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Skeletal Biology and Sow Longevity

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Summary.

A healthy skeletal system is essential for the longevity of a sow in a breeding herd. The purpose of this paper is to examine areas of research that may provide useful information for maintaining or improving the sow's skeleton. Genetic selection in many livestock species has not focused on skeletal health. Thus, leg problems have increased. As an example, the improved growth rates are associated with increased incidences of skeletal diseases such as osteochondrosis. Slowing growth can ameliorate those problems. In humans exercise is an important strategy for maintaining bone density, but in the livestock industry restricted movement (such as gestational crates) can be extensive. Also, the toll that continual involvement of a sow in the reproductive cycle has on the skeleton is not known. Fortunately, many new diagnostic tools are available to monitor bone health and new biotechnologies and nutraceuticals are being developed that may benefit the reproducing sow. If sow longevity is a priority, improving both bone and cartilage health is a requirement.

INTRODUCTION

Due to the adverse impact that skeletal diseases, such as osteoporosis and osteoarthritis, have on society, our understanding of skeletal biology has rapidly accelerated in the last 20 years. At the same time, skeletal health has become an issue of increasing concern in livestock and companion animal industries. Lameness is a significant problem with gilts and sows that will lead to premature culling. Others have identified skeletal problems as an important cause of mortality in breeding herds (Christensen et al., 1995). Keeping a reproductively active sow in the herd can be of substantial economic benefit, even if it delays the integration of improved genetics (Faust et al., 1993a, b). The purpose of this presentation is to highlight areas of skeletal biology that might provide useful information for those in the swine industry working to improve sow longevity.

GENETIC SELECTION/ STRUCTURE

Genetic selection has a played a critical role in improving growth efficiency, lean tissue mass, time to market weight, egg production, milk production, etc. in livestock. However, one of the consequences has been an increase in skeletal problems. For example, the percentage of broilers with tibial dyschondroplasia has increased dramatically over the last 40 years (Leach, 1996). Cage layer osteoporosis developed into a significant problem for the layer industry as egg production and quality improved. A likely reason for this is that focused selection on one trait has consequences on others. As an example, pigs selected for growth performance as well as gait had decreased growth performance improvements relative to pigs selected only for growth performance (Steenbergen et al., 1990). Turkeys selected for walking ability and shank width had reduced breast width size relative to commercial lines. Crossing the two lines produced turkeys with better walking ability than the commercial line but less breast width size (Ye et al., 1997). However, the most interesting example is that embryonic chick tibiae from a broiler strain did not respond to mechanical loading, suggesting that bones will not adapt to changes in force (Pitsillides et al., 1999). This could partially explain why birds reared for meat production have leg problems at heavier weights (more discussion will follow). In addition, meat-type birds have bones that are not as well mineralized relative to random bred birds at similar weights (Corr et al., 2003; Williams et al., 2000). By genetically selecting for certain characteristics, the skeleton of an animal may be compromised. As an example, nutrient partitioning may reduce the ability of an animal to increase bone strength by limiting available resources.

Genetic selection for larger animals has caused structural problems in the past. Instead of bigger cattle, taller cattle were produced by altering the joint angles in the hindlimbs and forelimbs. This led to problems regarding movement and joint health (H. Ritchie, personal communication). The skeletal conformation of an animal is critical to its longevity. In purebred dogs, canine hip and elbow dysplasia (both producing joint abnormalities) will increase the susceptibility of a dog to osteoarthritis (Burton-Wurster et al., 1999; Smith et al., 2001). Horses with various conformation defects, such as "knocked knees" and "bowed knees", have an increased risk of osteoarthritis (Auer and Fackelman, 1981). In the rear legs, horses that are straight in the angulation of the stifle and hock are predisposed to lameness (Goble, 1992). Similar problems are found in swine and may highlight the fact that visually evaluating gilts/sows for correct joint angles and structure is important for developing a sound herd.

REGULATION OF GROWTH RATE

One of the consequences of genetic selection has been improved growth rate, especially in animals reared for meat production. However, faster growing animals are more susceptible to skeletal diseases such as osteochondrosis. Fast growing horses and pigs have increased susceptibility to osteochondrosis (Hill, 1990; Jeffcott, 1991). In most livestock species with growth-related skeletal problems, slowing down growth will decrease the problems. Regulating growth rate for animals reared for meat production might not be advantageous, but restricting growth in a breeding herd may improve longevity. In biomedical research, a great deal of work has suggested that caloric restriction is beneficial to increasing longevity by protecting many physiological systems. In animals, this may be the case regarding the skeletal system. Labrador Retrievers fed 75% of the diet given to a full-fed group had a dramatic decrease in developmental joint diseases over the course of their life (Smith et al., 2002). Furthermore dietary restriction of obese dogs can decrease the impact of osteoarthritis (Impellizeri et al., 2000). Even in broilers, dietary restriction can improve mineralization of the skeleton (Corr et al., 2003). A recent study suggests that energy deficient diets in growing pigs will not adversely affect bone development. Barrows on a 7 d severe restriction diet had increased serum concentrations of osteocalcin (indicator of bone formation) and decreased TRAP activity (indicator of bone resorption), despite decreases in circulating IGF-1 levels (Weiler et al., 2003). This is likely due to the increased growth hormone levels seen during feed restriction. In growing lambs, bone growth was less affected than weight gain during periods of feed restriction (Kamalzadeh et al., 1998). Dietary restriction had minimal effects on bone quality in sows after 3 parities (Arthur et al., 1983). Thus, growing gilts faster might not be better and even caloric restriction later in life could help keep sows structurally sound.

EXERCISE/MOVEMENT

Bone is a very dynamic tissue that remodels constantly in an adult animal. Wolff's Law, a fundamental principle in bone biology, simply states that bone will adapt to the forces imposed on it. If increased forces are applied to bone, it will adapt to handle those forces. Conversely, if decreased forces are applied to bone, it will adapt by resorbing bone that is not needed for support. Bed rest increases bone resorption as early as one week after initiation (Inoue et al., 2000). The complex cell signaling needed to translate a biomechanical force into a physiological response is being elucidated (Bidwell et al., 2001; Pavalko et al., 2003). In theory, Wolff's law may not apply to an animal if during the genetic selection process the factors necessary to respond to loading are decreased or impeded. As mentioned earlier, broilers and quite possibly turkeys have potentially lost some of their ability to respond to increased loads (Pitsillides et al., 1999). This could be important in that as the birds grow their skeleton will not adapt fast enough to hold the weight, especially if they are taken to heavy weights. The U.S. Egg and Poultry Association lists leg problems for broilers and turkeys as an area of concern. One of the management practices in the livestock industry is to restrict the movement of animals relative to free ranging. Under some of these conditions, bone loss and reduced bone strength has been documented. In horses placed in stalls, bone formation was decreased and resorption increased over 28 d (Hoekstra et al., 1999). Laying hens in cages had reduced bone strength and mineral density in their humeri relative to hens in perchery systems (Fleming et al., 1994). Sows housed in stalls had only 66% of the humeri and femur breaking strength of group-housed sows (Marchant and Broom, 1996). Gilts and sows housed in gestational and/or farrowing crates would be susceptible to bone loss due to the lack of movement. Although the long-term effects of this bone loss in sows is not known, extrapolation from both laying hens and humans would suggest it is detrimental. In addition, a lack of movement is detrimental for articular cartilage, causing a loss of proteoglycan and thinning of the cartilage (Vanwanseele et al., 2002).

REPRODUCTION

A large amount of calcium is needed during gestation and lactation to help build the bone structure of the rapidly developing fetuses and newborns, respectively. How much of an effect that has on the long term skeletal health of the animal is not known. In humans, mothers will lose calcium during lactation but replace it over time. Healthy women within the ages of 20 to 40 returned to prelactational bone mineral densities despite their second pregnancy within two years (Sowers et al., 1995). Some cases of pregnancy-associated osteoporosis have been reported but they are relatively rare (Black et al., 2003; Honjo and Mizunuma, 2001; Liel et al., 1998). Even in these cases, patients tend to recover the bone mineral in a couple of years. In general, women do not have long term detrimental effects related to gestation and lactation (Sowers, 1996). The high turnover of bone that occurs, especially during lactation, might actually improve bone quality. However, women do not have reproductive/lactational pressures similar to a dairy cow or sow. Because a sow's value is based on her litter size and number of parities, she almost continuously will be resorbing or depositing a large amount of bone mineral. With teenage girls, health professionals will recommend they drink plenty of milk and exercise so they can optimize bone mass. In comparison, starting in effect as a young teenager, gilts are bred and then continuously rebred throughout their life. Many potential questions can be asked. Do sows ever attain an optimal skeletal mass? Does that pose a problem in terms of longevity? How much mineral do they lose through gestation and lactation? Does a sow with a consistent and optimal intake of calcium accelerate bone mineral recovery? Answers to these questions would take time and resources but could yield valuable information.

ANALYTICAL TOOLS TO MONITOR SKELETAL HEALTH

Many new biological tools and non-invasive techniques are now available to monitor bone health in humans that can also be used in livestock. A recent review summarizes their use in equine bone (Lepage et al., 2001). The tools can generally be classified as static or dynamic measures of bone health. Static measures of bone health include quantitative ultrasonagraphy (QUS), Dual-energy X-ray absorptiometry (DXA), radiographic absorptiometry (RA), and quantitative computed tomography (QCT). They will in general provide a measurement on a specific bone of mineral content, mineral density, or architecture. They are static in the sense that they produce a snapshot of the bone at that time. In order to get a comprehensive picture of metabolism in the specific bone, analyses would need to be done serially. In horses, QUS and DXA were used on ex vivo third metacarpal bones to determine their precision for evaluating bone quality (Carstanjen et al., 2003). Both methods perform well, although they do not analyze the same parameters. DXA (live scans) has been used to study bone development at various skeletal regions in pigs from 3 kg to 138 kg (Mitchell et al., 2001). Thus, equipment can be adapted for the use in swine and should become even more mobile and practical in the future.

On the other hand, biological molecules originating from bone cells or matrix and found in the urine or serum can elucidate the current status of systemic bone metabolism. Molecules that reflect bone formation include osteocalcin, alkaline phosphatase, and collagen I propeptides. Molecules that reflect bone resorption include hydroxyproline, collagen cross-links, tartrate-resistant acid phosphatase (TRAP), galactosyl hydroxylysine, and more recently osteoprotegerin (OPG). More information concerning these molecules can be found in review articles (Buckley and Fraser, 2002; Delmas, 1993; Withold, 1996). Many of these molecules can be measured in livestock species and are very useful in determining how managerial and dietary changes affect bone metabolism. As an example, my laboratory in conjunction with many faculty in the department and some at other universities have used them to analyze bone turnover in broilers, laying hens, pigs, horses, dairy cattle, and beef cattle. Specifically, we found that when horses were placed in stalls, bone formation, as measured by osteocalcin, was decreased while bone resorption, as measured by urinary collagen cross-links, was increased, suggesting a net bone loss that was confirmed after one month by RA (Hoekstra et al., 1999). Osteocalcin concentrations in the sera of beef cattle on varying levels of phosphorus supported the conclusion that even marginal levels of dietary phosphorus did not induce bone turnover during the finishing phase (Erickson et al., 2002). In growing pigs, osteocalcin was used to look at the effect of varying calcium and phosphorus levels in the diet and along with 1,25 dihydroxyvitamin D_3 was a good predictor bone mineralization (Carter et al., 1996). We found that both serum osteocalcin and pyridinoline concentrations during a 28 d pre-slaughter vitamin and mineral withdrawal were increased, which lead to decreased bone mineralization and strength in the metacarpal bone at the time of slaughter (Shaw, 2001). These are just a few examples of how bone biological markers can be used to monitor skeletal health. Ideally, coupling bone marker data (dynamic indicators) with bone quality data (static indicators) will provide the most comprehensive picture of skeletal health.

FUTURE TECHNOLOGIES AND OPPORTUNITIES

Because osteoporosis is only going to become a bigger problem in the next 20 to 30 years, research efforts will continue to produce technologies that can be used in the livestock industry. As an example, although growth hormone increases the development of osteochondrosis in growing pigs (He et al., 1994), the use of low doses of growth hormone could improve bone density in mature animals. Growth hormone is now being studied as a potential therapeutic agent for osteoporosis (Landin-Wilhelmsen et al., 2003). In addition, a high-efficiency growth-hormone releasing hormone plasmid vector inserted into skeletal muscle of young pigs improved not only lean body mass but bone mineral density (Draghia-Akli et al., 2003). Unique delivery systems for osteogenic molecules could overcome some of the stresses endured by the skeletons of breeding livestock. Besides biotechnology, nutraceuticals (factors found in food that have a pharmacological-like effect) are being studied to determine if they can prevent either osteoporosis or osteoarthritis. Phytoestrogens, such as isoflavones, in some experimental models prevent bone loss (Picherit et al., 2001). Recent studies suggest that certain plant oils and monoterpenes can reversibly prevent bone resorption (Muhlbauer et al., 2003). Conjugated linoleic acid (CLA) has anti-inflammatory effects in bone and cartilage cells (Watkins and Seifert, 2000). In cartilage, glucosamine and chondroitin sulfate have been studied in vivo and in vitro and can inhibit cartilage degeneration (Leffler et al., 1999; Orth et al., 2002). Nutraceutical therapy targeted at times of stress on the skeleton may benefit gilts and/or sows.

CONCLUSION

Genetic manipulation and animal husbandry can influence sow longevity. The purpose of the paper was to provide some more recent information regarding skeletal biology and to stimulate discussion on how this information could be used to improve skeletal health in gilts and sows. In many respects, the ideal meat-producing animal has the image of a "muscle-bound couch potato". Skeletal research focused on humans provides some help in understanding bone biology for livestock but there are too many different factors involved in gilt development to make the results immediately applicable. Ultimately, understanding the bone biology of today's gilts will be needed to guide future research and management directions.

LITERATURE CITED

Arthur, S. R., E. T. Kornegay, H. R. Thomas, H. P. Veit, D. R. Notter, K. E. Webb, Jr., and J. L. Baker. 1983. Restricted energy intake and elevated calcium and phosphorus intake for gilts during growth. IV. Characterization of metacarpal, metatarsal, femur, humerus and turbinate bones of sows during three parities. J. Anim. Sci. 57: 1200-1214.

Auer, J. A., and G. E. Fackelman. 1981. Treatment of degenerative disease of the horse: A review and commentary. Vet. Surg. 10: 80-89.

Bidwell, J. P., K. Torrungruang, M. Alvarez, S. J. Rhodes, R. Shah, D. R. Jones, K. Charoonpatrapong, J. M. Hock, and A. J. Watt. 2001. Involvement of the nuclear matrix in the control of skeletal genes: The nmp1 (yy1), nmp2 (cbfa1), and nmp4 (nmp4/ciz) transcription factors. Crit. Rev. Eukaryot. Gene Expr. 11: 279-297.

Black, A. J., R. Reid, D. M. Reid, A. G. MacDonald, and W. D. Fraser. 2003. Effect of pregnancy on bone mineral density and biochemical markers of bone turnover in a patient with juvenile idiopathic osteoporosis. J. Bone Miner. Res. 18: 167-171.

Buckley, K. A., and W. D. Fraser. 2002. Receptor activator for nuclear factor kappab ligand and osteoprotegerin: Regulators of bone physiology and immune responses/potential therapeutic agents and biochemical markers. Ann. Clin. Biochem. 39: 551-556.

Burton-Wurster, N., J. P. Farese, R. J. Todhunter, and G. Lust. 1999. Site-specific variation in femoral head cartilage composition in dogs at high and low risk for development of osteoarthritis: Insights into cartilage degeneration. Osteoarthritis Cart. 7: 486-497.

Carstanjen, B., F. Duboeuf, J. Detilleux, and O. M. Lepage. 2003. Equine third metacarpal bone assessment by quantitative ultrasound and dual energy x-ray absorptiometry: An ex vivo study. J. Vet. Med. A Physiol. Pathol. Clin. Med. 50: 42-47.

Carter, S. D., G. L. Cromwell, T. R. Combs, G. Colombo, and P. Fanti. 1996. The determination of serum concentrations of osteocalcin in growing pigs and its relationship to end-measures of bone mineralization. J. Anim. Sci. 74: 2719-2729.

Christensen, G., L. Vraa-Andersen, and J. Mousing. 1995. Causes of mortality among sows in danish pig herds. Vet. Rec. 137: 395-399.

Corr, S. A., M. J. Gentle, C. C. McCorquodale, and D. Bennett. 2003. The effect of the morphology on the musculoskeletal system on the modern broiler. Animal Welfare 12: 145-157.

Delmas, P. D. 1993. Biological markers of bone metabolism. Presse Med. 22: 263-268.

Draghia-Akli, R., K. M. Ellis, L. A. Hill, P. B. Malone, and M. L. Fiorotto. 2003. High-efficiency growth hormone-releasing hormone plasmid vector administration into skeletal muscle mediated by electroporation in pigs. Faseb J. 17: 526-528.

Erickson, G. E., T. J. Klopfenstein, C. T. Milton, D. Brink, M. W. Orth, and K. M. Whittet. 2002. Phosphorus requirement of finishing feedlot calves. J. Anim. Sci. 80: 1690-1695.

Faust, M. A., O. W. Robison, and M. W. Tess. 1993a. Genetic and economic analyses of sow replacement rates in the commercial tier of a hierarchical swine breeding structure. J. Anim. Sci. 71: 1400-1406.

Faust, M. A., O. W. Robison, and M. W. Tess. 1993b. Integrated systems analysis of sow replacement rates in a hierarchical swine breeding structure. J Anim Sci 71: 2885-2890.

Fleming, R. H., C. C. Whitehead, D. Alvey, N. G. Gregory, and L. J. Wilkins. 1994. Bone structure and breaking strength in laying hens housed in different husbandry systems. Br. Poult. Sci. 35: 651-662.

Goble, D. O. 1992. Medical evaluation of the musculoskeletal system and common integument relevant to purchase. Vet. Clin. North Am. Equine Pract. 8: 285-302.

He, P., F. X. Aherne, A. L. Schaefer, J. R. Thompson, T. Nakano, and S. D. M. Jones. 1994. Differentiation of the effects of somatotropin and enhanced growth rate on the occurrence of osteochondrosis in pigs. Can. J. Anim. Sci. 74: 251-255.

Hill, M. A. 1990. Economic relevance, diagnosis, and countermeasures for degenerative joint disease (osteoarthrosis) and dyschondroplasia (osteochondrosis) in pigs. J. Am. Vet. Med. Assoc. 197: 254-259.

Hoekstra, K. E., B. D. Nielsen, M. W. Orth, D. S. Rosenstein, H. C. n. Schott, and J. E. Shelle. 1999. Comparison of bone mineral content and biochemical markers of bone metabolism in stall- vs. Pasture-reared horses. Equine Vet. J. Suppl. 30: 601-604.

Honjo, S., and H. Mizunuma. 2001. Changes in biochemical parameters of bone turnover and bone mineral density in post-pregnancy osteoporosis. Am. J. Obstet. Gynecol. 185: 246-247.

Impellizeri, J. A., M. A. Tetrick, and P. Muir. 2000. Effect of weight reduction on clinical signs of lameness in dogs with hip osteoarthritis. J. Am. Vet. Med. Assoc. 216: 1089-1091.

Inoue, M., H. Tanaka, T. Moriwake, M. Oka, C. Sekiguchi, and Y. Seino. 2000. Altered biochemical markers of bone turnover in humans during 120 days of bed rest. Bone 26: 281-286.

Jeffcott, L. B. 1991. Osteochondrosis in the horse--searching for the key to pathogenesis. Equine Vet. J. 23: 331-338.

Kamalzadeh, A., W. J. Koops, J. van Bruchem, and G. A. Bangma. 1998. Effect of duration of feed quality restriction on body dimensions in lambs. J. Anim. Sci. 76: 735-742.

Landin-Wilhelmsen, K., A. Nilsson, I. Bosaeus, and B. A. Bengtsson. 2003. Growth hormone increases bone mineral content in postmenopausal osteoporosis: A randomized placebo-controlled trial. J. Bone Miner. Res. 18: 393-405.

Leach, R. M., Jr. 1996. Poultry industry should reconsider if bigger is better. Feedstuffs 68: 10.

Leffler, C. T., A. F. Philippi, S. G. Leffler, J. C. Mosure, and P. D. Kim. 1999. Glucosamine, chondroitin, and manganese ascorbate for degenerative joint disease of the knee or low back: A randomized, double-blind, placebo-controlled pilot study. Mil. Med. 164: 85-91.

Lepage, O. M., B. Carstanjen, and D. Uebelhart. 2001. Non-invasive assessment of equine bone: An update. Vet. J. 161: 10-22.

Liel, Y., D. Atar, and N. Ohana. 1998. Pregnancy-associated osteoporosis: Preliminary densitometric evidence of extremely rapid recovery of bone mineral density. South Med. J. 91: 33-35.

Marchant, J. N., and D. M. Broom. 1996. Effects of dry sow housing conditions on muscle weight and bone strength. Anim. Sci. 62: 105-113.

Mitchell, A. D., A. M. Scholz, and V. G. Pursel. 2001. Total body and regional measurements of bone mineral content and bone mineral density in pigs by dual energy x-ray absorptiometry. J. Anim. Sci. 79: 2594-2604.

Muhlbauer, R. C., A. Lozano, S. Palacio, A. Reinli, and R. Felix. 2003. Common herbs, essential oils, and monoterpenes potently modulate bone metabolism. Bone 32: 372-380.

Orth, M. W., T. L. Peters, and J. N. Hawkins. 2002. Inhibition of articular cartilage degradation by glucosamine-hcl and chondroitin sulphate. Equine Vet. J. 34: 224-229.

Pavalko, F. M., S. M. Norvell, D. B. Burr, C. H. Turner, R. L. Duncan, and J. P. Bidwell. 2003. A model for mechanotransduction in bone cells: The load-bearing mechanosomes. J. Cell. Biochem. 88: 104-112.

Picherit, C., C. Bennetau-Pelissero, B. Chanteranne, P. Lebecque, M. J. Davicco, J. P. Barlet, and V. Coxam. 2001. Soybean isoflavones dose-dependently reduce bone turnover but do not reverse established osteopenia in adult ovariectomized rats. J. Nutr. 131: 723-728.

Pitsillides, A. A., S. C. Rawlinson, J. R. Mosley, and L. E. Lanyon. 1999. Bone's early responses to mechanical loading differ in distinct genetic strains of chick: Selection for enhanced growth reduces skeletal adaptability. J. Bone Miner. Res. 14: 980-987.

Shaw, D. T. 2001. The impact of supplement withdrawal and wheat middling inclusion on pork nutrient content and bone quality. M.S. Thesis, Michigan State University, East Lansing

Smith, G. K., R. D. Kealy, D. N. Biery, and D. F. Lawler. 2002. Influence of body condition on canine osteoarthritis. Purina Nutrition Forum Proceedings 24: 8-12.

Smith, G. K., P. D. Mayhew, A. S. Kapatkin, P. J. McKelvie, F. S. Shofer, and T. P. Gregor. 2001. Evaluation of risk factors for degenerative joint disease associated with hip dysplasia in german shepherd dogs, golden retrievers, labrador retrievers, and rottweilers. J. Amer. Vet Med. Assoc. 219: 1719-1724.

Sowers, M. 1996. Pregnancy and lactation as risk factors for subsequent bone loss and osteoporosis. J. Bone Miner. Res. 11: 1052-1060.

Sowers, M., J. Randolph, B. Shapiro, and M. Jannausch. 1995. A prospective study of bone density and pregnancy after an extended period of lactation with bone loss. Obstet. Gynecol. 85: 285-289.

Steenbergen, E. J. v., E. Kanis, and H. A. M. v. d. Steen. 1990. Genetic parameters of fattening performance and exterior traits of boars tested in central stations. Livest. Prod. Sci. 24: 65-82.

Vanwanseele, B., F. Eckstein, H. Knecht, E. Stussi, and A. Spaepen. 2002. Knee cartilage of spinal cord-injured patients displays progressive thinning in the absence of normal joint loading and movement. Arthritis Rheum. 46: 2073-2078.

Watkins, B. A., and M. F. Seifert. 2000. Conjugated linoleic acid and bone biology. J. Am. Coll. Nutr. 19: 478S-486S.

Weiler, U., S. Finsler, and R. Claus. 2003. Influence of cortisol, gonadal steroids and an energy deficit on biochemical indicators of bone turnover in swine. J. Vet. Med. A Physiol. Pathol. Clin. Med. 50: 79-87.

Williams, B., S. Solomon, D. Waddington, B. Thorp, and C. Farquharson. 2000. Skeletal development in the meat-type chicken. Br. Poult. Sci. 41: 141-149.

Withold, W. 1996. Monitoring of bone turnover biological, preanalytical and technical criteria in the assessment of biochemical markers. Eur. J. Clin. Chem. Clin. Biochem. 34: 785-799.

Ye, X., J. W. Anderson, D. O. Noble, J. Zhu, and K. E. Nestor. 1997. Influence of crossing a line selected for increased shank width and a commercial sire line on performance and walking ability of turkeys. Poultry Sci. 76: 1327-1331.

Conception Enhancement

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Summary.

There are no "Silver Bullets" to enhance conception rate and litter size born alive. Pork producers need to utilize excellent management practices to: (1) enhance the transport of sperm cells to the oviduct during the first two hours after insemination, (2) prevent physical stress during the period of time the ova have to remain in oviduct [three to five days after onset of estrus], and (3) prevent physical stress and heat-stress during the time when the embryo sends a pregnancy signal to the sow [10 to 12 days of pregnancy] and during the time of implantation [12 to 18 days of pregnancy]. The addition of hormones (oxytocin or prostaglandin) to a dose of semen may or may not enhance conception rate and litter size. The use of Predil may or may not enhance farrowing rate and litter size. Sows should be inseminated with high quality semen that contains 3 to 5 billion sperm cells per dose.

Introduction.

To set the boundaries for writing this paper, I have defined conception enhancement as: Factors that make the act of becoming pregnant greater with a larger litter. At the farm level, it is economically important to not only increase the proportion of bred sows becoming pregnant, but also to increase the litter size born alive and the proportion of sows farrowing. It is beyond the scope of this Midwest Swine Nutrition Conference paper to discuss all the aspects affecting conception rate, farrowing rate and litter size; thus, this paper will focus on a few non-nutritional factors that might enhance conception rate and litter size during the first 28 days of gestation.

Critical periods.

The establishment of pregnancy in the pig is a complex process and occurs during several days after insemination (Figure 1). There are "biological" periods during the first 28 days after mating that can significantly affect conception rate and litter size.

Transport of sperm to oviduct.

One critical aspect for conception to occur is that an adequate number of high quality spermatozoa must be transported from the cervix to the sperm reservoir of the oviduct (1 to 2 cm of oviduct isthmus). Sperm cells that do not reach the oviduct are killed by phagocytosis (Woelders and Matthijs, 2001). Artificial insemination or natural mating of pigs triggers a massive influx of polymorphonuclear leucocytes into the lumen of the uterus. Large numbers of leucocytes have been found at 30 minutes (Lovell and Getty, 1968), 2 hours (Pursel et al., 1978), and 3 hours (Kohsaka et al., 2000) after insemination. Rozeboom et al. (1999) found greater numbers of leucocytes from 12 to 36 hours after sows were inseminated with 5 billion sperm cells compared to sows inseminated with 100 mL of seminal plasma or phosphate buffered saline. Although approximately 90% of the spermatozoa cannot be recovered from the uterus within 2 hours after a natural mating (Viring, 1980), a sufficient number of sperm cells (100 to 200 million) reach the uterotubal junction and the adjacent first isthmic segment of the oviduct (sperm reservoir) within 15 to 20 minutes after a natural mating (Hunter, 1990). *Take Home Message*: Although research has not been conducted to determine the affects of physical and environmental stress during the first three hours after insemination on efficiency of transporting sperm cells to the oviduct, it is advisable to minimize physical stress (mixing of animals) for two hours after insemination.

Are there products that can be used to enhance the transport of spermatozoa to the oviduct? The major mechanism for transporting spermatozoa to the oviduct is smooth muscle contraction of the uterus (Langendijk et al., 2002).

Oxytocin. The presence of a boar induces the release of oxytocin in the sow during mating (Langendijk, 2003). Several investigators have studied whether injecting oxytocin into semen before artificial insemination improves farrowing rate and litter size. The conclusions from a review of 12 studies by Levis (2000) were: 1) Adding 4 to 5 IU's of oxytocin to a dose of semen improves farrowing rate and litter size; 2) Use of oxytocin-treated semen is more effective in multiparous sows than gilts; 3) During the summer months, oxytocin-treated semen significantly increased farrowing rate and litter size; and 4) In most studies, the use of oxytocin at the time of insemination was profitable (Table 1). A recent study by Willenburg et al. (2003) reported that the number of healthy fetuses tended ($P < 0.06$) to increase in gilts inseminated with a single dose of semen containing 4 IU of oxytocin. However, a recent large-scale study on five farms by Levis (unpublished data) did not find a significant improvement in reproductive performance by adding 5 IU of oxytocin to a dose of semen during the summer months (Table 2). Juarez et al. (2002) did not find a significant improvement in farrowing rate (Oxytocin, 85.1% vs Control, 80.4%) or litter size born alive (9.9 vs 9.9 piglets) when adding 5 IU of oxytocin per dose of semen. *Take Home Message*: The addition of oxytocin to a dose of boar semen may or may not enhance reproductive performance.

Prostaglandin. Ejaculated boar semen contains prostaglandin $F_{2\alpha}$ (Cheng et al., 2003). Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) increases myometrical activity of the uterus and oviduct in pigs (Cheng et al., 2001; Rodriguez-Martinez and Einarrson, 1985). Mateusen et al. (2002) found that motility of spermatozoa was enhanced by adding .5 mL, 1 mL or 2 mL of $PGF_{2\alpha}$ to 100 mL of diluted semen. A study by Gil et al. (1998) found that injecting 5 mg of $PGF_{2\alpha}$ into the dose of semen used for the first insemination resulted in an improvement in farrowing rate, total number of piglets born per litter, and number of piglets born live per litter (Table 3). A study by Reicks (2000) found that placing 0.5 mL of Lutalyse® into an AI catheter (catheter attached to semen vessel first) just prior to insertion of catheter resulted in: (1) An increase in farrowing rate and less number of service periods per pregnancy in gilts and Parity 1 sows (Table 4), and (2) An increase in farrowing rate of gilts bred during a hotter season (June 1 to August 31) compared to a moderate season (after August 31) [Table 5]. Jimenez and Calderon (2000) found a significant ($P < 0.05$) increase in farrowing rate (86.6 vs 63.6%) and a numeric increase (9.7 vs 8.8 piglets) in litter size when 1 mL of dinoprost was added only to the first dose of semen.

It is well known that repeat breeders have a lower farrowing rate. The addition of 1 mL of Lutalyse® in the semen just prior to insemination did not enhance farrowing rate of repeat breeders (Wayne, 2000). Interestingly, the addition of Lutalyse® reduced farrowing rate of repeat breeders by 3.3 to 9.2 percentage points. In contrast, Horvat and Bilkei (2003) found that conception rate (94.1 vs 73.2%) and farrowing rate (89.9 vs 70.0%) of repeat breeders were significantly increased ($P < 0.001$) by adding 1 mL Dinolytic to an 80 mL dose of semen. However, adding prostaglandin to semen did not enhance litter size. *Take Home Message*: The addition of prostaglandin to a dose of boar semen may or may not enhance reproductive performance.

Estrogen. Although boar semen has a high concentration of estrogen, the addition of estrogen (5µg estradiol, 4.5µg estrone sulphate, 2µg estrone in 1 mL of vegetable oil) in 70 mL of semen did not show an improvement in conception rate or number of embryos at 30 days of gestation (Lambert et al., 1991a). The gilts were inseminated with 2.7 billion sperm cells per dose at 16 and 32 hours after detection of estrus. A small scale study in Canada did not find a statistical difference in pregnancy rate, farrowing rate, or litter size when gilts and sows were artificially inseminated with semen contain 10µg of estradiol (Kirkwood and Thacker, 1991). However, estradiol did have positive effects on the reproductive traits. Additional research needs to be conducted, whereby a larger number of sows are on each treatment, to determine the true effect of adding estrogen to boar semen on farrowing rate and litter size. *Take Home Message*: The addition of estrogen to a dose of boar semen did not enhance reproductive performance.

Predil MR-A®. Predil MR-A is a synthetic seminal plasma that is used to optimize reproductive performance in sows and gilts. The chemical composition is a trade secret of a company in Spain called KUBUS, SA (Calle E, 20-Pol. Ind. Europolis, 28230 Las Rozas [Madrid], Spain). A volume of 30 cc of Predil MR-A is passed through the AI catheter just prior to inseminating the females. The use of this product has produced positive effects on farrowing rate and litter size (Table 6 and 7; Lycznski et al., 2000; Martin Rillo et al., 1996). [Mention of a trade name, proprietary product, or specific equipment does not constitute endorsement or warranty of the product by The Ohio State University or imply its approval to the exclusion of other products that may also be suitable.] *Take Home Message*: The use of Predil may or may not enhance reproductive performance.

Ova in oviduct.

Porcine ova are fertilized in the ampulla of the oviduct, near the ampulla-isthmic junction. It has been documented that the fertilization rate of recovered ova of gilts inseminated twice (either AM and PM Day 1 of estrus or PM Day 1 and AM Day 2 of estrus) is 94.5% (Lambert et al., 1991b). The uterine horn is a hostile environment for several hours after the ova are fertilized; thus, the ova remain in the oviduct for about 48 to 56 hours after ovulation. The majority of embryos (4-cell stage) and unfertilized ova enter the uterus three to four days after the onset of estrus. Scientists have not investigated whether excessive physical stress (mixing and moving) at three to five days after onset of estrus causes the embryos to enter the uterine horns to early. *Take Home Message*: Additional research is need to study the effect of stress on transport of ova in the oviduct.

Pregnancy signals.

Between days 10 to 12 of pregnancy, blastocysts undergo a rapid period of elongation and start to synthesis estrogen. These estrogens appear to be the blastocysts signal for maternal recognition of pregnancy; plus, embryos must be present in both uterine horns to prevent luteolysis. Filamentous blastocysts can be easily dislodged by flushing of the reproductive tract on day 12 of pregnancy. Scientists have not investigated whether excessive physical stress (mixing and moving) or heat-stress at 10 to 12 days of pregnancy are capable of interfering with the signal for maternal recognition of pregnancy.

Blastocyts attachment.

Embryos start to attach to the uterus around days 13 to 14 of pregnancy with a loose contact between trophoblast and uterine membranes. The attachment process is complete by day 28 of pregnancy. Although Lambert et al. (1991b) suggested that the majority of embryonic death occurs prior to day 10 after mating, the general consensus is that embryonic death occurs mainly during the time of implantation at days 12 to 18 after mating (Pope 1994; van der Lende and Schoenmaker, 1990). The publications by Ashworth and Pickard (1998) and van der Lende et al. (1994) provide a comprehensive review of extrinsic and intrinsic factors influencing embryo survival. For this manuscript I want to emphasis that stress (high ambient temperatures, fighting during establishment of a social hierarchy in newly formed groups) can increase the embryo mortality.

Semen quality. The percentage of abnormal cells in a dose of semen can lower farrowing rate and litter size born. If the percentage of abnormal cells is greater than 20%, the dose of semen should be discarded. A procedure for evaluating boar semen for quality can be found at: http://porkinfo.osu.edu/Word%20Documents/evaluating.semen.doc.

Sperm cells per dose. The number of spermatozoa inseminated is an important factor that affects boar fertility. The results from a study of 40 to 50 ejaculates per boar (200 crossbred boars), over a period of 12 months, showed that the number of sperm cells per dose to optimize farrowing rate and litter size is quite variable between boars (Flowers, 2001). The majority of boars exhibited a fertility pattern with a plateau that occurred between 3 and 5 billion sperm per dose (homospermic dose). However, farrowing rate and litter size did not plateau at 9 billion sperm per dose for some of the boars (Figure 2 and 3). *Take Home Message*: It is most likely advantageous to use heterospermic semen.

Reference

Ashworth, C. J., and A. R. Pickard. 1998. Embryo survival and prolificacy. Pages 303-325 in Progress in Pig Science. J. Wiseman, M. A. Varley and J. P. Chadwick, editors. Nottingham University Press.

Cheng, H., G. C., Althouse, and W. H. Hsu. 2001. Prostaglandin F2α added to extended boar semen at processing elicits in vitro myometrial contractility after 72 hours of storage. Theriogenology 55:1901-1906.

Cheng, H., G. C., Althouse, and W. H. Hsu. 2003. Concentrations of endogenous prostaglandin $F_{2\alpha}$ in boar semen and effect of a 72-h incubation period on exogenous prostaglandin $F_{2\alpha}$ concentration in extended boar semen. Prostaglandins & other Lipid Mediators 70:285-290.

Flowers, W. L. 2001. Production of fertile insemination doses. Pages 157-165 in Manipulating Pig Production VIII. P. D. Cranwell, ed. S. R. Frankland Pty Ltd, Melbourne, Australia.

Gil, J., J. Chico, O. Gil, and A. Lopez. 1998. Increasing swine prolificacy by adding Dinolytic® to semen doses. Proc. 15[th] International Pig Veterinary Society Congress. Birmingham, England. p 216 (Abstr).

Horvat, G., and G. Bilkei. 2003. Exogenous prostaglandin $F_{2\alpha}$ at time of ovulation improves reproductive efficiency in repeat breeder sows. Theriogenology 59:1479-1484.

Hunter, R.H.F. 1990. Fertilization of Pig Eggs *in vivo* and *in vitro*. J. Reprod. Fert. (Suppl) 40:211-226.

Jimenez, E., and E. Calderon. 2000. Use of dinoprost in boar semen in the first service: two field cases. Proc. 16[th] International Pig Vet. Soc. Congr, Melbourne, Australia, p 408. (Abstr)

Juarez, A., J. Becerril, P. Granados, G. Ortega, and J. Izeta. 2002. A comparison of the effect of addition of Lutalyse® or oxytocin to extended boar semen on reproductive performance (fertility and prolificacy) in sows. Proc. International Pig Vet. Soc. Ames, IA. Paper 645. (Abstr).

Kirkwood, R. N., and P. A. Thacker. 1991. The influence of adding estradiol to semen on performance of sows. Can. J. Anim. Sci. 71:589-591.

Kohsaka, T., H. Sasada, K. Hashizume, F. Akita, J. Kobayashi, M. Kang, T. Kawaraski, K. Bamba, and E. Sato. 2000. Uterine inflammatory response after mating and in vitro detection of granulocyte-macrophage colony stimulating factor in pig semen. Theriogenology 53:475 (Abstr).

Lambert, E., D. H. Williams, P. B. Lynch, T. J. Hanrahan, T. A. McGeady, F. H. Austin, M. P. Boland, and J. F. Roche. 1991a. The effect of administration of gonadotrophins and estrogens on prenatal survival in gilts. Theriogenology 36:77-85.

Lambert, E., D. H. Williams, P.B. Lynch, T. J. Hanrahan, T. A. McGeady, F. H. Austin, M. P. Boland, and J. F. Roche. 1991b. The extent and timing of prenatal loss in gilts. Theriogenology 36:655-665.

Langendijk, P., E. G. Bouwwman, N. M. Soede, M. A. M. Taverne, and B. Kemp. 2002. Myometrial activity around estrus in sows: spontaneous activity and effects of estrogens, cloprostenol, seminal plasma and clenbuterol. Theriogenology 57:1563-1577.

Langendijk, P., E. G. Bouwan, D. Schams, N. M. Soede, and B. Kemp. 2003. Effects of different sexual stimuli on oxytocin release, uterine activity and receptive behavior in estrous sows. Theriogenology 59:849-861.

Levis, D. G. 2000. The Effect of Oxytocin at the Time of Insemination on Reproductive Performance - A Review. Pages 11-17 in Nebraska Swine Report EC00-219-A. D. Moser, ed. University of Nebraska Cooperative Extension.

Lovell, J. W., and R. Getty. 1968. Fate of semen in the uterus of the sow: histologic study of endometrium during the 27 hours after natural service. Am. J. Vet. Res. 29:609-625.

Lyczynski, A., T. Soczywko, S. Martin Rillo, and C. de Alba Romero. 2000. The effect of Predil-MRA synthetic seminal plasma used to inseminate sows and gilts on their reproductive efficiency. Proc. IV[th] International Conference on Boar Semen Preservation. Beltsville, MD. Allen Press, Inc., Lawrence, KS. pp 250 (Abstr).

Martin Rillo, S., S. Lapuente, R. Hernandez-Gil, J. A. Carcia Ruvalcaba, and C. Garcia Artiga. 1996. Improvement of fertility results by means of usage of synthetic seminal plasma before artificial insemination. Proc. 14[th] International Pig Veterinary Society Congress. Bologna, Italy. p 605 (Abstr).

Mateusen, B., A. Van Soom, D. Maes, M. Degezelle, M. Verdonck, and A. de Kruif. 2002. Effect of prostaglandin $F_{2\alpha}$ (DINOLYTIC®) added to extended boar semen on the motility of boar spermatozoa. Proc. International Pig Veterinary Society Congress. Ames, IA. Paper 57 (Abstr).

Pope, W. F. 1994. Embryonic mortality in swine. Pages 53-77 in Embryonic Mortality in Domestic Species. M. T. Zavy and R. D. Geisert, editors, CRC Press, Boca Raton, Florida.

Pursel, V. G., L. L. Schulman, and L. A. Johnson. 1978. Distribution and morphology of fresh and frozen-thawed sperm in the reproductive tract of gilts after insemination. Biol. Reprod. 19:69-76.

Reicks, D. 2000. Evaluation of the effect of adding Lutalyse® Sterile Solution to extended boar semen on farrowing rate, service periods per pregnancy and litter size. Proc. Allen D. Leman Swine Conference. University of Minnesota College of Veterinary Medicine. Vol. 27:13 (Abstr).

Rodriguez-Martinez, H., and S. Einarson. 1985. Influence of prostaglandins on the spontaneous motility of pig oviducts. Anim. Reprod. Sci. 8:259-279.

Rozeboom, K. J., M. H. Troedsson, T. W. Molitor, and B. G. Crabo. 1999. The effect of spermatozoa and seminal plasma on leukocyte migration into the uterus of gilts. J. Anim. Sci. 77:2201-2206.

Woelders, H., and A. Matthijs. 2001. Phagocytosis of boar spermatozoa *in vitro* and *in vivo*. Reproduction (Suppl) 58:113-127.

van der Lende, T., and G. J. W. Schoenmaker. 1990. The relationship between ovulation rate and litter size before and after Day 35 of pregnancy in gilts and sows: an analysis of published data. Livest. Prod. Sci. 26:217-229.

van der Lende, T., N. M. Soede, and B. V. Kemp. 1994. Embryo mortality and prolificacy in the pig. Pages 297-317 in Principles of Pig Science. D. J. A. Cole, J. Wiseman, and M. A. Varley, editors. Nottingham University Press.

Viring, S. 1980. Distribution of live and dead spermatozoa in the genital tract of gilts at different times after insemination. Acta Veterinaria Scandinavica 21:145-149.

Wayne, S. R. 2000. Effect of prostaglandin $F_{2\alpha}$ administered in semen on fertility in repeat breeding females. Proc. American Association of Swine Practitioners. 31st Annual Meeting. Indianapolis, Indiana. pp 33-37.

Willenburg, K. L., G. M. Miller, S. L. Rodrigeuez-Zas, and R. V. Knox. 2003. Influence of hormone supplementation to extended semen on artificial insemination, uterine contractions, establishment of a sperm reservoir, and fertility in swine. J. Anim. Sci. 81:821-829.

Table 1. Summary of benefit from using oxytocin in conjunction with artificial insemination.

Study	Farrowing rate	Piglets born live	FI[a] per 100 sows	Profit at $10/head	Cost of oxytocin[b]	Net gain per 100 sows
A	+5.8	+.82	+132	+$1,320	$2.00	+$1,318
B	+1.2	+.04	+15	+$150	$2.00	+$148
C	+3.0	-.20	+15	+$150	$2.00	+$148
D	-5.8	+.50	-14	-$140	$2.00	-$142
E (gilts)	+6.3	+.04	+54	+$540	$2.00	+$538
E (sows)	-.80	+.14	+5	+$50	$2.00	+$48
F (gilts)	-1.8	-.10	-22	-$220	$2.00	-$222
F (sows)	+4.2	+.20	+57	+$570	$2.00	+$568
G (gilts)	-.74	-.12	-14	-$140	$2.00	-$142
G (sows)	+3.5	+.54	+72	+$720	$2.00	+$718
H (gilts)	+.10	+.10	+9	+$90	$2.00	+$88
H (sows)	+1.1	+.11	+20	+$200	$2.00	+$198
I (gilts)	-1.36	-1.09	-102	-$1,020	$2.00	-$1,022
I (sows)	+4.08	+.85	+107	+1,070	$2.00	+$1,068
J (Inexp. person)	+12.1	+.8	+186	+$1,860	$2.00	+$1,858
J (Exp. person)	+5	+.4	+87	+$870	$2.00	+$868
K (old semen+oxy)	+17	+.07	+219	+$2,190	$2.00	+$2,188
L (oxy in semen)	+8.1	+1.84	+231	+$2,310	$2.00	+$2,308

[a] FI is fecundity index (FI = farrowing rate x litter size born live)
[b] $4.00 per 100 mL of oxytocin (20 IU per mL); 5 IU per dose; 1¢ per dose; 2 doses per sow

Table 2. Effect of adding 5 IU of oxytocin to a dose of boar semen on reproductive performance.

Item	Control	Oxytocin	Benefit of oxytocin	P value
Number bred	3189	3232		
Pregnant at day 34	87.43	88.24	.82	.60
Farrowing rate, %	77.40	78.62	1.22	.36
Total piglets born per litter	11.88	11.91	.03	.73

Levis (2003, unpublished data)

Table 3. Increasing swine prolificacy by adding Dinolytic™ to semen doses.

Item	Lutalyse	Control
Number of sows	433	420
PGF in semen	First dose of semen only	0
Amount of PGF per dose	5 mg (1 mL)	0
Number of sows farrowing	348	329
Farrowing rate, %	80.4*	78.3
Avg piglets born live per litter	10.21**	10.04
Avg total piglets born per litter	11.51***	11.17
Calculated total pigs born live	3553	3303
Difference in total pigs born live (Lutalyse - control)	250 piglets	
Assumed cost of PGF per dose	.429	0
Calculated total cost of PGF	$185.76	0

Different from control * ($P < .02$); ** ($P < .05$); *** ($P < .004$)

Table 4. Effect of Lutalyse® at time of insemination on reproductive performance.

	Gilts			Parity 1		
Item	Lutalyse[a]	Control	P value	Lutalyse	Control	P value
Number animals	769	806		373	326	
Farrowing rate, %	84.9	81.5	.02	85.8	81.4	.05
Total born	10.1	10.1	.17	10.2	10.1	.27
Service periods per pregnancy	1.26	1.38	.03	1.29	1.31	.06

[a] The catheter was attached to the vial of semen, Lutalyse (0.5 mL) was injected into the breeding catheter, the catheter was inserted into the cervix and then 80 mL of semen was inseminated.

Table 5. Effect of Lutalyse® at time of insemination on farrowing rate and number of service periods per pregnancy by season inseminated (Gilts).

Date of inseminations	Farrowing rate, %			Service periods per pregnancy		
	Lutalyse[a]	Control	P value	Lutalyse	Control	P value
June 1 to August 31	86.8	82.0	.03	1.17	1.31	.04
> August 31	82.9	80.1	.03	1.35	1.47	.05
	Seasonal effect		.03	Seasonal effect		.05

[a] The catheter was attached to the vial of semen, Lutalyse (0.5 mL) was injected into the breeding catheter, the catheter was inserted into the cervix and then 80 mL of semen was inseminated.

Table 6. Effect of Predil MR-A® on farrowing rate and litter size.

	Gilts			Sows		
Item	Predil	Control	Difference	Predil	Control	Difference
Number of animals	35	15	20	69	69	0
Farrowing rate, %	85.71	73.33	12.38	95.65	89.85	5.8
Avg live piglets per litter	9.28*	8.27	1.01	10.27*	9.50	0.77

* Significantly different from control (P < .05)

Table 7. Effect of Predil MR-A® on farrowing rate and litter size.

	Gilts (Farms 1 & 2)			Sows (Farms 1 & 2)		
Item	Predil	Control	Difference	Predil	Control	Difference
Number of animals	174	225	51	342	386	44
Farrowing rate, %	85.6*	81.3	4.3	87.4	84.9	2.5
Avg live piglets per litter	10.03	9.84	0.19	10.52	10.43	0.09
	All females (Farms 3, 4, 5, 6, & 7)					
Item	Predil		Control		Difference	
Number of animals	489		885		396	
Farrowing rate, %	82.6*		80.3		2.3	
Avg live piglets per litter	10.41		10.36		0.05	

* Significantly different from control (P < .05)

Figure 1. Schematic description of early pregnancy in the pig.

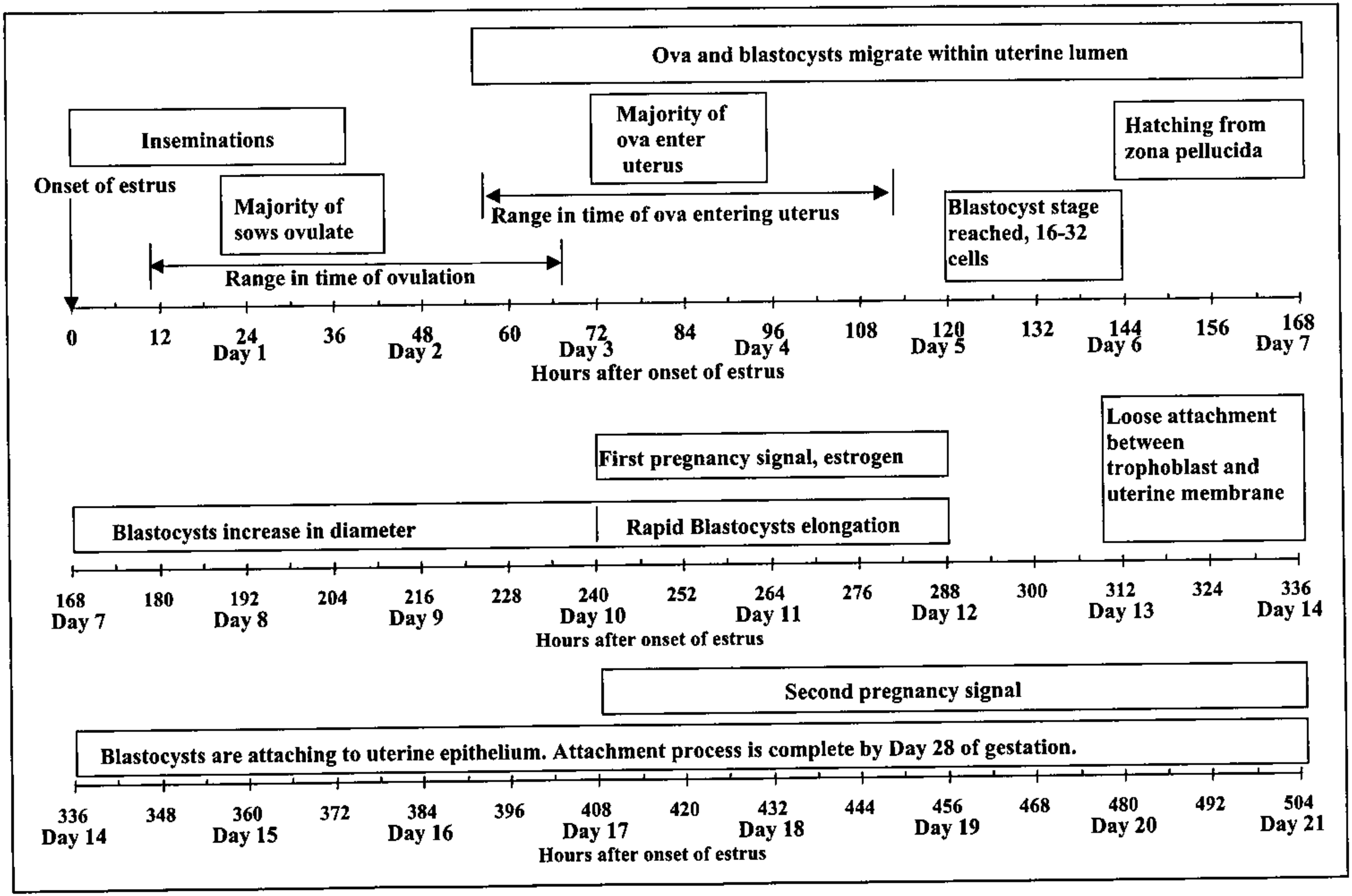


Figure 2. Fertility pattern for farrowing rate of boar semen by number of sperm per dose.

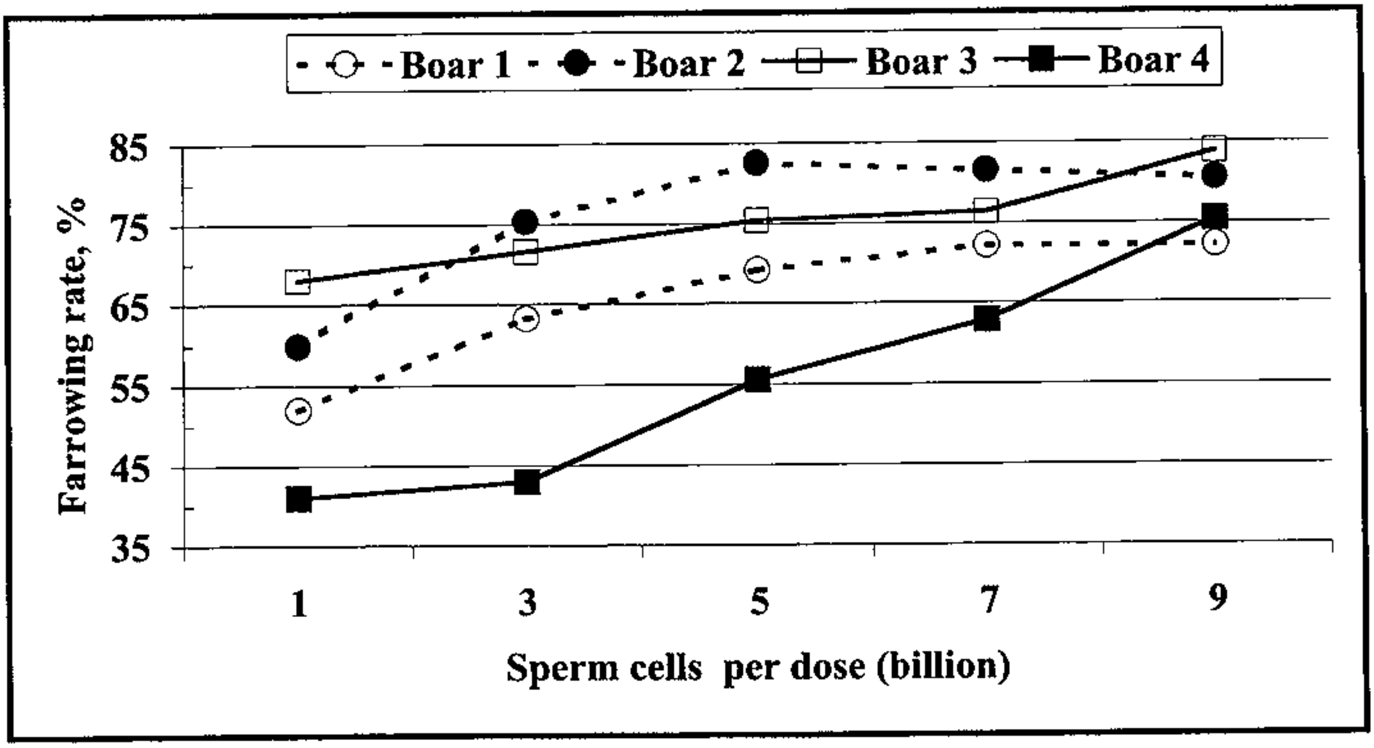
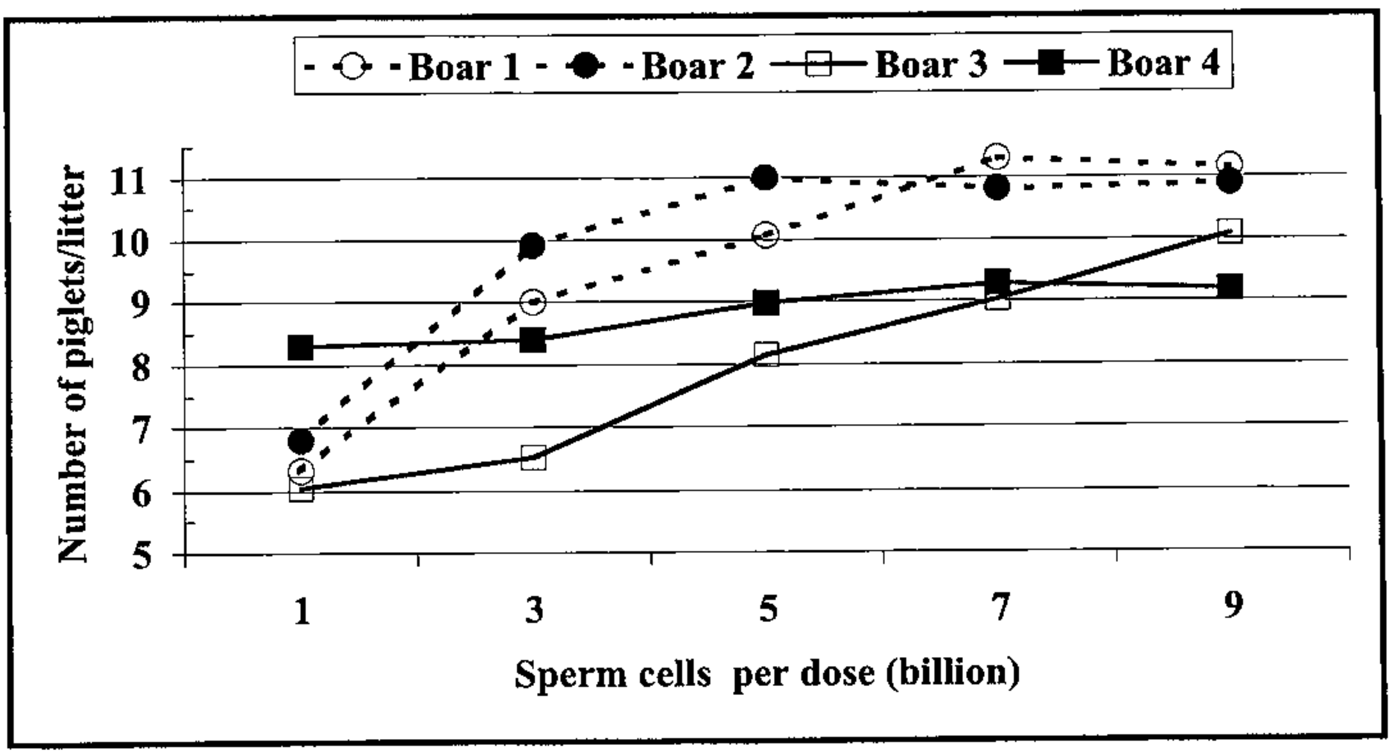


Figure 3. Fertility pattern for number of piglets born alive by number of sperm per dose.



Update on Carcass Modifiers

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Introduction

Broadly defined a dietary carcass modifier includes any component of the diet that modifies carcass composition. Therefore, the list of potential carcass modifiers is endless. This review is not exhaustive and will not try to detail the effects of all carcass modifiers. Instead, this review will briefly discuss carcass modifiers that have received attention in the last few years. Detailed reviews of this subject have been conducted in recent years (Berg, 2001; Pettigrew and Esnaola, 2001), and readers are directed to these reviews for additional details. Carcass modifiers may enhance lean tissue accretion rates, modify the ratio of protein to fat accretion, alter the fatty acid profile of meat, or alter post-mortem metabolism. Many carcass modifiers are vitamins or minerals essential for growth and maintenance of the pig, that have additional carcass benefits when added in excess of requirements (i.e. Cr, Mg, niacin, pantothenic acid, and vitamins E and D_3). Additional carcass modifiers include vitamin metabolites (betaine) and vitamin-like compounds (carnitine). Fats, conjugated linoleic acid, and β-agonists have also been investigated for potential carcass modifying effects. The effectiveness of some of these carcass modifiers is well documented and clear; however, many others are less well documented, and the results are variable. Each section below discusses a carcass modifier and attempts to provide an overview of available data.

Chromium

Trivalent Cr is an essential trace mineral for pigs, but a dietary requirement has not been set (NRC, 1998). Additional interest in Cr has surrounded reports of improved growth and reproductive performance, and carcass characteristics when trivalent Cr is supplemented to swine diets.

The most researched form of Cr in swine diets, to date, is chromium tripicolinate (CrPic). Research investigating the possible carcass modifying effects of this compound has resulted in variable results. Lindemann et al. (1995) reported an increase in longisimus muscle area which was in agreement with earlier research of Page et al. (1993), but contrasted the research of many others (Bolemann et al., 1995; Mooney and Cromwell, 1995). Likewise, improvements in carcass lean have been reported by several researchers (Page et al., 1993; Lindemann et al., 1995; Mooney and Cromwell, 1995), while others have reported no effect of CrPic supplementation on carcass lean (Evock-Clover et al., 1993; Mooney and Cromwell, 1999; Mathews et al., 2003).

Reasons for the variability of published results have been attributed to the length of time of Cr supplementation prior to slaughter and to the source and level of fat in the diet. Mooney and Cromwell (1997) reported the results of two experiments where Cr from CrPic (Exp. 1: 200 or 400 ppb Cr; Exp. 2: 200 ppb Cr) or chromium chloride ($CrCl_3$, Exp. 1: 5,000 or 25,000 ppb Cr; Exp. 2: 5,000 ppb Cr) were fed during the grower phase only (Exp. 1) or during the grower and finisher phases (Exp. 2). In Exp. 1, pigs averaged approximately 43 kg BW at the conclusion of the trial, and no improvements ($P > 0.10$) in carcass characteristics were observed. Pigs fed diets supplemented with 400 ppb Cr from CrPic tended to have lighter carcass weights ($P < 0.05$) than pigs fed diets supplemented with 200 ppb Cr from CrPic. In Exp. 2, pigs had an average final BW of approximately 108 kg and pigs fed diets supplemented with Cr tended ($P < 0.07$) to have larger longissimus muscle areas (30.63 vs. 28.82 cm^2) than pigs fed the control diet with no supplemental Cr. However, backfat measurements and carcass lean were not different between diets.

In 2002, van de Ligt et al. reported on two experiments examining the effects of various energy levels and sources with and without Cr (0 or 200 ppb) supplementation. No effects of Cr on backfat thickness or longissimus muscle area were reported for either experiment. A reduction ($P < 0.05$) in kidney weight (244 vs 232g) was observed in Exp. 2, and a similar numerical trend ($P < 0.12$) was observed in Exp. 1 when Cr was supplemented to the diet. In addition, in Exp. 2, a decreased carcass ash content ($P < 0.05$) and a tendency for a decreased protein accretion rate ($P < 0.10$) was observed when Cr was supplemented to the diet. It is important to note, that the pigs used in both of these experiments were slaughtered at approximately 70 kg BW, which is lighter than in other reported literature, and therefore supplementation length was relatively short. However, Mathews et al. (2003) supplemented grower and finisher pig diets with 200 ppb Cr from chromium propionate and reported no consistent effects of Cr supplementation on carcass characteristics.

Conjugated Linoleic Acid

Conjugated linoleic acid (CLA) consists of isomers of linoleic acid that contain conjugated double bonds, and it has been reported to reduce whole body fat accretion by repartitioning fat and lean tissue (Park et al., 1997; DeLany et al., 1999; Stangl, 2000). Additional interest in CLA surrounds its reported antioxidative and anticarcinogenic properties, which have been implicated in benefits for human health (Ip et al., 1995; Belury et al., 1996). As a result, there has been interest in increasing the CLA content of pork. Pork normally contains a small amount of CLA, around 0.6 mg/g of fat (Chin et al., 1992). Joo et al. (2002) demonstrated that this level is increased by feeding 1 to 5% CLA as a dietary supplement for 4 wk prior to slaughter. They also observed an increase in intramuscular fat, but this effect was only significant from pigs fed diets supplemented with 5% CLA. Wiegand et al. (2002) fed diets to finishing barrows supplemented with 0.75% CLA for the last 0, 29, 56, or 87 kg BW prior to slaughter. Increasing the length of CLA supplementation resulted in a linear decrease in carcass backfat ($P < 0.05$) and linear increases in loin muscle area ($P < 0.01$) and percentage lean ($P < 0.01$). Similar to the results of Joo et al. (2002), an increase in CLA content of the loin muscle ($P < 0.01$) was observed with CLA supplementation. In addition, the saturated fatty acid content of the loin increased ($P < 0.001$), while the polyunsaturated fatty acid content decreased ($P < 0.001$).

Vitamin E

Vitamin E refers to a group of fat soluble compounds consisting of tocopherol and tocotrienol derivatives with α-tocopherol like activity (Combs, 1998). Vitamin E acts as an antioxidant, serving as a free radical scanvenger and protecting cell membranes from oxidative damage. Requirements for vitamin E in swine decrease from 16 IU/kg of diet (3-10 kg BW) to 11 IU/kg of diet (10-120 kg BW), with 1 IU equal to 0.67 mg of D-α-tocopherol or 1 mg of DL-α-tocopheryl acetate (NRC, 1998). However, several researchers have fed diets containing levels of vitamin E at 10-75 times the requirement and have reported improvements in shelf life of meat products from these pigs. Berg (2001) reviews current research, which clearly demonstrates that supplementing pig diets with 100-200 mg Vitamin E per kilogram of diet results in decreased lipid oxidation, improved color retention and improved water holding capacity of pork during storage compared to pigs receiving only NRC levels of vitamin E in their diet. However, the increased cost of adding such high levels of vitamin E makes this practice cost prohibitive at the present time.

Betaine

Betaine is a degradation product of choline that results from the loss of one methyl group from choline. Like choline, betaine or trimethlyglycine can serve as a methyl donor. Specifically, betaine acts as a methyl donor to homocysteine forming methionine. As a result, supplementation of betaine to methionine deficient pig diets can overcome a methionine deficiency (Campbell et al., 1995). Additional interest in betaine supplementation to swine diets has occurred, because degradation of homocysteine, in the absence of methionine synthesis, can result in an increased production of acetyl CoA. Acetyl CoA has many metabolic functions and fates, including serving as a precursor for fatty acid synthesis. Therefore, in theory, supplementation of swine diets with betaine, might lead to an increased methionine synthesis, and a decreased acetyl CoA synthesis, resulting in decreased lipogenesis.

Supplementing swine diets with betaine has produced mixed results. Differences in the reported literature may be due to differences in the methionine, crude protein, choline and energy concentrations of the diets being fed. A review of nine studies at the University of Kentucky (Cromwell et al., 2003), concluded that betaine was most effective in low energy diets at improving carcass lean, and that betaine could partially over come the detrimental carcass characteristics of animals fed a low protein diet. Lawrence et al. (2002) reported an increased growth rate from d28 to 56 when betaine was supplemented to a low CP diet, but not to a higher CP diet. They also observed a decrease in backfat depth in barrows, but not in gilts.

Vitamin D_3

The active form of vitamin D, 1α,25-dihydroxyvitamin-D_3 (1,25$(OH)_2$-D_3), is created through the dihydroxylation of vitamin D_3 (cholecalciferol). Vitamin D_3 can be endogenously produced in the skin through the photoconversion of 7-dehydrocholesterol, or it can be obtained from the diet as cholecalciferol or synthetic vitamin D. The two hydroxylation steps that transform vitamin D to the active form of vitamin D occur in the liver and the kidney. In the liver, 25-hydroxylase catalyzes the addition of a hydroxyl group to carbon 25. In the kidney, 25-hydroxyvitamin-D_3-1α-hydroxylase catalyzes the addition of a hydroxyl group to carbon 1, creating the active form of vitamin D, 1,25$(OH)_2$-D_3. Alternatively, 25(OH)-D_3 can be hydroxylated at carbon 24 to create 24,25$(OH)_2$-D_3 which does not possess the metabolic activity of 1,25$(OH)_2$-D_3, and can ultimately be further metabolized and excreted in bile. The primary function attributed to 1,25$(OH)_2$-D_3 is Ca homeostasis in the blood.

Increases in muscle Ca levels postmortem activate Ca^{2+}-dependent proteases (calpains) resulting in increased tenderness. Postmortem injections of calcium chloride into beef carcasses increased meat tenderness . Beitz et al. (1998) proposed that feeding elevated levels of vitamin D prior to slaughter would increase blood Ca concentrations, and result in increased postmortem activation of calpains. However, research in support of this theory is limited. Wiegand et al. (2002) found no affects of vitamin D supplementation (500,000 IU D_3/d) on carcass quality or meat tenderness. However, an improvement in Hunter color values was observed after 14d of storage. Similarly, previous researchers (Enright et al., 1998) reported no effects of vitamin D supplementation (176,000 IU/kg) to swine diets on pork tenderness, but color and water holding capacity were improved. However, reductions in live weight or carcass weight have also been reported when high levels of vitamin D are supplemented to swine diets (Enright et al., 1998; Sparks et al., 1999; Wiegand et al., 2002). Therefore, the cost effectiveness of increasing the level of supplementary vitamin D must be questioned.

Niacin

Niacin is the generic description for nicotinic acid (pyridine-3-carboxylic acid) and its derivatives exhibiting similar biological activity to nicotinamide (nicotinic acid amide). The nicotinamide (NAD+) moiety of niacin acts as an electron receptor in many biological redox reactions, including several reactions in glycolysis and the Krebs cycle. In its reduced form, nicotinamide, serves as an electron carrier for intracellular respiration. Determining the dietary requirement for niacin is complicated because niacin can be synthesized in the pig from tryptophan. Studies investigating the effects of supplemental niacin, above the current recommended requirement (NRC, 1998) are limited. However, two recent studies have reported beneficial effects (Piva, 1995; Real et al., 2002). Piva (1995) observed higher marbling scores when 150 mg of niacin were supplemented per kilogram of diet compared with controls. At 75mg of niacin per kilogram of diet, higher muscle reflectance scores and a darker red color were observed. In support of this, Real et al. (2002) reported improvements in gain:feed, subjective color score, and ultimate pH when niacin was supplemented in the diet at levels up to 55mg/kg. They also reported a decreased carcass shrink and drip loss (%) with niacin supplementation. The mode of action of niacin on carcass characteristics still remains unclear. It has been theorized that it may act by enhancing the Krebs cycle and preventing ketosis (Real et al., 2002).

Creatine monohydrate

Creatine monohydrate provides energy for ATP synthesis in muscle. Studies in humans demonstrated that intramuscular creatine levels can be increased with only 5 d of creatine monohydrate supplementation (Greenhaff, 1996). By increasing the creatine level in muscle, it may be possible to slow the onset of anerobic glycolsis and subsequent lactic acid generation via the synthesis of ATP from creatine phosphate. As a result, the decline in muscle pH may be less rapid. In addition, it may be possible to increase lean tissue weight as a result of water movement into the cell. Additionally, Haussinger (1996) reported that increased hydration of muscle cells may lead to an increased rate of protein synthesis and a decreased proteolysis. Berg et al. (1999) found a numerical increase in ADG when pigs were supplemented with creatine monohydrate, but the results were not significant. In later work from this same group (Maddock et al., 2002), a significant increase in BW (4.4 vs 6.6 kg BW) was observed after 5 d of creatine monohydrate supplementation. In both studies, 45 min. pH in semimembranosus muscle was higher for creatine monohydrate supplemented pigs compared to controls. No differences were observed for longissimus muscle. It was suggested that differences between muscle types were the result of a higher concentration of glycolytic (a-white or Type II) muscle fibers in semimembranosus muscle compared to longissimus muscle.

Carnitine

Carnitine is a vitamin-like compound that plays a role in the transport of long- and medium-chain fatty acids across the mitochondrial membrane. It has been hypothesized that supplementing the diet of pigs with carnitine may shift the ratio of protein:fat accretion, yielding a leaner animal at slaughter. Early research seemed to support this hypothesis (Smith et al., 1994), but results have been inconsistent, with some studies finding no effect of carnitine on backfat thickness or carcass lean (O'Quinn et al., 1999). More recent results (Owen et al., 2001) reported no effects of L-carnitine on the performance of nursery pigs, and minimal effects on daily lipid accretion. However, in growing and finishing pigs, decreases in backfat and increases in carcass lean were reported (Owen et al., 2001).

Dietary Fat Source

Adipocytes store fat as triacylgycerol, which consists of a glycerol backbone and three attached fatty acids. Fatty acids may be synthesized in the body or may be obtained from the diet. Increased absorption of dietary fatty acids results in decreased de novo synthesis of fatty acids. Therefore, it is possible to shift the content of triacylglycerol within the body by shifting the fatty acid profile within the diet. Berg (2001) and Pettigrew and Esnaola (2001) do a good job of reviewing the effects of supplementing swine diets with various fat sources on body lipid composition. It is very clear, that carcass lipid content can be altered by altering the dietary lipid content. However, defining what carcass lipid profile provides the highest quality pork product is less clear. Consumers seem to desire increased polyunsaturated fatty acid content. However, this results in a less firm product with a lower water holding capacity. Therefore a healthy balance must be struck between consumer nutritional demands, product appeal, processibility of the product, and shelf-life concerns.

Pantothenic Acid

Pantothenic acid is a component of coenzymne A, and is required in the diet of pigs at concentrations of 7 to 12 ppm. However, recent interest has surrounded the over supplementation of this vitamin and its potential effects on carcass lean. Stahly and Lutz (2001) observed a quadratic increase in estimated fat free lean (%) as pantothenic acid supplementation to the diet of pigs from wean to finish was increased from 0 to 120 ppm added pantothenic acid. Further research from this same group (Lutz and Stahly, 2002) reported a linear increase in estimated fat free lean (%) as the level of supplemental pantothenic acid was increased from 0 to 45 ppm added from wean to finish. However, in more recent work, Radcliffe et al. (2003) found mixed results when pantothenic acid was added to the grower and finisher phase, but not in the nursery. They reported a significant improvement in carcass lean (%), however, pigs fed supplemental pantothenic acid (30 ppm to a basal containing 13 ppm) also had a slightly lighter carcass. It is unclear why these differences were observed, but there were many differences between the experiment of Radcliffe et al. (2003) and those of Lutz and Stahly (2001, 2002). Lutz and Stahly (2001, 2002) added supplemental pantothenic acid starting in the nursery, whereas Radcliffe et al. (2003) waited until the grower phase. In addition, Lutz and Stahly (2001, 2002) fed elevated levels of all B vitamins (6x NRC, 1998), and lysine. All of these differences may have contributed to differences in observed results. Additional research in this area has or is in the process of being conducted. Unfortunately, none of it has been presented to date. Pantothenic acid supplementation to swine diets may be a very cost-effective method of increasing carcass lean, but there is not enough conclusive evidence in this area to draw any conclusions, and that which does exist brings into question its efficacy in practical swine diets.

Amino Acid Levels

Amino acids are essential for protein synthesis. Feeding a diet deficient in essential amino acids will result in decreases in growth rate and carcass lean (Ellis and McKeith, 1999). However, feeding a diet deficient in essential amino acids will also increase marbling in swine (Ellis and McKeith, 1999). While effective, reductions in growth and increases in overall carcass fat make it unlikely that this will become a practiced method of increasing marbling in pork.

Magnesium

Magnesium is an important cofactor in over 300 reactions in intermediary metabolism, and may reduce the release of norepinephrine and epinephrine from nerve terminals. Increases in these catecholamines may increase the rate of glycogenolysis, which as discussed by Berg (2001) will have a detrimental effect of pork quality. As a result, interest surrounds the supplementation of Mg to swine diets above the requirement (NRC, 1998) in an attempt to down regulate the sympathetic nervous system prior to slaughter, and thereby enhance pork quality. Berg (2001), presented the results of three studies investigating the supplementation of Mg for 5 d prior to slaughter, and concluded that it was an effective method of reducing the incidence of PSE pork. In those studies, the work of D'Souza et al. (1998, 1999) had the most pronounced effect, where the incidences of PSE were completely eliminated by the inclusion of 3.2g of Mg/d to swine diets. Apple et al. (2000), in two experiments, investigated the supplementation of pig diets with Mg from magnesium mica from weaning to market and found mixed results. They reported an improvement in loin color scoring in Exp. 1, but not in Exp. 2. However, in Exp. 2, decreases in 10^{th} rib backfat were observed when Mg was supplemented to the diet. This area appears to have a lot of potential, but needs further investigation. The length of Mg supplementation needed to show consistent effects and the level of Mg need to be further refined. Based on available data, it appears that short (5d) term supplementation of relatively high levels of Mg (3.2 g/d) are sufficient to provide beneficial results.

Ractopamine

Ractopamine is perhaps the carcass modifier that has received the most attention in recent years. Ractopamine is a β-adrenergic agonist that acts as a growth repartitioning agent, increasing lean tissue accretion and decreasing fat accretion. Increases in lean accretion rate are substantial and result in the need for a much more nutrient dense diet. The intricacies of properly and most effectively feeding ractopamine are numerous and are beyond the scope of this paper. Readers are directed to several recent publications concerning ractopamine for more information (Mills, 2002; Schinckel et al., 2002).

Conclusions

Dietary carcass modifiers provide opportunities for enhancements in pork quality via a number of different avenues. Some require no additional changes in the diet other than the addition of the carcass modifier, whereas others require significant changes in the diet to meet the changing needs of muscle and fat accretion. Decisions on the use of carcass modifiers will require an evaluation of current research on the effectiveness of the modifier, the cost of adding the modifier to the diet, and the benefit obtained by feeding the modifier. The future will undoubtedly bring additional carcass modifiers which will further enhance the producer's ability to provide pork products, which are of high quality to the consumer.

Literature Cited

Apple, J. K., C. V. Maxwell, B. deRodas, H. B. Watson, and Z. B. Johnson. 2000. Effect of magnesium mica on performance and carcass quality of growing-finishing swine. J. Anim. Sci. 78:2135-2143.

Beitz, D. C., J. C. Sparks, B. R. Wiegand, F. C. Parish, R. C. Ewan, R. L. Horst, and A. H. Trenkle. 1998. Effects of vitamin D on pork quality. In: Proc. Pork Quality and Safety Summit, National Pork Producers Council, Des Moines, IA, pp. 137.

Belury, M. A., K. P. Nickel, C. E. Bird, and Y. Wu. 1996. Dietary conjugated linoleic acid modulation of phorbol ester skin tumor promotion. Nutr. Cancer 26:149-157.

Berg, E. P. 2001. Swine nutrition, the conversion of muscle to meat, and pork quality. In: Swine

Nutrition (Eds. A. J. Lewis and L. L. Southern). pp. 631-658.

Berg, E. P., J. Spencer, and G. L. Allee. 1999. Dietary supplementation of creatine monohydrate in swine finishing diets improves fresh pork quality. UMC Animal Sciences Departmental Report. pp. 99-103.

Boleman, S. L., S. J. Boleman, T. D. Bidner, L. L. Southern, T. L. Ward, J. E. Pontif, and M. M. Pike. 1995. Effect of chromium picolinate on growth, body composition, and tissue accretion in pigs. J. Anim. Sci. 73:2033-2042.

Campbell, R. G., D. J. CAdogan, W. C. Morley, R. Vusitale, an d E. Virtanen. 1995. Interrelationship between dietary methionine and betaine on the growth performance of pigs from 65 to 100 kg. J. Anim. Sci. 73(Suppl. 1):82(Abstr.).

Chin, S. F., W. Liu, J. M. Storkson, Y. L. Ha, and M. W. Pariza. 1992. Dietary sources of conjugated dienoic isomers on linoleic acid, a newly recognized class of anticarcinogens. J. Food Compos. Anal. 5: 185-197.

Combs, G. F. 1998. The Vitamins. Fundamental aspects in nutrition and health, 2nd. ed., Academic Press, New York.

D'Souza, D. N., R. D. Warner, B. J. Leury, and F. R. Dunshea. 1998. The effect of dietary magnesium aspartate supplementation on pork quality. J. Anim. Sci. 76:104-109.

D'Souza, D. N., R. D. Warner, F. R. Dunshea, and B. J. Leury. 1999. Comparisons of different dietary magnesium supplements on pork quality. Meat Sci. 51:221-225.

DeLany, J. P., F. Blohm, A. A. Truett, J. A. Scimeca, and D. B. West. 1999. Conjugated linoleic acid rapidly reduce body fat content in mice without affecting energy intake. Amer. J. Physiol. 276:1172-1179.

Ellis, M. and F. K. McKeith. 1999. Nutritional influence on pork quality. National Pork Producers Council Fact Sheet 04422, Des Moines, IA.

Enright, K. L., B. K. Anderson,. M. Ellis, F. K. McKeith, L. L. Berger and D. H. Baker. 1998. The effects of feeding high levels of vitamin D_3 on pork quality. J. Anim. Sci. 76(Suppl. 1):149(Abstr.).

Evock-Clover, C. M., M. M. Polansky, R. A. Anderson, and N. C. Steele. 1993. Dietary chromium supplementation with or without somatotropin treatment alters serum hormones and metabolites in growing pigs without affecting growth performance. J. Nutr. 123:1504-1512.

Greenhaff, P. L. 1996. Creatinine supplementation: Recent developments. Br. J. Sports Med. 30: 281-282.

Gromwell, G., M. Lindemann, R. Coffey, G. Parker, A. Pettey, J. Montegue, J. Randolph, and K. Laurent. 2003. Dietary supplement has marginal benefits. Available: http://nationalhogfarmer.com/ar/farming_nutrition_4/index.htm. Accessed July 9, 2003.

Haussinger D. 1996. The role of cellular hydration in the regulation of cell function. Biochem. J. 313:697-710.

Ip, C., J. A. Scimeca, and H. Thompson. 1995. Effect of timing and duration of dietary conjugated linoleic acid on mammary cancer prevention. Nutr. Cancer, 24:241-247.

Joo, S. T., J. I. Lee, Y. L. Ha, and G. B. Park. 2002. Effects of dietary conjugated linoleic acid on fatty acid composition, lipid oxidation, color, and water-holding capcity of pork loin. J. Anim. Sci. 80:108-112.

Lawrence, B. V., A. P. Schinckel, O. Adeola, and K. Cera. 2002. Impact of betaine on pig finishing performance and carcass composition. J. Anim. Sci. 80:475-482.

Lindemann, M. D., C. M. Wood, A. F. Harper, E. T. Kornegay, and R. A. Anderson. 1995. Dietary chromium piucolinate additions improve gain:feed and carcass characteristics, and accretion rates of carcass tissues in growing-finishing pigs and increase litter size in reproducing sows. J. Anim. Sci. 73:457-465.

Maddock, R. J., B. S. Bidner, S. N. Carr, F. K. McKeith, E. P. Berg, and J. W. Savell. 2002. Creatine monohydrate supplementation and the quality of fresh pork in normal and halothane carrier pigs. J. Anim. Sci. 80:997-1004.

Matthews, J. O., A. D. Higbie, L. L. Southern, D. F. Combs, T. D. Bidner, and R. L. Odgaard. 2003. Effect of chromium propionate and metabolizable energy on growth, carcass traits, and pork quality of growing-finishing pigs. J. Anim. Sci. 81:191-196.

Mills, S. E. 2002. Biological basis of the ractopamine response. J. Anim. Sci. 80(E.

Suppl. 2):E28-E32.

Mooney K. W. and G. L. Cromwell. 1997. Efficacy of chromium picolinate and chromium chloride as potential carcass modifiers in swine. J. Anim. Sci. 75:2661-2671.

Mooney, K. W. and G. L. Cromwell. 1995. Effects of dietary chromium picolinate supplementation on growth, carcass characteristics, and accretion rates of carcass tissues in growing-finishing swine. J. Anim. Sci. 73:3351-3357.

Mooney, K. W. and G. L. Cromwell. 1999. Efficacy of chromium picolinate on performance and tissue accretion in pigs with different lean gain potential. J. Anim. Sci. 77:1188-1198.

NRC. 1998. Nutrient Requirements of Swine (10th ed.). National Academy Press, Washington, DC.

O'Quinn, P. R., A. T. Waylan, R. D. Goodband, J. L. Nelssen, J. A. Unruh, J. C. Woodworth, M. D. Tokach, and K. Q. Owen. 1999. Effects of modified tall oil, chromium nicotinate, and L-carnitine on growth and carcass traits of finishing pigs. J. Anim. Sci. 77(Suppl. 1):67(Abstr.).

Owen, K. Q., H. Ji, C. V. Maxwell, J. L. Nelssen, R. D. Goodband, M. D. Tokach, G. C. Tremblay, and S. I. Koo. 2001. Dietary L-carnitine suppresses mitochondrial branched-chain keto acid dehydrogenase activity and enhances protein accretion and carcass characteristics of swine. J. Anim. Sci. 79:3104-3112.

Owen, K. Q., J. L. Nelssen, R. D. Goodband, M. D. Tokach, and K. G. Friesen. 2001. Effect of dietary L-carnitine on growth performance and body composition in nursery and growing-finishing pigs. J. Anim. Sci. 79:1509-1515.

Page, T. G., L. L. Southern, T. L. Ward, and D. L. Thompson, Jr. 1993. Effect of chromium picolinate on growth and serum and carcass traits of growin-finishin pigs. J. Anim. Sci. &1:656-662.

Park, Y., K. L. Albright, W. Liu, J. M. Storkson, M. E. Cook, and M. W. Pariza. 1997. Effect of conjugated linoleic acid on body composition in mice. Lipids 32:853-858.

Pettigrew, J. E. and M. A. Esnaola. 2001. Swine nutrition and pork qual;ity: a review. J. Anim. Sci. 79(E. Suppl.):E316-E342.

Piva, G. 1995. Tranquilizing effect of nicotinic acid and its effects on pork meat and Parma ham quality. In: Feed Additive News. Lonza, Inc., pp. 1-7.

Radcliffe, J.S., B.T. Richert, L. Peddireddi, and S.A. Trapp. 2003. Effects of supplemental pantothenic acid during all or part of the grow-finish period on growth performance and carcass composition. J. Anim. Sci. 81(Suppl. 1):255.

Real, D. E., J. L. Nelssen, J. A. Unruh, M. D. Tokach, R. D. Goodband, S. S. Dritz, J. M. DeRouchey, and E. Alonso. 2002. Effects of increasing dietary niacin on growth performance and meat quality in finishing pigs reared in two different environments. J. Anim. Sci. 80:3203-3210.

Schinckel, A. P., B. T. Richert, and C. T. Herr. 2002. Variation in the response of multiple genetic populations of pigs to ractopamine. J. Anim. Sci. 80(E. Suppl. 2):E85-E89.

Smith, J. W., II, K. Q. Owen, J. L. Nelssen, R. D. Goodband, M. D. Tokach, K. G. Friesen, T. L. Lohrmann, and S. A. Blum. 1994. The effects of dietary carnitine, betaine and chromium nicotionate supplementation on growth and carcass characteristics in growing-finishing pigs. J. Anim. Sci. 72(Suppl. 1):274(Abstr.).

Sparks, J. C., B. R. Wiegand, F. C. Parish, Jr., and J. A. Love. 1999. Effects of length of feeding conjugated linoleic acid (CLA) on growth and body composition of pigs. J. Anim. Sci. 77(Suppl. 1): 178(Abstr.).

Stahly, T.S. and T.R. Lutz. 2001. Role of pantothenic acid as a modifier of body composition in pigs. J. Anim. Sci. 79(Suppl. 1):68.

Stahly, T.S. and T.R. Lutz. 2002. Efficacy of pantothenic acid as a modifier of body composition in pigs. J. Anim. Sci. 80(Suppl. 1):163.

Stangl, G. I. 2000. Influence of dietary conjugated linoleic acid on volatile profiles, color and lipid oxidation of irradiated raw chicken meat. Meat Sci. 56:387-395.

Van de Ligt, C. P. A., M. D. Lindemann, and G. L. Cromwell. 2002. Assessment of chromium tripicoloinate supplementation and dietary energy level and source on growth, carcass, and blood criteria in growing pigs. J. Anim. Sci. 80:483-493.

Wiegand, B. R. , J. C. Sparks, F. C. Parrish, Jr. and D. R. Zimmerman. 2002. Duration of feeding cvonjugated linoleic acid influences growth performance, carcass traits, and meat quality of finishing barrows. J. Anim. Sci. 80:637-643.

Wiegand, B. R., J. C. Sparks, D. C. Beitz, F. C. Parish, Jr., R. L. Horst, A. H. Trenkle, and R. C. Ewan. J. Anim. Sci. 80:2116-2121.

Automated Sorting Technologies for Market Hogs

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Summary

The commercial swine finishing sector in the United States has shown great interest in the use of large group housing and automatic sorting technologies over the last few years. Fueled by efforts to improve production efficiency and improve economic returns, large groups have offered producers the opportunity to more effectively utilize barn space by reducing the area dedicated to alleys as well as an associated reduction in the cost of gating within the barn. In an effort to reduce labor associated with sorting and improve market value by meeting packer-designated carcass weight ranges, producers have adopted automatic sorting technologies in conjunction with large pen designs. Through this rapid adoption of these new technologies, additional opportunities to improve feed management, pig welfare, and labor efficacy have been identified. However, along with the opportunities, additional challenges and questions have become evident and require the attention and resources of researchers in conjunction with industry/commercial affiliates to fully answer. The following materials will briefly identify the opportunities and challenges related to large group and automatic sorting technologies.

Introduction

The concept of auto-sorting dates back many years, if not many generations, as entrepreneurial swine producers sought simple ways to sort market hogs that were often raised in relatively large groups of varying age and weight and housed in either large outside lots or on feeding floors. Early attempts at sorting generally included an adjustable gate separating either water from the feed, the sleeping area from the feeding area, or placements near the loading area. All of the early designs sorted only by stature, not weight, and were prone to errors in mechanical adjustment and (or) differences in physical stature (height and width) of pigs with similar weight. The driving force behind sorting gates was an attempt to save labor while maintaining relatively simple, low cost facilities.

Modern, "cookie cutter"-designed confinement housing systems, holding 1000 to 1200 pigs per barn in groups of 25 to 30 per pen, have been the trend since the industry moved toward two- or three-site production and all-in, all-out pig flow. Coupled with limited scientific data, producers routinely sorted pigs by sex and weight on entry to the finishing facility in an attempt to reduce (narrow) weight variation within a pen at a young age with the hope of more uniform growth through reduced competition within pens and the ability to program nutrition to more closely meet the pigs' needs as well as a reduction in sorting near market weight. However, too frequently uniformity of weight at entry does not result in uniform growth rate and subsequent weight uniformity throughout the finishing stage, resulting in labor intensive sorting to meet market targets and reduce carcass weight discounts (sort loss) at the packing plant. The surge in contract production, where one individual may be responsible for the daily management of 1 to 8 barns with limited outside labor availability, makes sorting a difficult, time-consuming, and expensive task that producers are now actively trying to avoid.

Recent interest in what may be termed "non-traditional, modern" finishing facilities has led to development and implementation of automatic sorting technology in new and retrofitted finishing facilities. The dynamics of automatic sorting technology revolve around two basic themes: 1) large group housing (100 to 500+ pigs per pen) and the intricacies of managing pigs in large groups, and 2) accurately and easily monitoring pig weight, weight gain, and weight variation to meet production and marketing targets while minimizing labor costs. The first finishing facilities using automatic sorting technology appear to have been started in the year 2000 in Pennsylvania (National Hog Farm, June 15, 2002) and now have spread quickly throughout the United States. Currently, the concepts of both large group finishing (>100 pigs per pen) and automatic sorting technology have limited scientific data to either support or refute the industry claims that are being made. In the context of the available scientific information, this paper will primarily serve to describe the current status of the technology and identify key areas for research and discovery.

Large Group Housing

The concept of large group housing has received great interest from producers involved in large-scale swine production. A weekly or bi-weekly flow of large groups of high-health, single source pigs has reduced the disease load substantially and reduced the (perceived) need to house pigs in small uniform groups to maximize management opportunities. Previous success with large groups (up to 1000 pigs per pen) housed in retrofitted poultry barns that provide low cost, less intense housing and "hoop-house" facilities that utilize deep-bedded, large group (100 – 200 pigs per pen) systems have been demonstrated to be at least partially successful in the U.S. These success stories have led innovators to attempt the concept of large groups in environmentally controlled, confinement facilities where a balance of cost control and pig performance play a large part in system economics.

A move toward large group housing involves a myriad of choices, questions, and concerns for the production system and managers including:

1) Projected impact on pig performance
   a. Feed intake
   b. Growth rate
   c. Feed efficiency
   d. Variability in weight and weight gain
   e. Nutrition program
2) Projected impact on pig well-being
   a. Social hierarchy
   b. Mortality rate
   c. Morbidity rate
   d. Injury incidence, severity, and proportion
3) Projected impact on caretakers
   a. Ability to identify problem pigs
   b. Ability to treat problem pigs
   c. Ability to sort market pigs
   d. Safety of workers in large groups of swine
4) Projected impact on facilities
   a. Determining optimal stocking density
   b. Gating requirements
   c. Feed and water location
   d. Feed and water allocation
   e. Flooring wear and tear
5) Project impact on Production Economics
   a. Margin over cost per square foot of building space

Peer-reviewed literature estimates of the impact of large group finishing on pig performance are somewhat contradictory. Kornegay and Notter (1984) summarized literature from previous studies and estimated that an 11 to 21% decrease in growth rate was predicted when pigs were housed in groups of 80 compared with groups of 10 pigs. Wolter et al. (2001, groups of 25, 50 and 100) a recent report comparing group size in a wean-to-finish production system reported pigs were lighter and had poorer feed conversion efficiency while maintaining similar daily feed intake for the first 8 weeks post-weaning when fed in groups of 50 and 100 when compared with groups of 25. However, by the end of the finishing period (255 ± 1.98 lbs) no differences in average daily gain, average daily feed intake, feed efficiency, or mortality rate. They also noted an increase in percent morbidity (pigs removed due to poor health or injury) in pigs housed in groups of 25 compared with group sizes of 50 or 100. Schmolke et al. (2003, groups of 10, 20, 40, or 80 pigs), in a study comparing group sizes for pigs started on test at 51 and ending at 209 pounds, reported no differences in average daily feed intake, average daily gain, feed efficiency, or variability in final body weight over the 12 week testing period.

Schmolke et al., 2003 also reported no group size differences for evidence of tail biting or spatial subgrouping as group size increased up to 80 pigs per pen. Scientists have often felt that the social structure of pigs housed in larger groups may differs from those housed in smaller groups that may influence the incidence of injuries and social vices that can be detrimental to animal health, welfare, and subsequent performance. Additional research to characterize social structure and optimal pen layout in very large groups of swine is needed prior to making definitive claims in this area of study.

Unfortunately, at this time, the scientific literature does not address a number of previously listed questions and concerns revolving around large pen designs, including the implications of very large groups on expected performance. Primary limitations to conducting unbiased research on very large groups is the lack of university and (or) commercial facilities and financial resources to adequately design, carryout, and replicate experiments across times and locations. Also, because many of the pig and non-pig issues are interrelated, careful experimental design must be used to avoid biases that can result from confounding of treatments with other extraneous factors. To many people the experimental design appears very simple, but in practice the issues are much more complex.

Automatic Sorting Technology

Marketing swine within packer specific, optimal weight and carcass lean categories allows producers to optimize a combination of production efficiency and revenue generation. Lightweight swine carcasses are usually severely discounted due to the difficulty or inability of packers to merchandise small cuts and the associated increase in the labor costs per pound of pork marketed from the lightweight carcass. Price discounts for heavy carcasses also occur but to a much lesser degree than for lightweight carcasses. Producers who market swine within the packer designated carcass weight and lean content matrix generate more revenue as a result of more pounds of lean marketed and a reduction in losses due to improper sorting based on weight. Sorting based on individual live weights taken prior to harvest would, theoretically, ensure minimal sort loss, but is not feasible in practice due to the cost associated with time, labor, and the physical attributes of facilities that prevent weighing of pigs in an efficient manner. In addition, as producers concentrate on pig flow, marketing's may be pulled forward or delayed to accommodate fluctuations in pig flow and pig performance. Thus, many marketing strategies in conventional facilities rely on eyeball estimation of live weights and the desire to fill transport trailers rather than defined weights, resulting in substantial sort losses and extensive labor costs associated with sorting.

Automatic sorting technologies used in the industry rely on the ability to reduce sort losses, a reduction in labor associated with sorting, the potential for more efficient building space utilization, and reliable equipment and scales situated in a large group setting as the key components for economic justification of an investment in the technology. Additional opportunities exist for producers to capture production efficiency through refined feeding strategies that focus on continuous sorting and feeding of pigs based on body weight. The sum of the potential advantages is the ability to efficiently market more pork, at a greater marginal value and with less labor cost, per square foot of finishing space.

Automatic sorting for market purposes can not only reduce sort loss, but also provide flexibility in meeting market needs for different packing plants. Opportunities to tailor marketing of the fast growing, fatter pigs (often barrows) to packers that desire lightweight carcasses improves lean content relative to weight and allows marketing prior to the point where feed conversion efficiency becomes poor. Likewise, marketing the slow growing, lean, often lightweight, closeout set of pigs within a group to a specific packer may improve revenue generation for the operation. In theory, specialty markets with even tighter weight restrictions may offer benefits to producers wishing to market targeted products. However, weight alone does not ensure biological uniformity of fat and muscle as they relate to cut size and trimmed weight. Industry estimates (Farmweld™, Inc., 2002) of the advantage of marketing pigs in a typical 1000 pig finishing facility range from $0.60 to 1.99 per pig depending on the aggressiveness of sorting.

Because automatic sorting facilities typically do not have a traditional alley for pig observation, a greater number of pigs can be finished within the constraints of the outside walls. Industry contacts estimate that that 75 to 90% of the space previously used by the alley can be turned into usable pig space, which, in a typical 1000 pig finishing facility, equates to approximately 55 to 68 more pigs per building (@ 8 square feet per pig). Observations of pigs in large group finishing facilities also show that dunging areas, sleeping areas, and feeding areas (by design) become more defined which might allow for further reduction in required pig space per pig. While the industry maintains guidelines of 7.5 to 8 square feet per pig, it is not uncommon for producers to stock at 7.2 square feet per pig or slightly lower when the first cut of market pigs are sold at a lighter weight. Research to study the appropriate pig space allocation in large-pen, auto-sort facilities must be completed to establish effective guidelines and to ensure that welfare of the pig is being adequately addressed.

Labor savings associated with automatic sorting technology result as function of pigs being sorted prior to loading and easier handling of pigs during the loading process. It is estimated that loading time is reduced by 50% (Farmweld™, Inc., 2002) in a typical 1000 pig finishing facility. Cost savings per pigs will be a function of labor rate and time required for sorting and loading compared to a traditional finisher. The importance of easier pig handling is attributed to pigs being familiar with movement through the scales and one-way gates that are similar to a loading chute. The importance of reduced stress at loading has potential implications on the number of dead or downer pigs in transit.

Enhanced feed management protocols are an added opportunity for producers utilizing automatic sorting technology. One proposed benefit is the ability to monitor and track weight of the pigs on a more continuous basis allowing for adjustment of dietary nutrient levels more precisely and by sorting on weight they will be able to feed two or more diets concurrently while maintaining the large group setting. A second benefit is the ability to fine-tune the use of growth promoting additives to help improve pig uniformity and barn flow.

Producers have also theorized that that feed usage can be reduced through removal of feed from sorted pigs prior to loading. The potential benefits of feed removal prior to loading include reduced waste in transport and at the packing plant and the associated reduction in labor required to clean and disinfect trailers. An additional claim of improved pork quality from pigs that have had feed removed prior to harvest has been made by some. However, research on the effectiveness of this process on improving fresh or processed muscle quality traits is not definitive and thus needs to be further evaluated in the context of large pens and auto sorting, where the length of time pigs are removed from feed will vary considerably as a function of how fast or slow the sorting process is completed for a barn of pigs.

The cornerstone of automatic sorting technologies is the ability to effectively train pigs to move across the scales. Many times this is accomplished through establishing a food court that is used to entice the pigs out of the lounging area. Adjustments to gates and gate configurations are used near the scale entrance to train pigs to the sorting device. Timing of training appears to be crucial to overall success of the system. Pig trained very near to the desired market weight may go off feed prior to learning the process of movement through the scale, resulting in reduced weight gain and the potential to increase the incidence of ulcers due to a feast and famine intake pattern. Some systems use water and feed separation to train pigs to the sorting device, a practice that must be thoroughly evaluated from the standpoint of the vital importance of adequate access to clean, fresh water on pig welfare and production efficiency.

Additional questions arise when analyzing the concepts of automatic sorting and the economic feasibility at the commercial level including: 1) pig eating behavior (frequency, speed, time spent in the food court, movement between the food court and lounging area, etc), 2) food court design (feeder space availability, overall size relative to total pen area, water access, etc), and 3) pig and group social behavior. Research to answer these and other questions in the context of the production systems is necessary to fully understand the implications on pig performance, social behaviors, and system-wide economic feasibility.

Conclusion

The concepts of large group housing and automatic sorting present new and unique challenges for both the industry and academia. From the industry standpoint, system-wide economics appears to drive the attention producers have in choosing to use large pens independent of, or in conjunction with, automatic sorting technology. Unfortunately, the industry field data currently available often lacks sufficient replication and is often confounded with multiple factors which may lead to erroneous conclusions over time.

From the academic standpoint, the cost of animal identification, scales, retrofitting facilities, and labor availability severely limit opportunities for large group research within an most institutions. Thus, to fully understand the implications of large group housing and automatic sorting technology on the very basic performance criteria for the pig, industry and academia will need to partner resources to conduct the necessary research that can replicated and free of confounding factors.

References

Connor, J. F. and J. F. Lowe. 2002. Economic analysis and discussion of automatic sorting technology. Farmweld, Inc. Teutopolis, IL.

Kornegay, E. T., and D. R. Notter. 1984. Effect of floor space and number of pigs per pen on performance. Pig News Info. 5:23-33.

Neutkens, D. 2002. Hands off sorting. National Hog Farmer. Vol. 47, No. 6. P 14-16.

Schmolke, S. A. Y. Z. Li, and H. W. Gonyou. 2003. Effect of group size on performance of growing-finishing pigs. J. Anim. Sci. 81:874-878.

Wolter, B. F. M. Ellis, S. E. Curtis, N. R. Augspurger, D. N. Hamilton, E. N. Parr, and D. M. Webel. 2001. Effect of group size on pig performance in wean-to-finish production systems. J. Anim. Sci. 79: 1067-1073.

Effects of Antibiotic Withdrawal on Resistance

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Summary

Antibiotic resistance is a reality. It seems that every time you watch TV or pick up a newspaper another study describes the increase in antibiotic resistance and the potential impact on human health. Certainly the livestock industry is very aware of the limitations on antibiotic use in Europe and the potential limitations here in the United States. Fortunately we are learning that livestock production can occur in the absence of antibiotic growth enhancers with changes in production practices and the inclusion of new growth promoters. Unfortunately the legacy of antibiotic resistance currently on the farm will not disappear with the withdrawal of antibiotics from livestock feeds. A thorough understanding of the impact of antibiotics on the ecology of the microflora is necessary before we can effectively eliminate the genetic determinates for antibiotic resistance.

What is the current state of antibiotic resistance?

The effectiveness of antibiotics at controlling infectious disease has been apparent since the early 1940's when penicillin was first available for commercial use. Unfortunately by 1947 penicillin resistance had already been documented (Tan et al., 2000). This observed adaptation indicated that bacteria had the potential to fight back but the significance of this resistance was not fully recognized. This ability of pathogens to become resistant to antibiotics is certainly a critical human health issue (Flaherty and Weinstein, 1996). The problem is perpetuated by the fact that the genes coding for antibiotic resistance can be transferred between species of bacteria in the population. In effect we are not only concerned with the antibiotic resistant bacteria themselves, but also the genetic determinants for antibiotic resistance and the possibility that they can be passed among the population.

The Center for Disease Control released a study where only 0.2% of the pneumococcus isolates collected from patients between 1979 and 1987 were resistant to penicillin, but the percentage had increased to 6.6% in 1994 (CDC, 1994). This was not a unique observation. In fact, all around the world with all classes of antibiotics. increased antibiotic resistance has been documented (Aaerstrup et al., 1998, Cohen, 1992, Neu, 1992). This is not only a significant issue in regards to human health, but also animal health and food production. There is strong evidence that the use of antibiotics as growth promotants in livestock feeds actually enhance the selection for antibiotic resistance. Europe has banned the use of most antibiotics on the farm as growth promoters and is strictly controlling their therapeutic use (European Commission/Scientific Steering Committee, 1999). This action may indeed reduce the propagation of new antibiotic resistant bacteria in the gastrointestinal tract and on the farm, but what happens to the antibiotic resistance that already exists?

How do bacteria become resistant to antibiotics?

Microorganisms can demonstrate either an intrinsic or an acquired resistance to antibiotics. An intrinsic resistance occurs as a result of a point mutation in the bacteria's DNA. This actually occurs very infrequently since the bacteria possess protective mechanisms to ensure that DNA replication is error free. This is an evolutionary process that requires several stepwise mutations to result in the microorganism becoming resistant to the antibiotic. This kind of resistance rarely occurs in the absence of the selective pressure of antibiotic treatment (MacManus, 1997).

Acquired antibiotic resistance may occur in the absence of antibiotic treatment but the use of antibiotics has been shown to promote the phenomena. When an antibiotic comes in contact with a group of bacteria the sensitive organisms will die, but the resistant organisms or those that can acquire resistance survive. The selective pressure of the antibiotic results in a bacterial population that is resistant to the effects of the antibiotic. Acquired resistance results from the transfer of resistant genes from one bacterium to another. These genes may confer resistance to one or several different antibiotics. Once the genes are transferred they may be incorporated into the bacterial chromosome or maintained extra-chromosomally on a plasmid (MacManus, 1997). Plasmids are included in a group of horizontal mobile elements that also include bacteriophages, intregons and transposons. This phenomenon was first described in livestock in the late 1960's, when a large reservoir of plasmid encoded oxytetracycline resistance was detected in farm animals and in the potential pathogen *Salmonella typhimurium* (Swann Committee, 1969). Often the antibiotic resistance genes in the plasmid are genetically linked to other survival genes which results in the selection of these genes in a bacterial population in the absence of the antibiotic (Hall and Collins, 1995; Salyers and Amabile-Cuevas, 1997). One of the most significant methods by which resistance is transferred between bacteria is with the plasmid (Lederberg, 1998). In Gram-negative bacteria such as *Salmonella* and *E.coli* this method relies on the presence of sex pili or fimbriae (Meynell, 1972). The tips of the pili initiates contact with specific receptors on a recipient cell via the carbohydrate residues of glycoproteins or glycolipids. Currently it is not clear if the pili acts as a channel for the movement of the plasmid or simply acts to bring the two cells together. Once the cells are attached the circular plasmid is replicated and one copy is transferred to the recipient cell, resulting in antibiotic resistance in both the donor and recipient cells (Willetts, 1972). Plasmids that can transport DNA to adjacent bacteria are known as conjugative plasmids. Berends and coworkers (2001) estimated that 85% of therapy failures experienced in human medicine from staphylococci (ß-lactams), *Enterobacteriaceae* (ampicillin, sulpha/trimethoprim, gentamicin and chloramphenicol) and enterococci (vancomycim) were a result of conjugation-induced antimicrobial resistance.

Dealing with antibiotic resistance in animal production

Widespread resistance to antimicrobials has been documented in several pathogens commonly associated with disease in both animals and humans. Perhaps the two most economically significant pathogens to the livestock industry are *Escherichia coli* and *Salmonella*. These pathogens, particularly *Salmonella*, are also associated with more than 1.3 billion dollars per year in cost to humans related specifically to hospitalization as a result of antibiotic resistance (Institute of Medicine, 1998). The potential for resistant bacteria to negatively impact livestock health and production is real.

The beneficial effects of using antibiotics as growth promotents, to the animal, producer, consumer, and environment have been well documented. We are all aware that antibiotics in the feed result in lower morbidity and mortality of the animal, shorter time to market, less expensive meat, and ultimately less manure (Rosen, 1995). The mode of action of these antibiotic growth promotants is somewhat less clear. Potential modes of action include elimination of pathogens and their potentially growth-suppressing toxins or metabolites, selection for a more efficient gastrointestinal microbial population, and direct effects on the gut metabolism and morphology of the host animal (Muir, 1985). The future of growth promotants depends on our ability to find ways to achieve the same beneficial effects in the absence of antibiotics, in addition to finding ways to reduce or eliminate the antibiotic resistance that has already been established.

Eliminate antibiotic use in animals and solve the problem?

The popular press and many consumer groups demand the removal of antibiotics from animal feeds, claiming that antibiotic use is responsible for the appearance and maintenance of resistance on the farm and in the human population. The premise is that resistant organisms are less physiologically fit and in the absence of the selective pressure will not be able to compete with the antibiotic susceptible bacterial population (Lee and Edlin, 1985). As a result the resistant bacteria will simply die off resulting in a return to the susceptible population that was present prior to antibiotic exposure. Unfortunately this belief is not founded since many resistant organisms survive extremely well in the absence of antibiotic selection (Gillespie, 2001). It is true that at least originally antibiotic resistant bacteria may pay a physiological price for resistance development, but many of these organisms have demonstrated the ability to recover physiological fitness in subsequent generations (Andersson, D.I. and B.R. Levin. 1999; Schrag et al., 1997). This recovery is often as a result of compensatory second-site mutations if the resistance is of chromosomal origin, or plasmid mutation if the resistance is plasmid mediated (Tables 1 and 2). This ability to recover from the physiological stress may be one reason that it takes a relatively long time to detect changes in prevalence of resistance following antibiotic withdrawal.

In May of 1972, the University of Kentucky discontinued the application of all antimicrobial agents for therapeutic, prophylactic, and subtherapeutic use in one of their swine herds. This was done in an attempt to evaluate the many factors associated with the development of antimicrobial resistant bacterial populations in pigs. A review of the research surrounding this herd indicates that the population of antibiotic-resistant bacteria established in this herd at the time when antibiotics were used routinely has survived from generation to generation for over 26 years even in the absence of antibiotic exposure (Langlois et al., 1986). This observation is contrary to many popular beliefs that antibiotic resistance is a physiological burden to the bacteria and will be easily lost when the resistance is no longer needed, as with the withdrawal of antibiotic supplementation (Salyers and Amabile-Cuevas, 1997). In reality a persistent and high frequency of antibiotic resistance can be found in the normal bacterial flora of humans and animals in the absence of antibiotic exposure (Calva et al., 1996; Gilliver et al., 1999).

Where does antibiotic resistance originate?

Although specific point mutations in an organism's genetic material may occur resulting in antibiotic resistance, the most significant source of resistance appears to be conjugation-induced (Berends et al., 2001). In many instances this transferable resistance appears to originate from mutated housekeeping genes that are responsible for the growth and survival of the organism or more commonly from natural resistance genes originating from antibiotic producing microorganisms as a self defense mechanism (e.g. *Streptomyces* spp.) (Davis, 1994). The genetic material coding for antibiotic resistance can be transferred between distinctly different organisms including bacterial, archaea and eukarya (Dröge et al., 1998). The gastrointestinal tract of humans and animals provides a nutrient rich environment in which the transfer of genetic material can occur. Shoemaker and co-workers (2001) demonstrated that a significant horizontal transfer of antibiotic genes occurs in the human colon. In addition, the resulting resistant organisms were highly stable maintaining resistance in the absence of antibiotic challenge. Perhaps one of the most important observations of this study was the role *Bacteroides* spp plays in the maintenance and transfer of antibiotic resistance genes in the gastrointestinal tract. Members of the genus *Bacteroides* are recognized as indigenous normal flora that may be opportunistic pathogens under predisposing conditions such as immunosuppression, aminoglycoside therapy, tissue damage, malignancy, etc. Some researchers estimate 50% of fecal matter is composed of *Bacteroides*. These organisms represent a much larger proportion of the gastrointestinal population than the often-studied coliform and *E. coli* populations, yet we know very little about the maintenance of antibiotic resistance in these organisms. Studies with antibiotic resistant *E.coli* isolated from swine and cattle have demonstrated that resistance maintenance varies depending on the antibiotic. Tetracycline and chloramphenicol resistant *E.coli* have been shown to maintain resistance in the absence of antibiotic challenge for longer periods of time than gentamicin resistant *E. coli* (Dubel et al., 1982; Dunlop et al., 1998; Langlois et al.1986). Similar data describing the maintenance of resistance in the intestinal *Bacteroides* is not available.

Resistance maintenance may also be associated with a co-selection process where the use of one antibiotic actually selects for the resistance of another. This multiple resistance may originate from the same plasmid or transposon that is carrying the antibiotic resistant determent (Barbosa and Levy, 2000). This phenomenon is not limited to just antibiotics but may include: heavy metals, disinfectants, and other antimicrobials that may stress the bacteria (Russel, 1997; Moken et al., 1998). Given the possibility that antibiotic resistance can be directly linked to bacterial survival in the presence of a variety of external stresses it is obvious why antibiotic resistance doesn't disappear in the absence of antibiotic use.

What other factors effect the maintenance of antibiotic resistance?

Resistant organisms that predominate in the bacterial population during antibiotic use are more likely to be transferred between hosts (Donskey et al., 2000). Similar selection in subsequent hosts would boost the possibility that these organisms or its resistance determinants could reach around the world. An example of this was first described in the 1950s when specific strains of *Staphylococcus aureus* demonstrating an inducible penicillinase enzyme which resulted in penicillin resistance through out the worlds hospitals and ultimately into communities in the 1960s (Williams, 1959). Today with the increased world travel and the movement of food over great distances the potential for resistance to originate in one part of the world and quickly spread is very real (Figure 1).

Stage of production or animal age appears to be an important factor in the persistence of antibiotic resistance in both man and animals. Langlois and coworkers (1988) demonstrated that the fecal coliform population of young pigs less than 6 months of age had a significantly greater proportion of antibiotic resistant bacteria than older animals. Studies in other young populations including children, calves and chickens had similar results (Wierup, 1975; Butaye et al., 1998). One explanation for these observations is that younger gastrointestinal tracts may be colonized more readily by the bacteria carrying antibiotic resistance than the more established populations generally found in the older animals. In addition, the composition of the gastrointestinal bacterial population may vary depending upon physical location. Moro and coworkers (2000) described a difference in antibiotic resistance patterns from organisms obtained from different parts of the digestive tract. This observation might play a role in explaining the elevated shedding of antibiotic resistant bacteria in the feces of animals following stress. Transportation, disease, temperature extremes and other physical challenges have been directly associated with increased prevalence of antibiotic resistant bacteria in the feces (Langlois and Dawson, 1999, Moro et al., 2000; Minton et al., 1983).

The environment also plays an important role in the presence and persistence of antibiotic resistance in an animal population. Since most antibiotics are products of microbial metabolism and are commonly produced in nature the genetic determinants of antibiotic resistance are present in the environment. The current concern about the use of antibiotics in agriculture is not limited to animal production, in fact several antibiotics are used in horticulture and aquaculture (Schnabel and Jones, 1999, Barbosa and Levy, 2000). These compounds, in addition to the unaltered antibiotics excreted in human or animal feces following administration of antibiotic treatment will find their way in to the soil and water and ultimately back to humans (Chee-Sanford et al., 2001: Schnabel and Jones, 1999).

Conclusion

Amid growing evidence and concern that the use of antibiotics on the farm will continue to exacerbate the dramatic increase in deaths and illness associated with antibiotic resistance, countries worldwide are examining the use of antibiotics in agriculture. In many instances it is believed that simply eliminating the antibiotics will solve the problem. In fact, once antibiotic resistance is established in a bacterial population it has been shown that it is maintained for long periods of time in the absence of the selective pressure of antibiotic supplementation. Because of the persistence of antibiotic resistance genetic determinants in the environment the potential for a pathogen to obtain resistance remains. If we are to salvage the effectiveness of antibiotics for future generations we must direct our efforts toward preventing the development of additional antibiotic resistance in animals and the environment. Before we can begin to significantly reduce the prevalence of antibiotic resistance we must seek a better understanding of the factors involved in the persistence of antibiotic resistance following the withdrawal of antibiotics. Hopefully with this understanding we can learn how to reduce antibiotic resistance in the animal and human populations.

References cited

Aaerstrup, F.M., F. Bager, N.E. Jensen, M. Madsen, A. Meyling, and H.C. Wegener. 1998. Surveillance of antimicrobial resistance in bacteria isolated from food animals to antimicrobial growth promoters and related therapeutic agents in Denmark. APMIS 106: 606.

Andersson, D.I. and B.R. Levin. 1999. The biological cost of antibiotic resistance. Curr. Opin. Microbiol. 2:489-493.

Barbosa, T,M, and S.B. Levy. 2000. The impact of antibiotic use on resistance and persistence. Drug Res. Updates 3:303-311.

Björkman, J., D. Hughes, and D.I. Andersson. 1998. Virulence of antibiotic resistant *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA 95:3949-3953.

Björkman, J., I. Nagaev, O.G. Berg, H.Hughes, and D.I. Andersson. 2000. Effects of environment on compensatory mutations to ameliorate costs of antibiotic treatment. Science 287:1479-1482.

Bouma, J.E. and R.E. Lenski. 1988. Evolution of a bacteria/plasmid association. Nature. 335:351-352

Berends, B.R., A.E. van den Bogaard, F. Van Knapen, and j.M. A. Snijders. 2001. Human health hazards associated with the administration of antimicrobials to slaughter animals. Part II. An assessment of the risks of resistant bacteria in pigs and pork. Veterinary Quarterly. 23:10-21.

Bryan, L.E.. 1984. Antimicrobial Drug Resistance. Academic Press. New York.

Center for Disease Control (CDC). 1994. http://www.fda.gov/fdac/features/795-antibio.html

Buddington, R.K. and E. Weiher. 1999. The application of ecological principles and fermentable fibers to manage the gastrointestinal tract ecosystem. J. Nutr. 129:1446S.

Calva, J.J., J. Sifuentes-Osornio, J. and C. Ceron. 1996. Antimicrobial resistance in fecal flora: longitudinal community-based surveillance of children from urban Mexico. Antimicrob. Agents Chemother. 40: 1699-1702.

Cohen, M.L. 1992. Epidemology of drug resistance: implications for a post-antimicrobial era. Science 257:1050.

Davis, J. 1994. Inactivation of antibiotics and dissemination of resistance genes. Nature 264:375-381.

Donskey, C.J., T.K. Chowdhry, M.T. Hecker, C.K. Hoyen, J.A. Hanrahan, A.M. Hujer, R.A. Hutton-Thomas, C.C. Whalen, R.A. Bonomo, and L.B. Rice. 2000. Effect of antibiotic therapy on the density of vancomycin-resistant entercocci in the stool of colonized patients. N. engl. J. Med. 343:1925-32.

Dröge, M., A. Pühler and W. Selbitschka. 1998. Horizontal gene transfer as a biosafety issue: A natural phenomenon of public concern. J. Bacteriol. 64: 75-90.

Dubel, J.R., D.L. Zink, L.M. Kelly, S.A. Naqi, and H.W. Rensham. 1982. Bacterial Resistance: frequency of gentamicin-resistant strains of *Escherichia coli* in the fecal microflora of commercial turkeys. Am. J. Vet. Res. 43:1786-1789.

Dunlop, R.H., S.A. McEwen, A.H. Meek, R.C. Clarke, W.D. Black, R.M. Friendship. 1998. Associations among drug treatments and antimicrobial resistance of fecal *Escherichia coli* of swine on thirty-four farrow to finish farms in Ontario, Canada. Pre. Vet. Med. 34:265-282.

European Commision/Scientific Steering Committee, DG XXIV. Opinion of the steering Committee of Antimicrobial Resistance. 28 May, 1999.

Flaherty, J. P. and R.A. Weinstein. 1996 Nosocomial infection caused by antibiotic-resistant organisms in the intensive-care unit. Infect. Control Hospital Epi. 17 (4): 236

Gillespie, S. H. 2001. Antibiotic resistance in the absence of selective pressure. Int. J. Antimicrob. Agents 17:171-176.

Gilliver M.A., M. Bennett, M. Begon, S.M.Hazel, and C.A. Hart. 1999. Antibiotic resistance found in wild rodents, Nature 401: 233-234

Godwin, D and J.H. Slater. The influence of the growth environment on the stability of a drug resistance plasmid in *Escherichia coli* K12. J. Gen. Microbiol. 111:201-210.

Hall, R.M. and C.M. Collis. 1995. Mobile gene cassettes and integrons: capture and spread of genes by site –specific recombination. Mol. Microbiol. 15: 593.

Institute of Medicine, Antimicrobial resistance: Issues and Options. 1998. Harrison, P.F. and J. Lederberg, eds. Washington, D.C.: National Academy Press.

Johanson, U., A. Aevarrson, A. Liljas, and D. Hughes. 1996. The dynamic structure of EF-G studied by fusidic acid resistance and internal revertents, J. Mol. Biol 258:420-432

Langlois, B.E. and K.A. Dawson. 1999. Antimicrobial resistance of gram-negative enteric bacteria from pigs in a nonantibiotic-exposed herd before and after transportation. J. Food Prot. 62:797-799.

Langlois, B.E., K.A. Dawson, G.L. Cromwell, and T.S. Stahly. 1986. Antibiotic resistance following a 13 year ban. J. Anim. Sci. 62 (suppl.3):18.

Langlois, B.E., K.A. Dawson, I. Leak and D.K. Aaron. 1988. Effect of age and housing location on antibiotic resistance of fecal coliforms from pigs in a nonantibiotic-exposed herd. Appl. Environ. Microbiol. 54:1341-1344.

Lederberg, J. 1998. Infectious agents, hosts in constant flux. ASM News. 64:18.

Lee, S.W. and G. Edlin, 1985. Expression of tetracycline resistance in pBR322 derivatives reduces the reproductive fitness of plasmid containing *Escherichia coli*. Gene 39:173-180.

Lenski, R.E., S.C. Simpson, and T.T. Nguyen. 1994. Genetic analysis of a plasmid encoded, host genotype-specific enhancement of bacterial fitness. J. Bacteriol. 176:3140-3147.

MacManus, M.C. 1997. Mechanisms of bacterial resistance to antimicrobial agents. Am.J. Health0Syst. Pharm. 54:1420.

McDermott, P.J., P. Gowland, and P.C. Gowland. 1993. Adaptation of *Escherichia coli* growth rates to the presence of pBR322. Lett. Appl. Microbiol. 17: 139-143

Meynell, G.G. 1972. Bacterial plasmids. M.I.T. Press. Cambridge, MA.

Minton, N.P., J. Marsh, and T. Atkinson. 1983. The R-factors of multiple antibiotic resistant fecal coliforms isolated from a domestic dog. J. Applied Bact. 55:445-452.

Modi, R.I. and J. Adams. 1991. Coevolution in bacteria-plasmid populations. Evolution 45:656-667.

Modi, R.I, L.H. Castilla, S. Puska-Rozsa, R.B. Helling, and J. Adams1992. Genetic changes accompanying increased fitness in evolving populations of *Escherichia coli*. Genetics 130:241-249.

Modi, R.I, C.M. Wilke, R.F. Rosenzweig, and J. Adams. 1991. Plasmid macroevolution: Selection of deletions during adaptation in a nutrient limited environment. Genetica 84:195-202.

Moken, M.C., L.M. McMurray, and S.B. Levy. 1997. Selection of multiple antibiotic resistant (Mar) mutants of Echerichia coli by using the disinfectant pine oli: roles of the mar and acrAB loci. Anitmicrob. Agents Chemother. 41:2770-2772.

Moro, M.H. , G.W. Beran, L.J. Hoffman, and R.W. Griffith. 2000. Effects of cold stress on the antimicrobial drug resistance of Escherichia coli on the intestinal flora of swine. J. App. Microbiol. 88: 836-844

Muir, L.A. 1985. Mode of action of exogenous substances on animal growth – an overview. J. Anim. Sci 61 (Suppl. 2) 154.

Neu, H.C. 1992. The crisis of antimicroibial resistance. Science 257:1064.

Reynolds, M.G. 2000. Compensatory evolution in rifampin-resistant *Escherichia coli*. Genetics 156: 1471-1481.

Rosen, G.D. 1995. Antibacterials in poultry and pig nutrition. In: Biotechnology In Animal Feeds And Feeding. VCH Publishers, New York NY. p. 143.

Russel, A.D. 1997. Plasmids and bacterial resistance to biocides. J. Applied Microbiol. 82(2): 155-165.

Schrag, S.J. and V. Perrot. Reducing antibiotic resistance. 1996. Nature 381:120-121.

Schrag, S.J., V. Perrot and B.R. Levin. 1997. Adaptation to the fitness costs of antibiotic resistance in *Escherichia coli*. Proc. R. Soc. Lond. B. Biol. Sci. 264:1287-1291.

Salyers, A.A. and C.F. Amabile-Cuevas. 1997. Why are antibiotic resistance genes so resistant to elimination? Antimicrob. Agents and Chemother. 54: 1232.

Schnabel, e.L. and A.L. Jones. 1999. Distribution of tetracycline resistance genes and transposons among phylloplane bacteria in Michigan apple orchards. Appl. Environ. Microbiol. 65(11)4898-4907.

Schrag, S.J., V. Perrot, B.R. Levin. 1997. Adaptation to the fitness costs of antibiotic resistance in *Escherichia coli*. Proc. R. Soc. London B.Biol. Sci. 264:1287-1291.

Shoemaker, N.B., H. Vlamakis, K. Hayes, and A.A. Salyers. 2001. Evidence for extensive resistance gene transfer among Bacteroides spp. and among Bacteroides and other genera in the human colon. Appl. Environ. Microbiol. 67 (2) 561-568.

Swann Committee, Her Majestys Stationary Office. 1969. Report of joint committee on the use of antibiotics in animal husbandry and veterinary medicine. London.

Tan, Y., D.J. Tillett and I.A. McKay. 2000. Molecular strategies for overcoming antibiotic resistance in bacteria. Mol. Med. Today. 6:309.

Wierup, M. 1975. Antibiotic resistance and transferable antibiotic resistance of *Escherichia coli* isolated from Swedish calves 6 and 30 days old. Nordic Vet. Med. 27:77-84.

Willetts, N. 1972. The genetics of transmissible plasmids. Ann. Rev. Genetics. 6:257.

Williams, R.E.O. 1949. Epidemic staphylococci. Lancet 1(1): 190-195.

Table 1. Compensatory second-site mutations of chromosomal resistance.

Organism	Resistance	Compensatory Mutation	Reference
Salmonella typhimurium	Streptomycin (*rpsl* mutation)	Intragenic, *rpsl* (maintained)	Björkman et al., 1998
	Streptomycin (*rpsl* mutation)	extragenic, *rpsl*D/E (maintained)	Björkman et al., 1998; Björkman et al., 2000
	Nalidixic acid (*gyr*A mutation)	Intragenic, *gyr*A (maintained)	Björkman et al., 1998
	Rifampicin (*Rpo*B mutation)	Intragenic, *Rpo*B (maintained)	Björkman et al., 1998
	Fusidic acid (*Fus*A mutation)	True reversion, *Fus*A (lost)	Björkman et al., 2000; Johnson et al., 1996
	Fusidic acid (*Fus*A mutation)	Intragenic, *Fus*A (often maintained)	Björkman et al., 2000; Johnson et al., 1996
Escherichia coli	Streptomycin (*rpsl* mutation)	extragenic, *rpsl*D/E (maintained)	Schrag and Perrot, 1996; Schrag et al., 1997
	Rifampicin (*Rpo*B mutation)	Intragenic, *Rpo*B (maintained)	Reynolds, 2000

Table 2. Compensatory mutations of extrachromosomal resistance.

Organism	Resistance	Location of mutation	Reference
Escherichia coli K12	Ampicillin, Tetracycline, Streptomycin, Sulfonamide (plasmid pTPI20)	Plasmid	Godwin and Slater, 1979
Escherichia coli B	Tetracycline, Chloramphenicol (plasmid pACYC184)	Chromosome	Bouma and Lenski, 1988
Escherichia coli B	Ampicillin, Tetracycline (plasmid pBR322)	Chromosome	Lenski et al., 1994
Escherichia coli K12	Ampicillin, Tetracycline (plasmid pBR322)	Chromosome	McDermott et al., 1993
Escherichia coli JA101	Ampicillin, Tetracycline (plasmid pBR322_5)	Chromosome	Modi and Adams, 1991
Escherichia coli RR1	Ampicillin, Tetracycline (plasmid pBR322)	Chromosome	Lee and Edlin, 1985
Escherichia coli	Ampicillin (plasmid pBR322:: Tn3)	Chromosome	Modi et al., 1992
Escherichia coli	Ampicillin, Tetracycline (plasmid pBR322)	Plasmid	Modi et al., 1991

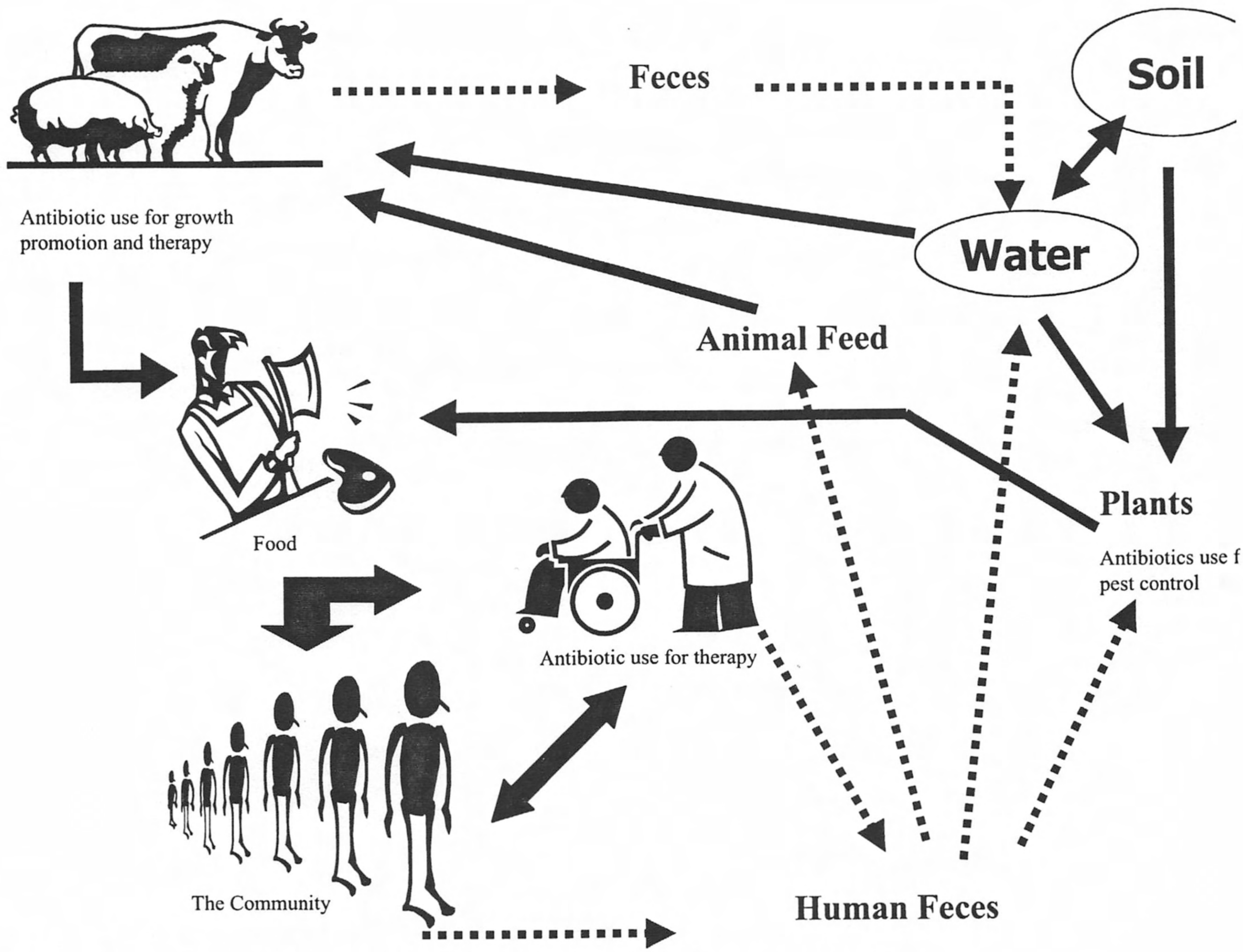

Figure 1. Routes of transmission of antibiotic resistant bacteria and resistance genes.

The Danish Experience After Termination of Use of Antimicrobial Growth Promoters

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Summary

Denmark stopped using antimicrobials as growth promoters in finishing pigs in early 1998. There followed a reduction in the total amount of antimicrobials used, reduction in the prevalence of resistance to antimicrobials in some commensal and zoonotic bacteria taken from pigs at slaughter, and little interruption of historic trends in pig performance. Nearly two years later, in late 1999, Denmark stopped using antimicrobial growth promoters in nursery pigs. This was followed by an apparent increase in the total amount of antibiotics used because of increased therapeutic use, including an increase in use of tetracyclines. The prevalence of tetracycline resistance then increased in *Salmonella typhimurium* taken from pigs at slaughter and from ill humans. The growth performance of nursery pigs was clearly impaired, and the mortality rate increased. Termination of antimicrobial use for growth promotion in finishing pigs was accomplished without obvious difficulty, but termination of use in nursery pigs caused significant problems and no obvious benefit.

Introduction

The world-wide pig industry is under increasing pressure to reduce the amount of antimicrobials used in pig production because of concerns about resistance to antimicrobials used in human medicine. Antimicrobials are now used in pig production for growth promotion, for disease prevention, and for treatment of diseased animals. There is a wide range of potential steps toward reduction of antimicrobial use, some much more severe than others. It may be unrealistic to attempt complete elimination of antimicrobial use from pig production on a wide scale, while elimination of their use for growth promotion is a significantly smaller step.

The European Union has tightened restrictions on antimicrobial use much more aggressively than has the United States. Sweden and Denmark have eliminated the use of antimicrobials for growth promotion in animals, and their experience may be instructive for us. Denmark's experience may be more relevant for us because it eliminated antimicrobial growth promoters more recently, and the aggressive Danish industry has more in common with the U.S. industry than does the Swedish industry.

This paper will follow the following outline:

- Chronology of restrictions on antimicrobial use in Denmark
- Amount of antimicrobials used
- Antimicrobial resistance
- Effects on pig production
- Implications

Chronology of Restrictions on Antimicrobial Use in Denmark

Denmark's termination of antimicrobial use for growth promotion occurred in a very different environment of antimicrobial use than now exists in the United States, as detailed below. All interpretation of the Danish experience should consider the fact that the Danish industry had a much smaller selection of antibiotics at its disposal than we have in the United States. For that reason, termination of antibiotic use for growth promotion would be a bigger step for us than it was for Denmark.

The following key events occurred in Denmark:

- 1995: Denmark started monitoring antibiotic resistance
- 1997: Denmark started an intensive research pro-

gram on the discontinuation of use of antimicrobial growth promoters, including study of alternatives
- December, 1997: The European Union (EU) banned avoparcin
- January, 1998: Denmark banned virginiamycin for growth promotion
- **February, 1998: Denmark stopped using antimicrobial growth promoters in cattle, poultry and finishing pigs (>35 kg)**
- July, 1999: The EU banned four antimicrobial growth promoters (tylosin, spiramycin, bacitracin, virginiamycin)
- September, 1999: The EU banned olaquindox and carbadox
- **December, 1999: Denmark stopped using antimicrobial growth promoters in weaners (<35 kg)**

When Denmark banned the use of antimicrobial growth promoters in nursery pigs (December, 1999), it effectively eliminated the use of only flavomycin and avilamycin. However, the timing of the events confounds the cessation of use of these products with the elimination of the quinoxalines (olaquindox and carbadox) and, to a lesser degree, with the elimination of the four growth promoters in July, 1999.

Amount of Antimicrobials Used

In Denmark, the pig industry is so much larger than the other livestock industries that it dominates the data on the use of antimicrobials in livestock production. The antimicrobial use data available historically are for all livestock industries combined, but they provide considerable information about use in pigs.

The total amount of antimicrobials used in livestock production in Denmark is now dramatically lower than before Denmark stopped using antimicrobial growth promoters in finishing pigs (Figure 1). The program has been remarkably successful in reducing total antimicrobial use.

The effect of stopping the use of antimicrobial growth promoters in nursery pigs is less clear. The use of antimicrobials for disease treatment has increased. In fact, it appears the total amount of antimicrobials used has increased since termination of the use of antimicrobial growth promoters in nursery pigs.

It may also be important that the antimicrobials eliminated as growth promoters are unrelated to compounds used in human medicine, but that several of the drugs used therapeutically are also used in human medicine, or are related to drugs used in human medicine. In other words, there was a shift from drugs considered "safer" to those considered "less safe". Note that this situation would not hold in the United States, because we use some of the drugs considered "less safe" (because they are related to drugs used in human medicine) as growth promoters.

Antimicrobial Resistance

The fundamental reason for terminating the use of antimicrobials as growth promoters is to reduce the threat of resistance to antimicrobials in human medicine. There is widespread agreement that the source of most antimicrobial resistance is use of antimicrobials in human medicine, but there are indications that use of antimicrobials in animals may also contribute to the problem in some small measure. Therefore, there is legitimate pressure to reduce antimicrobial use in animal production.

Denmark has an impressive system for monitoring antimicrobial resistance in bacteria in animals, in food produced from animals (pork and chicken), and in humans. Commensal bacteria that may serve as a reservoir of antimicrobial resistance, and also zoonotic bacteria (those that are pathogenic to both animals and man) are monitored. The commensal bacteria monitored are *Enterococcus faecium*, *Enterococcus faecalis*, and *Escherichia coli*; the zoonotics are *Salmonella* and *Campylobacter*. Care must be used in drawing general conclusions, because there is considerable variation among responses among various "bug-drug combinations".

All observations reported in this section are taken from DANMAP (2003).

There was a relatively high initial prevalence of resistance to several antimicrobials in *E. faecium*, and in general the prevalence declined after termination of use of antimicrobial growth promoters. Resistance to only erythromycin (related to tylosin) was prevalent in *E. faecalis*, and that resistance declined with time. There were too few isolates of *Enterococci* from either pork or people to draw conclusions about changes over time. There were no indications of changes in prevalence of resistance to any antimicrobial in *E. coli* as a result of termination of use of antimicrobial growth promoters.

In *Campylobacter coli* taken from pigs at slaughter, there was a decline in resistance to erythromycin after the termination of antimicrobial growth promoter use, presumably due to reduced use of the related antibiotic tylosin.

In *Salmonella typhimurium* collected from pigs at slaughter, there was a marked increase in prevalence of resistance to tetracyclines during 2000 and later, after termination of use of antibiotic growth promoters in nursery pigs. This increase is presumably related to the increased use of tetracyclines for disease treatment during these years. It also appears to be related to increased prevalence of resistance to tetracyclines in *S. typhimurium* taken from ill humans. On one hand, this observation suggests that termination of use of antibiotic growth promoters in Denmark had an unintended detrimental consequence. On the other hand, it confirms a cause-and-effect relationship between antibiotic use in pigs and antibiotic resistance in bacteria taken from ill humans.

It may be that the use of antimicrobial growth promoters during the finishing phase produces a higher prevalence of antimicrobial resistance in bacteria collected from the pig at slaughter than does use of the same antimicrobial during the nursery phase, several months before slaughter. The resistance data are consistent with this suggestion, but do not show it clearly.

Effects on Pig Production

The growth rate of finishing pigs in Denmark has increased steadily during the last several years, with no clear interruption associated with termination of antimicrobial growth promoter use (Table 1). There has been a less regular trend for improvement of feed efficiency, with some suggestion that termination of antimicrobial growth promotants use interrupted the trend slightly. The mortality rate of finishers has steadily increased, again with no clear effect of termination of antimicrobial growth promoters. Overall, the effects of terminating the use of antimicrobial growth promoters on finishing pig performance have been remarkably small.

The situation with nursery pigs is strikingly different (Table 2). Terminating the use of antimicrobial growth promoters had detrimental effects on growth rate, feed efficiency and mortality rate. These effects occurred at the same time as an apparent increase in enteric disease.

Pork producers and their advisors are creative, and we should expect that they would introduce adaptive mechanisms to compensate for the loss of antimicrobial growth promoters. There is an indirect indication that Danish producers did just that. Recent controlled experiments in Denmark showed an average growth rate response of 10.8% to antimicrobial growth promoters in nursery pigs (Kjeldsen, 2002), but the growth rate declined only 2.6% upon termination of antimicrobial growth promoter use. The difference may have occurred because producers introduced other compensating technologies. There are no data to confirm that suggestion, or to indicate what technologies may have been adopted beyond the increased use of therapeutic antimicrobials. I suggest that other possibilities include adoption of all in-all out pig flow, increased weaning age, more aggressive biosecurity and sanitation measures, and addition of organic acids to the diet, among other possibilities. Some of these measures are expensive. Given the lack of information about what changes producers actually made and the cost of those changes, I find it impossible to estimate with confidence the increased production cost associated with termination of antimicrobial growth promoters.

Implications

Antimicrobial growth promoters can be withdrawn from finishing pigs without clear detrimental effects on growth performance, but eliminating them from the diets of nursery pigs is remarkably unsuccessful.

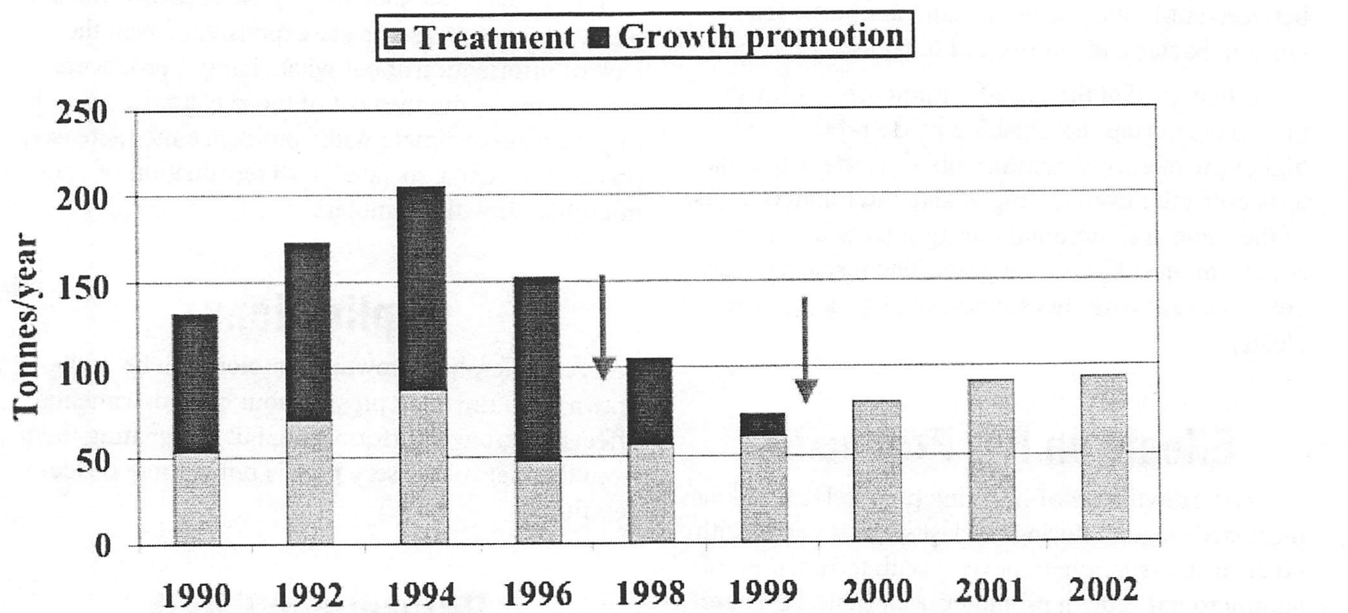
References Cited

Callesen, J. 2002. Effects of termination of AGP-use on pig welfare and productivity, In: International Invitational Symposium; Beyond Antimicrobial Growth Promoters in Food Animal Production, November 6-7 2002, Foulum, Demark.

DANMAP. 2003. DANMAP 2002- Use of antimicrobial agents and occurrence of antimicrobial resistance in bacteria from food animals, foods and humans in Denmark. ISSN 1600-2032. http://www.svs.dk.

Kjeldsen, N. 2002. Alternatives to AGP in pig production- practical experience, In: International Invitational Symposium; Beyond Antimicrobial Growth Promoters in Food Animal Production, November 6-7 2002, Foulum, Demark.

Figure 1. Antimicrobial Use in Denmark.



Data from DANMAP, 2003. Data before 2001 exclude most use in aquaculture.
Arrows show stop of antimicrobial growth promoters in finishers and weaners, respectively.

Table 1. Effect of termination of use of antimicrobial growth promoters in finishing pigs on finishing pig performance.[a]

Year	Average daily gain, lb.	Feed units/kg gain[b]	% Mortality
1995	1.64	2.94	3.0
1996	1.68	2.93	3.2
1997	1.72	2.89	3.2
1998	1.73	2.91	3.4
1999	1.76	2.89	3.6
2000	1.80	2.89	3.4
2001	1.82	2.89	3.5

[a]Average daily gain and mortality data are from Callesen, 2002. Feed units/kg gain data are from H. Wegener, *personal communication.* All data are from the Efficiency Control Program (production records system) to which about 1,500 Danish producers (11% of total producers) submit data monthly.

[b]A feed unit is a measure of dietary energy, approximately equal to a kg of feed.

Table 2. Effect of termination of use of antimicrobial growth promoters in nursery pigs on nursery pig performance.[a]

Year	Average daily gain, lb.	Feed units/pig to 66 lb.[b]	% Mortality
1995	.93	97.9	2.7
1996	.93	97.6	2.8
1997	.92	97.1	2.9
1998	.94	99.4	2.9
1999	.90	99.2	3.6
2000	.91	99.3	3.5
2001	.91	99.3	3.5

[a]Average daily gain and mortality data are from Callesen, 2002. Feed units/kg gain data are from H. Wegener, *personal communication.* All data are from the Efficiency Control Program (production records system) to which about 1,500 Danish producers (11% of total producers) submit data monthly.

[b]A feed unit is a measure of dietary energy, approximately equal to a kg of feed. Includes sow feed.

Viable Alternatives to Antibiotics

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Summary

Concern about the potential negative impact of usage of antibiotics for animals on the efficacy of those same antibiotics (or similar antibiotics) is shaping governmental policies and restricting antibiotic use for animals in some countries. When this occurs there is need for careful attention of the nutritionist to the impact of dietary ingredients on animal performance and interaction with gastrointestinal organisms. Various dietary ingredients have been been examined with respect to their impact on gastrointestinal physiology and host resistance to pathogenic organisms. Research is identifying those feed ingredients that function in extra-nutrient manners.

Introduction

During the past decade there has been a change in the global perspective toward antibiotic use in animal agriculture. Some countries now have total bans on the use of in-feed antibiotics, some have partial bans, and some have experienced very little change. Irrespective of the current status of antibiotic use, all countries have experienced fairly intense discussion about the issue of antibiotic use in animal agriculture as it relates to perceived impact on antibiotic resistance in humans and the effectiveness of antibiotics for humans. When discussions about antibiotic use take place in a non-agricultural setting, the conclusion often arrived at is that if there is any potential benefit to humans from removing them from agricultural use, then it probably should be done. While this conclusion is understandable to some extent, it is not a knowledgeable one that considers the potential impact of that decision on all affected parties – farmer/producers, feed manufacturers, and the farm animals themselves.

All production systems are set up based on some type of standard inputs in relation to some type of production/management system. When those inputs change, the production systems may not then be the most well-suited for maximizing animal health and, consequently, performance and profitability. For example, physical facilities can not be abandoned before their amortized life without a large negative impact on the producer; if facilities are designed for an animal density that assumes use of antibiotics as a health management tool, removal of that tool can adversely affect animal well-being. Therefore, if a standard input such as antibiotic use is altered, producers and every individual in their production teams (feed supplier, veterinarian, extension personnel) have to be creative in determining how to maintain animal health and resultant profitability if that enterprise is to remain a viable part of animal production.

With regard to antibiotics, there are two aspects of their use that need to be discussed. Those two aspects are their use as an aid for improved growth and efficiency of feed utilization and their use in disease protection/management/elimination. Because the improved performance responses are often markedly less in 'clean' environments, there is good reason to think that these two aspects of their use are closely related and that the improved performance is a result of improved health related to some degree, if not a large degree, of microorganism control. The public debate about the use of antibiotics for improvement of growth often misses this point; perhaps an alternative term such as 'growth permitter' would communicate better the concept that growth can be compromised by the particular profile and metabolism of the microorganisms and that the altered growth related to antibiotic use is a consequence of the impact of the antibiotic on the microorganisms and not directly on the animal. While this concept can conceivably be better communicated, it still remains a concept that may have relatively little impact on the debate at this point because of governmental positions that are already taken in some countries.

With regard to antibiotic use in disease protection/management/elimination there needs to be distinction between the use of antibiotics for treatment and for prophylaxis. With regard to their use for disease treatment, in many instances there is no alternative for antibiotics. Specific antibiotics developed/discovered to treat specific diseases are the fastest and most efficacious manner of facilitating animal health and well-being. If an operation is challenged by a particular organism or has a recurring problem with a particular organism, the manner of attacking and resolving that problem must be very focused. Fortunately, outbreaks of a disease can still be treated with antibiotics under a veterinarians care in even the most restricted use countries. With regard to the use of antibiotics for prophylaxis (or to prevent the spread of disease), public opinion would range between allowance and restriction for use. Irrespective of public opinion and governmental regulation, an important issue for the nutritionist and feed supplier are those non-antibiotic things that can be done through the feed that might resolve or, hopefully, prevent the health related problems.

For the purposes of this paper, the potential alternatives that might allow or facilitate growth enhancement in the absence of antibiotics and the alternatives that might serve in some prophylactic manner will be discussed. These are not offered as alternatives for antibiotics but items in addition to antibiotics. Without antibiotics, a producer simply has fewer tools in his arsenal for facilitating animal health and well-being. Antibiotics are a powerful tool. Its absence puts more pressure on the nutritionist and health professional to understand the development of gastrointestinal physiology, mechanisms to which it is responsive, the impact of feed ingredients and processing on the intestinal milieu, and how all of this interacts with an extremely dynamic ecosystem within the gut. In the absence of antibiotics there is a necessity for the host defense mechanisms to provide for the health of the animal itself. This will require an understanding of, and ability to regulate or manipulate, various physiological aspects within the animal. An understanding of these items has always been important in nutrition but whereas it is useful when antibiotics are available it becomes requisite in situations where antibiotics are not available.

This paper will not discuss items such as dietary fiber, lactose, zinc oxide or copper sulfate. All of these have been researched and reviewed extensively and do have roles that must not be ignored especially in young pig diets (fiber should be considered in diets of pigs of all ages). The items discussed in this paper will simply be examples of items that have demonstrated the capacity to affect the gastrointestinal system in a way that would suggest that they have potential to be of benefit in a production system that does not use, or that does not have access to, antibiotics. Because their mechanism of action may be different than that of antibiotics, they may have potential to provide an additive response to antibiotics in situations where antibiotics are available. But that additivity is not guaranteed. Also, simply because they have shown potential to be of benefit in a situation without antibiotics does not mean they will function that way in all situations; their ability to effect a positive impact will depend on a host of items that includes, but is not limited to, animal age and intestinal development, other dietary ingredients, and disease load or pressure. Some of these items are not new items to swine diet formulation but there may have been recent research that illustrates a new facet of their utility.

Existing technology

A feed ingredient that has transformed early weaning diets is plasma protein. There have been improvements in this product over the past two decades and there are a variety of products currently available. While product differences exist, it can be said that there is generally an increase in feed intake and, consequently, growth in weanling pigs consuming this product. Table 1 is from recent research in Spain and gives an example of the additivity in general feedlot performance that is possible with this product and an antibiotic. Later tables and discussion in this paper will provide additional information about plasma protein but at this point it should be noted that should antibiotics not be available to a producer, plasma protein is a major contributor to good performance in the young pig. In the absence of antibiotics, discussion and decisions about inclusion level of the plasma protein used will be even more critical as it relates to maintaining growth performance.

An item that has received quite a bit of attention for the past three decades is that of acidification of the diet. Early work in the US tended to be relatively simplistic in simply looking at graded additions of citric or fumaric acid. More recent work tends to look at dietary buffering capacity of the ingredients, age of pig, and alternate types of acids to explain or expand on early results. In general there is much more use of dietary acids in Europe. An example of alternate acids as well as salts of an acid are provided in results from German experiments in Table 2. In addition to growth effects, it can be seen that positive effects on the frequency and severity of diarrhea are possibilities. A summary of a variety of experiments was recently reported by Mroz (2003) with a statistical analysis of the pooled results (Table 3 and 4) that examines acids for various age pigs. While the specific experiments would need to be examined to determine items that may affect magnitude of responses, again it can be seen that the use of acids to affect the gastrointestinal tract can be of benefit beyond just the nursery phase (when presumably there is a limited acid secretory ability).

The addition of acids to the diet draws ones attention to pH in different segments in the gastrointestinal tract. It is sometimes forgotten that feed processing itself may affect gastrointestinal pH. A recent study (Table 5) presented at the *2003 Digestive Physiology in Pigs* meeting demonstrates that coarseness of grind and pelleting can impact organic acid production and microbial load in the stomach. Further aspects of pelleting are illustrated in Table 6. The results of the pelleting are presented in conjunction with the inclusion of a xylanase enzyme. In this study it is evident that there were differences in form of feed (pellet vs mash) in the potential to impact meat quality. In addition to these items related to feed processing, there is substantial interest in the potential benefits of fermented liquid feed in Europe to affect pig performance and health. Should liquid feed become a part of US production, then there may be aspects of its modification that would warrant further attention.

Alteration of the gastrointestinal flora by means of probiotics has received much attention during the past decade. A summary of some of the published literature that looked beyond the performance effects to actual gut health (as evidenced by diarrhea) is provided in Table 7. In addition to these probiotics, the potential of mannan oligosaccharides (MOS) to alter the microflora has been extensively studied and has been reviewed by Miguel et al. (2002). The results of a meta-analysis by those authors of studies invovling the commercial product Bio-Mos® has demonstrated that the best effects are when the product is included at 0.2% when pigs are weaned at an early age of 17-21 days and fed the product for 2 weeks.

Newer technology

The technologies previously discussed have existed and been the subject of research for many years. These will need to be considered more and exploited positively in any production setting devoid of routine antibiotic use. Additionally, there are newer technologies that have been developed that offer a producer tools that are directed specifically at disease-causing agents. Research from Canada is illustrated in Tables 8 and 9 that presents the possibilities from immunizing laying hens with specific disease causing agents and then using egg yolks from those immunized hens to deliver antibodies to young pigs. The results demonstrate again the benefits of plasma protein but also the benefit of the egg antibodies in reducing scour score and mortality associated with that scour bout.

Future technology

In addition to these technologies, there is growing interest in the ability of essential oils and other compounds to effect the microbiology of the gastrointestinal tract. Examples of the compounds that may have potential application specifically because they are active in vitro against bacteria, listeria, or certain fungi are provided in Table 10. In addition to the potential for essential oils, there is increasing interest in the value of herbs (reviewed by Wenk, 2002); some herbs may function as antioxidants while others may enhance palatability of the diet and thereby increase feed intake. Alteration in feed intake may not improve an animals ability to resist disease but it would affect growth performance. There is also potential for biologically active peptides to provide benefits (Power and Murphy, 1999). Peptides have been identified with that have antimicrobial activity, that are immunoactive or neuroactive, have antioxidant properties, or impart sensory or flavor properties to feeds.

Conclusion

In summation, antibiotics are valuable tools in the production of swine. If their use is changed with future legislation, there will be negative effects on swine health and well-being in the short term because current production systems are based on a system of inputs that includes antibiotics. But there are a variety of items that may improve normal swine physiology, development of physiology, or modify microflora that are at a nutritionists disposal as means to intervene and positively affect disease resistance and animal performance.

References cited

Bolduan, G. 1999. Feeding weaner pigs without in-feed antibiotics. In: T. P. Lyons and K. A. Jacques (Eds.) Proc. of Alltech's 15th Annual Feed Ingredient Symp. Pp. 223-230.

Hansen, C. F., K. E. B. Knudsen, B. B. Jensen. 2003. Effect of meal feed and xylanase on Salmonella, gastro-intestinal health and productivity of finishers. Proc. of 9th Internatl. Symp. on Digestive Phys. in Pigs, Banff, AB, Canada Vol. 2:308-310.

Kirchgessner, M., B. R. Paulicks and F. X. Roth. 1997. Effects of supplementation of diformate complexes on the growth and carcass performances of piglets and fattening pigs in response to application time. Agribiol. Res. 50:1.

Lis-Balchin, M. 2003. Feed additives as alternatives to antibiotic growth promoters: Botanicals. Proc. of 9th Internatl. Symp. on Digestive Phys. in Pigs, Banff, AB, Canada. Vol. 1:333-352.

Miguel, J. C., S. L.Rodriguez-Zas, J. E. Pettigrew. 2002. Practical response to Bio-Mos® in nusurey pigs: a meta-analysis. In: T. P. Lyons and K. A. Jacques (Eds.) Proc. of Alltech's 18th Annual Feed Ingredient Symp. Pp. Pp. 425-433.

Mikkelsen, L. L. and B. B. Jensen. 2003. The stomach as a barrier that reduces the occurrence of pathogenic bacteria in pigs. Proc. of 9th Internatl. Symp. on Digestive Phys. in Pigs, Banff, AB, Canada Vol. 2:66-71.

Mroz, Z. 2003. Organic acids of various origin and physico-chemical forms as potential alternatives to antibiotic growth promoters for pigs. Proc. of 9th Internatl. Symp. on Digestive Phys. in Pigs, Banff, AB, Canada. Vol. 1:267-293.

Owusu-Asiedu, A., Baidoo, S.K., Nyachoti, C.M., Marquardt, R.R. 2002. Response of early-weaned pigs to spray-dried porcine or animal plasma-based diets supplemented with egg-yolk antibodies against Eschericia coli. J. Anim. Sci. 80:2895-2903.

Owusu-Asiedu, A., Baidoo, S.K., Nyachoti, C.M., Marquardt, R.R. 2003. Response of early-weaned pigs to an enterotoxigenic Eschericia coli (K88) challenge when fed diets containing spray dried porcine plasma or pea protein isolate plus egg-yolk antibody. J. Anim. Sci. 81:1781-1789.

Partanen, K. H. 2001. Organic acids – their efficacy and modes of action in pigs. In: A. Piva, K.E. Bach Knudsen, and J.E. Lindberg, (Eds.) Gut Environment of Pigs. Pp. 201-218, University Press, Nottingham, UK.

Power, R. and R. Murphy. 1999. Biologically active peptides: sources, production and nutritional importance. In: T. P. Lyons and K. A. Jacques (Eds.) Proc. of Alltech's 19th Annual Feed Ingredient Symp. Pp. 435-447.

Simon, O., W. Vahjen, and L. Scharek. 2003. Micro-organisms as feed additives-probiotics. Proc. of 9th Internatl. Symp. on Digestive Phys. in Pigs, Banff, AB, Canada. Vol. 1:295-318.

Toralladona, D., M.R. Conde, I. Badiola, J. Polo, J. Brufau. 2003. Effect of fishmeal replacement with spray dried animal plasma and colistin on intestinal structure, intestinal microbiology and performance of weanling pigs challenged with Eschericia coli K99. J. Anim. Sci. 81: 1220-1226.

Wenk, C. 2000. Herbs, spices, and botanicals: 'Old fashioned' or the new feed additives for tomorrow's feed formulations? Concepts for their successful use. In: T. P. Lyons and K. A. Jacques (Eds.) Proc. of Alltech's 16th Annual Feed Ingredient Symp. Pp. 79-96.

Table 1. Performance of weaning pigs fed diets containing spray-dried animal plasma (SDAP) and/or colistin (0 or 300 g/kg).[a]

Item	0% SDAP		7% SDAP		SEM
	0	300	0	300	
Day 0-14 postweaning					
ADG, g/d [b,c]	101	214	193	230	20.7
ADFI, g/d [c,d]	227	298	268	286	10.8
Gain:Feed [b,c]	0.43	0.71	0.72	0.81	.069

[a] Adapted from Toralladona et al. (2003). Each mean represents three pens of four pigs; mean initial BW was 7.1 kg and mean initial age was 24d.
[b] SDAP effect, $P < 0.05$.
[c] Colistin effect, $P < 0.05$.
[d] SDAP x colistin interaction, $P < 0.05$.

Table 2. Relative performance (%) of piglets receiving acid supplements (piglets reared from 8-25 kg).[a]

Item	Acid Supplement:	Formic Acid	Sorbic Acid	Potassium Formate[b]
	Inclusion rate, %:	0.65	1.8	1.2/0.9[c]
Increase in body wt.		117	126	125
Feed conversion ratio		86	78	85
Frequency of diarrhea		36	23	39
Fraction of severe diarrhea		59	37	15

[a] From Kirchgessner et al. (1997) as presented by Bolduan (1999).
[b] Potassium diformate (Norsk Hydro, Norway).
[c] 1.2% in the week of weaning/0.9% during subsequent 5 weeks.

Table 3. A summary of studies with organic acids or salts for weanling pigs.[a]

	Formic Acid	Fumaric Acid	Citric Acid	Potassium diformate
Experiments:	6	18	9	3
Observations:	10	27	19	13
Acid levels, g/kg:	3-8	5-25	5-25	4-24
Dietary CP, g/kg:	234 +/- 22	208 +/- 27	216 +/- 21	222 +/- 5
Feed Intake, g/d				
Control	667	613	534	764
Experimental	710	614	528	823
P <[b]	0.01	0.42	0.14	0.001
Weight Gain, g/d				
Control	387	358	382	479
Experimental	428	374	396	536
P <[b]	0.001	0.01	0.01	0.02
Feed:Gain				
Control	1.64	1.59	1.67	1.60
Experimental	1.60	1.55	1.60	1.54
P <[b]	0.02	0.01	0.01	0.02

[a] Adapted from Mroz (2003) who analyzed the summary of Partenen (2001).
[b] Probability for a comparison with the non-acidified control diet.

Table 4. A summary of studies with organic acids or salts with grow-finish pigs.[a]

	Formic Acid	Propionic Acid	Fumaric Acid	Potassium Diformate
Experiments:	8	3	4	4
Observations:	15	6	10	8
Acid levels, g/kg:	5-16	5-25	6-25	6-20
Dietary CP, g/kg:	164 +/- 11	159 +/- 10	162 +/- 16	156 +/- 10
Weight gain, g/d				
Control	807	755	727	805
Experimental	857	784	753	842
P <[b]	0.001	0.001	0.01	0.001
Feed:gain				
Control	2.48	2.82	2.82	2.64
Experimental	2.36	2.71	2.74	2.59
P <[b]	0.001	0.01	0.01	0.01

[a] Adapted from Mroz (2003) who analyzed the summary of Partenen (2001).
[b] Probability for a comparison with the non-acidified control diet.

Table 5. Effect of feed processing on aspects of gastric physiology [a]

Response	Diet: [b] F-NP	C-NP	F-P	C-P	P =
pH	3.93	3.38	3.71	4.19	0.03
Organic acids, mM					
Acetic acid	2.7	10.7	0.0	4.5	0.02
Propionic acid	0.0	5.7	0.0	0.2	0.04
Butyric acid	0.0	1.6	0.0	0.2	0.12
Lactic acid	1.6	20.3	1.9	10.9	0.09
Anaerobic bacteria, log CFU/g	7.06	8.53	7.16	7.59	0.04

[a] Adapted from Mikkelson and Jensen, 2003.
[b] F-NP: fine, not pelleted; F-P: fine, pelleted; C-NP: coarse, non-pelleted; C-P: coarse, pelleted.

Table 6. A study examining effect of pelleting on finisher performance. [a,b]

Diet type:	Pellet feed		Mash feed		P=	
Response	Control	Xylanase	Control	Xylanase	Pellet vs Mash	Xyl
ADFI, kg	2.34	2.33	2.53	2.48	<0.05	0.24
ADG, g	882	901	828	839	<0.05	0.08
Feed:gain	2.66	2.58	3.06	2.96	<0.05	<0.05
Lean meat (%)	59.9	59.6	60.4	60	0.13	0.15
% Salmonella positive pigs[c]	30.6	24.1	17.6	13.0	<0.05	0.27

[a] Adapted from Hansen et al., 2003.
[b] Porzyme® 9300, Danisco Animal Nutrition, UK containing 4000U xylanase/g and added at 1 kg/ton of feed.
[c] Measured by the 'Danish MIX-ELISA test'. Pigs with an OD value >20 are categorized as Salmonella positive.

Table 7. Effects of probiotics from selected studies.[a]

Probiotic	Age or BW	Incidence of diarrhea	Stat. Sig.	Literature
B. cereus	8 weeks	Reduced	+	Kyriakis et al., '99
B. cereus	Day 1-85	Reduced	+	Iben u. Leibetseder, '89
B. cereus	Day 7-21	Reduced	+	Zani et al., '98
B. cereus	Day 24-66	No effect	-	Eidelsburger et al., '92
B. cereus	25 kg BW	No effect	-	Kirchgessner et al., '93
B. cereus	2 wks postwean	Reduced	+	Jadamus, '01
E. faecium	Day 1-70	Reduced	+	Manner u. Spieler, '97
E. faecium	8 days pre/post weaning	Reduced	+	Schumm et al., '90
P. acidilactici	Day 5-28	Reduced	+	Durst et al., '98
P. acidilactici S. cerevisiae	Day 5-28	Reduced	+	Durst et al., '98

[a] Adapted from Simon et al., 2003.

Table 8. Performance of 10-d-old weaned pigs when challenged with F18 enterotoxigenic E. coli (ETEC) and fed spray-dried animal (SDAP) or porcine (SDPP) plasma supplemented with egg yolk antibody (EYA) or egg yolk powder (EYP) in Exp. 2.[a]

	SDAP		SDPP			
Item	EYP	EYA	EYP	EYA	SEM	P=
Initial BW, kg	3.5	3.4	3.4	3.5		
ADG, g/d (n=18)						
D 1-7	74	84.1	91.4	97.1	5.4	0.04
D 1-14	146.1	175.3	176.1	180.1	12.5	0.22
ADFI, g/d (n=6)						
D 1-7	107.4	122.3	135.3	142.7	9.3	0.06
D 1-14	191.6	230.3	234.9	236.8	16.2	0.18
Gain/Feed, g/g (n=6)						
D 1-7	0.689	0.688	0.674	0.680	17.7	0.95
D 1-14	0.762	0.761	0.750	0.760	18.5	0.98

[a] Adapted from Owusu-Asiedu et al., 2002. Pigs were challenged on d 7 with 6 mL of 10^{10} cfu/mL ETEC (F18).

Table 9. Performance of piglets fed spray-dried porcine plasma (SDPP) of pea protein isolate (PPI) supplemented with egg-yolk antibody (EYA).[a]

Protein source:	PPI	PPI	SDPP	SDPP	PPI/ SDPP		
Egg-Yolk antibody:	-	+	-	+	-	SEM	P=
ADG, g (d 1-14)	84.5	123.4	127.4	129.9	132.3	11.2	0.04
G/F (d 1-14)	0.743	0.788	0.765	0.746	0.764	.018	0.80
Scours [b]	23/24	16/18	14/18	15/18	16/18	-	-
Scour score [b]	2.8	1.4	1.4	1.2	1.8	-	0.05
% Dead	33.3	11.1	0	5.5	11.1	-	0.01

[a] Adapted from Owusu-Asiedu et al., 2003.
[b] 24 and 48 h after E. coli challenge; pigs challenged on d 7 with K88 E. coli.

Table 10. Selected essential oil activity against a variety of disease agents [a]

	Antibacterial Activity		Antifungal Activity		
	Number affected of:				
Essential oil	25 Bacteria	25 Listeria	A. nig	A. och.	F. cul
Angelica Root	23	20	0	16	-18
Basil	15	20	94	76	71
Cedarwood, Atlas	2	0	0	0	0
Clove bud	23	20	95	94	73
Cinnamon Leaf	24	20	95	94	73
Fennel	6	0	95	78	66
Peppermint (20)	15-22	13-20	80-98	70-93	47-85
Rosemary	21	16	12	14	0
Sage, Dalmatian	16	6	0	53	33
Thyme	14-25	6-20	91-96	61-92	75-86

[a] Adapted from Lis-Balchin, 2003.

Practical Problems if Antibiotics are Banned

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Summary

Experiences in Europe and Scandinavia indicate that banning the use of antibiotics as growth promotants in livestock will result in; an increase in disease - particularly enteric in weaned and growing pigs, increased use of antibiotics for treatment and increased costs of production without any reduction in the prevalence of antibiotic-resistant infections in people.

Introduction

While lacking scientific support for their contention, various activist groups have taken the position that the use of antibiotics in livestock contributes to the development of resistance in bacteria that affect people. Their theory is that banning the use of growth promotants in food animals will reduce the problem of resistant infections in people. Lack of scientific validation did not prevent bans on AGP's from being imposed in Scandinavia and Europe. Limitations on the use of antibiotics, especially AGP's may be enacted in the U.S. in the near future. Fortunately, there is data on the effect on animals and resistant bacteria from these countries where AGP's were banned, and this information can be used to predict what would happen in the U.S. to swine productivity as well as both livestock and human resistance.

Effect on baseline growth rate and feed conversion

We have 50 or more years of data documenting the effect of AGP's on performance. AGP's have their greatest effect on the youngest pigs (Hays, 1981) and consequently this group will be affected the most, and older pigs to a lesser extent, if AGP's are banned. In the U.S. finishing herd we would expect to see an increase in sort loss.

This effect was clearly seen in Denmark when they instituted a two-stage withdrawal of AGP's. First they banned the use of AGP's in finishing pigs with some initial reduction in feed conversion efficiency and an increase in days to slaughter, however, no major impacts were obvious in the finishing stage. Later, they extended the ban to the nursery stage where disease and death loss increased dramatically.

The effects of eliminating AGP's will be greatest in swine housed in unsanitary conditions, overcrowded, inadequately nourished and managed in a continuous flow (Melliere et al, 1973).

Production in the U.S. is shifting towards greater use of specialized housing, and all-in, all-out management with cleaning between groups. Larger herd sizes make this possible. Smaller farms, unable to use all-in, all-out management and older facilities will be affected to the greatest extent if growth promotants are banned.

Figures from the Animal Health Institute (2002) show that antibiotic use is dropping. In 1999 the

Table 1 - Hays (1981) Summary of the Effect of Growth Promotants by Age of Pig.

Stage	Number of trials	Ave Daily Gain (g/day)			Feed Efficiency (feed/gain)		
		Control	AGP's	Improvement	Control	AGP's	Improvement
Starter 17-53 lb	378	.87	1.01	16.09 %	2.32	2.16	6.90 %
Grower 37-109 lb	276	1.31	1.45	10.68 %	2.91	2.78	4.47 %
Finisher 44-190 lb	279	1.51	1.57	3.97 %	3.37	3.30	2.08 %

Table 2 - Effect of Growth Promotants on Pigs Housed in Various Conditions. (Melliere et al, 1973)

Facility type	Number of trials	Ave Daily Gain (g/day)			Feed Efficiency (feed/gain)		
		Control	Tylan	Improvement	Control	Tylan	Improvement
Research	32	790	803	1.7 %	3.36	3.35	0.3 %
University	16	763	790	3.6 %	3.64	3.52	3.3 %
Field	24	713	754	5.7 %	3.84	3.66	4.7 %

industry used 24 million pounds of antibiotics compared to 23.7 M in 2000 and 21.8 M in 2001. This trend may reflect the growth of larger farms and the move to more customized facilities and management.

Task forces of U.S. researchers visited Sweden and Denmark to assess the effect that the ban on AGP's had on production costs. They estimated that compared to the effect in Sweden, that producers in the U.S. might expect a ban on AGP's initially to cost them $6.05/pig and $5.24/pig after 10 years (Hayes et al 1999). From a similar review of the situation in Denmark, investigators concluded that a ban might cost $4.50/pig. (Hayes et al 2003)

Effect on disease

Starting in 1986 several European and Scandinavian countries instituted bans against the use of antibiotic growth promotants (AGP's). Specific data on the effect on the health of swine has been described in various reports, but in general, after the ban on AGP's, observers noted: 1) increased disease among swine, especially weaned pigs, 2) greater amounts of antibiotics being used to treat clinical outbreaks, and 3) failure to reduce the prevalence of antibiotic-resistant infections in humans.

Sweden

After certain growth promoting antibiotics were banned in 1986, an increased incidence of diarrhea was seen in piglets, which rose dramatically after the ban and after 10 years still has not returned to pre-removal levels (Table 1 - Inborr 2000). Consequently more medicated feed was used for therapeutic purposes. In addition, there was a 1.5% increase in nursery pig mortality and an increase of 1 week in the time to reach 25 kg (Robertsson and Lundeheim, 1994).

Denmark

In 1998 Denmark banned the use of AGP's in the finishing stage as well as tightening the legislation on prescription of medicine for treatment of disease. In 2000 they also banned the use of AGP's in the nursery pigs. Pharmacies, veterinarians and feed mills are required to report all sales/use of medication for food animals on a monthly basis to VETSTAT (Jensen 2003)

Before the ban the prevalence of diarrhea in Danish herds was low, with only occasional post-weaning *E. coli* diarrhea, and while *Lawsonia intracellularis* was widespread, clinical disease was rare. After the ban in 1998 there was a dramatic increase in diarrhea which commonly was due to hemolytic *E. coli* or *L. intracellularis*, which precipitated an increase in the use of therapeutic antibiotics (Larsen, 2002) (Figures 2 and 3).

Holland

Increases in disease after removal of AGP's is reflected in the increase in use of therapeutic medications (Tijdshcrift voor diereneeskunde). Table 3 shows the increase in use of antibiotics for treatment of disease during years when animal numbers were decreasing.

United States

A few U. S. producers who are raising "antibiotic-free" pork for specialty markets report similar effects to those reported in Europe. For the majority of groups raised, feed efficiency and growth rates can be kept close to normal by absolute adherence to all-in, all-out, strict hygiene measures, provision of an optimal environment, and additional vaccinations. However, when disease does occur, outbreaks tend to be more severe and more difficult to treat. In addition, these pigs tend to have more abscesses at slaughter from the additional vaccines they received.

Table 3 - Use of Antibiotics for Therapy in Holland After a Ban on Growth Promotants.

Year	Tons of antibiotics used for therapy
1999	322.3
2000	351.3
2001	368.9

Effect on antimicrobial resistance

In livestock - Fifty years of using AGP's has not eliminated the therapeutic benefit of antibiotics in treatment of disease. It is not likely that antibiotics will become more effective in treatment of clinical outbreaks if subtherapeutic use is banned.

In people - SENTRY is a global network of healthcare facilities monitoring resistance levels in human bacteria, providing the world's largest database of antibiotic resistance. "Vancomycin resistant enterococci (VRE) and Synercid resistant *E. faecium* are becoming more prevalent as a cause of infections in humans in Europe at a time when these resistant organisms are becoming less prevalent in animals and food products following the antibiotic growth promoter ban. (Dr. Ronald N. Jones, Principal Investigator, SENTRY.)

Other experts have stated that banning antibiotics as growth promoters in animals will not solve or even impact the problem of antibiotic resistance in hospitals. (Acar et al 2000).

If antibiotics are banned for growth promotion on the basis of the information we now have at hand, we will have ignored the scientific process in the interest of appeasing activists. Many activist groups who promote against a certain issue actually have a greater interest in a hidden issue. If a ban on AGP's is enacted in the absence of convincing scientific data it may be an indication that other subsequent regulations regarding farming will be made in an arbitrary manner.

References cited

Citations are to follow the same format as in the Journal of Animal Science.

Ron Jones, MD. PI SENTRY

Acar J, Casewell M, Freeman J, Goossens H, Friis C. Avoparcin and virginiamycin as animal growth promoters: A plea for science in decision-making. Clinical Microbiology and Infections 2000; 6(9);477-482.

Hayes D, Jensen H and Backstrom L 2003 Analysis of a more restricted antimicrobial access policy in pork production, Proc AASV p 433-438.

Hayes DJ, Jensen HH, Backstrom L and Fabiosa J 1998. Economic impact of a ban on the use of over-the-counter antibiotics in U.S. swine rations. CARD Staff Report 99-SR90. Iowa State Univ, Dec 1999. Final report to National Pork Producers Council Project No 1998-235.

Hays V. The Hays Report

Jensen HM. Life After the Ban - Experiences of a Danish swine veterinarian. Proc AASV 2003, p 427-431

Conclusions

Changes in Swine Production

If AGP's are banned in the U.S. we can anticipate more disease, more vaccination, adoption of older weaning ages, the need for meticulous management of weaned pigs, the need for repalcement of older facilities, additional consolidation of the industry and greater use of alternatives such as copper and zinc.

Societal Implications

According to Jones (2002), the Principal Investigator of SENTRY, which provides the world's largest database of antibiotic effectiveness, antibiotic resistance in humans is growing around the world, but a global collection of human and animal resistant strains shows little significant association between animal and human patterns.

Larsen PB. Consequences of termination of AGP use for pig health and usage of antimicrobials for therapy and prophylaxis. Proc Int Invitational Symp " Beyond Antimicrobial Growth Promoptors in Food Animal Production" Foulum, Denmark, Nov 2002.

Melliere AL, Brown H and Rathmacher RP. 1973. Finishing swine performance and responses to tylosin. J Anim Sci 37:286

Robertsson JA and Lundeheim N, 1994 Prohibited Use of Antibiotics as a feed additive for growth promotion - Effects on Piglet health amd production parameters. In Proc IPVS Bankok, Thailand, p 282

Wierup M. 2001. The Swedish experience of the 1986 year ban of antimicrobial growth promoters, with special reference to animal health, disease prevention, productivity, and usage of antimicrobials. Microb. Drug Resistance 7(2):183-190.

Figure 1 - Incidence of Diarrhea in Swedish Pigs After the Ban on Growth Promotants in 1985 (Inborr, 2000)

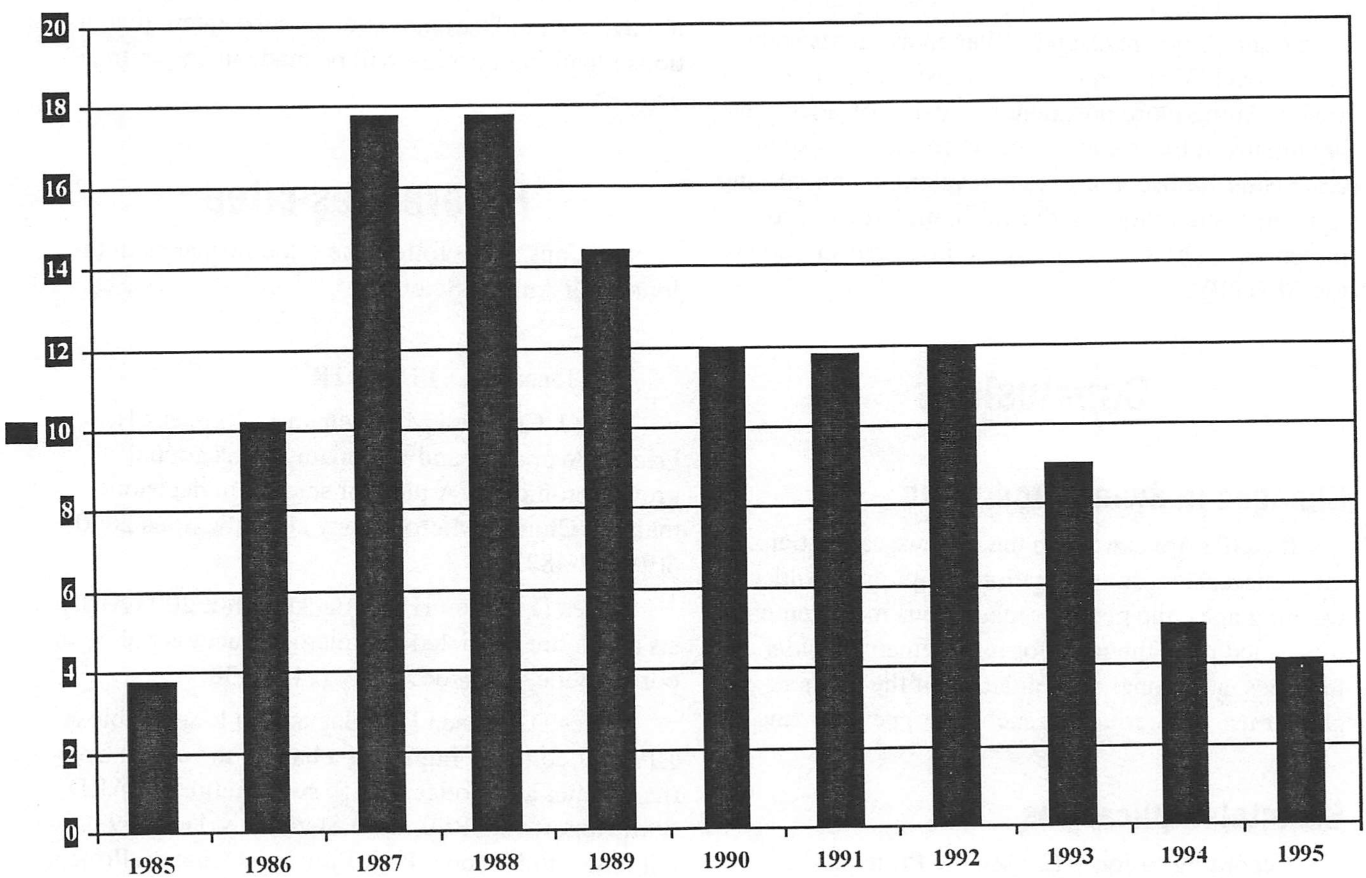
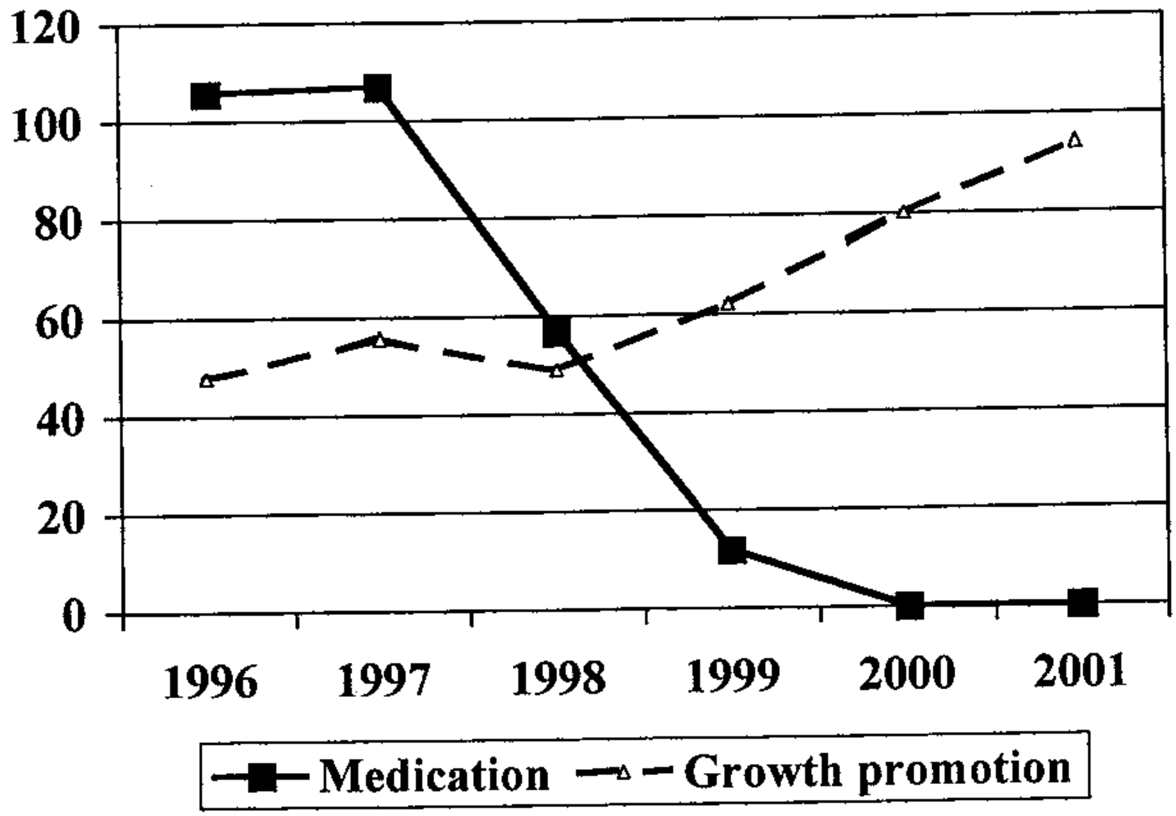


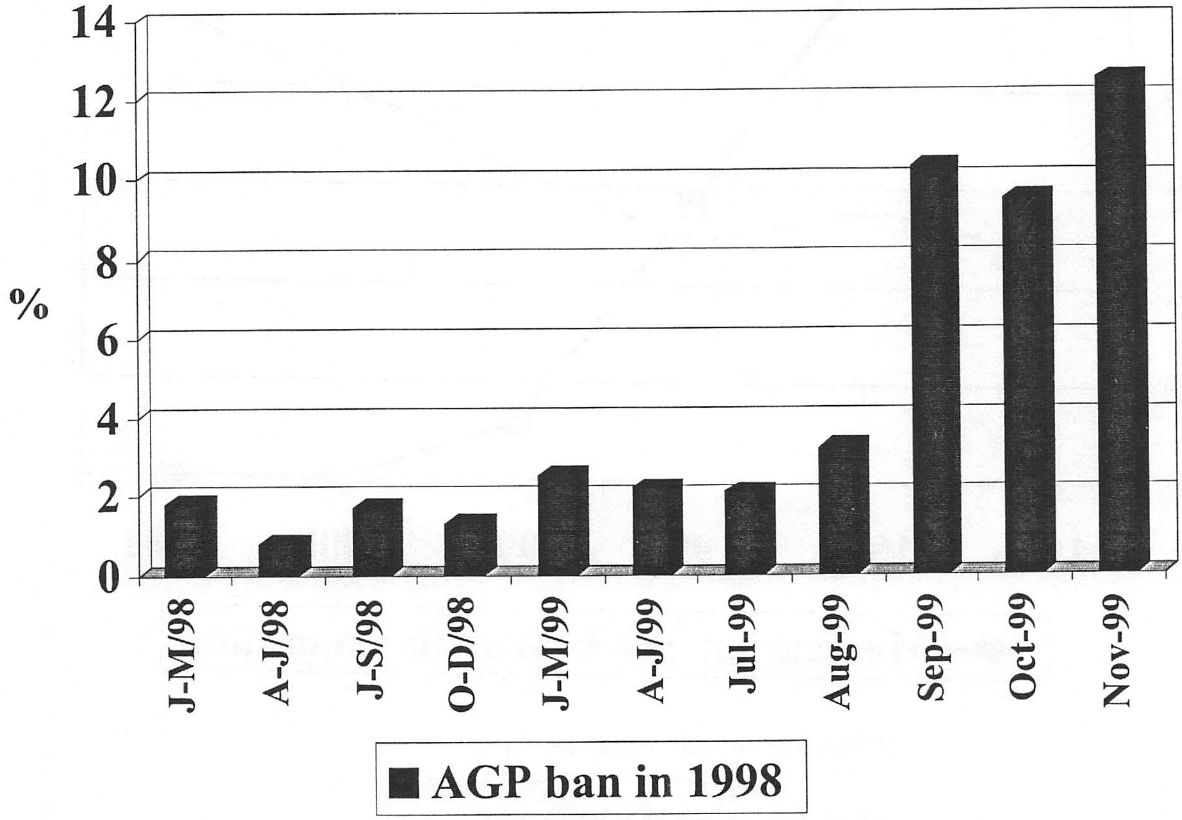
Figure 2 - Use of Antibiotics in Denmark (Danmap 1999 and Danmap 2000)



1998 - ban on use in finishing pigs

2000 - ban on use in nursery pigs

Figure 3 - Ileitis as a Percent of the Diagnoses Made at the Diagnostic Laboratory (DS Laboratory - Kjellerup)



Feeding Breeding and Gestating Sows in a Group-Housing Environment

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Summary

Societal pressures are slowly changing the method by which gestating sows can be housed in the United States. If the current pressure continues, gestating sows will have to be housed in a group environment. However, group-housing systems are not without their own welfare problems, such as, aggressive behavior between sows, health risks of low dominance sows, possible reduction in reproductive performance, occurrence of stereotypic behaviors, and inadequate feed intake per sow. The take home message from the research trials reviewed is: *A feeding system has to be utilized whereby each sow within the group can "peacefully" consume an appropriate amount of feed.* To meet this requirement when group-housing gestating sows, a system has to be designed whereby the sows are fed a specific amount of feed in an individual stall that can be locked either by a computer (Electronic Sow Feeder), by hand, or self-locking. Aggression during the time of eating can still occur when other types of feeding methods are used, such as, non-locking individual feeding stalls, Biofix feeding system, and floor feeding. Scientists and pork producers need to continually search for improved methods and diets to feed group-housed gestating sows that will meet future welfare requirements.

Introduction

The technology of housing gestating sows individually in stalls has received a substantial amount of criticism from animal welfare, animal rights, and vegetarian groups during the last several years, especially in Europe, United States and Canada. The BanCruelFarms organization has placed an article on the worldwide web that clearly indicates their position on housing sows in gestation stalls (www.bancruelfarms.org/evidence.htm). Their interpretation of 28 scientific publications reviewed lead them to the following conclusion:

The scientific evidence is clear: gestation stalls cause physical and mental suffering to sows. Stall-housed sows cannot exercise and are deprived of the basic necessity of living space. As a result, they are weak, suffer leg and joint problems, and experience difficulty carrying out simple movements. The barren sow stall does not meet the sow's social and cognitive needs and fails to allow for behaviors important to her species' way of life, and makes the animal depressed and frustrated to the point that she must perform repetitive actions in a pitiful appeal for mental stimulation. In scientific studies, sows have let us know themselves that they prefer environments that offer more space and complexity.

As indicated in Appendix I, legislation in Florida has banned the use of gestation crates.

The legislation to ban the use of gestation crates in Florida has provided "precedence" for activist groups to pursue their activity to ban gestation crates in other states. In California, the California Assembly Public Safety Committee has approved a bill (AB 732) to outlaw confinement stalls for pigs. In Rhode Island, the House of Representatives' Health, Education and Welfare Committee have received comments from various groups concerning a bill to ban gestation crates of pigs. A bill to ban gestation crates is under construction in Texas. There has also been activity to ban gestation crates in Oregon. On the positive side for pork producers who use gestation crates, Maryland voters defeated a bill to ban the use of gestation crates.

Effect of Feed Intake

If pork producers are mandated to house gestating sows in groups, what strategies will be used for feeding gestating sow? Will the feeding strategies of group-housed sows be welfare "friendly" and fulfills the nutritional requirements of each individual sow and gilt? Breeding females are traditionally fed to maintain a relatively constant body condition to enhance longevity, productivity and health (Dourmad et al., 1994). Thus, feed intake during gestation is restricted to prevent excessive body weight gain and fat deposition. It is known that excessive feed intake during early gestation increases embryonic death in gilts (Liao and Veum, 1994; Jindal et al., 1996, 1997; Cassar et al., 1994) but not in multiparous sows (Dyck and Cole, 1986; Toplis and Ginesi, 1983). Excessive feed intake during gestation deceases feed intake during lactation (Weldon et al., 1994; Xue et al., 1997). Excessive underfeeding of gestating sows can reduce piglet birth weight, piglet viability, and lower body fat reserves at farrowing and weaning. Research has indicated that food deprivation for 48 hours after ovulation is associated with: (a) changes in reproductive hormones (progesterone, estradiol 17β, and 15-keto-13,14-dihydro-$PGF_{2\alpha}$), (b) changes in metabolic hormones (insulin, triglycerides and free fatty acids), (c) a decrease in number of sperm transported to the sperm reservoir of the oviduct, (d) a lower cleavage rate of embryos, and (e) a delayed transport of ova (Mburu et al., 1998; Mwanza et al, 2000a,b). In addition, fasting of sows on days 10 and 11 after mating produced an elevation in maternal plasma cortisol, progesterone, prostaglandin metabolite levels and a concomitant fall in estradiol 17β levels (Tsuma et al., 1996). Therefore, excessive underfeeding or overfeeding of sows has detrimental affects on their reproductive performance.

Although pork producers have known that floor-feeding of gestating sows as a group allows aggressive activities during eating, scientists have documented that the highest incidence of aggression occurs during the first 30 minutes after delivery of the feed (Csermely and Wood-Gush, 1986). As expected, dominant sows "defend" the center of the pile of feed that contains a good supply of the feed (Csermely and Wood-Gush, 1990). Subordinate sows take the strategy of quickly grabbing food at the edges and moving only when forced to do so. Scientists have not determined what proportion of the feed dropped are consumed by dominant, intermediate and subordinate sows. Unequal feed intake between sows within the group has detrimental effects on body reserves, especially for the low-ranking sows (Bourns and Edwards, 1994; Edwards, 1993; Signornet et al., 1995). Body weight gain is significantly lower for low ranking sows when floor fed (Table 1).

Aggression over food is not totally eliminated by providing piles of feed at several locations within the feeding area of an indoor housing system. Because the total amount of space is limited with an indoor housing system, there is most likely a correlation between the level of aggression and floor space available per animal for feeding.

Feeding Methods.

The take home message from the research trials reviewed is: *A feeding system has to be utilized whereby each sow within the group can "peacefully" consume an appropriate amount of feed.* An excellent feeding system for group-housed sows would be one that allows all individuals to have simultaneous access to feed and allows each individual to completely consume their "specified" ration without being displaced by other aggressive sows. If the traditional gestation stall cannot be used, are there viable and economical options for individually feeding sows when housed as a group?

Option 1

Computerized Individual Feeding System. The computerized feeding system allows sows to be loose-housed in groups but fed individually. However, the sows must take turns to eat, as the group shares one or more computer-controlled feeding stations. Each sow carries a unique electronic identifier, in the form of a collar or ear tag. When the sow enters an enclosed station, she is identified and any feed due to her is measured out into the trough. The computer can also be used to alter the feed ration for each individual sow (Edwards and Riley, 1986). Also, the computer can be adjusted to give each sow her entire meal in one single visit or several smaller meals throughout the day (Eddison and Roberts, 1995). The suggested number of animals per electronic feeder is 40 to 50 sows (Edwards and Riley, 1986).

Aggressive physical acts do occur while sows are waiting for their turn to enter the feeder. A major concern has been the high incidence of vulva biting (Van Putten and Van De Burgwal, 1990). The United Kingdom has provided information on how to design and manage an electronic sow feeding system (Ministry of Agriculture, Fisheries and Food, 1997). There are essentially two management schemes. Option A is a static group of 35 to 40 sows per pen with only one electronic feeding station. All the animals of the group are in the same productive phase. Option B is a dynamic group of about 80 to 200 sows with two to five electronic feeding stations (Edwards, 1998).

Every week sows enter and leave the group; thus, the animals are in different productive phases. CAUTION: The introduction of bred sows to an existing group of sows eating from an ESF at one to eight days after mating has increased the incidence of bred sows returning to estrus (20% vs 10%) and reduced litter size (10.5 vs 10.7 piglets per litter) compared to introducing bred sows at 22 to 29 days after mating (Bokma, 1990). Although data presented by Simmins (1993) has to be interpreted with caution due to a difference in parity distribution between treatments, sows housed in a stable group (group established within 7 days of mating) had a lower farrowing rate (78 vs 85%) but a greater ($P < .05$) average number of total piglets born live per litter (16.9 vs 14.6) compared to sows housed in a dynamic group (removal and adding of 2 to 4 sows per week).

Although the use of an ESF system helps ensure that sows receive the correct allowance of feed, sows with low social rank have lower ($P < .001$) bodyweights, higher ($P < .01$) injury levels, lower ($P < .001$) position in the feeding order, and displaced more often ($P < .01$) from the drinkers than high-ranking sows (O'Connell et al., 2003).

Option 2

Biofix Feeding System. Another method to possibly limit aggression and feed intake by dominant sows is the trickle feeding or "Biofix" (biological fixation) system (Figure 1). Sows are usually kept in small stable groups and shoulder length barriers separate the feeding trough. An auger apparatus slowly delivers food (140 to 180 grams per minute) over a period of approximately 15 to 30 minutes. In the ideal system, there is no incentive for sows to move away from the feeder to bully other sows (Svendsen and Bengstsson, 1983). The slow rhythm of feed distribution encourages the sows to remain at the feed space for the duration of the feeding period. In other words, the sows are "biologically fixed" to the feed space. Because each sow can randomly enter any feeding space, individualized rationing is not possible with the Biofix feeding system.

Option 3

Self-Locking or Manual Locking Individual Feeding Stall. Many producers have developed a combination of group sow housing and individual feeding stalls (Figure 2). In this system, sows are generally free to roam around in a large pen with other sows except when they are being fed. The surface of the lying area has been total slats, partial slats, or solid concrete with or without bedding. The sows must enter body length individual feeding stations (one feeding stall per sow), where they are fed on the floor or in a trough that continues in front of all the stalls. The body length feeding stalls are also used as a resting area. If feeding stalls are not lockable and all the feed is dropped at one time, vulvae biting occurs because faster eating sows are trying to steal feed from slower eating sows. Because each sow can randomly enter any of the feeding stalls, individualized rationing is not possible with this feeding system unless each sow is fed by hand.

Option 4

Non-locking Individual Feeding Stall. Researchers have investigated the influence of the length of feeding stall partition (body, shoulder, or none) and type of food (wet or dry) on the amount of aggression, frequency of changing position at the trough and the time at trough in groups of pregnant sows (Table 2; Andersen et al., 1999). When sows were provided dry feed, it was reported that increasing the length of partitions resulted in a significant reduction in the number of bites, total aggressive behaviors and displacement at the trough; plus, the time at the trough increased (Figure 3 and 4). When sows were provided wet feed, there were no significant differences between body and shoulder partitions concerning the number of bites (Figure 3) or time feeding at the trough (Figure 4). Top ranked sows received significantly less bites toward head/shoulder and body ($P < .001$) and were less frequently displaced ($P < .05$) at the trough than sows with a lower rank when eating from trough with no partition or shoulder partition. Vulva bites were significantly greater when sows consumed feed (wet or dry) from a feeding stall with a body partition compared to a shoulder feeding stall or a stall with no partitions (Table 2). Vulva biting in breeding sows is an important welfare issue in group-housed sows (Rizvi et al., 2000). Individualized rationing is not possible with this feeding system.

Stereotypies

The Commission of the European Communities (1983) has defined stereotypies as: behavior patterns that are performed repetitively, in fixed order and without obvious function. The occurrence of stereotypies (vacuum chewing, bar-biting, chain-chewing, and excessive drinking) has been suggested as an indicator of the welfare status of gestating sows housed in individual stalls. Stereotypies have been seen in group-housed sows to the same extent as in individual gestation stalls (Den Hartog et al., 1993). Because a low feeding level has been linked to the occurrence of stereotypic activities, all the options previously presented are at risk of "attack" by animal activists groups. One option to "help" satisfy feeding motivation while maintaining sows on restricted energy supply is to provide diets with additional roughage. Meunier-Salaun et al. (2001) recently reviewed the scientific literature on feeding dietary fiber during gestation on the behavior and health of restricted fed sows. Incorporation of fiber in diets to increase bulk has been shown to result in at least a doubling of eating time, a 20% reduction in feeding rate, a 30% reduction in operant response to feeding motivation tests, a reduction of 7 to 50% in stereotypic behavior, and a decease in general restlessness and aggression.

Implications

The pork industry is still facing challenging aspects of how to best feed gestating sows in a group-housing environment to meet animal welfare requirements.

References

Table 1. Effect of dominance on average live weight gain during gestation when sows are floor-fed (12 sows per pen; 3.1 m^2 [32.7 ft^2] per sow)

Item	Pen 1	Pen 2
High ranking sows	46.3 kg (8 sows)	44.9 kg (9 sows)
Low ranking sows	28.3 kg (4 sows)	22.4 kg (2 sows)
SED	7.19	8.70
P value	.03	.03

Bourns and Edwards, 1994.

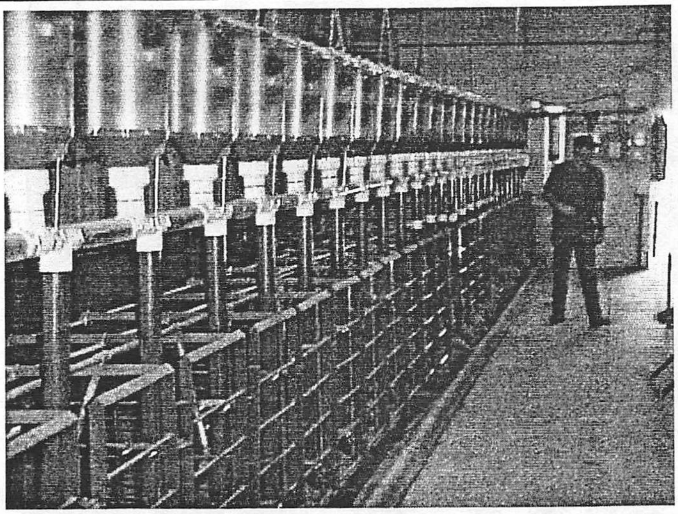
Table 2. Frequency of behavior (mean per group) delivered in different feeding arrangements and type of food

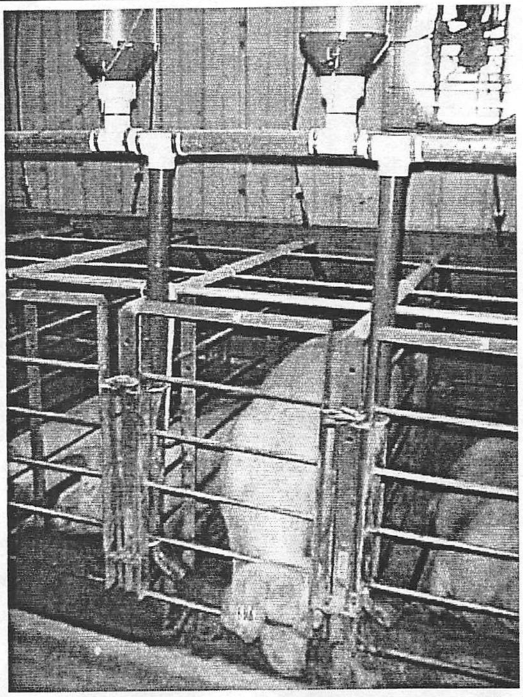
		Length of partition [a]			Food		P <		
Behavior	Activity	Body	Shoulder	None	Dry	Wet	Feeding method	Food	F x FM
Bite towards head/shoulder /body	Feeding	.70	1.94	3.44	2.44	1.62	.001	.01	.05
	After feeding	.21	.30	.42	.40	.22	.05	.01	ns
Bite towards Vulva	Feeding	.67	.13	.19	.40	.25	.001	ns	ns
	After feeding	.07	.02	.01	.04	.03	.05	ns	ns
Total aggressive behaviors	Feeding	2.99	4.41	4.63	4.50	3.52	.01	.05	.01
	After feeding	.47	.55	.55	.67	.38	ns	.01	ns
Displacement	Feeding	.44	1.15	1.83	1.14	1.14	.001	ns	ns
	After feeding	.04	.08	.03	.07	.03	ns	.07	.06
Leave feed trough voluntarily	Feeding	2.81	5.53	3.40	3.46	4.37	.001	.001	.001
	After feeding	1.63	2.63	1.18	1.85	1.78	.001	ns	.06

[a] Body partition (.5 m wide x 2 m long [19.5 in. x 78 in.]); Shoulder partition (.5 m wide x .40 m long [19.5 in. x 15.6 in]); None (no partition on feed trough).

Andersen et al., 1999.

Figure 1. Biofix feeding system





Feed quantity can be controlled by the hopper. At feeding time (controlled by a timer), the feed is either dropped (no second auger) or it enters the second auger that delivers the feed according to a fixed time schedule. The feed is trickled slowly so that the slowest sow can consume all of her feed and thus it prevents other sows from stealing their feed.

Pictures are from Texas Tech University, Lubbock, Texas.
www.depts.ttu.edu/porkindustryinstitute/SowHousing_files/Trickle%20Feeding%20pictures.htm

Figure 2. Self-locking individual feeding stalls.

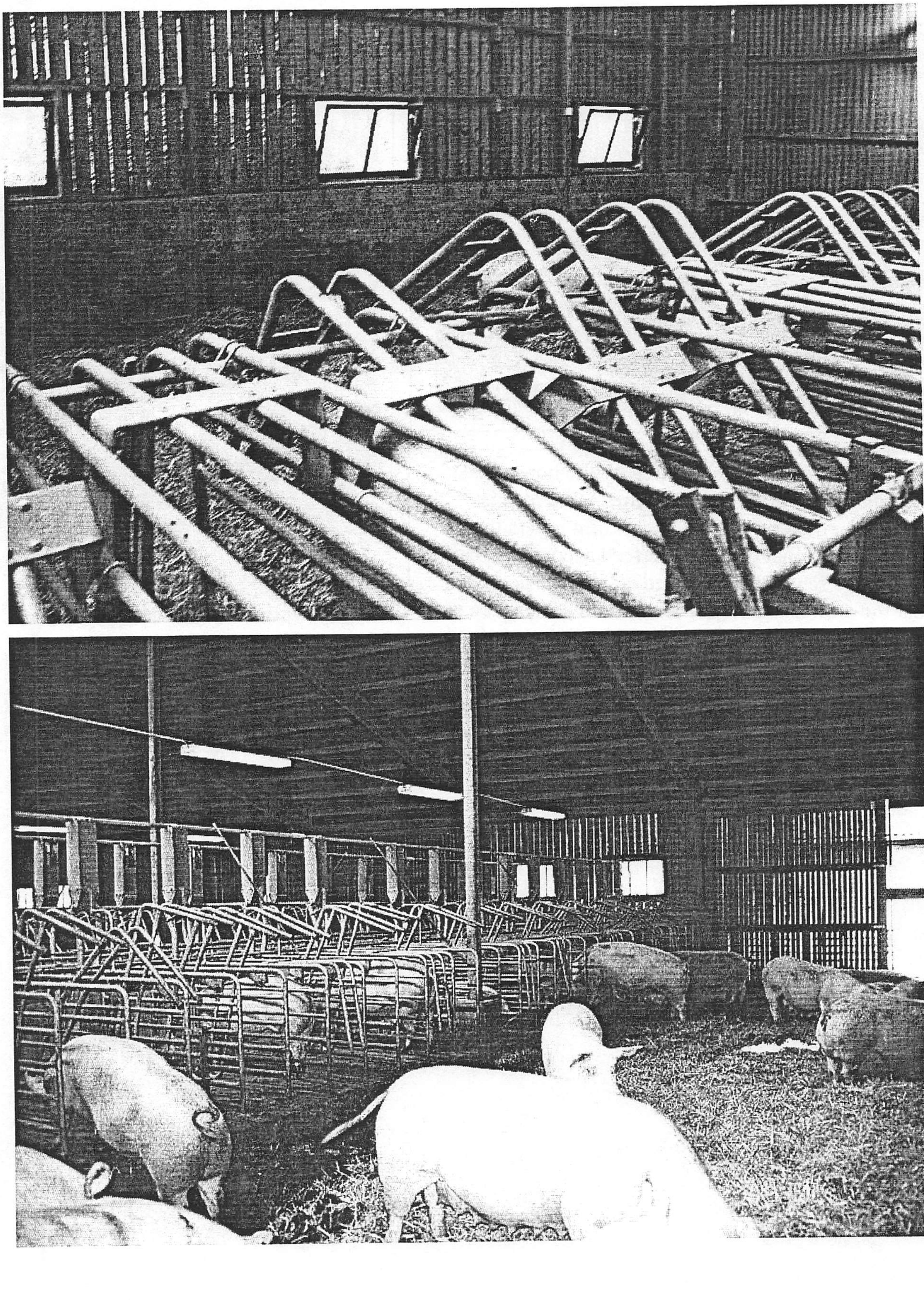
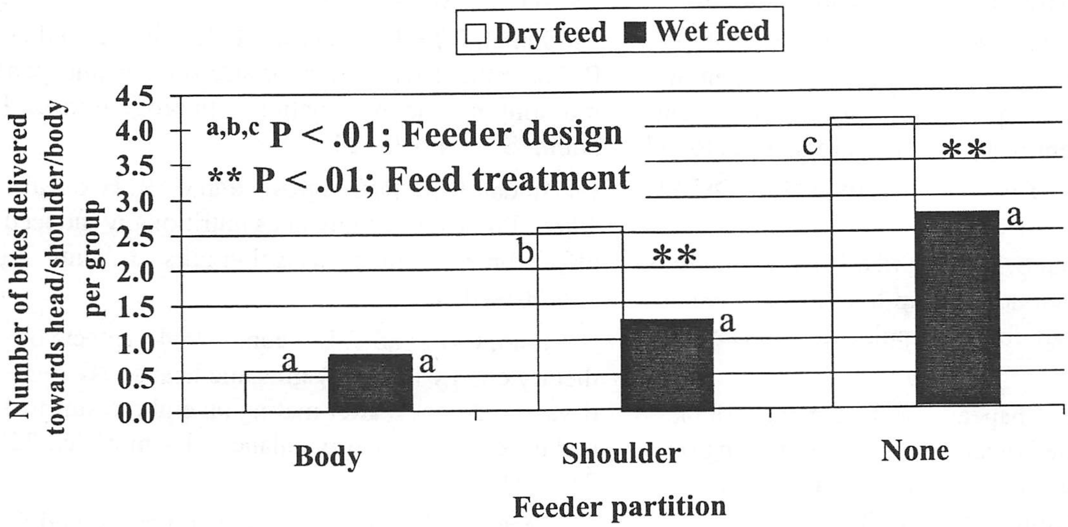
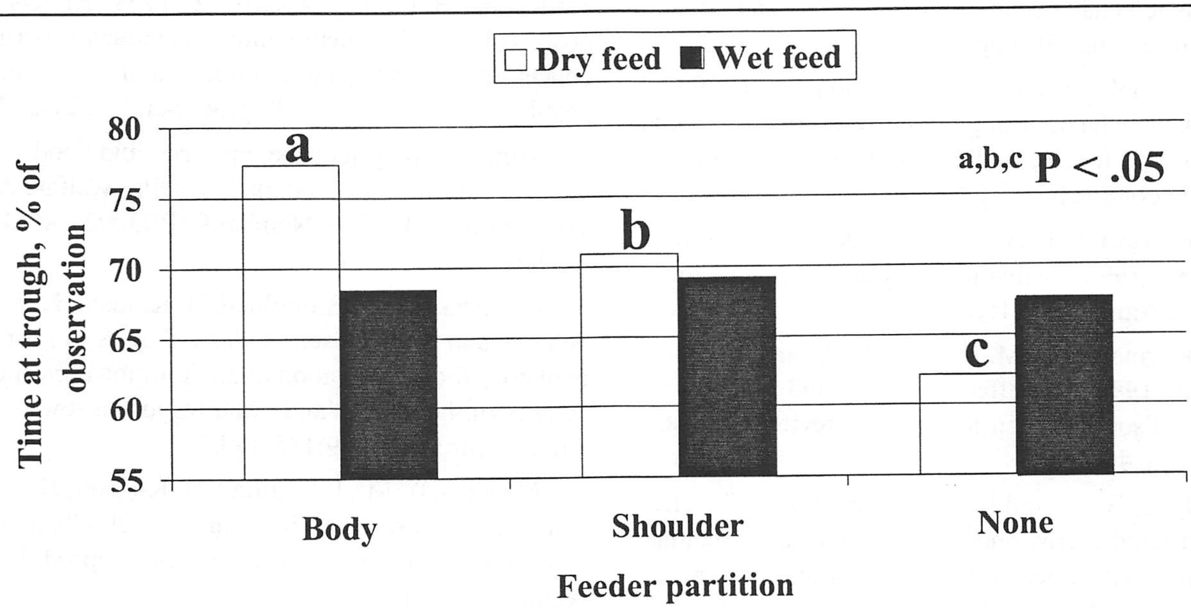


Figure 3. Effect of feeding arrangement and type of feed on number of bites delivered during first 15 minutes after dropping feed.



Appl. Anim. Behav. Sci. 65:91-104, 1999.

Figure 4. Effect of feeding arrangement and type of feed on time spent at trough during first 15 minutes after dropping feed.



Appl. Anim. Behav. Sci. 65:91-104, 1999.

Andersen, I. L., K. E. Boe, and A. L. Kristiansen. 1999. The influence of different feeding arrangements and food type on competition at feeding in pregnant sows. Appl. Anim. Behav. Sci. 65:91-104.

Bokma, Sj. 1990. Housing and management of dry sows in groups in practice: partly slatted systems. In: Electronic Identification in Pig Production. Royal Agricultural Society of England. Monograph Series No. 10, pp 37-46.

Bourns, F., and S. A. Edwards. 1994. Social rank and feeding behaviour of group-housed sows fed competitively or ad libitum. Appl. Anim. Behav. Sci. 39:225-235.

Cassar, G., C. Chapeau, and G. J. King. 1994. Effects of increased dietary energy after mating on developmental uniformity and survival of porcine conceptuses. J. Anim. Sci. 72:1320-1324.

Commission of the European Community. 1983. Abnormal Behaviours in Farm Animals. Brussels, 16 pages.

Csermely, D., and D. G. M. Wood-Gush. 1986. Agonistic behaviour in grouped sows. I. The influence of feeding. Biology of Behav. 11:244-252.

Csermely, D., and D. G. M. Wood-Gush. 1990. Agonistic behaviour in grouped sows. II. How social rank affects feeding and drinking behavior. Bollettino di zoologia 57:55-58.

Den Hartog, L. A., G. B. C. Backus, and H. M. Vermeer. 1993. Evaluation of housing systems for sows. J. Anim. Sci. 71:1339-1344.

Dourmad, J. Y., M. Etienne, A. Prunier, and J. Noblet. 1994. The effect of energy and protein intake of sows on their longevity: a review. Livest. Prod. Sci. 40:87-97.

Dyck, G. W., and D. J. A. Cole. 1986. The effect of restricted energy and nutrient intake after mating on reproductive performance of multiparous sows. Anim. Prod. 42:127-132.

Eddison, J.C. and N.E. Roberts 1995. Variability in feeding behaviour of group-housed sows using electronic feeders. Anim. Sci. 60:307-314.

Edwards, S. A.. 1993. The effect of feeding system on the longevity of sows. Proc. 44th Annual Meeting European Assoc. Anim. Prod. Aarhus, Denmark. P 34.

Edwards, S. A. 1998. Housing the breeding sow. In Practice 20:339-343.

Edwards, S.A. and J.E. Riley 1986. The application of the electronic identification and computerized feed dispensing system in dry sow housing. Pig News Info. 7(3):295-298.

Jindal, R., J. R. Cosgrove, F. X. Aherne, and G. R. Foxcroft. 1996. Effect of nutrition on embryonal mortality in gilts: Association with progesterone. J. Anim. Sci. 74:620-624.

Jindal, R., J. R. Cosgrove, and G. R. Foxcroft. 1997. Progesterone mediates nutritionally induced effects on embryonic survival in gilts. J. Anim. Sci. 75:1063-1070.

Liao, C-W, and T. L. Veum. 1994. Effects of dietary energy intake by gilts and heat stress from days 3 to 24 or 30 after mating on embryo survival and nitrogen and energy balance. J. Anim. Sci. 72: 2369-2377.

Meunier-Salaun, M. C., S. A. Edwards, and S. Robert. 2001. Effect of dietary fibre on the behaviour and health of the restricted fed sow. Anim. Feed Sci. and Tech. 90:53-69.

Mburu, J. N., S. Einarrsson, H. Kindahl, A. Madej, and H. Rodrigez-Martinez. 1998. Effects of post-ovulatory food deprivation on oviductal sperm concentration, embryo development and hormonal profiles in the pig. Anim. Reprod. Sci. 52:221-234.

Ministry of Agriculture, Fisheries and Food. 1997. Electronic sow feeders (ESF). Pig Welfare Advisory Group. Booklet Number 9 (PB3092). MAFF Publications, London.

Mwanza, A. M., P. Englund, H, Kindahl, N. Lundeheim, and S. Einarsson. 2000a. Effects of post-ovulatory food deprivation on the hormonal profiles, activity of the oviduct and ova transport in sows. Anim. Reprod. Sci. 59:185-199.

Mwanza, A. M., P. Englund, H, Kindahl, H. Rodrigez-Martinez, and S. Einarsson. 2000b. Effect of stressor on ova transport in the pig. Reprod. Dom. Anim. 35:188-189.

O'Connell, N. E., V. E. Beattie, and B. W. Moss. 2003. Influence of social status on the welfare of sows in static and dynamic groups. Anim. Welfare 12:239-249.

Rizvi, S., C. J. Nicol, and L. E. Green. 2000. A descriptive survey of the range of injuries sustained and farmer's attitudes to vulva biting in breeding sows in south-west England. Anim. Welfare 9:273-280.

Signornet, J. P., Y. Ramonet, and C. Yieuille-

Thomas. 1995. L'elevage en plein-air des truies gestantes: problemes poses par les relations sociales. J. Rech. Procine en France 27:11-18.

Simmins, P. H. 1993. Reproductive performance of sows entering stable and dynamic groups after mating. Anim. Prod. 57:293-298.

Svendsen, J., and A.C. Bengtsson 1983. Housing of sows in gestation. Paper presented at the Guelph Pork Symposium. pp 118-131. Guelph, Ontario, Canada.

Toplis, P., and M. F. J. Ginesi. 1983. The influence of high food levels in early pregnancy on embryo survival in multiparous sows. Anim. Prod. 37:45-48.

Tsuma, V. T., S. Einarsson, A. Madej, H. Kindahl, and N. Lundeheim. 1996. Effect of food deprivation during early pregnancy on endocrine changes in primiparous sows. Anim. Reprod. Sci. 41:267-278.

Van Putten, G., and J. A. Van De Burgwal. 1990. Vulva biting in group-housed sows: preliminary report. Appl. Anim. Behav. Sci. 26:181-186.

Weldon, W. C., A. J. Lewis, G. F. Louis, J. L. Kovar, M. A. Giesemann, and P. S. Miller. 1994. Postpartum hypophagia in primiparous sows: I. Effects of gestation feeding level on feed intake, feeding behaviour, and plasma metabolite concentrations during lactation. J. Anim. Sci. 72_387-394.

Xue, J-L, Y. Koktsu, G. D. Dial, J. Pettigrew, and A. Sower. 1997. Glucose tolerance, luteinizing hormone release, and reproductive performance of first-litter sows fed two levels of energy during gestation. J. Anim. Sci. 75:1845-1852.

Appendix I. Animal rights groups applaud Florida hog crate ban.

Reuters, 11.06.02, 3:37 PM ET By Bob Burgdorfer CHICAGO (Reuters) - Animal welfare groups, bolstered by a Florida vote that bans the popular swine industry practice of keeping pregnant pigs in small pens, said Wednesday they hope other states will follow to make hog farming more humane. Florida voters Tuesday approved a state constitutional amendment to prohibit commercial hog farmers from housing pregnant sows in cages that are too small to turn around in. The proposal, placed on the ballot by animal rights activists, passed 55percent to 45 percent. The measure will have only limited effect in Florida. Among the state's10 commercial hog farms, only two use the small stalls known as "gestation crates" to house pregnant pigs, and one of those is going out of business in December, the Miami Herald reported last week. Hog farmers say the stalls are necessary because sows are aggressive and will attack each other to get more food if housed together. Housed in separate cages, they receive individualized nutrition and cannot hurt each other, farmers have said. Animal welfare groups reject those arguments. "It is a national movement to prevent this type of cruelty on factory farms," said Wayne Pacelle, senior vice president of the Humane Society of the United States. "We won't be satisfied until all states ban the keeping of pigs in2-foot by 7-foot crates for the bulk of their lives," Pacelle said. Another group, People for the Ethical Treatment of Animals, also cheered the Florida vote and said use of the crates is one of several practices in the livestock industry that harm animals. "If a dog or cat were treated similarly, people could go to jail," said Bruce Friedrich, a PETA spokesman. Pacelle said the Florida amendment should boost efforts to ban the practice in top hog-producing states such as Iowa, North Carolina, Nebraska and Illinois, either with similar petition-based ballots or by working through state legislatures. "It certainly gives momentum to any effort that would be advanced to ban gestation crates in other states," Pacelle said. About 64 percent of the nation's roughly 80,000 hog farms use the gestation crates, pork industry officials said. "We've have both types of systems," said Missouri hog producer Kathy Chinn, who is also chairman of the National Pork Board's animal welfare committee. "We have moved progressively toward the stalled system as opposed to the group housing because we find it much better to care for our sows." The board is a farmer-funded industry group that works to promote pork and develop new pork products. In September, more than 900,000 sows on U.S. farms "farrowed" or gave birth to a litter of pigs, according to the latest monthly U.S. Agriculture Department data. Each litter usually consists of seven to nine piglets. Top corporate hog farmers include Smithfield Foods, Premium Standard Farms and Seaboard Corp. Last year, Virginia-based Smithfield owned 710,000 sows, Missouri-based Premium Standard 211,000 sows, and Kansas-based Seaboard 185,000 sows, according to industry statistics.

The scientific evidence is clear: gestation stalls cause physical and mental suffering to sows. Stall-housed sows cannot exercise and are deprived of the basic necessity of living space. As a result, they are weak, suffer leg and joint problems, and experience difficulty carrying out simple movements. The barren sow stall does not meet the sow's social and cognitive needs and fails to allow for behaviors important to her species' way of life, and makes the animal depressed and frustrated to the point that she must perform repetitive actions in a pitiful appeal for mental stimulation. In scientific studies, sows have let us know themselves that they prefer environments that offer more space and complexity.