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Changes in Feeding Behavior and Metabolism Associated with the Addition of Tallow to the Diet of Lactating Sows

by

Paul M. Ermer

A DISSERTATION

Presented to the Faculty of
The Graduate College in the University of Nebraska
In Partial Fulfillment of the Requirements
For the Degree of Doctor of Philosophy

Major: Animal Science

Under the Supervision of Professors
Austin J. Lewis and Phillip S. Miller

Lincoln, Nebraska

May, 2000

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GRADUATE COLLEGE
UNIVERSITY OF NEBRASKA

Changes in Feeding Behavior and Metabolism Associated with the Addition of Tallow to the Diet of Lactating Sows

Paul Michael Ermer, Ph.D.

University of Nebraska, 2000

Advisors: Austin J. Lewis / Phillip S. Miller

An experiment was conducted to examine the effect of immunoneutralization against CCK-8 on feed intake of lactating sows. Forty-eight sows were immunized against CCK-8 and received either a corn-soybean meal (C-SBM) or a corn-soybean meal-10% tallow (tallow) diet. Diet had no effect (P = .33) on metabolizable energy (ME) intake. No linear relationships between either feed or ME intake, or litter weight gain, and anti-CCK-8 titer were present (P = .18 to .94). These results indicate that immunization against CCK-8 does not increase sow feed intake or litter performance. Four experiments also were conducted to examine: 1) meal patterns, and 2) plasma concentrations of selected metabolites and hormones in lactating sows fed either a C-SBM or a tallow diet. There were 16, 16, 18, and 8 sows in Exp. 1 through 4, respectively. There was no effect of diet on ME (P = .34 to .94) intake in Exp. 1 through 4. Feed intake was reduced (P = .09) in sows fed tallow in Exp. 4. Tallow reduced (P = .01) meal size in Exp. 3, but there was no effect (P = .13 to .92) of diet on

meal size in Exp 1, 2, or 4. In Exp. 1 through 3, tallow reduced (P = .0001 to .05) the time spent consuming feed and increased (P = .0001 to .08) the rate of feed consumption. There was no effect of diet on the number of meals consumed (P = .29) or the time spent consuming feed (P = .79) in Exp. 4. In Exp. 2, plasma concentrations of glucose were increased (P = .01), while concentrations of non-esterified fatty acids (NEFA; P = .10), and glucagon (P = .02) were decreased in sows fed tallow. There was no effect (P = .17 to .61) of diet on the plasma concentration of metabolites or hormones in Exp. 3. In Exp. 4, the concentration of NEFA was increased (P = .06) in sows fed tallow. The effect of tallow on feeding patterns are consistent with the benefits of dietary fat observed during heat stress. Changes in plasma concentrations of metabolites and hormones were inconsistent regarding both parity and prandial state.

Acknowledgments

It is difficult to find the words to express my feelings for those who have been such an important part of my life. To Drs. Phil Miller and Austin Lewis, your unending patience with my progress on this project is undoubtedly worthy of a place in the Guinness Book of Records. The personal and professional guidance of Drs. Don Levis, Duane Reese, and Ted Doane is most greatly appreciated. I fear that I shall never be able to fully repay the support and friendship of all these men. Without the knowledge, ability, and insight of Dr. Jerry Pekas, this project would have never come to fruition. I am deeply indebted to you for your many investments into this project and my development. To Drs. Pekas, Miller, and Lewis, I offer my sincerest apology for the delay in this project. I would also like to thank Drs. Rodger Johnson and Rick Grant for serving on my reading committee and Drs. Roger Kittok and Jack Nienaber for serving on my committee.

To the many students I have come to know, particularly Drs. Bridgette Kirkpatrick-Fuhrmann, Andrea Cupp, Brad Frekking, and Scott Eilert, and Ms. Shawna Lodge, your friendship and camaraderie has provided for some great memories. I would like to thank many others, including Drs. Scott Tilton, Gary Louis, and Bill Weldon, and Mr. Darren

Critser for their friendship and many efforts in the completion of this project. The assistance of Cindy Wolverton, Joy Kovar, Ruth Diedrichsen, and Cindy Blankenship along with the crew at the swine farm at Mead is also greatly appreciated.

To my parents, Clarence and Jeanette, I offer thanks for the inspiration, prayers, love, and the values they instilled in me. Moreover, they have been a model of grace and character which I can only aspire to.

My wife, Robin, has been a beacon of hope and love. You are my love, my best friend, and my greatest inspiration. The best thing about me is you.

Paul M. Ermer

Table of Contents

Literature Review

Introduction

Suboptimal intake of nutrients by lactating sows has received a great deal of attention by researchers. The resultant lower nutrient intake may contribute to reproductive disorders, such as a prolonged weaning to estrus interval (King and Dunkin, 1986; Reese et al., 1984; King, 1987), or as less evident problems such as reduced embryo survival (Hughes et al., 1984) and subsequent decreased litter size (Kirkwood et al., 1988). In addition, suboptimal nutrient intake results in a reduction in precursors for milk synthesis and reduced weaning weights (Reese et al., 1982; Brendemuhl et al., 1989).

Nutrition and Reproductive Failure

Total culling rates for sows remain high. One Midwest database reported annual culling rates of 59% (Swine Graphics, Inc., 1990). The majority of sows were culled for reproductive reasons. In summarizing data from 28 Midwest swine herds, Lucia et al. (1995) found that reproductive failures accounted for the largest proportion (34%) of sows culled. Culling rates for reproductive failure were highest among primiparous sows.

Characterizing reproductive failures due to reduced

nutrient intake is confounded by variations in genetics, parity, and body composition. Furthermore, no critical amount of body fat or protein loss resulting in postweaning anestrus has been identified. Both dietary energy and protein affect the weaning to estrus interval. Collectively, the results of several experiments (Nelssen et al., 1985; Reese et al., 1982; Brendemuhl et al., 1987) indicate no detrimental effects of restricting metabolizable energy (ME) intake to 16 Mcal/d.

It has been suggested (Brendehmuhl et al., 1987) that suboptimal amino acid intake and associated losses of body protein during lactation may further reduce the weaning to estrus interval compared to suboptimal energy intake. Restricting protein (380 g CP/d) but not energy (8 Mcal/d) intake resulted in a decrease in the percentage of sows in estrus by d 7, 14, and 35 postweaning.

Other experiments indicated an interaction of energy and protein intake on the weaning to estrus interval (King and Williams, 1984). The mechanism by which energy and protein intake affect the weaning to estrus interval may be the secretion of luteinizing hormone (LH). Tokach et al. (1992) found that suboptimal lysine and energy intakes reduced both the concentration and number of pulses of LH in sows. More pronounced feed restriction (50%), during either the first three weeks or the last week of lactation,

resulted in reduced sow weight and backfat, diminished LH pulse frequency, and reduced ovulation rate while extending the weaning-to-estrus interval (Zak et al., 1997).

Nutritional Limitations during Lactation

Suboptimal feed intake and associated losses of body tissues in support of lactation remain a prevalent problem in the swine industry. In a summary of records from 30 swine herds encompassing 25,000 mixed-parity lactating sows, Koketsu et al. (1995) reported average daily feed intakes (ADFI) of 5.2 kg for sows producing litters weighing 52 kg after 19 d of lactation. Substantial losses in both body fat and protein would have occurred in these sows. Assuming these sows were consuming a 16% CP corn-soybean meal diet (3,200 kcal ME/kg) and an efficiency of dietary energy utilization for milk energy of 72% (Noblet and Etienne, 1987), the calculated energy deficit would require the mobilization of 6.2 kg of fat during lactation. Also, assuming a 70% efficiency of digestible CP for milk synthesis (ARC, 1981), the CP deficit of 3.1 kg would require the mobilization of 15.5 kg of muscle. Clearly, feed intake may place serious limitations on the lactation capacity of sows. Matzat et al. (1990) reported that

superalimentation of lactating sows (120% of ad libitum) resulted in reduced sow weight and backfat losses and increased litter weight gain.

Our understanding of feed intake regulation must advance to keep pace with the demands of increasing litter size. Sauber et al. (1994) have estimated that the lactational capacity of sows under maximal nursing demand is 1.5 to 2 times that estimated for sows nursing seven to nine pigs.

The Regulation of Feed Intake

Feeding is more than a behavioral index of hunger. Control of feed intake requires not only signals indicating the availability and utilization of fuels in the body, but also information regarding the nutritional quality of the diet. This requires learning and experience. In the past, feed intake research was driven by the search for the "switch," the ultimate signal involved in hunger. Hunger is now considered a highly integrated process related to both the availability of metabolic fuels and the rate of their utilization. Furthermore, satiety is specific to an individual diet. Animals form associations between the organoleptic properties of a diet and the consequences of

its ingestion in order to maintain energy balance.

In both scope and complexity, research into the control of feed intake is one of the most rapidly expanding areas of research. Our appreciation of feed intake regulation has expanded from experiments involving injections of various substances into, or ablation of, various brain regions and the associated feeding responses to experiments designed to identify interactions of behavioral and metabolic cues in the regulation of intake.

In this review, I will first describe the role of the central nervous system in controlling feed intake. The brain regulates both feed intake, by integrating central and peripheral signals into a feeding response, and nutrient utilization, through sympathetic and parasympathetic outputs. I will then discuss the origins and roles of the metabolic cues involved in feed intake regulation. Finally, I will discuss the effects of dietary fat on the secretion of pancreatic hormones involved in feed intake regulation and the effect on feed and energy intake.

Limited information regarding mechanisms involved in feed intake regulation has been obtained using swine. Unless stated otherwise, information in this review was obtained from experiments utilizing rats or mice. However, when possible, extrapolations to swine will be made.

Central Nervous System (CNS) Mechanisms Controlling Appetitive Behavior

Emphasis regarding CNS control of feed intake has focused primarily around the lateral (LHA) and ventral-medial (VMH) regions of the hypothalamus. Both electrical and(or) physical lesioning of the VMH results in hyperphagia, while hypophagia results from similar lesioning of the LHA. These effects are observed in a variety of mammalian species, including swine (Auffray , 1969; Khalaf and Robinson, 1972; Rohner-Jeanrenaud, 1995), and form the basis of the dual-center hypothesis of feed intake control. The basis of this hypothesis is that the LHA is designated the hunger center and the VMH is designated as the satiety center. Both the LHA and VMH contain neurons sensitive to changes in glucose concentration (Oomura et al., 1990). Identical changes in feed intake and body weight result from either injection of gold thioglucose (GTG) into, or electrical stimulation of, these regions (Le Magnen, 1985; Bernardis and Bellinger, 1993). These findings suggest that changes in body weight associated with hypothalamic lesion are a result of destruction of glucose-sensitive cells.

Physiologically relevant data regarding the reciprocity between the LHA and VMH in the regulation of feed intake

include the fact that subcutaneous administration of glucose, glycerol, or lysine result in decreased total ribonucleic acid (RNA) content in the VMH and increased total RNA in the LHA (Kuchar et al., 1984). Similarly, total RNA content was increased in the VMH and reduced in the LHA of rats fasted for either 12 or 24 h as compared to rats allowed ad libitum access to feed (Mozes et al., 1988).

Changes in body weight resulting from perturbation of hypothalamic regions (i.e., LHA, VMH) are primarily of a metabolic nature, rather than a passive change in body weight in response to manipulation of a hunger or a satiety center. The LHA and VMH play reciprocal roles in regulating the activity of the autonomic nervous system with concomitant changes in whole body metabolism and feed intake (Bray, 1986).

After damage to the VMH, there is hyperactivity of the parasympathetic nervous system and reduced activity of the sympathetic nervous system (Bray, 1986). The physiological implications of VMH lesion are multifaceted including direct effects on energy utilization, carbohydrate metabolism, and endocrine secretion. Lesioning of the VMH directly influences energy utilization by reducing sympathetic firing rate to brown adipose tissue (BAT) in rodents (Sakaguchi et al., 1988; Rohner-Jeanrenaud, 1995). Both noradrenaline turnover and guanosine diphosphate (GDP) binding to

mitochondria are reduced as is diet-induced thermogenesis in this tissue. Glucose metabolism is influenced both by direct effects on enzymes involved in glycogen metabolism as well as changes in endocrine hormone concentrations. Reduced sympathetic input to the liver results in increased activity of glycogen synthase (Bray, 1986). Increased vagal and reduced sympathetic tone are involved in increasing both basal and meal-stimulated insulin and glucagon secretion (King and Frohman, 1982). Both the circadian pattern and concentration of corticosterone are affected (Dallman, 1984). Plasma concentrations of corticosterone have been reported to be either elevated (King et al., 1983; Dallman, 1984) or reduced (Arase et al., 1989a) as a result of VMH lesion. Nearly all of the metabolic anomalies which occur with lesioning of the VMH are present in genetic models of obesity, including the *fa/fa* rat and *ob/ob* mouse (Rohner-Jeanrenaud, 1995). Metabolic changes occurring in these strains precede and(or) are independent of the onset of hyperphagia (Planche et al., 1983; Rohner-Jeanrenaud and Jeanrenaud, 1985).

Hypersecretion of insulin is critical in the etiology of VMH lesion. Hyperinsulinemia was attenuated in VMH-lesioned rats following vagotomy (King and Frohman, 1982). In rats with denervated pancreas (Inoue et al., 1988) or

treated with streptozotocin (York and Bray, 1972), the presence of VMH lesion did not result in either elevated insulin secretion or increased weight gain (Inoue et al., 1988). Corticosterone exhibits a permissive role in the development of obesity following VMH lesion. Following adrenalectomy, cessation or reversal of obesity has been observed (Dallman, 1984; Bray, 1988).

The metabolic consequences of LHA lesion are extensive, but opposite in effect to those of VMH lesion. The most salient feature of LHA lesion is reduced activity in the parasympathetic branch of the autonomic nervous system and increased activity in the sympathetic branch (Arase et al., 1987). Again, direct metabolic effects are observed in LHA lesion including increased oxygen consumption, higher body core temperature, and increased energy utilization in BAT (Bernardis and Bellinger, 1993). Lesioning of the LHA also results in chronic increases in adrenal weight and circulating concentrations of both catecholamines and corticosterone (Bernardis and Bellinger, 1993). Reduced plasma concentrations of insulin, hyperglycemia, and increased glucagon concentration are observed with LHA lesion (Bray, 1986).

Weight change induced by hypothalamic lesion can be characterized by the underlying change in metabolism. First, changes in feed intake alone cannot mimic the effects

of the lesion. Also, many of the metabolic anomalies resulting from hypothalamic lesion occur despite maintaining constant feed intake. For example, rats with LHA lesion lose weight faster and to a greater extent than non-lesioned rats consuming similar amounts of feed (Bernardis and Bellinger, 1993). Similarly, rats with VMH lesion pair-fed to non-lesioned rats exhibited increased body weight and fat content (King and Frohman, 1982; Sakaguchi et al., 1988). Also, characteristic of hypothalamic lesion is the establishment of a new body weight "set-point," rather than absolute weight change. Reductions in feed intake and body weight were less pronounced in rats that had weight reduction prior to LHA lesion (Bernardis and Bellinger, 1993). Changes in body weight may be suspended for short periods, indicating constancy of this set-point. Following a period of tube feeding to maintain body weight, LHA-lesioned rats reverted to anorexia to attain a body weight similar to that of non-tube fed controls (Bernardis and Bellinger, 1993).

Neurotransmitters Involved in Body Weight Regulation

A variety of neurotransmitters are involved in the central control of feed intake. Bombesin, cholecystokinin