson, 1989, Kyriakis et al., 1990). Anorexia in pigs develops after weaning as the wasting pig syndrome (WPS) or after farrowing and/or weaning as the thin sow syndrome (TSS). WPS and TSS are related mainly to social and environmental stressors during the very critical period of lactation and weaning and are widespread under some modern intensively pig husbandry systems. In the largest pig farm in Greece with around 3000 sows in production, stocked with a very well known European hybrid brand name of lean breed, the prevalence of the condition is 6% (Kyriakis et al. 1990).

The clinical signs observed in both the two syndromes show an uncanny resemblance to those of anorexia nervosa. In particular affected animals restrict their normal feed intake, although some consume large amounts of straw. The affected animals spend more time on non-nutritive hyperactive behaviour and they are easily recognized as they develop a prominent dorsal spine and their hair becomes coarse and long and many of them do not return to heat. Few abnormalities are found at post-mortem examination and these include enlargement of adrenal glands and thymus atrophy. The early separation of sow and piglets and mixing of unacquainted pigs appear to be the trigger. When pigs from different litters are placed together for the first time they start fighting and over the next 24 to 48 hours a new dominant hierarchy is established. MacLean (1968) in his detailed study reported the possibility of a hereditary relationship because certain family lines were more severely affected than others (MacLean 1968).

Having described WPS and TSS the outcome question is whether WPS and TSS could serve as an animal model for anorexia nervosa. There have been recent proposals to simplify the diagnostic criteria of anorexia nervosa, as it is recognized that the features of the psychopathology vary across time and cultures. A proposed definition included the following: "subjects who become emaciated through restricting their dietary intake for whatever reason, this restriction is deliberate, and the resulting state is positively valued by the subject" (Szmuckler and Patton, 1994). Another proposed description says: "the patient avoids food and induces weight loss by virtue of a range of psychosocial conflicts whose resolution she perceives to be within her reach through the achievements of thinness and/or the avoidance of fatness" (Russell 1994). Omitting the subjective attribution of motivation,

it could be argued that wasting pigs and thin sows fulfill these broader criteria (Treasure and Owen, 1997).

Accepting this proposition it can be said that there is an intriguing possibility that the condition has an analogous genetic basis in both species and that the condition has only emerged in pigs since the relatively recent intensive selective breeding for leanness (Treasure and Owen, 1997). Owen (1990, 1992) has argued, drawing evidence from farm animals, that anorexia nervosa may rise primarily as an extreme variant of body composition. Farmers selectively breed their stock to optimize carcass composition. In recent years, particularly in pigs, consumer aversion to fat has meant that leanness has been favoured over fatness. Such selective breeding has led to the uncovering of recessive traits that produce extremes like the halothane gene. The halothane gene is of interest because it is associated with leanness, sensitivity to stress and porcine stress syndrome and the condition is seen in both pigs and humans. This gene or genes nearby sensitize the animal to stress, and sometimes affect appetite and reproduction (Treasure and Owen, 1997). Finally, as we have already mentioned, body composition is reviewed as a composite of several traits each with their distinctive genetic basis, including major effects of genes at single loci (Owen, 1999).

One interesting feature regarding the treatment and/or the prevention of WPS and TSS, is that the syndrome is frequently successfully treated with amperozide (Kyriakis and Andersson, 1989, Kyriakis et. al., 1990, Kyriakis, 1991). It is not surprising that a drug that acts on central serotonin (5HT) has been found to be effective, suggestive the pivotal role of serotonin (in particular 5HT2 receptors) in central control of appetite. 5HT, is also implicated in the central control of locomotor activity and sexual behaviour, both of which are components of the clinical syndrome of anorexia nervosa. Therefore, abnormalities in central 5HT may result in develop anorexia nervosa. Unfortunately. vulnerability to pharmacological approaches to treatment of anorexia nervosa have failed to impress clinicians with their benefits. However it remains likely that psychotherapy will be essential to understand the difficulties that produce the stress response in order to help the patient develop more effective coping strategies and to combat the pervasive value placed on thinness in our society. It is ironic that pigs, which are not renowned for their anorexic eating attitudes, may stimulate several researches leading to determination of the etiology of anorexia nervosa (Treasure and Owen, 1997).

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