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EFFECT OF DIET AND SPACE ON BARROW PERFORMANCE – EXPERIMENTS AND MODEL DEVELOPMENT

by

Kuo-Wei Ssu

A DISSERTATION

Presented to the Faculty of

The Graduate College at the University of Nebraska

In Partial Fulfillment of Requirements

For the Degree of Doctor of Philosophy

Major: Animal Science

Under the Supervision of Professors Michael C. Brumm and Phillip S. Miller

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Kuo-Wei Ssu

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UNIVERSITY OF NEBRASKA

EFFECT OF DIET AND SPACE ON BARROW PERFORMANCE – EXPERIMENTS AND MODEL DEVELOPMENT

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University of Nebraska, 2001

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Three experiments were conducted using feather meal as a source of dietary excess protein and space allocation to manipulate feed intake of barrows to improve carcass leanness. The results of the three experiments indicated that feed intake of barrows was reduced for the first two to three weeks after feather meal diets were began and then feed intakes gradually increased to a level similar to control barrows. Carcass leanness of barrows was not consistently improved by feather meal diets. Results suggested that feeding feather meal diets to barrows after their maximum rate of protein deposition was reached resulted in leaner carcasses. However, the daily lean gain of barrows was reduced by feather meal diets when fed before the maximum protein deposition rate was reached. Use of feather meal also improved the apparent digestibilities of calcium and phosphorus, resulting in reduction of calcium and phosphorus. Reducing space allocation of barrows decreased their feed intake. Results also suggested that body weight was affected by space allocation earlier in the trial than feed intake of barrows. This indicated that the reduction of feed intake was the consequence of the reduction of body weight affected by crowding.

The evaluations of a swine growth model using two newly suggested growth parameters from literature (Whittemore, 1998; Emmans and Kyriazakis, 1999) resulted in very high growth rates and feed efficiencies. Therefore, a new set of growth parameters was suggested to lower the growth rate predictions of the model to improve its prediction precision. The evaluation of the swine growth model using the research data indicated that the model was unable to predict the responses of barrows fed feather meal diets. However, the model was capable of predicting the responses of barrows fed common corn-soybean meal diets in both crowded and uncrowded pens. Further research is needed to reveal the feed intake reduction mechanism for pigs fed feather meal diets before it can be implemented into the model.

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Literature Review

1. Introduction

The primary agriculture purpose of the pig is to provide human food, therefore, it is important for the pork industry to produce high quality pork with a high production efficiency. Many factors have been identified by researchers that affect pork production efficiency. Feed intake and energy intake regulation in finishing pigs are problems of primary importance for lean-meat production. Increasing dietary protein concentration was effective in improving carcass leanness of pigs, however, feed intake was also reduced when pigs are fed high-protein diets. Pigs have the ability to consume more feed than needed to support their maximum lean growth in the finishing phase, while their protein accretion rate reached a plateau and was gradually decreased as body weight increases, especially for barrows. Barrows typically eat more feed, grow faster, and reach market weight 10 to 14 days faster than littermate gilts. Because of the increased feed intake, barrows have fatter carcasses than gilts. Thus, it is possible to feed high-protein diets to barrows to improve their carcass leanness and also reduce their feed intake during the finishing phase to slow down their growth rate.

As more producers adopt all-in-all-out (AIAO) systems, the difference in growth potential between barrows and gilts has become a concern affecting the efficiency of production because barrows grow faster than gilts. Pig flow and uniformity of pigs are improved when the growth rate of barrows is decreased, therefore barrows can reach market weight at similar time as their littermate gilts. The improvement of uniformity of pigs also means an improvement of profitability, especially for less than one thousand

head production systems where they don't have enough barrows or gilts along in each batch to fill a 2.5 tons truck to reduce their transportation cost. There were 101.7 million pigs slaughtered in the United States in 1999 (USDA, 2000). If one-half of the slaughtered pigs were barrows and 20% of the barrows were fed high-protein diets to improve their carcass leanness, there would be 10.2 million barrows with improved carcasses and producers would have higher value in return.

A computerized pig growth simulation model is an effective tool in assisting swine production managers in making decisions to improve production efficiency. However, a good pig growth model needs a comprehensive test and on-going validation to improve its accuracy in predicting growth performance of pigs over a wide range of conditions. A well-tested simulation model can provide reliable information to producers and researchers in several situations including testing alternative management systems, testing changes of marketing strategy, testing hypothesizes, and testing treatment effects before a new experiment is conducted.

In this literature review, feed intake control mechanisms, effect of high protein diets, use of feather meal as dietary protein source, and pig growth modeling will be reviewed for their potential roles in improving the efficiency of pork production.

2. Feed Intake Control

Feed intake control in animals is a complex function involving nutritional, physiological, and psychological factors. When voluntary feed intake is too high, excessive energy intake will be deposited as body fat. When voluntary feed intake is too low, the growth of animals will be depressed. Therefore, it is best to match an animal's

voluntary feed intake to their requirements in order to improve the efficiency of production. For finishing pigs, feed intake and energy intake exceeding their requirement for maximum lean growth has lowered the production efficiency by increasing feed cost and reducing feed efficiency and carcass values. It is important to regulate or manipulate feed intake of finishing pigs, especially barrows, to reduce their feed intake while maintaining the maximum lean growth to improve production efficiency. To lower the feed intake of finishing barrows to a level not affecting protein growth will reduce the energy intake of barrows and then reduce the body fat deposition, thus improve the carcass leanness of barrows. The high carcass values of barrows will lead to higher profit for producers, therefore, it will improve the efficiency of production.

A nutritionist is most interested in providing feeds that match the animals' voluntary feed intake, nutrient requirements, and are econimical. However, when using cheaper alternative feed ingredients in diets, it is a concern that their characteristics may change an animals' appetite and voluntary feed intake. Therefore, it is important for nutritionists to understand the process of feed intake control in animals in order to successfully achieve their goals.

2.1 Central Nervous System Control

The central nervous system (CNS) is an integrative component in animals that processes information collected from external and internal environments. Thirst, appetite, temperature regulation, and autonomic nervous system functions are all regulated by hypothalamus nuclei (Berne and Levy, 1993). It was first observed by Hetherington and Ranson (1940) that when the hypothalamus of a rat's brain was damaged, they overate and became over fat. The damaged areas of the rat's brain

were in the region of the ventromedial hypothalamus (**VMH**). The VMH of injected mice was damaged by injection of gold thioglucose and resulted in overeating and excessive fat deposition (Brecher and Waxler, 1949). The overeating behavior of rats with damaged or destroyed VMH was confirmed by other researchers which suggested that VMH was sensitive to glucose uptake and might play a role as a "satiety center". Similar results were also obtained from studies using pigs (Auffray, 1969; Khalaf, 1969; Baldwin, 1985).

The lateral hypothalamic area (**LHA**) is a part of the medial forebrain bundle that receives information both from the visceral receptors and from higher centers of the brain (Berne and Levy, 1993). When the chemical damages were on the areas of the LHA, rats (Teitelbaum and Epstein, 1962) and pigs (Khalaf and Robinson, 1972) were not interested in eating and died. Rats would resume their desire for eating if they were force-fed to keep them alive, but pigs would not. The area of the LHA became known as the "hunger center" (Teitelbaum and Epstein, 1962; Khalaf and Robinson, 1972).

Although feed intake of animals is controlled by brain, dietary nutrients are also playing a role in affecting feed intake of animals because they can interact with brain mechanisms or neurotransmitters to influence their nutritional intake. Peng et al. (1972) reported that a possible brain mechanism was involved in regulating the feed intake of animals fed amino acid imbalanced diets. Therefore, this indicates a possible method to manipulate feed intake of pigs via nutrient content of the diets. Since feed intake and energy intake of finishing pigs is a very important factor affecting the production efficiency of pork production, an effective method to

manipulate feed intake and energy intake of finishing pigs will have significant impact on improving efficiency of pork production.

Acetylcholine (Cholinergic agents): Acetylcholine acts as a neurotransmitter for sending impulses to the hypothalamus and receiving impulses from the hypothalamus to the median eminence (Drivers and Forbes, 1982). Acetylcholine is not considered to affect feed intake because when atropine, a cholinergic antagonist, was injected into the hypothalamus, it had no effect on feed intake in sheep (Forbes and Baile, 1974). When sheep were injected with carbachol, a slowly metabolized cholinergic agent, into the lateral ventricle, feed intake was inhibited. The effect of carbachol could be prevented by previous injection of atropine (Driver et al., 1979; Driver and Forbes, 1982).

Bombesin-Like Peptides: Bombesin, an amphibian tetradecapeptide (Anastasi et al., 1971), is a peptide which can inhibit feed intake without toxic side effects (Lieverse et al., 1993; Muurahainen et al., 1993; Gutzwiller et al., 1994). Although bombesin is not present in mammals, two families of mammalian homologues of bombesin have been identified, gastrin-releasing peptide and neuromedin B. When bombesin and gastrin-releasing peptide were injected into brain, feed intake was inhibited and this effect appeared to be nonspecific because water intake was also inhibited in rats, hamsters, and pigs (Morley and Levine, 1981; Kulkosky et al., 1982a, 1982b; Parrott and Baldwin, 1982; Miceli and Malsbury, 1985). The effect of bombesin on feed intake reduction may work via cholecystokinin (**CCK**) in animals. When bombesin was administrated

peripherally to rats, CCK was released from the small intestine (Erspamer et al., 1974). It was confirmed later that CCK had a satiating effect (Gibbs et al., 1979).

Cholecystokinin (CCK): Cholecystokinin is a gastrointestinal hormone that is produced by discrete endocrine cells of the upper small intestine. Cholecystokinin is secreted from the intestine in response to the ingestion of food (Liddle, 1994). Cholecystokinin has been reported to reduce feed intake in rats, dogs, pigs, monkeys, and humans (Gibbs et al., 1973; Gregory et al., 1989; Reidelberger et al., 1989; Della et al., 1990; Ebenezer et al., 1990; Drewe et al., 1992; Moran et al., 1993; Weatherford et al., 1993; Ballinger et al., 1995; Lieverse et al., 1995b; Voigt et al., 1996). The functions of CCK include inhibition of food intake and gastric emptying, stimulation of insulin secretion, pancreatic enzyme secretion, pancreatic growth, gallbladder contraction, and intestinal motility (Walsh, 1987; Rayner and Miller, 1993; Liddle, 1997). Cholecystokinin receptors have been identified throughout the brain and peripheral tissues including stomach, pancreas, gallbladder, pyloric sphincter, and afferent vagal fibers.

Because of the wide distribution of CCK receptors in the brain, research has been conducted to identify the mechanism of the satiety effect of CCK (Smith and Gibbs, 1992; Reidelberger, 1994; Brenner and Ritter, 1995). Results from these studies suggest CCK contributes to the satiety. The satiety effect of CCK is mainly mediated by the type A receptor which is primarily distributed in periphery tissues because central administered CCK-receptor antagonists do not

reduce food intake (Asin and Bednarz, 1992; Parrott, 1993, 1994; Weatherford et al., 1993; Brenner and Ritter, 1995; Ebenezer et al., 1996; Corp et al., 1997).

It has also been observed that when infusing CCK into blood vessel at physiological levels (basal level ~ 1pmol/L, after meal 3-8 pmol/L), food intakes were depressed in rats and humans (Cunningham et al., 1991; Lieverse et al., 1995a, b; Corp et al., 1997). Other evidence is that rats with chemical lesions of vagal afferent nerves and efferent nerves increased their food intake and with arterial infusions of CCK suppressed their food intake (Schwartz et al., 1995; Chavez et al., 1997; Moran et al., 1997).

Dietary carbohydrate has no effect on regulating feed intake or stimulating CCK release (Lieverse et al., 1995a, b). Dietary fat or intraluminal infusion of lipid was effective in suppressing feed intake and elevating plasma CCK level (Cunningham et al., 1991; Drewe et al., 1992; Read et al., 1994). Although the mechanism of how dietary fat stimulates CCK release from the gut remains unclear, it is clear that increasing dietary fat concentration can reduce feed intake and the size of next meal (Cunningham et al., 1991).

Evidence demonstrates that the down regulation of dietary fat on feed intake is a short-term effect. When a high fat diet was fed to obese rats and humans, plasma CCK was increased. After a two-week adaptation period, food intake of obese rats and humans was not affected by increased dietary fat while plasma CCK remained high. Consequently, energy intake of these obese subjects increased after the adaptation period and their CCK sensitivity was decreased (French et al., 1993, 1995; Read, 1994).

Soybean trypsin inhibitor has been reported to be a potent stimulus for pancreatic enzyme secretion and CCK release (Green and Lyman, 1972; Lu et al., 1989; Garlicki et al., 1990; Weller et al., 1990). Dietary protein has drawn some attention for its role in CCK regulation because both soybean trypsin inhibitor and dietary protein bind to trypsin (Owyang et al., 1986a, 1986b). It is believed that trypsin inhibitors stimulate pancreatic enzyme secretion indirectly by binding or neutralizing trypsin and therefore removing its feedback inhibition. This feedback control system has been found in chickens (Chernick et al., 1948), pigs (Corring, 1973), and humans (Owyang et al., 1986a; 1986b). In humans, this inhibitory effect is protease specific because food intake suppression was not observed with intraduodenal perfusion of lipase or amylase (Owyang et al., 1986a).

The mechanism of how trypsin suppresses the release of CCK is still unclear. Owyang (1994) has demonstrated a mechanism whereby the increased pancreatic enzyme secretion following pancreatic juice diversion is mediated by a trypsin-sensitive peptide secreted by the small intestine that stimulates release of CCK. A trypsin-sensitive CCK-releasing peptide was later purified and tested by researchers who found this CCK-releasing peptide mediated postprandial release of CCK and feedback regulation of pancreatic secretion (Spannagel et al., 1996, 1998; Li et al., 2000). Most importantly, it was found that intact protein was the only stimulant of CCK release in rats (Liddle et al., 1986) and cats (Backus et al., 1997). The possible action is that dietary protein is bound to trypsin in the intestine which then activates the CCK-releasing peptide because there is no