EVALUATION OF BETA-GLUCAN, ANTIBIOTICS, AND ANTIMICROBIAL ALTERNATIVES ON GROWTH PERFORMANCE AND IMMUNOLOGICAL PARAMETERS IN WEANLING PIGS

By

RODEL PUNZALAN CUENO

Bachelor of Science in Agriculture

University of the Philippines Los Baños

Laguna, Philippines

2000

Submitted to the Faculty of the Graduate College of the Oklahoma State University in partial fulfillment of the requirements for the Degree of MASTER OF SCIENCE July, 2005

EVALUATION OF BETA-GLUCAN, ANTIBIOTICS, AND ANTIMICROBIAL ALTERNATIVES ON GROWTH PERFORMANCE AND IMMUNOLOGICAL PARAMETERS IN WEANLING PIGS

Thesis Approved:

Dr. Scott D. Carter Thesis Adviser

Dr. Stanley E. Gilliland

Dr. Robert G. Teeter

Dr. A. Gordon Emslie Dean of the Graduate College

ACKNOWLEDGEMENTS

I would like to extend my heartfelt gratitude to the following important people, who have made valuable contributions for the accomplishment of this research study:

Dr. Scott D. Carter, my research adviser and mentor. He unselfishly shared his time and expertise, which made this research endeavor an effective learning process;

Dr. Bob Teeter and Dr. Stan Gilliland, my committee members, for their innovative lectures and insightful ideas, which greatly improved my work;

Dong-Ahm BT (Seoul, Korea), for partial financial support and for supplying the beta-glucan preparation used in all experiments;

The Fulbright-Philippine Agriculture Scholarship Program (FPASP), for the chance they have given me to do my graduate studies here in the US and to make my country proud;

Dr. Esmeralda Cunanan, Ms. Angela Dizon, and the Philippine-American Educational Foundation staff for all the help and support you have given me at the very start of the FPASP;

Paetra Hauck, Marie Ward, and David Hadley of Institute of International Education (Houston), and Randy Beckloff, Ruth Loffi, and Brenda Dean of International Students and Scholars, for the all the assistance, guidance and support;

Kim Brock, Cecil Hooper, and the swine barn crew (Reed, Travis, John, Sara, Emily, Chad, Todd, James, Nick) for assisting me in the conduct of my experiments and data collection;

iii

Jason Schneider, Dr. Jin-Seong Park, Theresa Buhay, Mariela Lachmann, and Sherrita Jenkins, my co-workers, for the camaraderie and for sharing their time and abilities to me on my research;

My fellow animal science graduate students (Dan, Rebecca, Kristin, Amy, Jason R., Jason B., Luis, Morgan, Patrick, Nathan, Pauline, Monica, Stanley, Dustin, Vicky, Ivette, Mayte, Sashi, Francis, Russell) in the department, for the friendship and good company;

My former professors in the Department of Animal Science, for the knowledge I acquired which enhanced my appreciation for this field;

Julian, my housemate, and the OSU Filipino community (Adel, Al, Joel, Rose, Yoli, Mel, Ed, Mike, Mader, Menchu, Wang, Brenda, Edwin, Leon, Ritchie, Arnold, Gina, Grace, Norma, Yusuf, and Tina) for their hospitality and good nature;

My fellow Fulbright grantees (Ervin, Eugene, Jerome, Jhoe', Joy, Lori, Melanie, Melvin, Myrene, Pam, Reynold, Sam, Serge, and Willie) for all the support and encouragement;

John, Elaina, Menggai, Bill, Donna, K-K, Cesar, and Len, Lylee, Tinay, Dobbie, Bunny, Claire, Rona for the friendship;

Tatay, Nanay, Kuya RV, Vhim, Rouvel and Roniel, my beloved family, for their unwavering encouragement despite being far from home;

Sarah, my fiancée, for her love and patience that inspired me to do the best I can. I can't imagine spending the rest of my life without her;

And of course, our Almighty God for the guidance, abundant blessings and unique opportunities that came into my life.

iv

TABLE OF CONTENTS

Chapter	Page
I. INTRODUCTION	1
II. LITERATURE REVIEW	2
Introduction Concern on antibiotic resistance European Union ban on antibiotics as growth promotants Effects of a ban in the United States	2 3 6 7 8 11 12 14 15 18 19 21 23 25 26 27
III. EFFECTS OF BETA-GLUCAN AS AN ALTERNATIVE TO ANTIBIOTIC IN THE DIETS OF WEANLING PIGS	28 28 28 30 32 35
IV. EFFECTS OF BETA-GLUCAN AND ANTIBIOTICS ON GROWTH PERFORMANCE AND CARCASS TRAITS OF WEANLING AND FINISHING PIGS	39

Introduction Materials and Methods Results	
Discussion	
V. EFFECTS OF BETA-GLUCAN, ANTIBIOTIC, AND ACIDIFIER ON GROWTH PERFORMANCE OF WEANLING PIGS	
Introduction	
Materials and Methods	
Results	
Discussion	
Conclusions	
VI. EFFECTS OF BETA-GLUCAN, ANTIBIOTIC, AND PROBIOTIC ON GROWTH PERFORMANCE OF WEANLING PIGS	
Introduction	
Materials and Methods	
Results	
Conclusions	
VII. SUMMARY	
Results	
Discussion	
VIII. CONCLUSION	
REFERENCES	
APPENDIX TABLES	

LIST OF TABLES

Chapter II

2.1 Response of weaned piglets to dietary organic acids	20
2.2 Effects of promicrobial and antimicrobial agents in starting diets for	
weanling pigs	22
2.3 Growth responses to Bio-Mos in starter diets for pigs	24

CHAPTER III

3.1 Composition of diets in Experiment 1 (as fed-basis)	36
3.2 Growth performance of weanling pigs (Exp. 1)	37
3.3 Hematology and serum CRP of weanling pigs (Exp. 1)	38

Chapter IV

4.1 Composition of diets in Experiment 2 (as fed-basis)	47
4.2 Growth performance of pigs during the nursery phase (Exp. 2)	48
4.3 Growth performance of pigs during the growing-finishing phase (Exp. 2)	49
4.4 Growth performance and carcass traits of pigs for the overall experiment	
(Exp. 2)	50

Chapter V

5.1 Composition of diets in Experiment 3 (as fed-basis)	60
5.2 Growth performance of weanling pigs (Exp. 3)	61

Chapter VI

6.1 Composition of diets in Experiment 4 (as fed-basis)	73
6.2 Growth performance of weanling pigs (Exp. 4)	74
6.3 Serum immune proteins (Exp. 4)	75

Chapter VII

7.1 Growth performance of weanling pigs from Exp. 1 to Exp. 4 (26 reps)	79
---	----

LIST OF FIGURES

Fi	gur	e
	\mathcal{O}	

CHAPTER II

2.1 Some routes of transmission of antibiotic-susceptible or –resistant gastrointestinal or normal intestinal flora between animals and humans	5
2.2 Universe of bacteria	6
2.3 Diagram of the proposed effects of antibiotics mediated through their effects on small intestinal microflora	16
2.4 Four major biochemical mechanisms of antibiotic resistance	18

CHAPTER VII

7.1 Percentage improvement in ADG due to carbadox or beta-glucan during the	
nursery phase from Experiment 1 to Experiment 4	80
7.2 Percentage improvement in GF due to carbadox or beta-glucan during the	
nursery phase from Experiment 1 to Experiment 4	80

APPENDIX TABLES

Tab	le	Page
1	Means for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 1	97
2	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 1	98
3	Means for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 1	99
4	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 1	100
5	Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period – Experiment 1	101
6	Analysis of variance for average daily gain, average daily feed intake, and gain: feed for the entire 42-d period. – Experiment 1	102
7	Means for average daily gain, average daily feed intake, and gain:feed for Nursery Phase 1 and Phase 2 – Experiment 2	103
8	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Nursery Phase 1 and Phase 2 – Experiment 2	104
9	Means for average daily gain, average daily feed intake, and gain:feed for Nursery Phases 1 & 2 combined and Phase 3 – Experiment 2	105
10	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Nursery Phases 1 & 2 combined and Phase 3 – Experiment 2	106
11	Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period – Experiment 2	107
12	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period. – Experiment 2	108

13	Means for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 1 and Phase 2 – Experiment 2	109
14	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 1 and Phase 2 – Experiment 2	110
15	Means for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 3 and the entire grow-finish stage – Experiment 2	111
16	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 3 and the entire grow-finish stage – Experiment 2	112
17	Means for average daily gain, average daily feed intake, and gain:feed from nursery to finisher stage – Experiment 2	113
18	Analysis of variance for average daily gain, average daily feed intake, and gain:feed from nursery to finisher stage – Experiment 2	114
19	Means for hot carcass weight, 10 th rib backfat, longissimus muscle area, and fat-free lean carcass of pigs – Experiment 2	115
20	Analysis of variance for hot carcass weight, 10 th rib backfat, longissimus muscle area, and fat-free lean carcass of pigs – Experiment 2	116
21	Means for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 3	117
22	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 3	118
23	Means for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 3	119
24	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 3	120
25	Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period – Experiment 3	121
26	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period. – Experiment 3	122
27	Means for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 4	123

28	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 4	124
29	Means for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 4	125
30	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 4	126
31	Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period – Experiment 4	127
32	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period. – Experiment 4	128
33	Means for the IgA serum proteins – Experiment 4	129
34	Analysis of variance for IgA serum proteins – Experiment 4	130
35	Means for the IgG serum proteins – Experiment 4	131
36	Analysis of variance for IgG serum proteins – Experiment 4	132
37	Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 from Experiment 1 to Experiment 4 (26 reps)	133
38	Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 from Experiment 1 to Experiment 4 (26 reps)	134
39	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 1 to Experiment 4 (26 reps)	135
40	Means of average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined from Experiment 1 to Experiment 4 (26 reps)	136
41	Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 from Experiment 1 to Experiment 4 (26 reps)	137
42	Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined – Experiment 1 to Experiment 4 (26 reps)	138

CHAPTER I

Introduction

Antibiotics and other antimicrobial agents have been used in swine diets for more than 50 years to improve growth performance and overall health status of pigs. Significant improvements in growth rate, feed efficiency, and economic gains are observed in all phases of growth. However, a growing concern over the development of antibiotic resistance with the use of antimicrobial agents, in particular at subtherapeutic levels, has been raised by consumers and medical groups. Although numerous studies have been conducted that prove otherwise, the pressure by such groups could lead to the potential banning of antibiotics for growth promotion, which is now being implemented in European Union countries. In the event of a ban, growth performance would likely be reduced and the cost of production would be increased. Thus, there is a need to evaluate alternatives that would mimic the positive response of antibiotic growth promotants.

The alternatives currently on the market have varying mechanisms that focus on gut microflora and immunity of pigs. Some of these alternatives, such as acidifiers, probiotics, mannan oligosaccharides, and beta-glucan, have the potential to substitute for antibiotics in swine diets. However, improvements in swine growth performance due to these alternatives usually have been variable and inconsistent. Thus, there is a need to continue to evaluate these alternatives and their combination as a replacement to antibiotic growth promotants.

CHAPTER II

Literature Review

Introduction

For more than 50 years, antibiotics and other antimicrobial agents have been incorporated in animal feeds to improve overall health and growth performance of farm animals, in particular swine. Swine feeds often contain antimicrobial agents with an estimated presence of 80% to 90% in starter diets, 70% to 80% in grower diets, 50% to 60% in finisher diets, and 40% to 50% in sow diets (Cromwell, 2001). These antimicrobial agents are used therapeutically, to treat, control and prevent diseases, and subtherapeutically, to improve overall growth rate and efficiency (Cromwell, 2001; Shea, 2004; Harper, 2004; Mathews, 2001; Mathew and Ebner, 2004). Moreover, subtherapeutic use of antibiotics reduces mortality and morbidity, improves reproductive performance, and increases profit (Cromwell, 2001).

Over 1,000 experiments have been conducted in the United States from 1950 to 1985 that showed improvements in growth rate and feed efficiency in pigs, in all phases of growth, with the use of antibiotics. The addition of antibiotics in diets increased growth rate by 16.4% in young pigs (7 to 25 kg), 10.6% in growing pigs (17 to 49 kg), and 4.2% in the growing-finishing stage (24 to 89 kg), and feed efficiency also was improved by 6.9%, 4.5% and 2.2%, respectively (Cromwell, 2001). Using these parameters, Cromwell (2002) calculated a net return of \$2.99 per pig from weaning to

market weight using chlortetracycline as the antibiotic in the diet at a cost of \$0.20 per pig in starter and \$0.50 per pig in grow-finish. For gestating sows fed with chlortetracycline at a cost of \$0.49 per litter, a conservative assumption of a 5% improvement in farrowing rate and 1/3 pig per litter increase translated to a very significant net return of \$7.12 per litter.

Concern on antibiotic resistance

Despite the significant benefits of antibiotic usage for farm animals, concerns related to the development of antibiotic or antimicrobial resistance in animal and human medicine have increased in particular with subtherapeutic use of antibiotics. Of the 45% of antibiotics used in all animals in the U.S., 14% are used subtherapeutically; thus, approximately 6% of all antibiotics are used for growth promotion (Messenger, 2003; Smith, 2002). A survey by the Animal Health Institute in February 2000 reported that in 1998, 17.8 million pounds of antimicrobials were used in animal production of which 14.7 million pounds or 83% were used as therapeutics and only 3.1 million pounds or 17% were used as growth promotants (McEwen and Fedorka-Cray, 2002). Smith (2002) stated the WHO estimates that 40% of all antibiotics used in human medicine are unnecessary where millions of antibiotic prescriptions are written for colds, bronchitis and other respiratory infections caused by viruses that do not respond to antibiotics. He further added that pound for pound, humans use about 10 times more antibiotics than farm animals. Nevertheless, because the majority of antibiotics being used today in animal feeds are the same as that used in human medicine (Mathews, 2001; Shea, 2004; Chee-Sanford et al., 2001; Phillips et al., 2004), an overwhelming concern and pressure from health specialists and consumer-activist groups (Braude, 1978; Kunin, 1993;

Cassell, 1995) to ban the use of antibiotics as growth promotants in farm animals have arisen.

In a more recent report by Florini et al. (2005), they estimated that 70% of the antibiotics used in the United States each year are used for growth promotion due to overcrowding, stressful, and unsanitary conditions of commercial farms. Moreover, they reported that of the 26.5 million pounds of total antibiotic feed additives used in the United States, 42% are accounted for by swine. However, this percentage goes up to 69% if the medically important antibiotics, such as penicillins, aminoglycosides, macrolides, sulfonamides, tetracyclines, streptogramins, and clindamycin/lincomycin, are considered. They also stated in their report that with the use of antibiotic feed additives, it resulted in an annual excretion of 13.5 million pounds of antibiotics in animal wastes, of which swine account for 47% of all antibiotics feed additives, whereas it is 72% for the medically important antibiotics.

The subtherapeutic use of antibiotics creates selection pressure on the microbial population (Shea, 2004), and the chronic exposure of bacteria to low doses of broad-spectrum antimicrobial agents leads to the development of resistant genes for both animals and humans, as evidenced by several studies (Levy et al., 1976; Aarestrup and Carstensen, 1998; Mathew and Garner, 2003; Langlois et al., 1983). As outlined in Figure 2.1, the resistant genes from the farm animals could be transferred to humans in three pathways; through the food chain by consuming the meat, through contamination from the sewage, and through contamination from the animal feeds (Philips et al., 2004). However, only a small fraction of harmful bacteria affects both animals and humans with even a lesser percentage of resistant bacteria (Mathews, 2001; Figure 2.2). Furthermore,

a long-term study done by Langlois et al. (1986) on the use of tetracycline at subtherapeutic levels, resulted in a negligible increase in antibiotic resistance.



Figure 2.1. Some routes of transmission of antibiotic-susceptible or –resistant gastrointestinal or normal intestinal flora between animals and humans (Philips et al., 2004).

Cromwell (2001) also compiled several animal studies from Hays (1977) and Zimmerman (1986) on antibiotics that have been used more than 50 years and found that there was no significant change in their effectiveness. A recent antibiotic research assessment sponsored by Elanco Animal Health (2003) was performed using a semiquantitative mathematical model for two macrolide animal antibiotics (tylosin and tilmicosin) on their impact on food safety when used in food animal production. These researchers found that a person has an overwhelming low risk of acquiring food-borne bacteria resistance from eating the meat of animals treated with either of the two macrolides. Eating pork treated with tylosin and tilmicosin has a probability of acquiring resistant infection resulting in treatment failure of less than one out of 53 million people per year for resistant *Campylobacter*, and less than one out of 21 billion people per year for *Enterococcus faecium* (Elanco Animal Health, 2003).



Figure 2.2. Universe of bacteria (Mathews, 2001).

European Union ban on antibiotics as growth promotants

Due to the growing concern of antibiotic resistance transmission from animals to humans, several countries already have banned the use of antibiotics and other antimicrobial agents as growth promotants. Sweden started the ban in 1986, followed by Denmark in 1995, and based on the Precautionary Principle, where regulatory action was implemented to control potentially hazardous substances in the absence of established scientific evidence (Animal Health Institute, 2005), five antibiotic growth promoters have been banned in European Union countries since 1997 (Casewell et al., 2003).

Following the ban, piglet production in Sweden experienced significant clinical problems, such as a two-fold increase in post-weaning diarrhea, which resulted in a 75% increase in the therapeutic use of antibiotics (Wierup, 2001; Krause and Graham, 2004). Stein (2002) further reported a 1.5% increase in mortality, 2 to 3 kg increase in feed consumption, and a reduction in daily gain. But since the ban, there was a decrease in the total use of antibacterial drugs administered to animals by 55% and antimicrobial resistance has been maintained to relatively low levels of prevalence.

In Denmark, the use of antibiotic growth promoters decreased by 50% but the therapeutic use has not changed (Hayes and Jensen, 2003). In a review by WHO (2002), weanling pigs had an increase in mortality by 0.5% and a 2.6% reduction in weight gain when antimicrobial growth promoters were terminated. Hayes and Jensen (2003) further added that the cost of the antibiotic ban in Denmark ranged from \$3 to \$4.50 per pig.

Effects of a ban in the United States

The U.S. food-animal industry is under extreme scrutiny from medical and environmental groups, the legislative body, and the corporate and public consumers for its use of antimicrobials for growth promotion (Pork News Source, 2005, 2003; Messenger, 2002). Even the World Health Organization in August 2003 recommended a worldwide ban on the use of growth-antibiotics in animal feed in spite of the absence of a risk-based evaluation (Kaufman, 2003; Messenger, 2004). Although a single case of antibiotic use in food-producing animals causing human antibiotic resistance has yet to be proven from over 40 years of research (Avery, 2002), the U.S. FDA (2003) released a

document (Guidance for Industry #152) for safety assessment of new antimicrobial drugs with regard to their microbiological effects on bacteria of human health concern. The guidance was made in spite of the decline in antibiotic usage in the U.S. from 23.7 million pounds in 2000 to 21.8 million pounds in 2001 (Pork News Source, 2002). In addition, the Preservation of Antibiotics for Medical Treatment Act of 2005 may potentially eliminate novel drug development as new treatments for emerging animal diseases (Pork News Source, 2005).

In the likelihood of an antibiotic ban for growth promotion, Hayes et al. (2002) reported that feed efficiency would be reduced by 1.5%; an increase of 1.5% in the post-weaning mortality; a decline in yearly sow productivity by 4.82%; and an additional \$0.25 cost per pig for veterinary and therapeutic drug use. These most-likely case scenarios would increase the cost per head by \$6.05 in the first year and by \$5.24 per head by the end of a 10-year period, which would result in a decline in the net profit per head of \$4.17 and \$0.79 in the first year period and at the end of a 10-year period, respectively (Hayes et al., 1999). It would also increase the retail price of pork by \$0.052 per pound resulting in an extra cost nationally of \$748 million per year. Mathews (2001) also predicted a net loss of \$45.5 million for the U.S. swine industry as a result of not using antimicrobial drugs in swine production.

Alternatives to antibiotics in swine diets

As a consequence of the ban on antibiotic usage for growth promotion in the European Union countries, swine producers altered management and feeding strategies, applying basic biological and physiological principles to improve pig performance and reduce economic loss (Stein, 2002). Some of the alternative feed and management

strategies incorporated were: a) increase in nutrient concentration of the diet; b) improvement in substrate digestibility and availability; c) modification of gut acidity; d) use of probiotic organisms or competitive exclusion technology; e) manipulation of the immune system; f) application of antimicrobial property concepts; g) use different management and environmental controls; and h) change direction of genetic selection (Hardy, 2002).

Of the modifications and strategies mentioned above, the alternatives given the most consideration were those that focused on the gut microbiota and immunity of the pigs, especially at the nursery stage (Krause, 2003; Mathew, 2002). These alternatives included acidifiers, probiotics, mannan oligosaccharides, and immune enhancers, wherein their addition to the diet would provide improvements in pig growth performance and overall health (Mathew, 2002). The digestive tract, especially for younger animals, needs to have an acidic, low pH environment for proper protein digestion and prohibition of bacterial growth (Dinsmore et al., 1997; Hardy, 2003). Thus, acidifiers, either organic, inorganic, or a combination of both may help young pigs overcome post-weaning stress and diseases caused by pathogens (Hardy, 2002; Hardy, 2003).

Probiotics, on the other hand, are live-microbial feed supplements that improve intestinal microbial balance of the animal (Kelly, 2004). However, their results are rather inconsistent due to differences in strain of organism used, dosage level, diet composition, feeding strategy, feed form and interaction with other dietary feed additives (Chesson, 1994). Changes in daily gain ranges from -8.5% to +10.5% and feed efficiency from -1.4% to +21.4% (Pollmann, 1992; Hardy, 2002).

Another product that is gaining considerable attention as a potential alternative for antibiotic growth promotants is mannan oligosaccharides (MOS). They are made up of complex polymers of mannose derived from yeast cell walls (Tizard et al., 1989). Mannan oligosaccharides act as a prebiotic that beneficially affects the host by selective stimulation of favorable bacteria in the lower gastrointestinal tract, thus improving the health of the animal (Gibson and Roberfroid, 1995). Moreover, MOS bind on specific sites of the pathogenic bacteria, allowing the beneficial microorganisms to colonize the gut (Cromwell, 2001). But as with other alternatives, the effects of MOS on animal performance are inconsistent. Supplementation of MOS in three different nursery facilities resulted in variable outcomes on growth performances of weanling pigs which may be due to differences in sanitation, disease history, and health status of the pigs (Rozeboom et al., 2001; Turner et al., 2001).

For the immune enhancers, a product known as beta-glucan can be used to stimulate non-specific defense mechanism in animals (Hardy, 2003). Present in some plants, and yeast cell walls, it activates both innate and adaptive immune responses that could decrease the animal's susceptibility to disease and increase growth performance (Blecha and Charley, 1990). Previous experiments performed with weanling (Dritz, et al., 1995; Decuypere et al., 1998; Hiss and Sauerwein, 2003; van Nevel et al., 2003) and finishing pigs (Fortin et al., 2003) using different beta-glucan sources, reported variable effects on growth and immune parameters.

Currently, there is an on-going debate as to the extent of the effect of antimicrobials being used at subtherapeutic levels in the development of antibiotic resistance in animals and humans. But with the increasing pressure from consumer

groups and the government, the swine industry must be prepared to address a ban on antibiotic growth promotants. Aside from using alternative feed ingredients, nonnutritional strategies must be employed to compensate for the ban on antibiotic growth promotants and maintain the growth and performance of pigs with a product that is healthy, safe and acceptable to the consumer.

Gut mechanism and competitive exclusion

The gastrointestinal tract functions not only as a site for digestion and absorption of nutrients, but it also aids in the immune response of the animal, since it serves as a host of intestinal microflora that maintain gut health (Hardy, 2003). The intestinal epithelium of the gastrointestinal tract, with its high cell turnover rate and constant production of a protective mucus coat, provides not only an extensive surface area for absorption of digested nutrients, but also serves as a barrier to pathogenic bacteria and antigens (Gaskins, 2001; Gaskins and Kelly, 1995; Webel et al., 2003). On the other hand, the microbial ecology has an important role in the maintenance of integrity of the enterocyte, modulation of metabolic and immunologic processes, and protection against colonization by invasive pathogens (Levy, 2000). This relationship between the intestine and the microflora was explained by a study of Hooper et al. (2001), where the commensal bacterium influenced the gene expression of the host's intestine functions (i.e. nutrient absorption, mucosal barrier fortification, xenobiotic formation, angiogenesis, and postnatal intestinal maturation).

Despite the high population density, extensive diversity, and complexity of interaction, the microflora that reside in the gastrointestinal tract of an animal can be distinctly categorized between indigenous and nonindigenous bacteria (Gaskins, 2001).

The indigenous bacteria are those that were present during the animal's evolution, which are ubiquitous in the community, and the true pathogens that have been accidentally acquired and are capable of persisting in the gastrointestinal tract, while the nonindigenous bacteria are those that are derived from the environment but do not colonize the gastrointestinal tract (Dubos et al., 1965; Savage, 1977). In all of these, a balance between beneficial and pathogenic bacteria and their interaction with the gastrointestinal tract must be established to maintain the integrity and health of the animal (Gaskins, 2001).

Competitive exclusion, as defined by Gaskins (2001) and Genovese (2003), is an applied application of oral supplements of either defined or undefined mixed bacterial cultures derived from a normal gut bacterial microflora given to animals to prevent intestinal colonization by pathogens that cause food born disease and disease affecting the animal. It has been shown that the competitive exclusion cultures, when administered to piglets, were effective against *Salmonella* and *Escherichia coli* infections in swine with decreased shedding, intestinal colonization, and reduced mortality and morbidity (Genovese et al., 2003). Baum and Harris (2000) also reported a reduction in the number of infected pigs that were culture-positive for *Salmonella typhimurium* and a reduction in the duration of *S. typhimurium* shedding from tonsil and fecal samples when fed with *Lactobacillus* spp. cultures.

Post weaning lag

At weaning, piglets experience stress from nutritional, environmental, and social changes resulting in post weaning lag, a period of little or no growth (Pieterse, 2000; Pluske et al., 1997). This period is further accompanied with a reduction in feed intake

due to the change of the diet from liquid milk provided by the sow to a solid feed that leads to scouring or diarrhea of the weanling pig with its limited digestive enzyme capacity and immature immune system (van Heugten, 1997; Coffey and Cromwell, 2001). The decrease in feed intake reduces the protein mass and DNA content of small intestine (Burrin and Stoll, 2003) affecting its integrity. This, in turn, could compromise the pig's ability to properly digest and absorb nutrients, and also to resist enteric pathogens (Webel et al., 2003). Furthermore, dietary restrictions would also decrease the thickness of the mucosa, villous height and width, and villous surface area (Nunez et al., 1996). These changes were described by Pluske et al. (1997) as villous atrophy and crypt hyperplasia, which are evident at weaning.

There are several factors leading to villous atrophy and crypt hyperplasia in weanling pigs. Cera et al. (1988) studied the effect of age and weaning on small intestinal growth and morphology of piglets and found that the jejunal villous were shorter in weaned pigs compared to that of pigs that remained suckling with the sow. Exposure to pathogens after weaning also resulted in a reduction in villous height (Vellenga et al., 1992), and net absorption of fluid and electrolytes in the small intestine (Nabuurs et al., 1994). The type of ingredients, especially the protein source, in the diet of the young pig also has an effect on the small intestine. For example, soybean meal decreased villous height, deformed the villi shape, and increased lamina propria depth as a result of less enterocyte maturation on the villi, and the presence of antigenic materials and other antinutritional factors (Dunsford et al., 1989; Li et al., 1991).

The stress associated with post-weaning lag can be prevented with good nutritional management using highly digestible and palatable ingredients, with proper

consideration given to nutrient levels and feeding methods, that aim to increase feed intake for faster development of digestive enzymes (van Heugten, 1997). Carbohydrate sources containing 20 to 25% lactose, such as dried whey and lactose, and protein sources, such as fish meal, skim milk, soy protein concentrate and plasma protein, can be used in weanling pig diets, along with other additives like zinc oxide, copper sulfate, synthetic amino acids, acidifiers, and other growth-promoting additives (van Heugten, 1997). Among these ingredients, the use of spray-dried animal plasma (SDAP) has been used more frequently in weanling pig diets. A review by van Dijk et al. (2001) reported that SDAP at 6% in the diet increased the average daily gain, average daily feed intake and improves feed efficiency. A similar extensive review by Coffey and Cromwell (2001), using around 7% of SDAP in 79 experiments involving more than 8,000 weaned pigs, reported an average improvement in growth rate and feed intake of 25% and 21%, respectively, and improvement in feed efficiency by 4%.

Definition and response of antibiotic usage

Antimicrobial agents have been used in swine diets to improve overall health, growth and performance, and carcass quality. As defined by Cromwell (2001), antimicrobial agents are substances that kill or suppress the growth of bacteria, which include antibiotics and chemotherapeutics. He further defined antibiotics as substances produced by living organisms, such as yeast and molds, while chemotherapeutics are substances that are chemically synthesized. These antimicrobial agents are administered via injection, feed, and water, and used as therapeutics, prophylactics, and as growth promoters (USDA, 1999). Therapeutic use of antibiotics is administered at dosage levels sufficient to treat, control, or prevent clinical disease of bacteria origin, while

prophylactic application is the use of small, subtherapeutic doses that prevent or limit the occurrence of bacterial disease, which leads to growth promotion (Animal Health Institute, 2005).

Along with the improvement in the growth and health of pigs, antibiotics have been more cost effective due to the decrease in price to \$20 to \$40 per kg from a high of \$200 to \$220 per kg (Cromwell, 2001). With a total feed cost for all U.S. hogs of about \$5 billion in 1999, a 1.25% improvement in feed efficiency would save the swine industry approximately \$63 million in feed costs (Mathews, 2001). In young pigs, the use of antibiotics as compared to non-inclusion not only improved daily gain and feed conversion ratio by 26% and 10%, respectively, but it also reduced mortality from 4.3% to 2.0%, with a more pronounced effect on farms with high-disease level (15.6% to 3.1%). These changes translate to a net-return per pig of \$1.51 (Cromwell, 2002). For grow-finish pigs, a conservative improvement in daily gain and feed efficiency would result in a net return per pig of \$1.48 (Cromwell, 2002). Another report estimated the breakeven production cost of using subtherapeutic antibiotics of \$44.52/ 100 lb gain compared to \$42.36/ 100 lb gain in non-usage for a difference of \$2.16/ 100 lb gain or \$5.39 per 250 lb market weight of pig (Holden et al., 2002).

Antibiotic mechanism on growth and development of resistance

Antibiotics treat, prevent and maintain overall health of the animal by its action with the bacteria either thru 1) interference with cell wall synthesis, 2) interference with peptide initiation and/or elongation, 3) interference with DNA replication, or 4) interference with the folic acid synthesis pathway (Garold et al., 1973; Plumb, 1995; Prescott et al., 2000). As growth promotants, antibiotics exert their effects by 1)

hindering sub-clinical infections, 2) lessening growth-depressing microbial metabolites, 3) lessening microbial use of nutrients, and 4) increasing uptake and use of nutrients (Francois, 1962; Visek, 1978; Anderson et al., 1999; Gaskins et al., 2002). The disease control, metabolic, and nutritional effect of antimicrobial growth promoters affects not only the bacteria/pathogens but the animal as well. The effects of antibiotics in the gut of the animal are shown in Figure 2.3.



Figure 2.3. Diagram of the proposed effects of antibiotics mediated through their effects on small intestinal microflora (Anderson et al., 1999).

Despite the advantages of using antibiotics for growth promotion, there is a strong

pressure from consumers and the government to ban the use due to fear of food-

producing animals developing antibiotic resistance that can be transferred to humans.

Antibiotic resistance, as defined by the Animal Health Institute (2005), is the ability of

microorganisms, such as bacteria, to withstand antibiotic treatment due to selective pressure. This selective pressure causes the development, acquisition, and spread of the resistance gene or factor itself or by specific biochemical mechanism of the resistance gene or factor (USDA, 1999). Moreover, Bach Knudsen (2001) reported that antibiotic growth promoters also exert a selective pressure on the commensal microorganisms since antibiotics are weakly absorbed in the gastrointestinal tract. Thus, the bacteria develop reduced susceptibility, where it become less susceptible to a particular antibiotic that can lead to the development of resistance, and a reduction or elimination in the effectiveness of that antibiotic to both animal and human medicine (Yan and Gilbert, 2004).

There are many mechanisms in the development of resistance with subtherapeutic use of antibiotics. Hawkey (1998) classified these mechanisms into four basic types: 1) modification of the antibiotic, 2) prevention of antibiotic from penetrating the cell wall, 3) production of an alternative target, and 4) alterations in the primary site (Figure 2.4). The first three mechanisms prevent the antibiotic (e.g. β -lactam antimicrobials, aminoglycosides and chloramphenicol) from having an effect on the bacteria due to drug inactivation, which involves hyperproduction of an enzyme that is unaffected by the antibiotic action (McManus, 1997). Antibiotic resistant bacteria also may alter their structure by natural selection, random mutation, and DNA swapping through transduction, transformation and transposition, which inhibit the action of antibiotics not only affects the bacteria or pathogen, but it also affects the entire microflora of the gastrointestinal tract creating an imbalance and can lead to the development of resistance.

This resistance can be transferred potentially to animals and humans as well (van den Bogaard and Stobberingh, 1999).



Figure 2.4. Four major biochemical mechanisms of antibiotic resistance (Hawkey, 1998).

Alternatives to antibiotics – mechanism and studies

Alternatives have been developed and used to replace antibiotic growth promoters with the objective of mimicking the effects of antibiotics in growth promotion, by altering the proportions of specific gut bacterial species and limiting the numbers of unfavorable bacteria while promoting the colonization of more favorable species (Verstegen and Williams, 2002; Mathew, 2002). Moreover, Hardy (2003) proposed three distinct approaches to improve animal performance with the use of alternative products. These approaches are: 1) providing the optimum conditions for digestive functions by supporting the intestinal environment with the nutrients available to the animal, 2) manipulating the microbial population directly, and 3) enhancing the immune system with the use of supplements.

Modifying gut acidity with acidifiers

Aside from the direct effect on digestive enzyme activities (Mathew, 2002), maintaining an optimum acidic pH in the gut is one of the important defenses against intestinal colonization by harmful bacteria (Dinsmore et al., 1997). This may be attained with the use of dietary acidifiers (e.g. organic and inorganic acids), which have been used primarily in weanling pig diets. With the reduction of pH in the stomach, both the gastric proteolysis and nutrient digestibility would increase and the beneficial bacteria would proliferate against the pathogens, thus, making the acidifiers exert some antimicrobial activities like that of antibiotics (Close, 2000). The acids, in particular the organic acids, have the ability to change to the dissociated form from its undissociated form (Partanen and Mroz, 1999). The dissociated form of the organic acid is responsible for the modification of the pH in the gut, while the undissociated form of the organic acid can penetrate the bacterial cell wall leading to the disruption of cellular DNA formation and protein synthesis (Hardy, 2003). Thus, organic acids may improve growth and performance by reducing microbial competition with the pig for nutrients, by lowering the occurrence of subclinical infections, by lowering the intestinal immune response, and by reducing the production of destructive microbial metabolites (Dibner and Buttin, 2002).

Several studies have been performed to evaluate the antimicrobial and growth performance effects of acidifiers, mainly organic acids, in the diets of pigs. Partanen and Mroz (1999) summarized the response of dietary organic acids in weaned piglets (Table

2.1). Although no differences were found among the different kinds of organic acids, supplementation of dietary organic acids increased average daily bodyweight gain and improved the feed:gain ratio as compared to the non-acidified control diet.

Table 2.1. Response	of weaned pig	lets to dietary	organic acids	(adapted f	irom
Partanen and Mroz,	1999).				

	No. of	Range of acid level		
Organic acid	Exp.	(mequiv/kg)	ADG ^a	Feed:gain ^a
Formic acid and formates	11	46-444	0.269	-0.721
Fumaric acid	15	86-431	0.409	-0.899
Citric acid	9	78-391	0.255	-0.829

^a P < 0.04, probability that acidified diets differ from non-acidified control diet.

A study performed by De Rodas et al. (1995) reported that using a blend of organic and inorganic acids at around 3 kg/ton improved daily gains in pigs by 27% during the first two wk after weaning. The addition of 1% citric acid also improved daily gain and feed conversion ratio in weanling pigs (Burnell et al., 1988). In another study,

supplementation of 1% to 3% fumaric acid to starter diets during the first 3 to 4 wk after weaning improved the apparent ileal amino acid digestibilities by 4.9% to 12.8% (Blank et al., 1999). In various studies, the inclusion of organic acids in the diets of pigs reduced the coliform incidence in the gastrointestinal tract, scouring, and piglet mortality (Cole et al., 1968; Bolduan et al., 1988; Thomlinson and Lawrence, 1981). However, some studies showed negligible effects of adding organic acids on bacterial infection. A study done by Risley et al. (1992) using 1.5% fumaric or citric acid did not change the intestinal bacterial populations in piglets. This variability may be due to the age of pigs, amount of milk by-products in the diet, and the presence or absence of antibiotics (Holden et al., 2002). Hardy (2003) further added that the inconsistent results can be attributed to the differences in levels and types of organic acids, the acid buffering capacity of the dietary ingredients, and the ability of bacteria to develop an acid resistance (Hardy, 2003).

Probiotics and competitive exclusion

The gastrointestinal microflora can be modified to improve the health and performance of the piglets by minimizing the adverse effects of pathogenic bacteria through the increase in the number of favorable organisms in the gut (Hardy, 2002). This alteration of gut microflora can be achieved with the use of probiotics or direct-fed microbials, which are live microorganisms added to animal feed to restore the balance of microflora in favor of the beneficial microorganisms (Fuller, 1989; Cromwell, 2001). Some of the major bacterial organisms used as probiotics are Lactobacilli spp., Streptococci spp., Bacillus spp., Bifidobacteria spp., and yeasts (Hardy, 2002). Numerous probiotic preparations and cultures are available in the market but to be effective in improving the performance of the animal. Collins and Gibson (1999) stated that probiotics should 1) exert a beneficial effect on the host, 2) be nonpathogenic and nontoxic, 3) contain a large number of viable cells, 4) be capable of surviving and metabolizing in the gut, 4) remain viable during storage and use, 5) have good sensory properties, and 6) be isolated from the same species as its intended host. Thus, probiotics modify the intestinal microflora by competing against pathogenic bacteria for nutrients in the gut, by producing compounds that are toxic to pathogens, and by competing with pathogens for binding sites on the intestinal wall (Hentges, 1992).

The application of probiotics has been widely used in humans and its gaining considerable interest in food-producing animals. Cromwell (2001) summarized the effects of probiotics and antimicrobial agents in the diets of weanling pigs (Table 2.2).

Although the addition of promicrobials did not improve growth performance as compared to the addition of antimicrobials, it has an additive effect when combined with antibiotics.

		, ,		
	None	Promicrobials ^b	Antimicrobials ^b	Both
Daily gain, g ^c	247	237	306	310
Daily feed, g ^c	467	460	540	550
Feed:gain, g ^c	1.92	1.96	1.77	1.75

Table 2.2. Effects of promicrobial and antimicrobial agents in starting diets for weanling pigs^a (adapted from Cromwell, 2001).

^a A summary of five experiments involving 764 pigs weaned at 4 weeks of age (7.4 kg BW); 4-wk test period.

^b Promicrobials were various combinations of *Saccharomyces cerevisiae*, *Lactobacillus acidophilus*, and *Streptococcus faecium*. Antimicrobials were a mixture of chlortetracycline, penicillin, and sulfamethazine.

^c Effect of antimicrobials (P < 0.001).

However, a study by Fedorka-Cray et al. (1999) showed piglets fed mucosal competitive exclusion cultures reduced the incidence of Salmonella compared to control pigs (28% vs 79%). In a separate study, the administration of a competitive exclusion culture to neonatal pigs reduced mortality and the incidence of fecal shedding and gut colonization of *E. coli* as compared to control pigs (Genovese et al., 2000). However, the improvement in animal performance with the use of probiotics is variable. Young pigs that were given either a low or high dose of *Lactobacillus acidophilus* L23 improved average daily gain compared to a control group without significant differences in feed efficiency (Lee et al., 2001). Kyriakis et al. (1999) evaluated the effect of feeding a probiotic to newly-weaned piglets and demonstrated that at a level of 10^7 viable spores of *Bacillus licheniformis*, the incidence and severity of diarrhea was reduced along with a reduction in mortality. Furthermore, the piglets fed the probiotic had improved growth rate and feed efficiency compared to control piglets. But a study done by De Cuper et

al. (1992) reported that supplementation of *Bacillus cereus*, *Lactobacillus* spp., and *Streptococcus faecium* did not prevent mortality and clinical symptoms in young pigs infected with *E. coli*. Average daily gain, feed intakes, and feed efficiency also were not improved in pigs fed a *Lactobacillus acidophilus* culture (Harper et al., 1983). The variability in results may be due to the viability of microbial cultures, strain differences, dosage level and frequency of feeding, and medicine interactions (Holden et al., 2002).

Mannan oligosaccharides (MOS) as a performance enhancer alternative

Addition of MOS in nursery pig diets has been proven to improve animal performance by two mechanisms. First, MOS prevent bacterial colonization in the gut by binding to bacterial cell walls (Spring et al., 2000). This hinders the bacteria from attaching to the epithelial cells of the intestines and the bacteria attached to MOS are washed out (Spring et al., 2000; Pettigrew, 2000). The second mechanism involves the enhancement of the immune system by increasing immunoglobulin levels resulting in a reduction in mortality (Newman and Newman, 2001; O'Quinn et al., 2001; Pettigrew, 2000).

Pettigrew (2000) reviewed the effects of a commercial mannan oligosaccharide (Bio-Mos) in 17 comparisons involving 13 experiments and they are summarized in Table 2.3. There were numerical gains in 14 out of the 17 comparisons and an overall improvement in ADG and feed efficiency by 4.4% and 1.47%, respectively. In other studies, addition of MOS in early-weaned pigs improved growth approximately one-half of that obtained with high inclusions of copper (Davis et al., 1999; Cromwell, 2001), whereas there was no response of MOS addition in weanling pig diets (Davis et al., 2000; Cromwell, 2001). In more recent studies, weanling pigs fed diets with 0.20% Bio-Mos

had improved growth performance, but this response was dependent on copper sulfate (Davis et al., 2002) or zinc oxide levels in the diet (LeMieux et al., 2003). Dietary supplementation of mannan oligosaccharides also modulated the immune function with an increase in IgG levels (White et al., 2002) and a decrease in the percentage of neutrophils (Davis et al., 2004).

	sponses e		os in starter	uicus it	n pigs	(adapted)		ingi en	, 2000).
Authors,	Year	Reps	Bio-Mos	AI	DG	%	Feed	/gain	%
description		_	level (%)	C ^a	$\mathbf{B}^{\mathbf{b}}$	Diff ^c	C ^a	B^{b}	Diff ^c
van der Beke	1997	12	0.2	243	261	7.33	1.90	1.80	-5.26
Dvorak & Jacques	1998	4	0.2	309	341	10.30 ^d	1.30	1.26	-3.08
Kumprecht & Zoba	1999	3	0.2	NA	NA	8.50			
LeMieux et al.,									
High Zn	1999	5	0.2-0.3	307	318	3.48^{f}	1.45	1.46	0.34
LeMieux et al.,									
Low Zn	1999	5	0.2-0.3	262	291	11.04	1.48	1.46	-1.35
Stockland, Trial 1	1999	4	0.1	243	258	6.17	1.18	1.20	1.69
Stockland, Trial 2	1999	5	0.1-0.4	163	189	16.07	1.40	1.25	-11.07
Stockland, Trial 3	1999	6	varied	418	427	2.18 ^d	1.23	1.27	2.85
Stockland, Trial 4	1999	6	0.2/0.1	452	439	-2.68	1.24	1.31	5.65
Brendemuhl and									
Harvey	1999	4	0.1-0.2	639	649	1.56	1.75	1.69	-3.43
Davis et al.	1999	18	0.2	402	427	6.30 ^e	1.49	1.41	-5.37 ^e
Harper & Estienne,									
No antibiotic	2000	5	0.3/0.2	445	450	1.03	1.73	1.72	-0.58
Harper & Estienne,									
Mecadox	2000	5	0.3/0.2	490	490	0.00	1.72	1.69	-1.74
Maxwell et al.,									
Low Zn	1999	9	0.3/0.2	406	423	4.23	1.47	1.43	-2.72
Maxwell et al.,									
High Zn	1999	9	0.3/0.2	446	439	-1.59	1.41	1.39	-1.42
Maxwell et al.,									
Low Zn	2000	6	0.2-0.3	413	406	-1.62	1.32	1.37	3.79 ^g
Maxwell et al.,									
High Zn	2000	6	0.2-0.3	427	437	2.44	1.33	1.31	-1.88
	total	112	Means	379	390	4.40	1.46	1.44	-1.47

Table 2.3. Growth responses to Bio-Mos in starter diets for pigs (adapted from Pettigrew. 2000).

^a Control.

^b Bio-Mos.

^c Bio-Mos minus control.

^d Statistical significance level, P < 0.01. ^e Statistical significance level, P < 0.04.

^f Bio-Mos by zinc level interaction, P < 0.07.

^g Quadratic effect of Bio-Mos level, P < 0.01.

Beta-glucan as an immunomodulator

Because piglets at the time of weaning experience great stress in addition to having a reduced digestive capacity and underdeveloped immune system, supplementation of immune modulators could enhance the immune system of the piglets. This effect of immunomodulators on the immune function of the weanling pig, which is exposed to various stressors and pathogenic organisms, could decrease the susceptibility to diseases and reduce economic loss (Blecha and Charley, 1990). One of the immunomodulators that can be fed to young pigs is beta-glucan.

Beta-glucan is a polysaccharide made up of 1,3- and 1,6-glucose linkages that is present in some plants (oat and barley bran), fungi (Lentinus edodes), mushrooms (Grifola frondosa), and from cell walls of brewers' and bakers' yeast (Saccharomyces spp.) (Baur and Geisler, 1996; Brown and Gordon, 2003; Tokunaka et al., 2000; Bacon et al., 1969; Borek, 2001). As an immunostimulatory substance, beta-glucan acts through the macrophage and eventually produces interleukin-1 thus, enhancing both innate and adaptive immune responses of the animal (Hardy, 2003). In vitro studies have been performed using beta-glucan from Grifola frondosa to determine the stimulatory effect on macrophages (Adachi et al., 1994; Okazaki et al., 1995). Results from these studies showed that beta-glucan stimulated the macrophages and produced cytokines (e.g. interleukin-1, interleukin-6 and tumor necrosis factor alpha). Beta-glucans also can induce the release of nitric oxide. A study performed by Jung et al. (2004) looked at the effects of *Saccharomyces cerevisiae* beta-glucan in increasing the production of nitric oxide and interferon- γ in piglets infected with swine influenza virus. They found that the concentrations of nitric oxide and interferon- γ were significantly higher in infected
neonatal pigs supplemented with beta-glucan than the piglets that were infected with the virus but were unsupplemented with beta-glucan. These effects of beta-glucan as immunomodulators are affected by their degree of branching, polymer lengths, and tertiary structures (Brown and Gordon, 2003), where only those that consist of (1-3)-linked beta-glucan backbone with (1-6)-linked beta-D-glucopyranosyl units as branches would have the immunomodulating capacity (Bohn and BeMiller, 1995).

Several experiments have evaluated the effects of beta-glucans on growth performance and overall health of pigs. Dritz et al. (1995) reported that weanling pigs fed with 0.025% beta-glucan had an increase in average daily gain and feed intake. In another study, young pigs raised from sows treated with beta-glucan had a higher weight gain compared to the control group due to the increase in antibody titer levels in the sow's milk (Decuypere et al., 1998). Piglets that received beta-glucan, with *Lentinus edodes* as the source, had an increase in villous length and had a lower bacterial load as a result of lower turnover rates of the intestinal epithelial cells (van Nevel et al., 2003). However, Hiss and Sauerwein (2003) reported in their study that the inclusion of betaglucan in the diets of piglets did not influence average daily gain and feed efficiency and lymphocyte proliferation indices also were not significantly different from the control group. In finishing pigs, beta-glucan supplementation in the diet did not improve growth performance or carcass quality (Fortin et al., 2003).

Non-nutritional strategies and alternative husbandry practices

Although the use of antibiotic growth promoters and its alternatives improve the performance and health of animals, better feeding strategies, optimum management, and environmental controls must also be addressed, especially with the possibility of an

antibiotic ban. Provision of good quality water, maintenance of ambient temperature, and implementation of strict biosecurity and hygiene practices can be implemented on commercial farms (Hardy, 2002), as well as effective feeding practices, proper animal flow (all-in, all-out), and timely vaccination programs would all contribute to improvements in animal health and well being (Mathew, 2002; Doyle, 2001). Genetic improvements can also be done by changing the capability of the animal to initiate an immune response to infection (Holden et al., 2002). Furthermore, reliance on antibiotic growth promotants can be reduced by appropriate maintenance of ventilation rate and stockings rates, and careful record keeping (Doyle, 2001). Implementation of these strategies would result in more-profitable swine farming for the producer and safe, healthy food-products for the consumer.

Summary

In the event of the ban on the use of antibiotics as growth promoters, the use of alternative growth promotants will be of great consideration as a replacement to maintain and further improve growth performance and overall health of animals. However, current alternatives that have been studied resulted in inconsistent results. Thus, the following experiments were conducted to evaluate the effects of beta-glucan and other alternatives compared with a standard antibiotic on growth performance and immunity of weanling pigs.

CHAPTER III

Experiment 1

Effects of beta-glucan as an alternative to antibiotics in the diets of weanling pigs.

Introduction

Beta-glucan is commonly derived from the cell wall of baker's yeast, *Saccharomyces cerevisiae*. As an immunomodulator, it helps boost the immune system by stimulating a cascade of pathways that enhance both innate and adaptive immune responses (Hardy, 2003). An active immune system would help the animal fight disease challenges, help control clinical infection, and maintain growth processes.

Several studies have been performed with weanling pigs using beta-glucan but results have been variable on growth and immune parameters. This may be due to differences in the composition and concentration of beta-glucan. Thus, the objective of this experiment was to determine the optimum dose of beta-glucan and its effect on growth performance and immune response of weanling pigs.

Materials and Methods

One-hundred seventy-six pigs (average initial BW = 5.8 kg) were weaned at approximately 21 d and housed (5-6 pigs/pen) in a temperature-controlled nursery rooms for 42 d. Pigs were blocked by weight and randomly allotted to four dietary treatments (8 pens/trt). Cornstarch was replaced, as needed, by carbadox (Mecadox®, Pfizer Animal Health, New York, NY), or beta-glucan (Dong-Ahm BT, Seoul, South Korea) to provide

the four dietary treatments within each phase as follows: 1) negative control (NC), 2) NC with 0.25% carbadox, 3) NC with 0.20% beta-glucan and 4) NC with 0.40% beta-glucan. All diets were corn-soybean meal-based (Table 3.1) and fed in meal form. Pigs were fed in three dietary phases. Phase 1 diets (1.60% tLys) were fed from d 0 to d 14 and contained dried whey, lactose, spray-dried animal plasma, and fish meal. Phase 2 diets (1.40% tLys) were fed from d 14 to d 28 and contained dried whey, spray-dried blood meal and fish meal. Phase 3 diets (1.20% tLys) were fed from d 28 to 42 and were simple corn-soybean meal diets. Feed and water were provided on an ad libitum basis throughout the experiment. Pigs and feeders were weighed on d 0, 7, 14, 21, 28, 35, and 42 to determine average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F ratio).

Blood collection and analysis - Blood was collected from two randomly selected pigs (one male and one female) per pen. Blood samples were taken via jugular venipuncture on d 14, 28, and 42 using vacutainer tubes with anticoagulant for hematology, or without anticoagulant for C-reactive protein (CRP) determination. Hematology (white blood cells [WBC], lymphocytes, and neutrophils) was performed within an hour after collection using colorimetric procedures (ABX Pentra). The tubes without anticoagulant were centrifuged and serum was frozen until CRP determination. C-reactive protein was determined using a colorimetric method (Alfa Wasserman Clinical Analyzer). *Statistical Analysis* – All data were analyzed as a randomized complete block design using analysis of variance procedures as described by Steel and Torrie (1997). The model included the effects of replication, treatment, and replication x treatment (error).

Treatment means were separated using Least Significant Difference. The pen served as the experimental unit.

Results

For Phase 1, there were no differences (P > 0.10) in ADG, ADFI, and G:F ratio of pigs fed diets containing carbadox or 0.20% beta-glucan compared to pigs fed the negative control diet (Table 3.2). However, pigs fed diets with 0.40% beta-glucan gained slower (P < 0.04) and consumed less (P < 0.04) feed as compared with pigs fed carbadox. Moreover, pigs fed 0.40% beta-glucan tended to have decreased ADG (P < 0.09) and ADFI (P < 0.04) as compared with pigs fed either 0.20% beta-glucan or the negative control diet. Although G:F did not differ (P > 0.10) among the dietary treatments, pigs fed 0.40% beta-glucan had the poorest feed efficiency. In Phase 2, pigs fed diets with carbadox tended to have greater ADG (P < 0.10) than pigs fed either 0.40% beta-glucan or the negative control diet. The growth performance response of pigs fed the diet with 0.20% beta-glucan was intermediate between pigs fed either carbadox or the negative control diet.

For Phases 1 and 2 combined, pigs fed diets with 0.20% beta-glucan had similar (P > 0.10) ADG, ADFI, and G:F as those pigs fed either carbadox or the negative control diet. Incorporation of 0.40% beta-glucan in the diet of weanling pigs resulted in lower (P < 0.02) ADG and ADFI as compared with pigs fed carbadox. Pigs supplemented with 0.20% beta-glucan tended to have greater ADG (P < 0.08) and greater ADFI (P < 0.03) than pigs fed diets with 0.40% beta-glucan. Numerically, pigs fed carbadox had the greatest growth performance. The growth response of pigs fed 0.20% beta-glucan was intermediate to that of pigs fed the negative control diet and those fed carbadox.

There were no differences (P > 0.10) in ADG, ADFI and G:F ratio during Phase 3 for all treatment groups. Overall (in all three phases), there were no differences (P > 0.10) in ADG, ADFI and G:F of pigs fed diets containing carbadox or 0.20% beta-glucan. Pigs fed diets with 0.40% beta-glucan had lower (P < 0.05) ADG compared with pigs fed diets containing carbadox although they did not differ (P > 0.10) in G:F. At the same time, growth performance was not different (P > 0.10) for pigs fed 0.20% beta-glucan compared with pigs fed the negative control diet. Although not statistically significant, ADG, ADFI, and G:F were greater for pigs fed carbadox than for pigs fed 0.20% betaglucan or the negative control diet. In addition, the response of pigs to 0.20% betaglucan was intermediate to that of pigs fed carbadox or the negative control diet.

Table 3.3 shows the hematology values and CRP levels, with some differences noted among treatment groups. Pigs fed the negative control diet or those with carbadox had lower (P < 0.10) WBC on d 14 and 28. However, WBC was increased (P < 0.10) with beta-glucan supplementation on d 14. By d 28, WBC was greater (P < 0.10) for pigs fed beta-glucan compared to pigs fed carbadox. No differences (P > 0.10) were noted on d 42. Lymphocyte count on d 14 tended to follow a similar trend as WBC. Pigs fed beta-glucan tended to have higher (P < 0.10) lymphocytes on d 14, followed by carbadox and then pigs fed the negative control diet. There were no differences (P > 0.10) in lymphocytes on d 28 and 42. Neutrophils followed a similar trend in that pigs fed 0.40% beta-glucan tended to have the greatest (P < 0.10) neutrophils. The levels of CRP were lower (P < 0.10) on d 14 for pigs fed diets containing carbadox or 0.20% beta-glucan compared to those in the negative control group and 0.40% beta-glucan. There were no differences in CRP on d 14 and 28 and CRP tended to increase with age.

Discussion

Schoenherr et al. (1994) performed an experiment evaluating the effects of feeding different levels of beta-glucan on growth performance of weanling pigs (19 d of age). They found no improvements in growth parameters among the different levels of beta-glucan (0, 0.025, 0.05, 0.075, 0.10, and 0.125% beta-glucan) during the first 2 wk postweaning, but on d 34 after weaning, beta-glucan improved (P < 0.01) overall ADG (0.498, 0.548, 0.528, 0.538, 0.507, and 0.495 kg/d, respectively). With these results, they concluded that to maximize the growth performance of pigs in the nursery period, the optimum inclusion level of beta-glucan in diets was between 0.025% and 0.05%. In another study, Dritz et al. (1995) evaluated the influence of dietary beta-glucan on growth performance of weanling pigs. Pigs (14-21 d of age) were fed diets with different levels of beta-glucan (0, 0.025, 0.05%, and 0.10% beta-glucan). They reported that the addition of 0.10% beta-glucan decreased growth performance of weanling pigs during the first 7 d post-weaning. However, they found that 0.025% inclusion of beta-glucan increased ADG of pigs at d 28 after weaning due to increased feed intake. The addition of 0.05%beta-glucan also increased ADG of pigs but was lower as compared to 0.025% betaglucan. Thus, they suggested that the optimum inclusion level of beta-glucan to improve growth performance of weanling pigs was at 0.025% of the diet.

The present experiment used beta-glucan at 0.20% inclusion level and the ADG of weanling pigs was not decreased, although it was at the 0.40% inclusion level. This could be attributed to a lower feed intake exhibited by pigs fed 0.40% beta-glucan as compared to pigs fed 0.20% beta-glucan. Moreover, since beta-glucan is being recognized by the pig's immune system as foreign, it generated an immune response,

which is energetically costly (Demas et al, 1997). In their study, Demas et al. (1997) reported that mice injected with an antigen had higher (P < 0.05) O₂ consumption, than mice injected with saline, and the authors suggested that the increase in O_2 consumption indicates increased metabolic heat production, which could have affected feed intake. There were minimal differences in growth parameters of pigs fed with either the negative control diet, carbadox treated diet, or 0.20% beta-glucan treated diet, except in Phase 2. However, ADG and G:F were consistently higher in pigs fed diets with carbadox than pigs fed diets with either 0.20% beta-glucan or the negative control diet. Moreover, the ADG of pigs fed 0.20% beta-glucan was intermediate between the pigs fed carbadox and negative control diets. In all three phases, ADG was improved by 4.9% for pigs treated with carbadox. This response was much lower than the average improvement in growth performance attributed to antibiotic supplementation to weanling pig diets (4.9% vs 16.4%) as reported by Cromwell (2001). However, the response to antibiotic in research facilities is often much less than that observed in commercial facilities (16.9% vs 28.4% for ADG; 7.0% vs 14.5% for feed/gain) due to differences in disease level (Cromwell, 2001). The average improvement in ADG due to beta-glucan was 1.8%, which was about half the response of carbadox.

Hardy (2003) stated that beta-glucan stimulates the macrophages to produce cytokines that activate lymphocytes. As constituents of white blood cells, lymphocytes and neutrophils function in the innate and acquired immunity of pigs by producing inflammatory response mediators, phagocytosis and destruction of bacteria (Johnson et al., 2001) and their numbers are increased during immune reaction to stress and disease conditions. This was demonstrated in this experiment, where pigs fed diets with 0.40%

beta-glucan had the highest values of WBC and lymphocytes on d 14, 28 and 42 and neutrophils on d 14. However, in a study by Dritz et al. (1995), the authors reported that neither the addition of 0.025% nor 0.05% beta-glucan influenced the neutrophils count or macrophage function. The differential response in WBC in our study as compared to that of Dritz et al. (1995) could be attributed to differences in the dosage levels of beta-glucan that were used.

C-reactive protein (CRP) is one of the major acute phase proteins in swine and its concentration is increased rapidly in response to infection, inflammation, or trauma (Chen et al., 2003; Eckersall et al., 1996). Moreover, CRP levels are increased in porcine serum within the first 5 days after challenge but subsequently decreased to normal levels thereafter (Heegaard et al., 1998; Eckersall et al., 1996). In this experiment, CRP levels were the lowest for pigs fed either carbadox or 0.20% beta-glucan diets, implying that those pigs were healthier than pigs fed either 0.40% beta-glucan diet or the negative control diet. However, CRP levels in pigs might be more important as a stress indicator (Burger et al., 1998) rather than health status (Burger et al., 1992). In a similar study by Dritz et al. (1995), they determined the influence of adding two levels of beta-glucan on the concentration of plasma haptoglobin, which is another major acute phase protein in swine. They reported that pigs fed either 0.025% or 0.05% beta-glucan had lower plasma haptoglobin concentrations, which translated to greater ADG, than pigs fed the control diets on d 14. In this experiment, pigs fed either 0.20% beta-glucan or carbadox had lower CRP levels and higher ADG than pigs fed either 0.40% beta-glucan or the negative control diet, which agreed with the results of Dritz et al. (1995).

Conclusions

The inclusion of 0.20% beta-glucan in the diets of weanling pigs appears to have a growth performance response that is intermediate to that of carbadox inclusion or feeding a negative control diet. Moreover, the addition of beta-glucan in the diet increased WBC, lymphocyte and neutrophil count, and decreased concentrations of CRP as compared to non-addition. However, the addition of 0.40% beta-glucan depressed growth performance of pigs.

Ingredient, %	Phase 1	Phase 2	Phase 3
Corn grain	28.23	48.79	56.58
Soybean meal (48% CP)	22.91	28.15	34.86
Whey, dried	20.00	10.00	0
Lactose	10.00	0	0
Plasma, spray-dried	6.00	0	0
Blood cells, spray dried	0	2.50	0
Fish meal, menhaden	5.00	2.50	0
Soybean oil	5.00	5.00	5.00
DL-methionine	0.22	0.06	0.00
Dicalcium phosphate	0.60	1.12	1.33
Limestone, ground	0.87	0.80	0.90
Salt	0.35	0.25	0.50
Trace mineral mix ^a	0.15	0.15	0.15
Vitamin mix ^b	0.25	0.25	0.25
Ethoxyquin	0.03	0.03	0.03
Cornstarch ^c	0.04	0.04	0.04
Calculated Analysis:			
ME, kcal/kg	3,506	3,534	3,549
Lysine, %	1.60	1.40	1.20
Ca, %	0.95	0.85	0.75
P, %	0.75	0.70	0.65

Table 3.1. Composition of diets in Experiment 1 (as fed-basis).

^a Supplied per kg of diet: 16.5 mg of Cu (copper sulfate); 165 mg of Fe (ferrous sulfate); 0.30 mg of I (calcium iodate); 40 mg of Mn (manganese oxide); 0.30 mg of Se (sodium selenite); and 165 mg of Zn (zinc oxide).

^b Supplied per kg of diet: 11,013 IU of vitamin A; 1,652 IU of vitamin D₃; 44 IU of vitamin E; 4.4 mg of vitamin K (menadione activity); 55 mg of niacin; 10 mg of riboflavin; 33 mg of pantothenic acid (D-calcium pantothenate); and 44 μ g of vitamin B₁₂.

^c Cornstarch (CS) was replaced by carbadox or beta-glucan, as needed, to provide the treatments within each phase as follows: 1) negative control (0.40% CS), 2) 0.25% carbadox^d + 0.15% CS, 3) 0.20% beta-glucan + 0.20% CS, and 4) 0.40% beta-glucan.

^d Provided 55 mg of carbadox per kg of complete feed.

	Treatment ^b							
Item	NC	AB	0.20% BG	0.40% BG	SE			
Number of pigs	44	44	44	44				
Initial weight, kg	5.83	5.82	5.81	5.82	0.03			
Final weight, kg	19.54 ^{cd}	20.47 ^c	20.00 ^{cd}	19.38 ^d	0.37			
Phase 1								
ADG, kg	0.172 ^c	0.177 ^c	0.176 ^c	0.150 ^d	0.01			
ADFI, kg	0.275 ^c	0.274 ^c	0.278 ^c	0.241 ^d	0.01			
G:F	0.631	0.652	0.623	0.622	0.03			
Phase 2								
ADG, kg	0.360 ^d	0.392 ^c	0.375 ^{cd}	0.357 ^d	0.01			
ADFI, kg	0.520 ^{cd}	0.556 ^c	0.550 ^{cd}	0.516 ^d	0.02			
G:F	0.686	0.701	0.681	0.697	0.02			
Phases 1 & 2								
ADG, kg	0.266 ^{cd}	0.285 ^c	0.275 ^c	0.252 ^d	0.01			
ADFI, kg	0.393 ^{cd}	0.411 ^c	0.407 ^c	0.371 ^d	0.01			
G:F	0.673	0.689	0.671	0.679	0.01			
Phase 3								
ADG, kg	0.455	0.462	0.456	0.450	0.02			
ADFI, kg	0.790	0.772	0.782	0.772	0.02			
G:F	0.578	0.602	0.587	0.587	0.02			
Overall								
ADG, kg	0.327 ^{cd}	0.343 ^c	0.333 ^{cd}	0.316 ^d	0.01			
ADFI, kg	0.526	0.534	0.532	0.505	0.01			
G:F	0.620	0.645	0.625	0.627	0.01			

Table 3.2. Growth performance of weanling pigs (Exp. 1)^a.

^a Least square means for 8 pens (5-6 pigs/pen) per treatment. ^b NC = negative control, AB = NC + carbadox^e, 0.20% or 0.40% BG = NC + 0.20% or 0.40% beta-glucan.

^{c,d} Within a row, means without a common superscript letter differ (P < 0.10).

^e Provided 55 mg of carbadox per kg of complete feed.

Item	NC	AB	0.20% BG	0.40% BG	SE
Number of pigs	16	16	16	16	
$WBC^{c}, 10^{3}/mm^{3}$					
d 14	16.17 ^d	15.61 ^d	17.01 ^{de}	19.11 ^e	1.17
d 28	14.36 ^{de}	12.94 ^d	15.59 ^e	15.73 ^e	0.87
d 42	17.73	17.41	19.66	19.94	1.21
Lymphocytes ^c , absolute					
d 14	8.59 ^d	9.34 ^{de}	9.73 ^{de}	10.56 ^e	0.55
d 28	8.08	8.55	9.67	8.92	0.76
d 42	12.39	12.51	13.41	12.45	0.95
Neutrophils ^c , absolute					
d 14	6.71 ^{de}	5.53 ^d	6.41 ^{de}	7.52 ^e	0.73
d 28	5.55 ^e	3.72 ^d	5.16 ^e	6.01 ^e	0.40
d 42	4.31 ^d	3.90 ^d	5.06 ^d	6.29 ^e	0.49
C-reactive protein, mg/dL					
d 14	7.89 ^d	4.75 ^e	3.44 ^e	5.82 ^d	1.01
d 28	6.33	6.87	6.05	6.76	0.59
d 42	8.78	8.44	8.62	9.50	0.58

Table 3.3. Hematology and serum CRP of weanling pigs (Exp. 1)^a.

^a Least square means for 8 pens (2 pigs/pen) per treatment. ^b NC = negative control, AB = NC + carbadox^f, 0.20% or 0.40% BG = NC + 0.20% or 0.40% beta-glucan.

^c Normal ranges: WBC: 11-22 x 10³/mm³; Lymphocytes:6-10; Neutrophils: 4.0-7.5 (CCAC, 1993).

^{d,e} Within a row, means without a common superscript letter differ (P < 0.10).

^f Provided 55 mg of carbadox per kg of complete feed.

CHAPTER IV

Experiment 2

Effects of beta-glucan and antibiotics on growth performance and carcass traits of weanling and finishing pigs

Introduction

Results observed in Experiment 1 suggested that growth performance of weanling pigs fed 0.20% beta-glucan was intermediate to that of pigs fed carbadox or a negative control diet. In addition, carbadox addition consistently resulted in the greatest improvement in growth performance among the dietary treatments used. However, inclusion of 0.40% beta-glucan reduced growth performance of pigs as compared to pigs fed the negative control diet. Previous studies also indicated that higher inclusion levels of beta-glucan may depress growth performance. Yet, results of Experiment 1 suggested that beta-glucan can be added in the diet up 0.20% without reduction in growth performance.

A study performed by Fortin et al. (2003) evaluated the effects of beta-glucan supplementation to the diet on the growth performance of growing-finishing pigs. These authors reported that beta-glucan did not improve growth performance or carcass quality. Moreover, several studies have been performed to evaluate the effect of combining antimicrobial agents with alternatives on growth performance of weanling and finishing pigs. Thus, this experiment was performed to determine the effects of beta-glucan and antibiotics on growth performance and carcass traits of weanling and finishing pigs, and

also to determine if an interaction exists between the inclusion of beta-glucan and antibiotic in the diet.

Materials and Methods

A total of one-hundred forty pigs (average initial BW = 5.4 kg) was weaned at approximately 20 d of age for the nursery study. Pigs were blocked by weight and allotted randomly to four dietary treatments (6 pens/trt) in a 2 x 2 factorial design with two levels of carbadox supplementation (0 vs 0.25%) and two levels of beta-glucan (0 vs 0.20%). All diets were corn-soybean meal-based (Table 4.1) and fed in meal form. Pigs were fed in three dietary phases. Phase 1 diets (1.60% tLys) were fed from d 0 to d 14 and contained dried whey, lactose, spray-dried animal plasma, and fish meal. Phase 2 diets (1.40% tLys) were fed from d 14 to d 28 and contained dried whey, spray-dried blood meal, and fish meal. Phase 3 diets (1.20% tLys) were fed from d 28 to 42 and were simple corn-soybean meal diets. Cornstarch was replaced, as needed, by carbadox (Mecadox[®], Pfizer Animal Health, New York, NY), or beta-glucan (Dong-Ahm BT, Seoul, South Korea) to provide the four dietary treatments. Pigs were housed (5-6 pigs/pen) in a temperature-controlled room and were allowed to have ad libitum access to feed and water throughout the experiment. Pigs and feeders were weighed on d 0, 7, 14, 21, 28, 35, and 42 to determine average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F ratio).

Following the nursery phase, the experiment was continued to the growingfinishing phase. Pigs were fed one week of their respective diets before being transferred to the finisher room. Four pigs from each nursery pen were randomly chosen, allotted to pens, and fed the same four dietary treatments in a 2 x 2 factorial design. In the finisher

phase, carbadox was replaced by chlortetracycline (0.10%). Dietary treatments (Table 4.1) were also fed in three dietary phases (Phase 1, initial weight in the finisher to 50 kg; Phase 2, 50 kg to 77 kg; Phase 3, 77 kg to 105 kg BW) and contained 1.05%, 0.90% and 0.75% tLys, respectively. All diets were corn-soybean meal-based and fed in meal form. Pigs and feeders were weighed every two weeks to determine rate and efficiency of gain until the pigs reached approximately 105 kg. At 105 kg, two pigs per pen were transported to a commercial packing plant, humanely slaughtered, and the carcasses were split along the dorsal midline, weighed and placed in a chiller overnight. The following day, carcasses were ribbed and 10th rib fat depth and longissimus muscle area (LMA) were measured. Using these measures, the percentage of lean in the carcass was calculated according to NPPC (2001).

Statistical Analysis – Data were analyzed as a 2 x 2 factorial arrangement in a randomized complete block design using procedures described by Steel and Torrie (1997). The main effects of antibiotic, beta-glucan and their interaction were tested using orthogonal contrasts. The pen served as the experimental unit.

Results

Nursery phase – There were no interactions (P > 0.10) between carbadox or beta-glucan inclusion in the nursery phase (Table 4.2). In Phase 1, carbadox inclusion improved (P < 0.04) ADG, but had no effect on ADFI and G:F, whereas the inclusion of beta-glucan tended to improve (P < 0.07) G:F. For Phase 2, the inclusion of carbadox improved ADG, and G:F (P < 0.02), and ADFI (P < 0.07). On the other hand, beta-glucan inclusion tended to improve (P < 0.10) G:F of pigs. For Phases 1 and 2 combined, beta-glucan and carbadox inclusion improved (P < 0.04) G:F. The ADG of pigs was increased

(P < 0.01) with carbadox addition to the diets, and inclusion of beta -glucan tended to improve (P < 0.10) ADG of pigs, as well. Furthermore, pigs fed carbadox consumed more (P < 0.03) feed. In Phase 3, ADG, ADFI, and G:F of pigs were not improved (P >(0.10) with addition of carbadox in the diet. However, beta-glucan tended to increase (P < 0.10) ADG of pigs although no improvements (P > 0.10) were seen in ADFI and G:F. Overall, carbadox and beta-glucan increased (P < 0.05) ADG of pigs. In addition, carbadox increased (P < 0.05) feed intake of pigs. However, the inclusion of either betaglucan or carbadox in the diet had no effect (P > 0.10) on G:F of weanling pigs. Addition of either beta-glucan or carbadox increased (P < 0.04) the final weights of the pigs. *Finisher phase* – During the finisher phase, there were no interactions (P > 0.10) between chlortetracycline and beta-glucan except on overall ADG (P < 0.06) (Table 4.3). In Phase 1, inclusion of chlortetracycline improved (P < 0.03) G:F of pigs while betaglucan tended to improve (P < 0.07) G:F. However, neither the addition of beta-glucan nor chlortetracycline improved (P > 0.10) ADG or ADFI of pigs. For Phase 2, there was no effect (P > 0.10) of adding beta-glucan or chlortetracycline on growth performance of pigs. In Phase 3, beta-glucan inclusion in the diets of pigs tended to increase (P < 0.09) ADG and ADFI but did not improve G:F. Overall, growth parameters were not affected by the inclusion of either beta-glucan or chlortetracycline in the diet. However, pigs fed beta-glucan numerically had the greatest ADG, followed by pigs fed carbadox, and then pigs fed the combination of beta-glucan and carbadox. The G:F was also the highest in pigs fed beta-glucan, followed by pigs fed the combination of beta-glucan and carbadox, and then pigs fed carbadox. The inclusion of beta-glucan in the diets tended to improve (P < 0.08) the final weight of finishing pigs while the inclusion of chlortetracycline in the

diet had no effect (P > 0.10) on the final weight. Numerically, pigs fed the combination of beta-glucan and carbadox had the highest market weight (110.51 kg), followed by pigs fed beta-glucan (109.30 kg), and then pigs fed carbadox (108.01 kg).

Overall and carcass traits – For the entire growing period (Table 4.4; nursery to finishing), the inclusion of beta-glucan increased (P < 0.04) ADG of pigs while antibiotic addition had no effect (P > 0.10) on ADG, ADFI, and G:F of pigs. For the carcass traits (Table 4.4), the inclusion of antibiotics in the diet tended to reduce (P < 0.06) 10th rib backfat, although the combination of beta-glucan and carbadox produced the thinnest 10th rib backfat (2.12 cm). The LMA of pigs was not affected with either beta-glucan or carbadox inclusion in the diet, although pigs fed the combination of beta-glucan and carbadox had the greatest LMA (44.16 cm²). Fat-free lean percentage also followed a similar trend as 10th rib backfat. Antibiotic addition improved (P < 0.05) the percentage fat-free lean but the combination of beta-glucan and carbadox had the highest fat-free lean percentage (52.4%). Overall, the inclusion of beta-glucan in the diet (alone or in combination with antibiotic) minimally affected (P > 0.10) the carcass traits measured.

Discussion

Based on the results of the first experiment, the addition of 0.40% beta-glucan in diets of weanling pigs was not as effective as the inclusion of 0.20% beta-glucan; thus, 0.20% beta-glucan was used in this experiment. Several studies have been performed to evaluate the effects of antimicrobial agents and alternatives on growth performance of weanling and growing-finishing pigs. One such alternative is mannan oligosaccharides (MOS).

Mannan oligosaccharides act as a growth promoter by preventing bacterial colonization in the gut and by enhancing the immune system (Pettigrew, 2000). Harper and Estienne (2000) evaluated the effect of carbadox (0 vs 55 ppm) and MOS (0 vs 0.30% for wk 1 and 0.20% for wk 2-5) as growth promoters for weanling pigs (24 d of age). They found no interactions of feeding the two additives for the duration of the trial. Mannan oligosaccharides inclusion also did not influence growth performance for the entire study. However, the addition of carbadox improved growth rate and increased feed intake by wk 3. In another study by LeMieux et al. (2003), the effects of MOS (0 vs (0.20%) and supplemental zinc oxide (0 vs 3,000 ppm) on growth performance of weanling pigs were inconsistent. In a 3-wk study, the addition of MOS improved overall ADG and G:F of pigs without zinc oxide (ZnO), but these responses were decreased with added ZnO in the diet. In 4-wk study (Exp. 1 and 2), inclusion of ZnO increased ADG and ADFI. The addition of MOS decreased ADG and G:F, but these responses were increased when ZnO was included in the diet. A study by Davis et al. (2002) determined the effect of MOS (0 vs 0.20%) and copper sulfate (0 vs 175 and 125 ppm) on growth performance of weanling and finishing pigs. In the 38 d nursery study, pigs fed either supplemental copper sulfate or MOS had greater overall ADG and G:F than pigs fed a basal diet without supplemental copper sulfate or MOS. In the grow-finish stage, addition of supplemental copper sulfate increased overall ADG, ADFI, and G:F, while MOS addition only improved overall ADG. However, growth performance of pigs was reduced when MOS was added to diet containing 125 ppm supplemental copper sulfate.

It has been documented that the addition of antibiotics in nursery pig diets improves growth performance and reduce mortality and morbidity (Cromwell, 2002).

Results of present experiment revealed that pigs fed nursery diets containing either carbadox or beta-glucan generally had higher weight gain and better overall feed efficiency than those pigs fed the negative control diet. These results were again similar with previous studies (Dritz et al., 1995; Schoenherr et al., 1994), where pigs fed either 0.025% or 0.05% beta-glucan had greater growth performance response than pigs fed the control diet without beta-glucan. Between the two additives, pigs fed carbadox had a greater response than pigs fed beta-glucan, as has been shown in MOS studies. Similarly, in the two experiments that LeMieux et al. (2003) conducted, weanling pigs fed diets supplemented with ZnO had the highest overall ADG as compared to pigs fed 0.20% MOS (366 vs 308 in Exp. 1; 365 vs 308 in Exp. 2). However, pigs fed carbadox and beta-glucan combined numerically had the greatest overall ADG and G:F among the dietary treatments used in our study.

There were only minimal improvements on growth performance associated with chlortetracycline or beta-glucan supplementation to the diets of pigs in the growing-finishing stage. Addition of beta-glucan only influenced growth performance of pigs in Phase 3. The response was also similar between chlortetracycline and beta-glucan. Davis et al. (2002) reported that pigs fed ZnO had higher ADG and G:F than pigs fed MOS or a combination of MOS and ZnO (1.100, 1.067, and 1.045 kg/d, respectively; 0.344, 0.329, and 0.328, respectively). Furthermore, Fortin et al. (2003) reported that the inclusion of beta-glucans from oats at different levels (1.6% to 4.1%) did not improve the growth performance of pigs as compared to the control diet.

The addition of beta-glucan did not influence the carcass traits of pigs. Similarly, Fortin et al. (2003) reported that pigs fed a control diet, or 1.6% or 4.1% beta-glucan had

similar fat thickness and longissimus muscle area. On the other hand, inclusions of antibiotic reduced 10th rib fat and increased fat-free lean percentage. Stahly et al. (1996) hypothesized that the addition of carbadox in the diet improves muscle growth due to the production of a growth-promoting factor and/or the reduction of cytokines and other inhibitory factors. They further reported that the inclusion of carbadox in diets of grow-finish pigs resulted in higher weight gain and better feed efficiency, lower backfat thickness, and greater loin eye area as compared to non-inclusion. These findings agree with the results of our study, where pigs fed chlortetracycline had lower backfat and higher fat-free lean percentage than pigs fed the negative control diet (2.20 cm vs 2.37 cm backfat; 52.1% vs 51.0% fat-free lean).

Conclusions

Overall, the addition of either beta-glucan or carbadox in the diet improved the growth performance response of weanling and growing-finishing pigs. Beta-glucan slightly improved ADG in all phases of nursery and finisher stages as compared with the other dietary treatments. No significant interactions were found in combining beta-glucan and carbadox in the diet.

<u> </u>	Nursery ^d			Í	Finisher ^{ef}			
	Phase	Phase	Phase	Phase	Phase	Phase		
Ingredient, %	1	2	3	1	2	3		
Corn grain	28.17	48.74	56.53	65.18	70.86	76.49		
Soybean meal (48% CP)	22.91	28.15	34.86	29.16	23.70	18.25		
Whey, dried	20.00	10.00						
Lactose	10.00	0						
Plasma, spray-dried	6.00	0						
Blood cells, spray dried	0	2.50						
Fish menhaden meal	5.00	2.50						
Soybean oil	5.00	5.00	5.00	3.00	3.00	3.00		
DL-methionine	0.22	0.06						
Dicalcium phosphate	0.60	1.12	1.33	0.85	0.89	0.65		
Limestone, ground	0.87	0.80	0.90	0.96	0.70	0.76		
Salt	0.35	0.25	0.50	0.25	0.25	0.25		
Trace mineral mix ^a	0.15	0.15	0.15	0.15	0.15	0.15		
Vitamin mix ^{bc}	0.25	0.25	0.25	0.15	0.15	0.15		
Ethoxyquin	0.03	0.03	0.03					
Cornstarch ^d	0.45	0.45	0.45	0.30	0.30	0.30		
Calculated Analysis:								
ME, kcal/kg	3,506	3,534	3,550	3,479	3,488	3,497		
Lysine, %	1.60	1.40	1.20	1.05	0.90	0.75		
Ca, %	0.95	0.85	0.75	0.65	0.55	0.50		
P, %	0.75	0.70	0.65	0.55	0.50	0.45		
Analyzed Composition:								
Crude Protein (as fed), %	23.38	22.29	20.83	19.04	17.67	12.75		

Table 4.1. Composition of diets in Experiment 2 (as fed-basis).

^a Supplied per kg of diet: 16.5 mg of Cu (copper sulfate); 165 mg of Fe (ferrous sulfate), 0.30 mg of I (calcium iodate); 40 mg of Mn (manganese oxide); 0.30 mg of Se (sodium selenite); and 165 mg of Zn (zinc oxide).

^b Supplied per kg of diet in the nursery phase: 11,013 IU of vitamin A; 1,652 IU of vitamin D₃; 44 IU of vitamin E; 4.4 mg of vitamin K (menadione activity); 55 mg of niacin; 10 mg of riboflavin; 33 mg of pantothenic acid (D-calcium pantothenate); and 44 μ g of vitamin B₁₂.

^c Supplied per kg of diet in the finisher phase: 6,608 IU of vitamin A; 991 IU of vitamin D₃; 26.4 IU of vitamin E; 2.6 mg of vitamin K (menadione activity); 33 mg of niacin; 6 mg of riboflavin; 20 mg of pantothenic acid (D-calcium pantothenate); and 26.4 μ g of vitamin B₁₂.

^d Within each nursery phase, cornstarch (CS) was replaced with either 0.20% beta-glucan or 0.25% antibiotic to provide the dietary treatments: 1) 0.45% CS, 2) 0.25% carbadox^g + 0.20% CS, 3) 0.20% beta-glucan + 0.25% CS, and 4) 0.20% beta-glucan plus 0.25% carbadox.

^e Finisher Phase 1 was from the initial weight in the finisher to 50 kg; Phase 2 was from 50 to 77 kg; and Phase 3 was from 77 kg to market weight (105 kg).

^f Dietary treatments during the grower-finisher phase were similar to the nursery phase with the exception that chlortetracycline^g (0.10%) was used in place of carbadox

^g Provided 55 mg of carbadox per kg of complete feed in the nursery phase and 110 mg of chlortetracycline per kg of complete feed in the finisher phase.

	Treatment							
		beta-glu	ucan, %					
	0	0	0.2	0.2			P < :	
		carba	adox ^b			AB ^c	BG^d	ABxBG
Item	_	+	-	+	SE	effect	effect	
Number of pigs	35	35	35	35				
Initial weight, kg	5.38	5.36	5.42	5.38	0.02	0.33	0.19	0.65
Final weight, kg	19.23	19.91	19.6	20.56	0.23	0.01	0.04	0.56
Phase 1								
ADG, kg	0.145	0.154	0.149	0.169	0.01	0.04	0.2	0.46
ADFI, kg	0.218	0.233	0.221	0.233	0.01	0.13	0.86	0.86
G:F	0.665	0.666	0.674	0.724	0.02	0.17	0.07	0.19
Phase 2								
ADG, kg	0.343	0.385	0.361	0.387	0.01	0.01	0.35	0.47
ADFI, kg	0.509	0.548	0.528	0.53	0.01	0.07	0.96	0.11
G:F	0.673	0.697	0.687	0.733	0.01	0.02	0.1	0.44
Phases 1 & 2								
ADG, kg	0.244	0.27	0.255	0.278	0.01	0.01	0.1	0.78
ADFI, kg	0.363	0.391	0.374	0.381	0.01	0.03	0.91	0.19
G:F	0.672	0.69	0.685	0.73	0.01	0.02	0.04	0.27
Phase 3								
ADG, kg	0.501	0.5	0.503	0.528	0.01	0.2	0.1	0.14
ADFI, kg	0.803	0.826	0.819	0.846	0.02	0.14	0.28	0.9
G:F	0.624	0.605	0.617	0.626	0.01	0.64	0.54	0.24
Overall								
ADG, kg	0.33	0.346	0.338	0.361	0.01	0.01	0.05	0.55
ADFI, kg	0.51	0.536	0.522	0.536	0.01	0.05	0.48	0.52
G:F	0.646	0.646	0.65	0.675	0.01	0.22	0.13	0.22

Table 4.2. Growth performance of pigs during the nursery phase (Exp. 2)^a.

^a Least square means for 6 pens (5-6 pigs/pen) per treatment.

^b Provided 55 mg of carbadox per kg of complete feed.

^c Antibiotic

^dBeta-glucan

		Treatment						
		beta-glucan, %						
	0	0 0 0.20 0.20			_		P < :	
		chlortetra	acycline ^b		_	AB^d	BG ^e	ABxBG
Item	-	+	-	+	SE	effect	effect	
Initial weight, kg	22.11	23.668	22.97	24.52	0.34	0.01	0.02	0.98
Final weight, kg	102.67	108.01	109.30	110.51	2.43	0.20	0.08	0.41
Phase 1 ^c								
ADG, kg	0.724	0.726	0.726	0.724	0.02	0.99	0.99	0.92
ADFI, kg	1.573	1.630	1.512	1.585	0.06	0.28	0.38	0.90
G:F	0.461	0.446	0.481	0.457	0.01	0.03	0.07	0.60
Phase 2 ^c								
ADG, kg	0.742	0.807	0.803	0.823	0.04	0.25	0.29	0.54
ADFI, kg	2.114	2.265	2.211	2.347	0.10	0.19	0.40	0.94
G:F	0.351	0.357	0.361	0.353	0.01	0.86	0.74	0.46
Phase 3 ^c								
ADG, kg	0.751	0.803	0.849	0.813	0.03	0.79	0.08	0.15
ADFI, kg	2.549	2.641	2.796	2.684	0.08	0.90	0.09	0.22
G:F	0.295	0.303	0.303	0.305	0.01	0.56	0.57	0.70
Overall								
ADG, kg	0.738	0.776	0.794	0.768	0.02	0.73	0.15	0.06
ADFI, kg	2.199	2.210	2.219	2.222	0.06	0.46	0.38	0.49
G:F	0.349	0.351	0.358	0.353	0.01	0.85	0.34	0.57

Table 4.3. Growth performance of pigs during the growing-finishing phase $(Exp. 2)^{a}$.

^a Least square means for 6 pens (4 pigs/pen) per treatment.

^b Provided 110 mg of chlortetracycline per kg of complete feed.
^c Finisher Phase 1 was from the initial weight in the finisher to 50 kg; Phase 2 was from 50 to 77 kg; and Phase was from 77 kg to market weight (105 kg).

^d Antibiotic

^eBeta-glucan

	Treatment				_			
		beta-glucan, %						
	0	0	0.20	0.20	_		P < :	
		antibiotic ^b				AB ^c	BG^{d}	ABxBG
Item	-	+	-	+	SE	effect	effect	
Initial weight, kg	5.38	5.36	5.42	5.38	0.02	0.33	0.19	0.65
Final weight, kg	102.67	108.01	109.30	110.51	2.43	0.20	0.08	0.41
ADG, kg	0.652	0.689	0.711	0.705	0.02	0.36	0.04	0.22
ADFI, kg	1.609	1.681	1.710	1.698	0.04	0.49	0.19	0.33
G:F	0.406	0.410	0.415	0.416	0.01	0.61	0.15	0.72
Carcass								
10th rib fat, cm	2.37	2.20	2.58	2.12	0.14	0.06	0.65	0.33
LMA, cm^2	43.24	43.84	43.63	44.16	1.04	0.62	0.74	0.98
Fat-free lean, %	51.0	52.1	50.1	52.4	0.71	0.05	0.69	0.45

Table 4.4. Growth performance and carcass traits of pigs for the overall experiment (Exp. 2)^a.

^a Least square means for 6 pens (4-6 pigs/pen) per treatment.

^b Provided 55 mg of carbadox per kg of completed feed in the nursery phase and 110 mg of chlortetracycline per kg of complete feed in the finisher phase

^c Antibiotic

^dBeta-glucan

CHAPTER V

Experiment 3

Effects of beta-glucan, antibiotic, and acidifier on growth performance of weanling pigs

Introduction

Based on the results of the two previous experiments, the addition of beta-glucan improved growth and performance of pigs. However, overall performance was better with the inclusion of antibiotics. The addition of beta-glucan in the diet containing antibiotic numerically improved growth performance of weanling and growing-finishing pigs.

Acidifiers (inorganic and organic acids) have been used in weanling pig diets not only as a feed preservative but more so as an antimicrobial alternative (Mathew, 2002). Acidifiers lower the pH of the gut and disrupt the cellular mechanisms of the pathogenic bacteria thus favoring the proliferation of beneficial bacteria. Thus, this experiment was performed to determine the synergistic effect of combining beta-glucan and a commercial acidifier (Kem-Gest[™]) on growth and performance of pigs and comparing this effect with a standard antibiotic.

Materials and Methods

Two-hundred sixty pigs (average initial BW = 5.6 kg) were weaned at approximately 20 d of age. Pigs were blocked by weight and randomly allotted to five

dietary treatments (8 pens/trt). The five dietary treatments consisted of 1) negative control (NC), 2) NC with 0.25% carbadox (AB), 3) NC with 0.20% beta-glucan, 4) NC with 0.20% acidifier, and 5) NC with 0.20% beta-glucan and 0.20% acidifier. Carbadox (Mecadox[®]) was provided by Pfizer Animal Health (New York, NY), beta-glucan was provided by Dong-Ahm BT (Seoul, South Korea) and the acidifier was composed of inorganic (phosphoric acid) and organic acids (fumaric, lactic, and citric acid) and was provided by Kemin Industries (Des Moines, IA). All diets were corn-soybean mealbased (Table 5.1) and fed in meal form. Pigs were fed in three dietary phases. Phase 1 diets (1.60% tLys) were fed from d 0 to d 14 and contained dried whey, lactose, fish meal, and soy protein concentrate. Phase 2 diets (1.40% tLys) were fed from d 14 to d 28 and contained dried whey, fish meal, and soy-protein concentrate. Phase 3 diets (1.20% tLys) were fed from d 28 to 42 and were simple corn-soybean meal diet. Pigs were housed (5-8 pigs/pen) in a temperature-controlled room and were allowed to have ad libitum access to feed and water throughout the experiment. Pigs and feeders were weighed on d 0, 7, 14, 21, 28, 35, and 42 to determine average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F ratio).

Statistical Analysis – All data were analyzed as a randomized complete block design using analysis of variance procedures as described by Steel and Torrie (1997). The model included the effects of replication, treatment, and replication x treatment (error). Treatment means were separated using Least Significant Difference. The pen served as the experimental unit.

Results

In Phase 1, there were no differences (P > 0.10) in ADG, ADFI, and G:F of pigs fed carbadox, beta-glucan, or the negative control diet (Table 5.2). However, the addition of acidifier tended to decrease (P < 0.08) ADG as compared to pigs fed beta-glucan. Moreover, pigs fed acidifier tended to consume less feed (P < 0.07) than pigs fed the negative control diet. However, pigs fed acidifier had similar (P > 0.10) response as those pigs fed carbadox. Pigs fed the combination of beta-glucan and acidifier had similar (P > 0.10) growth performance response as those pigs fed beta-glucan, carbadox, and the negative control diet. However, the addition of beta-glucan in diets with acidifier tended to increase (P < 0.09) feed intake of pigs as compared to pigs fed the acidifier alone. There were no differences (P > 0.10) in feed efficiency among the dietary treatments, although pigs fed beta-glucan numerically had the highest G:F (0.790).

In Phase 2, pigs fed carbadox tended to have greater (P < 0.09) ADG than pigs fed the negative control diet, although the two dietary treatments had similar (P > 0.10) ADFI and G:F. Addition of beta-glucan to the diet suppressed (P < 0.04) feed intake as compared to pigs fed either carbadox or the negative control diet, but it did not affect (P >0.10) ADG. However, G:F of pigs fed beta-glucan tended to improve (P < 0.06) compared with pigs fed the negative control diet. In addition, pigs fed beta-glucan had similar (P > 0.10) ADG, ADFI, and G:F to that of pigs fed the acidifier. But the inclusion of acidifier reduced (P < 0.05) feed intake of pigs as compared to pigs fed the negative control diet. Combining both beta-glucan and acidifier in the diet resulted in similar (P > 0.10) ADG, ADFI, and G:F as compared to pigs fed carbadox, beta-glucan,

and acidifier. However, G:F of pigs fed beta-glucan and acidifier combined was improved (P < 0.04) as compared with pigs fed the negative control diet.

For Phases 1 and 2 combined, G:F of pigs fed carbadox was greater (P < 0.04) compared with pigs fed the negative control, although ADG and ADFI were similar (P > 0.10). Pigs fed beta-glucan had a similar (P > 0.10) response as those pigs fed carbadox. However, pigs fed beta-glucan had greater (P < 0.001) G:F than pigs fed the negative control diet. Addition of acidifier decreased ADG (P < 0.06) and ADFI (P < 0.05) of pigs as compared to those pigs fed carbadox. Furthermore, pigs fed acidifier had lower (P < 0.04) feed intake than pigs fed the negative control diet. Pigs fed beta-glucan tended to have greater weight gain (P < 0.10) and better feed efficiency (P < 0.03) than pigs fed acidifier. The combination of beta-glucan and acidifier resulted in similar (P > 0.10) ADG and ADFI of pigs as compared to the other dietary treatments. Feed efficiency of pigs fed beta-glucan and acidifier combined tended to be greater (P < 0.06) than pigs fed the negative control diet, but tended to be lower (P < 0.09) than pigs fed beta-glucan.

In Phase 3, there were no differences (P > 0.10) in ADG and ADFI of pigs fed carbadox, beta-glucan or the negative control diet. However, pigs fed carbadox had greater (P < 0.02) G:F than pigs fed the negative control diet. The inclusion of acidifier in the diet tended to lower (P < 0.10) ADG of pigs as compared to those pigs fed either beta-glucan or carbadox. However, ADFI and G:F of pigs fed acidifier were similar (P >0.10) as compared to pigs fed either beta-glucan, carbadox or the negative control diet. Pigs fed the combination of beta-glucan and acidifier had similar (P > 0.10) ADG and ADFI as compared to pigs fed either beta-glucan, carbadox, acidifier or the negative control diet. But G:F was lower (P < 0.002) in pigs fed beta-glucan and acidifier

combined as compared to pigs fed carbadox. Furthermore, pigs fed either beta-glucan or acidifier tended to have higher (P < 0.10) G:F than pigs fed the combination of beta-glucan and acidifier.

Overall, no differences (P > 0.10) in ADG and ADFI were observed in pigs fed either beta-glucan, carbadox or the negative control diet. However, pigs fed either betaglucan or carbadox had greater (P < 0.02) G:F than pigs fed the negative control diet. The addition of acidifier lowered (P < 0.04) ADG of pigs as compared to pigs fed either beta-glucan or carbadox. Moreover, pigs fed acidifier tended to consume less (P < 0.10) feed than pigs fed the negative control diet. However, G:F of pigs fed acidifier was similar (P > 0.10) to pigs fed either beta-glucan, carbadox or the negative control diet. Pigs fed the combination of beta-glucan and acidifier had similar (P > 0.10) ADG and ADFI to pigs fed either beta-glucan, carbadox, acidifier or the negative control diet. But the addition of beta-glucan to diets with acidifier decreased (P < 0.04) G:F of pigs as compared to pigs fed carbadox. In addition, pigs fed the combination of beta-glucan and acidifier tended to have lower (P < 0.07) G:F than pigs fed beta-glucan. The final weight was higher (P < 0.04) for pigs fed either beta-glucan or carbadox as compared to pigs fed acidifier alone.

Discussion

From previous experiments, it was shown that pigs fed beta-glucan had an intermediate growth performance response with pigs fed either carbadox or the negative control diet. But in some phases, the effect of beta-glucan was greater than carbadox. Several alternatives claim to have antimicrobial activity, such as acidifiers, that have

comparable results with antibiotic. Thus, this study compared the effect of combining beta-glucan and acidifier against antibiotic on growth performance of weanling pigs.

Partanen and Mroz (1999) reviewed several studies evaluating the effect of dietary organic acid addition to diets and they reported that for weaned piglets, fumaric and citric acid improved the average daily bodyweight gain and feed:gain ratio. Moreover, a study by De Rodas et al. (1995) evaluated the effect of diet acidification using a complex diet, in 20 to 26 d old pigs during a 6-wk study. Phase 1 diets were composed of corn-soybean meal diet with 5% plasma protein, 1.5% spray-dried porcine blood meal, 5% fishmeal and 20% dried whey; Phase 2 diets were composed of cornsoybean meal diet with 2% spray-dried porcine blood meal, 2.5% fishmeal and 5% dried whey; Phase 3 diets were simple corn-soybean meal diets. They reported improvements in daily gains of pigs by 27% and 25% when pigs were fed with a 0.35% mixture of organic and inorganic acids, and 2% fumaric acid, respectively, as compared to pigs fed the control diet. In addition, feed:gain ratio was also improved by 16% and 14% when pigs were fed the mixture of acidifiers and fumaric acid, respectively, as compared to pigs fed the control diet. An experiment was conducted by Walsh et al. (2004b) evaluating the effect of acidifiers on growth performance of nursery pigs (18 d of age). They compared diets using treatments with a basal diet (no antibiotics, no acidifiers), basal diet with 50 ppm carbadox, basal diet with 0.40% organic acid blend, basal diet with 0.20% inorganic acid based blend, and basal diet with the combination of both organic and inorganic acids (0.60%). They reported that the pigs fed carbadox had the highest ADG and G:F, although it was not significantly different from pigs fed either the

organic or inorganic acid blend. But pigs fed diets with the combination of organic and inorganic acid blend consumed the least amount of feed.

Similarly, in this experiment, the inclusion of acidifier resulted in the lowest growth performance among the dietary treatments. Although the inclusion level of acidifier used in this experiment was not as high as compared to the study by Walsh et al. (2004b) (0.20% vs 0.60%), the levels and types of acids used, such as fumaric acid, could have change the palatability of the diet, thus, suppressing feed intake and depressing growth performance of pigs. Moreover, the type of diets used could also have affected the growth performance response of feeding dietary organic acids (Partanen and Mroz, 1999). Burnell et al. (1988) reported in their study that addition of organic acid to cornsoybean meal diet containing 15% dried whey resulted in greater weight gain and feed intake of weanling pigs than in simple corn-soybean meal diet, although no differences were observed in feed/gain response. The study done by De Rodas et al. (1995) used only 20% and 5% dried whey in Phase 1 and Phase 2, respectively, while the study of Walsh et al. (2004b) used 25% and 15% dried whey in Phase 1 and Phase 2, respectively. In our study, 20% dried whey and 10% lactose were used in Phase 1 diet and 15% dried whey was used in Phase 2 diet. However, pigs fed acidifier generally had lower growth performance response as compared to pigs fed the other dietary treatments. These variable responses may be attributed to differences in dietary buffering capacity of ingredients (Bolduan et al., 1988; Partanen and Mroz, 1999), such as the source of proteins and minerals, which could affect the level of pH in the gut and efficiency of protein digestion (Blank et al., 1999), and eventually growth performance. Makkink et al. (1994) reported in their study that pigs fed skim milk powder had higher protein

breakdown than pigs fed soybean protein concentrate, although no difference was seen on digesta pH. This suggests that newly-weaned pigs lack the ability to digest vegetable protein sources compared to animal protein sources, such as milk by-products (Walsh et al, 2004a).

Growth performance of pigs fed carbadox was consistently higher than pigs fed acidifier. These results were also similar with the results by Walsh et al. (2004b) where carbadox treated pigs had the highest ADG and better feed efficiency than pigs fed either the acid blends or the combination. However, Virtanen et al. (2004) reported that a 0.65% acid blend (formic acid, Na formate, phosphoric acid, lactic acid, citric acid in a diatomaceous earth carrier) improved ADG and feed efficiency of weanling pigs (25 d of age) by 19% and 4%, respectively, which was almost similar with pigs fed diets with 0.40% avilamycin.

In Phase 1 and Phases 1 and 2 combined, pigs fed beta-glucan had the greatest G:F. The ADG and G:F of pigs fed the combination of beta-glucan and acidifier were lower than pigs fed either beta-glucan or carbadox in the same period. However, pigs had better growth performance when beta-glucan was added to diets with acidifier as compared to pigs fed the acidifier alone. Overall, pigs fed carbadox had the greatest ADG and G:F, followed by pigs fed beta-glucan. The inconsistent results from previous studies and the present experiment, with regard to acidifier, may be due to the existing levels of performance where conditions were still favorable for the normal growth of pigs (Ravindran and Kornegay, 1993).

Conclusions

The addition of acidifiers was reported in other studies to improve growth performance of pigs, although it was not shown in this experiment. Pigs fed acidifier had a reduction in feed intake, but this was alleviated when beta-glucan was added to the diet. Pigs fed beta-glucan had similar growth performance response as that of pigs fed carbadox. However, pigs fed beta-glucan had greater growth performance response than pigs fed either acidifier or the combination of beta-glucan and acidifier.

Ingredient, %	Phase 1	Phase 2	Phase 3
Corn grain	30.01	46.72	57.49
Soybean meal (48% CP)	15.46	20.19	35.13
Whey, dried	20.00	15.00	0
Lactose	10.00	0	0
Fish menhaden meal	6.00	6.00	0
Soy protein concentrate	12.00	6.00	0
L-lysine HCL	0.10	0	
DL-methionine	0.12	0.05	
Soybean oil	4.00	4.00	4.00
Dicalcium phosphate	0.45	0.21	1.04
Limestone, ground	0.65	0.62	0.93
Zinc oxide	0.28	0.28	
Copper sulfate			0.49
Salt	0.50	0.50	0.50
Trace mineral mix ^b	0.15	0.15	0.15
Vitamin mix ^c	0.25	0.25	0.25
Ethoxyquin	0.03	0.03	0.03
Calculated Analysis:			
ME, kcal/kg	3,506	3,534	3,550
Lysine, %	1.60	1.40	1.20
Ca, %	0.95	0.85	0.75
P, %	0.75	0.70	0.65
Analyzed Composition:			
Crude Protein (as fed), %	24.49	22.95	20.54

Table 5.1. Composition of diets in Experiment 3 (as fed-basis)^a.

^a Within each nursery phase, either 0.25% carbadox^d, 0.20% beta-glucan or 0.20% acidifier^e was added to the basal diet to provide the five dietary treatments: 1) basal diet (BD), 2) BD + 0.25% carbadox, 3) BD + 0.20% beta-glucan, 4) BD + 0.20% acidifier, and 5) BD + 0.20% beta-glucan + 0.20% acidifier.

^b Supplied per kg of diet: 16.5 mg of Cu (copper sulfate); 165 mg of Fe (ferrous sulfate), 0.30 mg of I (calcium iodate); 40 mg of Mn (manganese oxide); 0.30 mg of Se (sodium selenite); and 165 mg of Zn (zinc oxide).

^c Supplied per kg of diet: 11,013 IU of vitamin A; 1,652 IU of vitamin D₃; 44 IU of vitamin E; 4.4 mg of vitamin K (menadione activity); 55 mg of niacin; 10 mg of riboflavin; 33 mg of pantothenic acid (D-calcium pantothenate); and 44 μ g of vitamin B₁₂.

^d Provided 55 mg of carbadox per kg of complete feed.

^e Combination of inorganic (phosphoric acid) and organic acids (fumaric, lactic, and citric acid).

	Treatment ^b						
	NC	AB	0.20% BG	0.20% AC	0.20% BG	SE	
Item					+ 0.20% AC		
Number of pigs	52	52	52	52	52		
Initial weight, kg	5.58	5.59	5.58	5.58	5.56	0.02	
Final weight, kg	16.18 ^{cd}	16.90 ^c	16.80 ^c	15.74 ^d	16.40 ^{cd}	0.34	
Phase 1							
ADG, kg	0.170 ^{cd}	0.158 ^{cd}	0.172 ^c	0.147 ^d	0.169 ^{cd}	0.01	
ADFI, kg	0.225 ^c	0.213 ^{cd}	0.218 ^{cd}	0.197 ^d	0.224 ^c	0.01	
G:F	0.762	0.752	0.790	0.744	0.755	0.02	
Phase 2							
ADG, kg	0.320 ^d	0.353 ^c	0.331 ^{cd}	0.320 ^d	0.340 ^{cd}	0.01	
ADFI, kg	0.551 ^c	0.557 ^c	0.509 ^d	0.516 ^d	0.529 ^{cd}	0.01	
G:F	0.582^{d}	0.633 ^{cd}	0.646 ^c	0.620 ^{cd}	0.654 ^c	0.02	
Phases 1 & 2							
ADG, kg	0.245 ^{cd}	0.256 ^c	0.252 ^c	0.231 ^d	0.247 ^{cd}	0.01	
ADFI, kg	0.388 ^c	0.385 ^c	0.363 ^{cd}	0.353 ^d	0.372 ^{cd}	0.01	
G:F	0.633 ^e	0.666 ^{cd}	0.691 ^c	0.654^{de}	0.664 ^d	0.01	
Phase 3							
ADG, kg	0.267 ^{cd}	0.297 ^c	0.298 ^c	0.251 ^d	0.261 ^{cd}	0.02	
ADFI, kg	0.580	0.567	0.610	0.523	0.577	0.04	
G:F	0.466 ^{de}	0.525 ^c	0.493 ^{cd}	0.486 ^{cd}	0.448 ^e	0.02	
Overall							
ADG, kg	0.252 ^{cd}	0.269 ^c	0.267 ^c	0.238 ^d	0.251 ^{cd}	0.01	
ADFI, kg	0.452 ^c	0.445 ^{cd}	0.446 ^{cd}	0.408 ^d	0.439 ^{cd}	0.02	
G:F	0.563 ^d	0.604 ^c	0.600 ^c	0.585 ^{cd}	0.574 ^d	0.01	

Table 5.2. Growth performance of weanling pigs (Exp. 3)^a.

^a Least square means for 8 pens (5-8 pigs/pen) per treatment. ^b NC = negative control, AB = NC + carbadox^f, 0.20% BG = NC + 0.20% betaglucan, 0.20% AC = NC + 0.20% acidifier^g, 0.20% BG + 0.20% AC = NC + 0.20%beta-glucan + 0.20% acidifier.

^{c,d,e} Within a row, means without a common superscript letter differ (P < 0.10).

^f Provided 55 mg of carbadox per kg of complete feed.

^g Combination of inorganic (phosphoric acid) and organic acids (fumaric, lactic, and citric acid).
CHAPTER VI

Experiment 4

Effects of beta-glucan, antibiotic, and probiotic on growth performance of weanling pigs

Introduction

In the previous experiments, a synergistic effect of beta-glucan addition with the inclusion of an acidifier in the diet was not established in improving the growth and performance of weanling pigs. However, a consistent response to beta-glucan was shown on growth performance, and in some phases, it was even greater than that obtained with carbadox inclusion in the first three experiments. Thus, the possibility of further improving the performance of weanling pigs with the use of beta-glucan in combination with another antibiotic growth promotant alternative, such as probiotics, was investigated.

One of the probiotics or direct-fed microbial supplements is *Lactobacillus acidophilus*. A previous study performed by Lee et al. (2000), using a culture of *Lactobacillus acidophilus* L23, improved average daily gain of young pigs. Because probiotics may modify the intestinal microflora, this experiment was performed to determine the effects of *Lactobacillus acidophilus* L23 in combination with beta-glucan on growth performance.

Materials and Methods

A total of one-hundred pigs (average initial BW = 5.2 kg) was weaned at approximately 20 d of age. Pigs were blocked by weight and randomly allotted to five dietary treatments (4 pens/trt). The five dietary treatments consisted of: 1) negative control (NC), 2) NC with 0.25% carbadox (AB), 3) NC with 0.20% beta-glucan, 4) NC with probiotic, and 5) NC with 0.20% beta-glucan and probiotic. Carbadox (Mecadox®) was provided by Pfizer Animal Health (New York, NY), beta-glucan was provided by Dong-Ahm BT (Seoul, South Korea) and the probiotic was a dried culture of Lactobacillus acidophilus L23 prepared by Culture Systems, Inc. (Granger, IN). The probiotic culture was given to the pigs as a top-dress $(1 \times 10^8 \text{ cfu/pig})$ each morning in the nursery pen feeders. All diets were corn-soybean meal-based and fed in meal form (Table 6.1). Pigs were fed in three dietary phases. Phase 1 diets (1.60% tLys) were fed from d 0 to d 14 and contained dried whey, lactose, fish meal, and soy protein concentrate. Phase 2 diets (1.40% tLys) were fed from d 14 to d 28 and contained dried whey, fish meal, and soy-protein concentrate. Phase 3 diets (1.20% tLys) were fed from d 28 to 42 and were a simple corn-soybean meal diet. Pigs were housed (5 pigs/pen) in a temperature-controlled room and were allowed to have ad libitum access to feed and water throughout the experiment. Pigs and feeders were weighed on d 0, 7, 14, 21, 28, 35, and 42 to determine average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F ratio).

Blood collection and analysis - Blood was collected from three randomly selected pigs per pen. Blood samples were taken via jugular venipuncture on d 0, 28 and 42 for the same three pigs using vacutainer tubes without anticoagulant for serum protein

determination. The samples were centrifuged at approximately 1500 x g for 20 min and the serum was harvested and stored at -20°C. The serum samples were later thawed for 30 min at room temperature for serum protein quantification. Serum samples were pooled within pen and IgA and IgG were quantified using the Pig ELISA Quantitation kits (Bethyl Laboratories, Inc., Montgomery, TX). The serum proteins were then measured using the AD LD Analysis Software 1.6 and the AD 340 Absorbance Detector (Beckman Coulter, Inc., Fullerton, CA).

Statistical Analysis – All growth performance and blood data were analyzed as a randomized complete block design using analysis of variance procedures as described by Steel and Torrie (1997). The model included the effects of replication, treatment, and replication x treatment (error). Treatment means were separated using Least Significant Difference. The pen served as the experimental unit. For the serum proteins, the effects of treatment, replication, and day were analyzed along with appropriate interactions using the PROC MIXED procedure of SAS.

Results

In Phase 1, the ADG, ADFI, and G:F of pigs were similar (P > 0.10) between the negative control and carbadox diets (Table 6.2). Pigs fed beta-glucan also had a response similar (P > 0.10) to those pigs fed either carbadox or the negative control diet. The ADG and G:F of pigs fed diets top-dressed with probiotic did not differ (P > 0.10) as compared to those pigs fed beta-glucan, carbadox, or the negative control diet. However, pigs fed probiotic tended to have greater (P < 0.06) ADFI than pigs fed the negative control diet. Addition of probiotic to the diet with beta-glucan did not influence (P > 0.10) the response of pigs.

In Phase 2, pigs fed carbadox had similar (P > 0.10) ADG, ADFI, and G:F as pigs fed the negative control diet. The ADG, ADFI, and G:F also were similar (P > 0.10) among pigs fed either beta-glucan, carbadox, and the negative control diet. Pigs fed the diet top-dressed with probiotic had similar (P > 0.10) growth performance to those pigs fed either beta-glucan, carbadox, or the negative control diet. The same trend was also noted for the addition of probiotic to the diet with beta-glucan where ADG, ADFI, and G:F were similar (P > 0.10) among pigs fed beta-glucan, carbadox, or probiotic. However, pigs fed beta-glucan top-dressed with probiotic tended to have greater (P < 0.09) G:F than pigs fed the negative control diet.

In Phases 1 and 2 combined, pigs fed carbadox had similar (P > 0.10) ADG, ADFI, and G:F as those pigs fed the negative control diet. In addition, growth performance of pigs was similar (P > 0.10) among pigs fed either beta-glucan, carbadox, or the negative control diet. Addition of probiotic to the diet tended to improve (P < 0.10) ADG of pigs as compared to those pigs fed the negative control diet. Pigs fed betaglucan top-dressed with probiotic had similar (P > 0.10) ADG as compared to those pigs fed either the negative control, carbadox, beta-glucan, or probiotic. However, ADFI of pigs tended to be reduced (P < 0.10) when beta-glucan was added to the diet top-dressed with probiotic.

In Phase 3, pigs fed carbadox had greater (P < 0.04) ADG and G:F than pigs fed the negative control diet, although ADFI of pigs was similar (P > 0.10) between pigs fed carbadox and the negative control diet. The ADG and ADFI of pigs fed beta-glucan were similar (P > 0.10) to pigs fed either carbadox or the negative control. However, G:F of pigs fed beta-glucan tended to be lower (P < 0.08) than pigs fed carbadox. Moreover,

pigs fed beta-glucan had similar (P > 0.10) G:F compared with pigs fed the negative control diet. Pigs fed probiotic tended to have lower (P < 0.09) ADG than pigs fed carbadox, although ADFI and G:F of pigs fed probiotic were similar (P > 0.10) to pigs fed either beta-glucan, carbadox or the negative control diet. The ADG of pigs fed the combination of probiotic and beta-glucan was similar (P > 0.10) to those pigs fed either beta-glucan, carbadox, probiotic or the negative control diet. However, pigs fed betaglucan top-dressed with probiotic had lower (P < 0.05) ADFI than pigs fed carbadox or the negative control diet. The G:F was similar (P > 0.10) in pigs fed the combination of beta-glucan and probiotic as compared with those pigs fed carbadox. However, betaglucan and probiotic combined improved (P < 0.03) G:F of pigs as compared to those pigs fed either beta-glucan, probiotic or the negative control diet.

Overall, pigs fed carbadox had greater (P < 0.03) ADG and G:F compared with pigs fed the negative control diet. The ADG, ADFI, and G:F of pigs fed beta-glucan were similar (P > 0.10) to pigs fed either carbadox or the negative control diet. Pigs fed the probiotic tended to have greater (P < 0.07) ADG than pigs fed the negative control diet, although they were similar (P > 0.10) to pigs fed either beta-glucan or carbadox. The ADFI and G:F of pigs fed probiotic also were similar (P > 0.10) to pigs fed either beta-glucan, carbadox or the negative control diet. Pigs fed the combination of betaglucan and probiotic had similar (P > 0.10) ADG to pigs fed either beta-glucan, carbadox, probiotic or the negative control diet. However, ADFI was decreased (P < 0.05) in pigs fed beta-glucan and probiotic combined as compared to pigs fed either probiotic or the negative control diet. Moreover, pigs fed the combination of betaglucan and probiotic combined as compared to pigs fed either probiotic or the negative control diet. Moreover, pigs fed the combination of beta-glucan and probiotic or the negative control diet. Moreover, pigs fed the combination of beta-glucan and probiotic or the negative control diet. Moreover, pigs fed the combination of beta-glucan and probiotic or the negative control diet. Moreover, pigs fed the combination of beta-glucan. The G:F of pigs fed the combination of beta-glucan and probiotic was similar (P > 0.10) to pigs fed carbadox, although it was greater (P < 0.05) than pigs fed either beta-glucan, probiotic, or the negative control diet. The final weight of pigs tended to be higher (P < 0.09) in pigs fed carbadox than those pigs fed the negative control, although it was similar (P > 0.10) to pigs fed either beta-glucan, probiotic or the combination of betaglucan and probiotic.

A day effect was observed (P < 0.01) on immunoglobulin levels (Table 6.3) but there was no day by treatment effect (P > 0.10). For the serum proteins, IgA levels at d 0 was similar (P > 0.10) in pigs fed either the negative control diet, carbadox, beta-glucan, or beta-glucan and probiotic combined. However, pigs fed the probiotic tended to have higher (P < 0.07) initial IgA levels than pigs fed beta-glucan. On d 28 and 42, there were no differences (P > 0.10) in IgA levels of pigs fed the negative control diet, carbadox, beta-glucan or probiotic. However, pigs fed the combination of beta-glucan and probiotic tended to have increased (P < 0.10) IgA levels compared with pigs fed beta-glucan.

Initially, at d 0, IgG levels were similar (P > 0.10) in pigs fed either the negative control diet, carbadox, probiotic or the combination of beta-glucan and probiotic. However, pigs fed beta-glucan tended to have lower (P < 0.10) initial IgG levels than pigs fed either carbadox or the combination of beta-glucan and probiotic. On d 28, IgG levels were similar in pigs fed either the negative control, carbadox, probiotic, or betaglucan and probiotic combined. Still, beta-glucan addition to the diet tended to have reduced (P < 0.06) IgG levels than inclusion of probiotic. On d 42, pigs fed carbadox had similar (P > 0.10) IgG levels as those pigs fed beta-glucan. In the same period, IgG levels were increased (P < 0.01) with probiotic addition to the diet, as compared to the

negative control diet. Moreover, pigs fed either the negative control diet or beta-glucan and probiotic combined tended to have increased (P < 0.10) IgG levels as compared to pigs fed the negative control diet.

Discussion

Weanling pigs experience stress from their diets and new environment which could lead to a depression in growth performance. These stresses leading to the reduction in performance may be alleviated in young pigs with supplementation of probiotics. This was evidenced in this experiment, where pigs fed the probiotic culture, Lactobacillus acidophilus L23, had greater ADG and G:F than pigs fed the negative control diet. A study by Lee et al. (2001) used the same probiotic culture, with a different strain, preparation, and administration (orally administered with milk), in weanling pigs fed corn-soybean meal diet with low lactose whey protein concentrate (10% in Phase 1, none in Phase 2) and 3% soybean protein concentrate (both in Phase 1 and Phase 2). In their study, pigs that received a low or high dose of L. acidophilus L23 (3 x 10^8 lactobacilli/day vs 3 x 10⁹ lactobacilli/day) had higher ADG and better feed:gain ratio than pigs in the control group as a result of a possible increase in starch digestibility. But it should be noted that the probiotic culture used by Lee et al. (2001) was prepared using a different strain, method, and purpose, thus, further investigation could be done to determine the effect of that probiotic culture on growth performance of weanling pigs.

Pollmann et al. (1980) performed two experiments (4-wk study) to evaluate the effects of adding a probiotic product of lactobacillus origin to the diet of weanling pigs. In Exp. 1, weanling pigs (28 d old) were fed a complex corn-soybean meal diet with 10% dried whey and growth performance was compared using a commercial probiotic,

Probios (*L. acidophilus*), different kinds of antibiotics (ASP-250, tylosin or lincomycin) and a control diet. The authors reported that probiotic supplementation improved ADG and feed conversion ratio of pigs by 4.5% and 7.2%, respectively, as compared to non-supplementation. In Exp. 2, the probiotic was compared to lactic acid, virginiamycin, and another commercial probiotic from *Streptococcus faecium* (Feed-Mate 68). Pigs (average initial BW = 7 kg) were fed a less complex corn-soybean meal diet without dried whey. Results were more pronounced this time, with improvements in ADG by 9.7% and feed conversion ratio by 21.4%, as compared to pigs fed the control diet.

However, a study by Harper and Estienne (2002) reported that a probiotic culture from *Bacillus licheniformis* and *B. subtilis* (BioMate-2B, Chris Hansen Biosystems) fed to nursery pigs resulted in lower ADG, ADFI, and poorer feed conversion ratio. In their 5-wk study, pigs were fed a complex corn-soybean meal diet with dried whey and were compared to pigs fed either the control or carbadox. These authors reported that inclusion of the probiotic culture reduced ADG and ADFI of pigs by -5.2% and -2.6%, respectively, and increased feed conversion ratio by 2.5%, when compared to pigs fed the control diet. However, ADG and ADFI of pigs fed carbadox were improved by 7.9% and 5.0%, respectively, and feed conversion ratio was decreased by 3.1%.

In the present study, probiotic addition to the diet of pigs resulted in greater feed consumption than pigs fed either carbadox or the negative control diet, which resulted in comparable improvements in ADG and feed efficiency. However, ADG and G:F of pigs fed probiotic were improved by 6.14% and 7.51%, respectively, unlike in other probiotic studies, where growth performance was reduced. This was anticipated because probiotics

promote the colonization of beneficial microorganisms colonization in the gut, thus, inhibiting the growth of pathogenic bacteria (Abe et al., 1995).

As in the previous experiments, beta-glucan supplementation to the diet improved growth performance of pigs. The ADG and G:F of pigs were improved by 5.42% and 7.51%, respectively, with addition of beta-glucan in the diet as compared to non-addition. Although no synergistic effects were observed in combining beta-glucan with acidifiers in Experiment 3, addition of probiotic to the diet with beta-glucan in this experiment resulted in higher growth performance of pigs compared with pigs fed the diet containing carbadox. Moreover, the greatest overall G:F was observed in pigs fed the combination of beta-glucan and probiotic (0.593). The ADG and G:F of pigs fed beta-glucan and probiotic combined were increased by 5.05% and 17.19%, respectively, as compared to pigs fed the negative control diet.

Serum protein levels generally indicate the immune status of the animals, where higher numbers indicate greater immune activation. Immunoglobulins or antibodies have binding sites for both antigens and pathogens and these promote interaction with the immune system that leads to the destruction of pathogens (Yaqoob and Calder, 2003). Aside from this neutralization process, immunoglobulins also activate complement plasma proteins that can also destroy pathogens (Yaqoob and Calder, 2003). White et al. (2002) stated that an antigenic response from fungi, e.g. *Saccharomyces cerevisiae*, not only activates the B-lymphocytes that produce the immunoglobulins, but it also activates the complement system, thus, increasing the response of neutrophils and activation of macrophages. In their study, the authors reported that weanling pigs fed 3% brewers

yeast had higher serum IgG levels as compared to a basal or carbadox treated diet, but serum IgA levels were not affected by yeast or carbadox treatment.

In the present experiment, pigs fed beta-glucan had the lowest IgA and IgG levels for the whole duration of the experiment. The IgA levels of pigs for all dietary treatments were increased in each period, with the greatest percentage increase in pigs fed beta-glucan and probiotic combined at 41% from d 0 to 28. From d 28 to 42, pigs fed the negative control diet had the greatest percentage increase in IgA levels at 68%. Overall (d 0 to d 42), pigs fed the combination of beta-glucan and probiotic had the highest percentage increase in IgA levels at 707%, whereas pigs fed carbadox had the lowest (360%). On the other hand, IgG levels for all treatments were decreased on d 28, where pigs fed carbadox had the greatest reduction at -47%, and pigs fed the probiotic had the least reduction (-8%). The levels of IgG in all treatments were increased again on d 42, and pigs fed carbadox had the greatest percentage increase (41%). From d 0 to d 42, pigs fed the combination of beta-glucan and probiotic had the greatest reduction in IgG levels at -27%, whereas pigs fed the negative control had the greatest increase at 10%.

Based on the study of White et al. (2002), it was expected that beta-glucan, which originated from yeast cell wall, would increase immunoglobulin levels of pigs, particularly that of IgG. But in the present experiment, beta-glucan only had an intermediate response compared to other dietary treatments. The low response of beta-glucan in this experiment could have been due to a lower concentration of beta-glucan used as compared to the study by White et al. (0.20% vs 3%), which might not be enough to stimulate immunoglobulin production. On the other hand, pigs fed carbadox had the lowest percentage increase in IgA and IgG levels, which was similar to the study by

White et al. (2002). One of the mechanisms of antibiotics is the suppression of pathogens (Gaskins et al., 2002) and the low levels of immunoglobulins might be a result of lower immune system activation due to carbadox addition in the diet.

Conclusions

The addition of probiotic to diets with or without beta-glucan improved growth performance of pigs. Pigs fed beta-glucan had similar growth performance response as that of probiotic addition in the diet. The addition of either beta-glucan or carbadox influenced serum protein levels. The combination of beta-glucan and probiotic in diets of pigs further improved growth response. By altering the intestinal microflora and/or by stimulating the immune system, these alternatives hold a promise in replacing antibiotic growth promotants, which could result in the decrease of antibiotic usage in diets of pigs. However, this synergistic effect needs further confirmation in future studies.

Ingredient, %	Phase 1	Phase 2	Phase 3
Corn grain	30.01	46.72	57.49
Soybean meal (48% CP)	15.46	20.19	35.13
Whey, dried	20.00	15.00	0
Lactose	10.00	0	0
Fish menhaden meal	6.00	6.00	0
Soy protein concentrate	12.00	6.00	0
L-lysine HCL	0.10	0	
DL-methionine	0.12	0.05	
Soybean oil	4.00	4.00	4.00
Dicalcium phosphate	0.45	0.21	1.04
Limestone, ground	0.65	0.62	0.93
Zinc oxide	0.28	0.28	
Copper sulfate			0.49
Salt	0.50	0.50	0.50
Trace mineral mix ^b	0.15	0.15	0.15
Vitamin mix ^c	0.25	0.25	0.25
Ethoxyquin	0.03	0.03	0.03
Calculated Analysis:			
ME, kcal/kg	3,506	3,534	3,550
Lysine, %	1.60	1.40	1.20
Ca, %	0.95	0.85	0.75
P, %	0.75	0.70	0.65
Analyzed Composition:			
Crude Protein (as fed), %	24.66	22.83	20.79

Table 6.1. Composition of diets in Experiment 4 (as fed-basis)^a.

^a Within each nursery phase, either 0.25% carbadox^d, 0.20% beta-glucan or probiotic^e was added to the basal diet to provide the five dietary treatments: 1) basal diet ((BD), 2) BD + 0.25% carbadox, 3) BD + 0.20% beta-glucan, 4) BD + probiotic, and 5) BD + 0.20% beta-glucan + probiotic.

^b Supplied per kg of diet: 16.5 mg of Cu (copper sulfate); 165 mg of Fe (ferrous sulfate), 0.30 mg of I (calcium iodate); 40 mg of Mn (manganese oxide); 0.30 mg of Se (sodium selenite); and 165 mg of Zn (zinc oxide).

^c Supplied per kg of diet: 11,013 IU of vitamin A; 1,652 IU of vitamin D₃; 44 IU of vitamin E; 4.4 mg of vitamin K (menadione activity); 55 mg of niacin; 10 mg of riboflavin; 33 mg of pantothenic acid (D-calcium pantothenate); and 44 μ g of vitamin B₁₂.

^d Provided 55 mg of carbadox per kg of complete feed.

^e Added Lactobacillus acidophilus L23 top-dressed in pen-feeders.

	Treatment ^b					
Item	NC	AB	0.20% BG	NC + PB	0.20% BG + PB	SE
Number of pigs	20	20	20	20	20	
Initial weight, kg	5.23	5.23	5.25	5.24	5.24	0.04
Final weight, kg	17.05 ^d	17.93 ^c	17.50 ^{cd}	17.62 ^{cd}	17.47 ^{cd}	0.33
Phase 1						
ADG, kg	0.163	0.176	0.178	0.188	0.187	0.01
ADFI, kg	0.268 ^d	0.294 ^{cd}	0.278 ^{cd}	0.302 ^c	0.293 ^{cd}	0.01
G:F	0.608	0.598	0.638	0.624	0.646	0.03
Phase 2						
ADG, kg	0.389	0.405	0.401	0.403	0.376	0.02
ADFI, kg	0.648	0.654	0.630	0.656	0.591	0.03
G:F	0.598 ^d	0.623 ^{cd}	0.636 ^{cd}	0.616 ^{cd}	0.642 ^c	0.02
Phases 1 & 2						
ADG, kg	0.272^{d}	0.288 ^{cd}	0.290 ^{cd}	0.294 ^c	0.282^{cd}	0.01
ADFI, kg	0.451 ^{cd}	0.470 ^{cd}	0.454 ^{cd}	0.476 ^c	0.442 ^d	0.01
G:F	0.601	0.615	0.637	0.618	0.644	0.02
Phase 3						
ADG, kg	0.285 ^d	0.321 ^c	0.296 ^{cd}	0.292 ^d	0.310 ^{cd}	0.01
ADFI, kg	0.740 ^c	0.659 ^{cd}	0.705 ^c	0.676 ^{cd}	0.600^{d}	0.03
G:F	0.386 ^e	0.490 ^{cd}	0.422 ^e	0.432 ^{de}	0.521 ^c	0.02
Overall						
ADG, kg	0.277 ^d	0.299 ^c	0.292 ^{cd}	0.294 ^c	0.291 ^{cd}	0.01
ADFI, kg	0.546 ^c	0.532 ^c	0.538 ^c	0.542 ^c	0.495 ^d	0.01
G:F	0.506 ^e	0.563 ^{cd}	0.544 ^{de}	0.544 ^{de}	0.593 ^c	0.02

Table 6.2. Growth performance of weanling pigs (Exp. 4)^a.

^a Least square means for 4 pens (5 pigs/pen) per treatment. ^b NC = negative control, AB = NC + carbadox^f, 0.20% BG = NC + 0.20% beta-glucan, NC + PB = NC + 0.20% probiotic^g, 0.20% BG + PB = NC + 0.20% beta-glucan + probiotic.

^{c,d,e} Within a row, means without a common superscript letter differ (P < 0.10).

^f Provided 55 mg of carbadox per kg of complete feed.

^g Added *Lactobacillus acidophilus* L23 top-dressed in pen-feeders.

	Treatment ^{bc}					
					0.20%	
Item	NC	AB	0.20% BG	NC + PB	BG + PB	SE
Number of pigs	12	12	12	12	12	
IgA, mg/100 mL						
Day 0	8.79 ^{de}	9.88 ^{de}	7.50 ^e	10.51 ^d	8.44 ^{de}	1.03
Day 28	35.01 ^{de}	33.43 ^{de}	30.55 ^e	35.77 ^{de}	41.32 ^d	4.23
Day 42	58.87 ^{de}	45.43 ^{de}	43.37 ^e	55.48 ^{de}	68.14 ^d	9.47
IgG, mg/100 mL						
Day 0	440.6 ^{de}	623.1 ^d	416.9 ^e	525.9 ^{de}	636.8 ^d	71.44
Day 28	346.2 ^{de}	329.6 ^{de}	283.2 ^e	482.2 ^d	388.9 ^{de}	65.47
Day 42	485.3 ^d	463.3 ^{de}	353.9 ^e	544.1 ^d	463.9 ^d	43.29

Table 6.3. Serum immune proteins (Exp. 4)^a.

^a Least square means for 4 pens (3 pigs/pen) per treatment. ^b NC = negative control, AB = NC + carbadox^f, 0.20% BG = NC + 0.20% beta-glucan, NC + PB = NC + 0.20% probiotic^g, 0.20% BG + PB = NC + 0.20% beta-glucan + probiotic.

^c Day effect (P < 0.01), Day*Treatment (P > 0.10).

^{d,e} Within a row, means without a common superscript letter differ (P < 0.10).

^f Provided 55 mg of carbadox per kg of complete feed.

^g Added *Lactobacillus acidophilus* L23 top-dressed in pen-feeders.

CHAPTER VII

SUMMARY

Experiment 1 to Experiment 4

Introduction

From the previous four experiments that were conducted, 26 replicates were summarized to evaluate the effects of beta-glucan and antibiotic on growth and performance of weanling pigs. Three dietary treatments were considered: 1) negative control (NC), 2) NC + 0.20% carbadox (AB), and 3) NC + 0.20% beta-glucan (BG). In each treatment, a total of 26 replications was used to evaluate ADG, ADFI, and G:F through the end of Phase 2 (28 days). Data were analyzed as a randomized complete block design using analysis of variance procedures as described by Steel and Torrie (1997). The model included the effects of replication, treatment, and experiment x treatment. Replication-within-experiment was considered a random effect. Treatment means were separated using Least Significant Difference. The pen served as the experimental unit.

Results

An experiment effect (P < 0.01) was observed in all growth parameters measured but there was no experiment and treatment interaction noted (P > 0.10). In Phase 1, there were no differences (P > 0.10) in growth performance among the dietary treatments, although numerically, pigs fed beta-glucan had the greatest ADG and G:F (0.169 kg and

0.681, respectively) (Table 7.1). In Phase 2, ADG of pigs was greater (P < 0.01) with carbadox inclusion in the diet as compared to pigs fed the negative control diet. The ADG of pigs fed beta-glucan was intermediate to that of pigs fed either carbadox or the negative control diet. Carbadox addition to the diet tended to increase (P < 0.10) feed intake of pigs as compared to beta-glucan and the negative control diet. Pigs fed either carbadox or beta-glucan had greater (P < 0.02) G:F than pigs fed the negative control diet. In Phases 1 and 2 combined, pigs fed carbadox had greater (P < 0.01) ADG than pigs fed the negative control diet. The inclusion of beta-glucan in the diet tended to improve (P < 0.07) ADG of pigs as compared to those pigs fed the negative control diet. Pigs fed carbadox had greater (P < 0.04) ADFI than pigs fed the negative control diet. The G:F of pigs was increased (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs were fed either carbadox or beta-glucan had greater (P < 0.04) when pigs fed carbadox.

Discussion

In all phases, inclusion of carbadox resulted in greater ADG, ADFI, and G:F of pigs as compared to pigs fed the negative control diet. These growth performance responses agree with a report by Cromwell (2001), where addition of antibiotic in starter diets resulted in improvements in growth rate and feed efficiency (16.4% and 6.9%, respectively). However, the response to antibiotic addition in this study improved ADG and G:F by 7.06% and 3.39%, respectively, when compared to the negative control diet (Figure 7.1). This lower growth response could be attributed to the existing disease level at the experiment station. Cromwell (2001) stated that antibiotics prevent proliferation of pathogenic microorganisms, which cause specific and non-specific diseases that could

hinder growth performance. However, Cromwell (2001) also stated that the effect of antibiotic is less pronounced in a "clean", more hygienic environment than in "dirty", unsanitary environment. The nursery rooms that were used in this study were properly cleaned and disinfected, which could have eliminated some growth-depressing pathogens. Moreover, the response to antibiotics in research facilities is usually lower than that in commercial farm settings due to the same reasons listed above (Cromwell, 2001).

Beta-glucan supplementation consistently improved ADG and G:F of pigs, as compared to pigs fed the negative control diet. The response of beta-glucan was improvements in ADG by 4.31% and G:F by 4.01% (Figure 7.2), when compared to pigs fed the negative control diet. Other alternatives, such as MOS, had similar improvements in growth performance. Pettigrew (2000) stated in his review that supplementation of MOS improved ADG and feed efficiency by 4.4% and 1.47%, respectively, when compared to diets without MOS. Although the growth performance response of betaglucan was smaller than that of antibiotic, it is still clear that beta-glucan improves growth performance. The overall mean percentage response of pigs in ADG to betaglucan addition was approximately half of the response obtained with carbadox inclusion in the diet. For G:F of pigs, it was slightly improved with beta-glucan supplementation as compared to carbadox addition in the diet. This summary was consistent with previous experiments, where beta-glucan had an intermediate response on growth performance between carbadox or the negative control diet.

Table 7.1. Orowin perior manee	or wearing	Sigs if our Exp	- I to Exp. 4 (2	<i>i</i> (10)
_				
Item	NC	AB	BG	SE
Number of pigs	151	151	151	
Initial weight, kg	5.50	5.50	5.51	0.01
Final weight, kg ^f	18.14 ^e	18.80 ^d	18.48 ^{de}	0.17
Phase 1				
ADG, kg	0.163	0.167	0.169	0.01
ADFI, kg	0.247	0.253	0.249	0.01
G:F	0.666	0.667	0.681	0.01
Phase 2				
ADG, kg	0.353 ^e	0.384^{d}	0.367 ^{de}	0.01
ADFI, kg	0.557	0.579	0.554	0.01
G:F	0.635 ^e	0.664 ^d	0.662 ^d	0.01
Phases 1 & 2				
ADG, kg	0.257 ^e	0.275^{d}	0.268 ^{de}	0.01
ADFI, kg	0.399 ^e	0.414^{d}	0.400 ^{de}	0.01
G:F	0.645 ^e	0.665 ^d	0.671 ^d	0.01

Table 7.1. Growth performance of weanling pigs from Exp. 1 to Exp. 4 (26 reps)^{ab}.

^a Least square means for 26 pens (5-8 pigs/pen) per treatment.

^b Experiment effect (P < 0.01), Experiment*Treatment (P > 0.10).

^c NC = negative control, $AB = NC + carbadox^{g}$, BG = NC + 0.20% beta-glucan.

^{d,e} Within a row, means without a common superscript letter differ (P < 0.10).

^f Final weight of pigs at the end of Phase 3.

^g Provided 55 mg of carbadox per kg of complete feed



Figure 7.1. Percentage improvement in ADG due to carbadox or beta-glucan during the nursery phase from Experiment 1 to Experiment 4.



Figure 7.2. Percentage improvement in G:F due to carbadox or beta-glucan during the nursery phase from Experiment 1 to Experiment 4.

CHAPTER VIII

CONCLUSION

Based on the results of the four experiments, addition of beta-glucan in the diet improved the growth performance response of weanling pigs. Pigs fed beta-glucan appeared to have a growth performance response that was intermediate to that of carbadox or feeding the negative control diet. The inclusion of acidifier and probiotic resulted in variable responses on growth performance when compared to pigs fed betaglucan or carbadox. No interaction was observed in combining beta-glucan and carbadox. However, slight improvements in ADG, ADFI, and G:F were noted when beta-glucan was combined with either acidifier or probiotic. The immune response of pigs generally was not affected with supplementation of either beta-glucan, carbadox, acidifier, or probiotic. These results suggest that beta-glucan can be used as an alternative to antibiotic growth promotants in weanling pig diets.

REFERENCES

- Aarestrup, F. M. and B. Carstensen. 1998. Effect of tylosin used as a growth promoter on the occurrence of macrolide resistant enterococci and staphylococci in pigs. Microbial Drug Resistance 4:307-312.
- Abe, F., N. Ishibashi, and S. Shimamura. 1995. Effect of administration of Bifidobacteria and lactic acid bacteria to newborn calves and piglets. J. Dairy Sci. 78:2838-2846.
- Adachi, Y., M. Okazaki, N. Ohno, and T. Yadomae. 1994. Enhancement of cytokine production by macrophages stimulated with (1-3)-beta-D-glucan, grifolan (GRN), isolated from *Grifola frondosa*. Biological and Pharmaceutical Bulletin 18:1554-1560.
- Anderson, D. B., V. J. McCracken, R. I. Aminov, J. M. Simpson, R. I. Mackie, M. W. A. Verstegen, and H. R. Gaskins. 1999. Gut microbiology and growth-promoting antibiotics in swine. Nutrition Abstracts and Reviews, Series B. Livestock Feeds and Feeding. Chapter 70:101-188.
- Animal Health Institute.2005.Keep Animals Healthy. AHI Resources, Glossary. http://www.ahi.org/resources/glossary.asp.
- Avery, A. 2002. Antibiotic use in food-animal production: Faith in science isn't going to win this one for us. Vance Publishing Corp. Lenexa, KS. October 7.
- Bach Knudsen, K. E. 2001. Development of antibiotic resistance and options to replace antimicrobials in animal diets. Proceedings of the Nutrition Society 60:291-299.
- Bacon, J. S., V. C. Farmer, D. Jones, and I. F. Taylor. 1969. The glucan component of the cell wall of baker's yeast (Saccharomyces cerevisiae) considered in relation to its ultrastructure. Biochem. J. 114:557-67.
- Baum, C. L., and D. L. Harris. 2000. Effect of feeding *Lactobacillus* to pigs infected with *Salmonella typhimurium*. Swine Research Report. Iowa State University, Ames, IA.
- Baur, S.K., and G. Geisler. 1996. Variability of the beta-glucan content in oat caryopsis of 132 cultivated oat genotypes and 39 wild oat genotypes. J. Agr. Crop Sci. 176:151-7.

- Blank, R., R. Mosenthin, W. C. Sauer, and S. Huang. 1999. Effect of fumaric acid and dietary buffering capacity on ileal and fecal amino acid digestibilities in earlyweaned pigs. J. Anim. Sci. 77:2974-2984.
- Blecha, F., and B. Charley. 1990. Rationale for using immunopotentiators in domestic food animals. Adv. Vet. Sci. Comp. Med. 35:3-19.
- Bohn, J. A., and J. N. BeMiller. 1995. $(1\rightarrow 3)$ - β -glucans as biological response modifiers: a review of structure-functional activity relationships. Carbohydrate Polymer 28:3-14.
- Bolduan, V.G., H. Jung, R. Schneider, J. Block, and B. Klenke. 1988. Influence of proprionic and formic acid on piglets. J. Anim. Phys. Anim. Nutr. 59:72-78.
- Borek, C. 2001. Beta-glucan boosts immunity. Nutrition Science News. New Hope Natural Media. Boulder, CO. January.
- Braude, R. 1978. Antibiotics in animal feeds in Great Britain. J. Anim. Sci. 46:1425-1436.
- Brown, G. D., and S. Gordon. 2003. Fungal β-glucans and mammalian immunity. Immunity 19:311-315.
- Burger, W., C. Ewald, and E. M. Fennert. 1998. Increase in C-reactive protein in the serum of piglets (pCRP) following ACTH or corticosteroid administration. Zentralbl Veterinarmed B. 45:1-6.
- Burger, W., E. M. Fennert, M. Pohle, and H. Wesemeier. 1992. C-reactive protein a characteristic feature of health control in swine. Zentralbl Veterinarmed A. 39:635-638.
- Burnell, T. W., G. L. Cromwell, and T. S. Stahly. 1988. Effects of dried whey and copper sulfate on the growth responses to organic acid in diets for weanling pigs. J. Anim. Sci. 66:1100-1108.
- Casewell, M., C. Friis, E. Marco, P. McMullin, and I. Phillips. 2003. The European ban on growth-promoting antibiotics and emerging consequences for human and animal health. J. Antimicrobial Chemotherapy 52:159-161.
- Cassell, G. H. 1995. ASM Task Force Urges Broad Program on Antimicrobial Resistance. ASM News 61(3):116-120.
- CCAC (Canadian Council on Animal Care) 1993. Guide to the Care and Use of Experimental Animals, Vol. 1, 2nd ed. E. D. Olfert, B. M. Cross, and A. A.
 McWilliam, eds. Canadian Council on Animal Care. Ontario Canada. Appendix IV.

- Cera, K. R., D. C. Mahan, R. F. Cross, G. A. Reinhart, and R. E. Whitmoyer. 1988. Effect of age, weaning and postweaning diet on small intestinal growth and jejunal morphology in young swine. J. Anim. Sci. 66:574-584.
- Chee-Sanford, J. C., R. I. Aminov, I. J. Krapac, N. Garrigues-Jeanjean, and R. I. Mackie. 2001. Occurrence and diversity of tetracycline resistance genes in lagoons and groundwater underlying two swine production facilities. Appl. Environ. Microbiol. 67:1494-1502.
- Chesson, A. 1994. Probiotics and other intestinal mediators. Pages 197-214 in Principles of Pig Science. D. J. A. Cole, J. Wiseman, and M. A. Varley, eds. Nottingham University Press, Nottingham, UK.
- Chen, H.-H., J.-H. Lin, H.-P. Fung, L.-L. Ho, P.-C. Yang, W.-C. Lee, Y.-P. Lee, and R.-M. Chu. 2003. Serum acute phase proteins and swine health status. Can. J. Vet. Res. 67(4):283-290.
- Close, W. H. 2000. Producing pigs without antibiotic growth promoters. Advances in Pork Production 11:47.
- Coffey, R. D., and G. L. Cromwell. 2001. Spray-dried animal plasma in diets for weanling pigs. The Farmer's Pride, Vol. 12, No. 37. March 14.
- Cole, D.J.A., R.M. Beal, and J.R. Luscombe. 1968. The effect on performance and bacterial flora of lactic acid, proprionic acid, calcium propionate and calcium acrylate in the drinking water of weaned pigs. Vet. Rec. 83:459-464.
- Collins, M. D., and G. R. Gibson. 1999. Probiotics, prebiotics and synbiotics: approaches for modulating the microbial ecology of the gut. Am. J. Clin. Nutr. 69(Suppl):1052S-1057S.
- Cromwell, G. L. 2001. Antimicrobial and Promicrobial Agents. Page 401-426 in Swine Nutrition, 2nd ed. A. J. Lewis and L. L. Southern, eds. CRC Press LLC. Boca Raton, Fl.
- Cromwell, G. L. 2002. Why and how antibiotics are used in swine production. Animal Biotechnology. 13:7-27.
- Davis, M. E., C. V. Maxwell, E. B. Kegley, B. Z. de Rodas, K. G. Friesen, D. H. Hellwig, and R. A. Dvorak. 1999. Efficacy of mannan oligosaccharide (Bio-Mos) addition at two levels of supplemental copper on performance and immunocompetence of early weaned pigs. J. Anim. Sci. 77(Suppl. 1):63 (Abstr.).

- Davis, M. E., C. V. Maxwell, E. B. Kegley, B. Z. de Rodas, K. G. Friesen, D. H. Hellwig, D. C. Brown, and R. A. Dvorak. 2000. Effect of mannan oligosaccharide (Bio-Mos) supplementation with and without zinc oxide on performance and immunocompetence of weanling pigs. J. Anim. Sci. 78(Suppl.2):61 (Abstr.).
- Davis, M. E., C. V. Maxwell, D. C. Brown, B. Z. de Rodas, Z. B. Johnson, E. B. Kegley, D. H. Hellwig, and R. A. Dvorak. 2002. Effect of dietary mannan oligosaccharides and (or) pharmacological additions of copper sulfate on growth performance and immunocompetence of weanling and growing/finishing pigs. J. Anim. Sci. 80:2887-2894.
- Davis, M. E., C. V. Maxwell, G. F. Erf, D. C. Brown, and T. J. Wistuba. 2004. Dietary supplementation with phosphorylated mannans improves growth response and modulates immune function of weanling pigs. J. Anim. Sci. 82:1882-1891.
- De Cupere, F., P. Deprez, D. Demeulenaere, and E. Muylle. 1992. Evaluation of the effect of 3 probiotics on experimental Escherichia coli enterotoxaemia in weaned piglets. Zentralbl Veterinarmed B. 39:277-284.
- Decuypere, J., N. Dierick, and S. Boddez. 1998. The potentials for immunostimulatory substances (b-1,3/1,6 glucans) in pig nutrition. J. Anim. Feed Sci. 7(Suppl. 1):259-265.
- De Rodas, B. Z., C. V. Maxwell and K. S. Brock. 1995. Diet acidification effects on performance of early-weaned pigs. 1995 Oklahoma State University Animal Science Research Report.
- Dibner, J. J., and P. Buttin. 2002. Use of organic acids as a model to study the impact of gut microflora on nutrition and metabolism. J. Appl. Poult. Res. 11:453-463.
- Dinsmore, J. E., R. J. Jackson, and S. D. Smith. 1997. The protective role of gastric acidity in neonatal bacterial isolation. J. Pediatric Surgery 32(7):1014-1016.
- Doyle, M. E. 2001. Alternatives to antibiotic use for growth promotion in animal husbandry. Food Research Institute. University of Wisconsin-Madison. April 2001:1-17
- Dritz, S. S., J. Shi, T. L. Kielian, R. D. Goodband, J. L. Nelssen, M. D. Tokach, M. M. Chengappa, J. E. Smith, and F. Blecha. 1995. Influence of dietary β-glucan on growth performance, nonspecific immunity, and resistance to *Streptococcus suis* infection in weanling pigs. J. Anim. Sci. 73:3341-3350.
- Dubos, R., R. W. Schaedler, R. Costello, and P. Hoet. 1965. Indigenous, normal and autochthonous flora of the gastrointestinal tract. J. Exp. Med. 122:67.

- Dunsford, B. R., D. A. Knabe, and W. E. Haensly. 1989. Effect of dietary soybean meal on the microscopic anatomy of the small intestine in the early-weaned pig. J. Anim. Sci. 67:1855-1863.
- Eckersall, P. D., P. K. Saini, and C. McComb. 1996. The acute phase response of acid soluble glycoprotein, alpha(1)-acid glycoprotein, ceruloplasmin, haptoglobin and C-reactive protein, in the pig. Vet. Immunol. Immunopathol. 51:377-385.
- Elanco Animal Health. 2003. New antibiotic risk assessment concludes macrolides can be safely used in food animal production. Elanco Animal Health News. September 16, 2003. Elanco Animal Health. Greenfield, IN.
- Fedorka-Cray, P. J., J. S. Bailey, N. J. Stern, N. A. Cox, S. R. Ladely, and M. Musgrove. 1999. Mucosal competitive exclusion to reduce Salmonella in swine. J. Food Prot. 62:1376-1380.
- Florini, K., R. Denison, T. Stiffler, T. Fitzgerald, and R. Goldburg. 2005. Resistant bugs and antibiotic drugs. Pages 1-48 in State and County Estimates of Antibiotics in Agricultural Feed and Animal Waste. Environmental Defense. New York, NY.
- Food Systems Insider. 2002. Antibiotic use in food-animal production. Putting antibiotic use into perspective. Vance Publishing Corp. Lenexa, KS. October 7.
- Fortin, A., W. M. Robertson, S. Kibite, and S. J. Landry. 2003. Growth performance, carcass and pork quality of finisher pigs fed oat-based diets containing different levels of β-glucans. J. Anim. Sci. 81:449-456.
- Francois, A. C. 1962. Mode of action of antibiotics on growth. World Review of Nutrition and Dietetics 3:21.
- Fuller, R. Probiotics for Farm Animals. In probiotics A critical review; Tannock, G.W. Ed. Horizon Scientific Press: Wymondham, UK, 1999. 15-22.
- Fuller, R. 1989. Probiotics in man and animals. J. Appl. Bacteriol. 66:365.
- Garrod, L. P., H. P. Lambert, F. O'Grady, and P. M. Waterworth. 1973. Antibiotic and Chemotherapy. Churchill Livingstone Press. London, UK.
- Gaskins, H. R. 2001. Intestinal bacteria and their influence on swine growth. Pages 585-608 in Swine Nutrition, 2nd ed. A. J. Lewis and L. L. Southern, eds. CRC Press LLC. Boca Raton, Fl.
- Gaskins, H. R., and K. W. Kelly. 1995. Immunology and neonatal mortality. The Neonatal Pig Development and Survival. M. A. Varley, Ed. CAB International. Oxon, UK. 39.

- Gaskins, H. R., C. T. Coller, and D. B. Anderson. 2002. Antibiotics as growth promotants: mode of action. Animal Biotechnology 13:29-42.
- Genovese, K. 2003. Competitive exclusion/colonization resistence. Is there a future for antibiotics in animal agriculture? Interpretative summary, Seventh Discover Conference on Food Animal Agriculture. Nashville, IN. September 21-24.
- Genovese, K. J., R. C. Anderson, R. B. Harvey, and D. J. Nisbet. 2000. Competitive exclusion treatment reduces the mortality and fecal shedding associated with enterotoxigenic *Escherichia coli* infection in nursery-raised neonatal pigs. Canadian Journal of Veterinary Research 64(4):204-207.
- Gibson, G. R., and M. B. Roberfroid. 1995. Dietary modulation of the human colonic microbiota: introducing the concept of prebiotics. J. Nutr. 125:1401-1412.
- Hardy, B. 2002. The issue of antibiotic use in the livestock industry: What have we learned? Animal Biotechnology. 13:129-147.
- Hardy, B. 2003. Nutraceutical concepts for gut health in pigs. 2003 Arkansas Nutrition Conference. Fayetteville, AR. September.
- Harper, A. F., and M. J. Estienne. 2000. Efficacy of carbadox antibiotic and a mannan oligosaccharide source as growth promoters for weanling pigs. J. Anim. Sci. 78(Suppl. 2):12.
- Harper, A. F., and M. J. Estienne. 2002. Efficacy of three potential alternatives to antimicrobial feed additives for weanling pigs. Prof. Animal Scientist 18:343-350.
- Harper, A. 2004. Antimicrobial feed additives for swine: Past, present and future trends. Virginia Cooperative Extension Livestock Update. Tidewater AREC. Suffolk, VA. February.
- Harper, A. F., E. T. Kornegay, K. L. Bryant, and H. R. Thomas. 1983. Efficacy of virginiamycin and a commercially available lactobacillus probiotic in swine diets. Anim. Feed Sci. Tech. 8:69-76.
- Hawkey, P. M. 1998. The origins and molecular basis of antibiotic resistance. BMJ 317:657-660.
- Hayes, D. J., H. H. Jensen, L. Backstrom, and J. Fabiosa. 1999. Economic impact of a ban on the use of over-the-counter antibiotics. Center for Agricultural and Rural Development. Iowa State University. Ames, IA. Staff Report 99-SR 90. December.

- Hayes, D. J., H. H. Jensen, and J. Fabiosa. 2002. Technology choice and the economic effects of a ban on the use of antimicrobial feed additives in swine rations. Food Control 13:97-101.
- Hayes, D. J., and H. H. Jensen. 2003. Lessons from the Danish ban on feed-grade antibiotics. Choices 3rd Quarter. American Agricultural Economics Association. Texas A&M University. College Station, TX.
- Hays, V. W. 1977. Effectiveness of feed additive usage of antibacterial agents in swine and poultry production. Office of Technology Assessment, U.S. Congress, Washington, D.C. (Edited version: Hays, V. W. 1981. The Hays Report, Rachelle Laboratories, Inc., Long Beach, CA).
- Heegaard, P. M. J. Klausen, J. P. Nielsen, N. Gonzalez-Ramon, M. Pineiro, F. Lampreave, and M. A. Alava. 1998. The porcine acute phase response to infection with *Actinobacillus pleuropneumoniae*. Haptoglobin, C-reactive protein, major acute phase protein and serum amyloid A protein are sensitive indicators of infection. Comp. Biochem. Physiol. B. Biochem. Mol. Biol. 119:365-373.
- Hentges, D. J. 1992. Gut flora and disease resistance. Pages 87-110 in Probiotics: the scientific basis. R. Fuller, Ed. Chapman and Hall. London, UK.
- Hiss, S., and H. Sauerwein. 2003. Influence of dietary β-glucan on growth performance, lymphocyte proliferation, specific immune response and haptoglobin plasma concentrations in pigs. J. Anim. Physiol. and Anim. Nutr. 87:2-11.
- Holden, P., J. Carr, M. Honeyman, J. Kliebenstein, J. McKean, J. Harmon, J. Mabry, and S. Hoyer. 2002. Minimizing the use of antibiotics in pork production. IPIC 8. Continuing Education and Communication Services. Iowa State University. Ames, IA. October
- Hooper, L. V., M. H. Wong, A. Thelin, L. Hansson, P. G. Falk, and J. I. Gordon. 2001. Molecular analysis of commensal host-microbial relationships in the intestine. Science. 291:881-884.
- Johnson, R. W., J. Escobar, and D. M. Webel. 2001. Nutrition and Immunology of Swine. Pages 545-562 in Swine Nutrition, 2nd Ed. A. J. Lewis and L. L. Southern, Eds. CRC Press LLC, Boca Raton, Fl.
- Jung, K., Y. Ha, S.-K. Ha, D. U. Han, D.-W. Kim, W. K. Moon, and C. Chae. 2004. Antiviral effect of *Saccharomyces cerevisiae* β-glucan to swine influenza virus by increased production of interferon-γ and nitric oxide. Journal of Veterinary Medicine Series B 51:72-76.
- Kaufman, M. 2003. WHO urges end to use of antibiotics for animal growth. Washington Post. August 13:A01.

- Kelly, T. 2004. Feeding to fight disease. Pork Magazine. Vance Publishing Corp. Lenexa, KS. March 9.
- Krause, D. 2003. Antibiotic in swine diets: where have we been? Part I. Council Research News. Manitoba Pork Council. Winnipeg, MB. December.
- Krause, D., and K. Graham. 2004. Antibiotic in swine diets: consequences of withdrawal? Part II. Council Research News. Manitoba Pork Council. Winnipeg, MB. March.
- Kunin, C. M. 1993. Resistance to antimicrobial drugs: a worldwide calamity. Ann. Intern. Med. 118:557-561.
- Kyriakis, S. C., V. K. Tsiloyiannis, J. Vlemmas, K. Sarris, A. C. Tsinas, C. Alexopoulos, and L. Jansegers. 1999. The effect of probiotic LSP 122 on the control of postweaning diarrhoea syndrome of piglets. Research in Veterinary Science 67:223-228.
- Langlois, B. E., G. L. Cromwell, T. S. Stahly, K. A. Dawson and V. W. Hays. 1983. Antibiotic resistance of fecal coliforms after long-term withdrawal of therapeutic and subtherapeutic antibiotic use in swine herd. Applied and Environmental Microbiology 46:1433-1434.
- Langlois, B. E., K. A. Dawson, G. L. Cromwell, T. S. Stahly. 1986. Antibiotic resistance in pigs following a 13 year ban. J. Anim. Sci. 62(Suppl. 3):18.
- Lee, H. S., S. E. Gilliland, and S. D. Carter.2001.Amylolytic cultures of *Lactobacillus acidophilus*: Potential probiotics to improve dietary starch utilization. J. Food Sci. 66:338-344.
- LeMieux, F. M., L. L. Southern, and T. D. Bidner. 2003. Effect of mannan oligosaccharides on growth performance of weanling pigs. J. Anim. Sci. 81:2482-2487.
- Levy, J. 2000. The effects of antibiotic use on gastrointestinal function. Am. J. Gastroenterol. 95(Suppl. 1):S8-S10.
- Levy, S. B., G. B. Fitzgerald, and A. B. Macone. 1976. Changes in intestinal flora of farm personnel after introduction of a tetracycline-supplemented feed on a farm. N. Engl. J. Med. 295(11):583-588.
- Li, D. F., J. L. Nelssen, P. G. Reddy, F. Blecha, R. D. Klemm, D. W. Giesting, J. D. Hancock, G. L. Allee, and R. D. Goodband. 1991. Measuring suitability of soybean products for early-weaned pigs with immunological criteria. J. Anim. Sci. 69:3299-3307.

- Makkink, C. A., P. J. M. Berntsen, B. M. L. op den Kamp, B. Kemp, and M. W. A. Verstegen. 1994. Gastric protein breakdown and pancreatic enzyme activities in response to two different dietary protein sources in newly weaned pigs. J. Anim. Sci. 72:2843-2850.
- Mathew, A. 2002. Seeking alternatives to growth promoting antibiotics. Vol. 16 in Sharing Ideas and Information for Efficient Pork Production: Manitoba Swine Seminar. Manitoba Agriculture and Food. Winnipeg, Manitoba.
- Mathew, A. G., and P. D. Ebner. 2004. Issues of drug use and antibiotic resistance in pig production. Pig News and Information 25:133N-147N.
- Mathews Jr., K. H. 2001. Antimicrobial drug use and veterinary costs in U.S. livestock production. In Agriculture Information Bulletin 766. Economic Research Service. May.
- Mathew, A. G., and K. N. Garner. 2003. Effects of feeding oxytetracycline to sows on antibiotic resistance of bacteria in pigs. Department of Animal Science Annual Report. The University of Tennessee, Knoxville, TN.
- McEwen, S. A. and P. J. Fedorka-Cray. 2002. Antimicrobial use and resistance in animals. Clinical Infectious Disease 34(Suppl. 3):S93-S106.
- McManus, M. C. 1997. Mechanisms of bacterial resistance to antimicrobial agents. American Journal Health-Systems Pharmacology 54:1420-1433.
- Messenger, J. 2002. Will consumers pay up? Pork Magazine. Vance Publishing Corp. Lenexa, KS. July 7.
- Messenger, J. 2003. Food safety is the top concern. Pork Magazine. Vance Publishing Corp. Lenexa, KS. November 10.
- Messenger, J. 2004. Animal antibiotics: What does the future hold? Pork Magazine. Vance Publishing Corp. Lenexa, KS. February 9.
- Nabuurs, M. J., A. Hoogendorn, and F. G. van Zijderveld. 1994. Effects of weaning and enterotoxigenic *Escheria coli* on net absorption in the small intestine of pigs. Res. Vet. Sci. 56:379-385.
- Newman, K. E., and M. C. Newman. 2001. Evaluation of mannan oligosaccharide on the microflora and immunoglobulin status of sows and piglet performance. J. Anim. Sci. 79(Suppl. 1):189 (Abstr).
- NPPC. 2001. Procedures for estimating pork carcass composition. National Pork Producers Council. Des Moines, IA.

- Nunez, M. C., J. D. Bueno, M. V. Ayudarte, A. Almendros, A. Rios, M. D. Suarez, and A. Gil. 1996. Dietary restriction induces biochemical and morphometric changes in the small intestine of nursing piglets. J. Nutr. 126:933-944.
- Okazaki, M., Y. Adachi, N. Ohno, and T. Yadomae. 1995. Structure-activity relationship of (1-3)-beta-D-glucans in the induction of cytokine production from macrophages, in vitro. Biological and Pharmaceutical Bulletin 18:1320-1327.
- O'Quinn, P. R., D. W. Funderburke, and G. W. Tibbetts. 2001. Effects of dietary supplementation with mannan oligosaccharides on sow and litter performance in a commercial production system. J. Anim. Sci. 79(Suppl. 1):212 (Abstr.)
- Partanen, K. H., and Z. Mroz. 1999. Organic acids for performance enhancement in pig diets. Nutrition Research Reviews 12:117-145.
- Pettigrew, J. E. 2000. Bio-Mos effects on pig performance: A review. Pages 31-44 in Proc. of Alltech's 16th Annu. Symp.: Biotechnol. in the Feed Industry. T. P. Lyons and K. A. Jacques, ed. Nottingham Univ. Press. Nottingham, U.K.
- Phillips, I., M. Casewell, T. Cox, B. De Groot, C. Friis, R. Jones, C. Nightingale, R. Preston, and J. Waddell. 2004. Does the use of antibiotics in food animals pose a risk to human health? A critical review of published data. J. Antimicrob. Chemother. 53:28.52.
- Pieterse, E. 2000. Protein sources for weaner piglets soya, fishmeal or milk products. Animal Feed Manufacturers Association Matrix. Centurion, South Africa. September.
- Plumb, D. C. 1995. Veterinary Drug Handbook. Pharma Vet Publishing. White Bear Lake, MN.
- Pluske, J. R., D. J. Hampson, and I. H. Williams. 1997. Factors influencing the structure and function of the small intestine in the weaned pig: a review. Livestock Production Science 51:215-236.
- Pollmann, D. S., D. M. Danielson, and E. R. Peo, Jr. 1980. Effects of microbial feed additives on performance of starter and growing finishing pigs. J. Anim. Sci. 51:577.
- Pollman, D. S. 1992. Probiotics in swine diets. Pages 65-74 in Proceedings of the International Round Table on Animal Feed Technology – Research and Scientific Regulation. D. A. Leger and S. K. Ho, ed. Agriculture Canada. Ottawa, Canada.
- Pork News Source. 2002. Antibiotic use in animals declines. Pork Magazine. Vance Publishing Corp. Lenexa, KS. September 30.

- Pork News Source. 2003. McDonald's sets antibiotic use policy. Pork Magazine. Vance Publishing Corp. Lenexa, KS. June 19.
- Pork News Source. 2003. Bill to ban certain antibiotics will be introduced. Pork Magazine. Vance Publishing Corp. Lenexa, KS. July 21.
- Pork News Source. 2004. Peers deem antibiotic safe. Pork Magazine. Vance Publishing Corp. Lenexa, KS. May 3.
- Pork News Source. 2005. Medical groups target animal antibiotics. Pork Magazine. Vance Publishing Corp. Lenexa, KS. April 11.
- Pork News Source. 2005. Industry opposes livestock antibiotic ban. Pork Magazine. Vance Publishing Corp. Lenexa, KS. April 15.
- Prescott, J. F., J. D. Baggot, and R. D. Walker. 2000. Antimicrobial drug action and interaction: an introduction. Pages 3-11 in Antimicrobial therapy in veterinary medicine. Iowa State University Press. Ames, IA.
- Ravindran, V., and E. T. Kornegay. 1993. Acidification of weaner pig diets: a review. J. Sci. Food Agric. 62:313-322.
- Risley, C.R., E.T. Kornegay, M.D. Lindemann, C.M. Wood, and W.N. Eigel. 1992. Effect of feeding organic acids on selected intestinal content measurements at varying times postweaning in pigs. J. Anim. Sci. 70:196-206.
- Rozeboom, D. W., D. T. Shaw, J. E. Pettigrew, and A. Connolly. 2001. Comparative effects of mannanoligosaccharide and an antibiotic in nursery diets on performance of pigs reared on three different farms. J. Anim. Sci. 79(Suppl. 2):79.
- Savage, D. C. 1977. Microbial ecology of the gastrointestinal tract. Ann. Rev. Microbiol. 31:107-133.
- Schoenherr, W. D., D. S. Pollmann, and J. A. Coalson. 1994. Titration of MacroGard[™]-S on growth performance of nursery pigs. J. Anim. Sci. 72(Suppl. 2):57 (Abstract).
- Shea, K. M. 2004. Nontherapeutic use of antimicrobial agents in animal agriculture: Implications for pediatrics. Pediatrics 114:862-868.
- Smith, M. 2002. Antibiotic use in food-animal production: Breathing new life into an old food safety issue. Food Systems Insider. Vance Publishing Corp. Lenexa, KS. October 7.

- Spring, P., C. Wenk, K. A. Dawson, and K. E. Newman. 2000. The effects of dietary mannanoligosaccharides on cecal parameters and the concentrations of enteric bacteria in the ceca of salmonella-challenged broiler chicks. Poult. Sci. 79:205-211.
- Stahly, T. S., N. H. Williams, and S. G. Swenson. 1996. Growth response to carbadox in pigs with a high or low genetic capacity for lean tissue growth. ISU Swine Research Report-1996. ASL-R1368. ISU Coop. Ext. Serv. Ames, IA.
- Steel, R. G. D., and J. H. Torrie. 1997. Principles and procedures of statistics: A biomedical approach. 3rd ed. McGraw-Hill Book Company. New York, NY.
- Stein, H. H. 2002. Experience of feeding pigs without antibiotics: a European Perspective. Animal Biotechnology 13:85-95.
- Thomlinson, J.R., and T.L.J. Lawrence. 1981. Dietary manipulation of gastric pH in the prophylaxis of enteric disease in weaned pigs. Some field observations. Vet. Rec. 109:120-122.
- Tizard, I. R., R. H. Carpenter, B. H. McAnalley, and M. C. Kemp. 1989. The biological actitivities of mannans and related complex carbohydrates. Mol. Biother. 1:290-296.
- Tokunaka, K., N. Ohno, Y. Adachi, S. Tanaka, H. Tamura, and T. Yadomae. 2000. Immunopharmacological and immunotoxicological activities of a water soluble (1-3)β-D-glucan, CSBG, from *Candida* spp. Int. J. Immunopharmacol. 22:383-394.
- Turner, J. L., S. S. Dritz, and J. E. Minton. Review: Alternatives to conventional antimicrobials in swine diets. Prof. Anim. Scientist 17:217-226.
- U.S. FDA (United States Department of Health and Human Services, Food and Drug Administration, Center for Veterinary Medicine). 2003. Guidance for Industry #152. Evaluating the safety of antimicrobial new animal drugs with regard to their microbiological effects on bacteria of human health concern. Washington, D.C. October 23.
- USDA (Animal and Plant Health Inspection Service, Veterinary Services, Centers for Epidemiology and Animal Health, Center for Emerging Issues). 1999. Antimicrobial resistance issues in animal agriculture. Washington, D. C. C10.1299. December.
- van den Bogaard, A. E. and E. E. Stobberingh. 1999. Antibiotic usage in animals: impact on bacterial resistance and public health. Drugs 58:589-607.

- van Dijk, A. J., H. Everts, M. J. A. Nabuurs, R. J. C. F. Margy and A. C. Beyen. 2001. Growth performance of weanling pigs fed spray-dried animal plasma: a review. Livestock Production Science 68:263-274.
- van Heugten, E. 1997. Feeding the early weaned pig. In Proceedings of the North Carolina Healthy Hogs Seminar. November 5-7, 2000. Greenville and Fayetteville, NC.
- van Nevel, C. J., J. A. Decuypere, N. Dierick, and K. Molly. 2003. The influence of *Lentinus edodes* (shiitake mushroom) preparations on bacteriological and morphological aspects of the small intestine in piglets. Arch. Anim. Nutr. 57(6):399-412.
- Vellenga, L, H. J. Egberts, T. Wensing, J. E. van Dijk, J. M. Mouwen, and H. J. Breukink. 1992. Intestinal permeability in pigs during rotavirus infection. Am. J. Vet. Res. 53:1180-1183.
- Verstegen, W. M. A. and B. A. Williams. 2002. Alternatives to the use of antibiotics as growth promoters for monogastric animals. Animal Biotechnology 13:113-127.
- Virtanen, E., K. GrowHow, B. Nilsson, and J. Khajarern. 2004. Organic acids in pig nutrition – novel solutions. In Animal Feed Manufacturers Association Forum. Centurion, South Africa.
- Visek, W. J. 1978. The mode of growth promotion by antibiotics. J. Anim. Sci. 46:1447-1469.
- Walsh, M. C., L. Peddireddi, and J. S. Radcliffe. 2004a. Acidification of nursery diets and the role of diet buffering capacity. 2004 Midwest Swine Nutrition Proceedings. Indianapolis, IN. August 31.
- Walsh, M., D. Sholly, D. Kelly, M. Cobb, S. Trapp, R. Hinson, B. Hill, A. Sutton, S. Radcliffe, J. Smith, and B. Richert. 2004b. The effects of supplementing weanling pig diets with organic and inorganic acids on growth performance and microbial shedding. J. Anim. Sci. 82(Suppl. 2):75.
- Webel, D. M., L. S. Brown, and J. D. Spencer. 2003. Recent advances in swine nutrition. XI Congresso Brasileiro de Veterinarios Especialistas em Suinos.
- White, L. A., M. C. Newman, G. L. Cromwell, and M. D. Lindemann. 2002. Brewers dried yeast as a source of mannan oligosaccharides for weanling pigs. J. Anim. Sci. 80:2619-2628.

- WHO (World Health Organization). 2002. Impacts of antimicrobial growth promoter termination in Denmark: The WHO international review panel's evaluation of the termination of the use of antimicrobial growth promoters in Denmark. WHO, Foulum, Denmark. November 6-9.
- 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 Resist. 7(2):183-190.
- Yan, S. S., and J. M. Gilbert. 2004. Antimicrobial drug delivery in food animals and microbial food safety concerns: an overview of in vitro and in vivo factors potentially affecting the animal gut microflora. Adv. Drug Del. Rev. 56:1497-1521.
- Yaqoob, P., and P. C. Calder. 2003. Nutrition and immune function. Pages 349-367 in Molecular Nutrition. J. Zempleni and H. Daniel, eds. CABI Publ. Cambridge, MA.
- Zimmerman, D. R. 1986. Role of subtherapeutic antimicrobials in animal production. J. Anim. Sci. 62(Suppl. 3):6.

APPENDIX TABLES

	Phase 1			Phase 2			
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F
1	1	0.144	0.262	0.550	0.355	0.548	0.649
2	1	0.155	0.304	0.510	0.431	0.612	0.704
3	1	0.194	0.300	0.646	0.445	0.681	0.654
4	1	0.160	0.261	0.615	0.398	0.680	0.586
1	2	0.150	0.228	0.658	0.370	0.531	0.696
2	2	0.118	0.183	0.641	0.342	0.498	0.687
3	2	0.141	0.219	0.644	0.330	0.469	0.702
4	2	0.143	0.245	0.585	0.298	0.444	0.672
1	3	0.166	0.291	0.572	0.257	0.411	0.625
2	3	0.130	0.320	0.408	0.296	0.455	0.651
3	3	0.116	0.292	0.398	0.303	0.450	0.673
4	3	0.080	0.221	0.364	0.347	0.428	0.810
1	4	0.135	0.307	0.439	0.236	0.383	0.617
2	4	0.168	0.240	0.698	0.328	0.492	0.667
3	4	0.106	0.239	0.442	0.260	0.395	0.659
4	4	0.122	0.245	0.499	0.213	0.313	0.681
1	5	0.207	0.302	0.687	0.408	0.565	0.723
2	5	0.256	0.323	0.793	0.537	0.687	0.781
3	5	0.296	0.370	0.800	0.504	0.666	0.757
4	5	0.198	0.294	0.674	0.417	0.559	0.746
1	6	0.215	0.264	0.813	0.443	0.625	0.709
2	6	0.213	0.286	0.743	0.400	0.602	0.664
3	6	0.209	0.294	0.710	0.434	0.611	0.710
4	6	0.198	0.243	0.813	0.445	0.646	0.689
1	7	0.179	0.253	0.707	0.415	0.542	0.766
2	7	0.217	0.306	0.709	0.415	0.579	0.716
3	7	0.183	0.271	0.674	0.379	0.612	0.619
4	7	0.171	0.216	0.792	0.372	0.546	0.681
1	8	0.182	0.291	0.625	0.392	0.556	0.705
2	8	0.162	0.226	0.715	0.387	0.522	0.741
3	8	0.159	0.238	0.668	0.346	0.517	0.670
4	8	0.128	0.202	0.633	0.365	0.513	0.710

Appendix Table 1. Means for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 - Experiment 1.

Trt 1: negative control diet (0.40% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.15% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.20% cornstarch.

Trt 4: diet + 0.40% beta-glucan.
		Mean Squares						
			Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	31							
Error	21	0.00063	0.00089	0.00546	0.00139	0.00204	0.00215	
Repetition	7	0.00652	0.00383	0.05308	0.01902	0.03227	0.00401	
Treatment	3	0.00129	0.00241	0.00158	0.00211	0.00331	0.00073	
Coefficient of								
Variation, %		14.81	11.16	11.69	10.04	8.43	6.70	

Appendix Table 2. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 - Experiment 1.

	Phases 1 & 2				Phase 3			
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F	
1	1	0.250	0.405	0.617	0.510	0.804	0.634	
2	1	0.293	0.458	0.640	0.545	0.793	0.688	
3	1	0.320	0.491	0.651	0.567	0.845	0.670	
4	1	0.279	0.470	0.594	0.444	0.861	0.516	
1	2	0.260	0.380	0.685	0.546	0.823	0.664	
2	2	0.230	0.341	0.675	0.536	0.754	0.711	
3	2	0.236	0.345	0.684	0.451	0.704	0.641	
4	2	0.217	0.339	0.639	0.483	0.800	0.604	
1	3	0.212	0.339	0.625	0.394	0.659	0.598	
2	3	0.213	0.350	0.610	0.481	0.745	0.646	
3	3	0.209	0.330	0.635	0.399	0.649	0.615	
4	3	0.214	0.296	0.722	0.434	0.679	0.639	
1	4	0.183	0.316	0.578	0.375	0.637	0.589	
2	4	0.248	0.373	0.664	0.460	0.758	0.607	
3	4	0.183	0.298	0.613	0.435	0.670	0.650	
4	4	0.168	0.264	0.634	0.371	0.578	0.642	
1	5	0.308	0.434	0.710	0.472	0.838	0.564	
2	5	0.396	0.505	0.784	0.360	0.881	0.408	
3	5	0.400	0.518	0.772	0.511	0.911	0.561	
4	5	0.308	0.427	0.721	0.438	0.800	0.548	
1	6	0.329	0.444	0.740	0.498	0.939	0.530	
2	6	0.306	0.444	0.689	0.429	0.713	0.601	
3	6	0.321	0.453	0.710	0.466	0.829	0.562	
4	6	0.321	0.445	0.722	0.466	0.829	0.562	
1	7	0.297	0.398	0.748	0.379	0.818	0.464	
2	7	0.316	0.442	0.714	0.408	0.774	0.527	
3	7	0.281	0.442	0.635	0.422	0.891	0.474	
4	7	0.271	0.381	0.713	0.481	0.820	0.587	
1	8	0.287	0.424	0.677	0.465	0.799	0.582	
2	8	0.274	0.374	0.733	0.475	0.757	0.627	
3	8	0.253	0.377	0.670	0.398	0.758	0.525	
4	8	0.235	0.343	0.685	0.481	0.806	0.597	

Appendix Table 3. Means for average daily gain, average daily feed intake, and gain: feed for Phases 1 & 2 combined and Phase 3 - Experiment 1.

Trt 1: negative control diet (0.40% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.15% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.20% cornstarch.

Trt 4: diet + 0.40% beta-glucan.

		Mean Squares						
		Р	hases 1 &	2		Phase 3		
Source	d.f.	ADG	ADFI	G:F		ADG	ADFI	G:F
Total	31							
Error	21	0.00065	0.00086	0.00134		0.00236	0.00352	0.00286
Repetition	7	0.01151	0.01479	0.00783		0.00584	0.02066	0.01104
Treatment	3	0.00158	0.00264	0.00005		0.00019	0.00061	0.00078
Coefficient of								
Variation, %		9.50	7.42	5.40		10.66	7.61	9.09

Appendix Table 4. Analysis of variance for average daily gain, average daily feed intake, and gain: feed for Phases 1 & 2 combined and Phase 3 - Experiment 1.

0			Overall	
Trt	Ren	ADG (kg)	ADFL (kg)	GF
1	1	0 336	0 538	0.625
2	1	0.330	0.550	0.663
2	1	0.396	0.507	0.659
5 4	1	0.334	0.600	0.557
	1	0.355	0.528	0.557
2	2	0.331	0.328	0.693
2	2	0.307	0.478	0.653
5 4	2	0.296	0.476	0.621
1	2	0.250	0.470	0.621
2	3	0.200	0.507	0.597
3	3	0.272	0.367	0.597
<u></u>	3	0.272	0.443	0.500
1	4	0.239	0.429	0.557
2	4	0.319	0.497	0.642
3	4	0.267	0.434	0.614
4	4	0 235	0.378	0.622
1	5	0.363	0.568	0.638
2	5	0.384	0.630	0.610
3	5	0.437	0.649	0.673
4	5	0.351	0.551	0.638
1	6	0.385	0.609	0.632
2	6	0.347	0.533	0.650
3	6	0.370	0.578	0.639
4	6	0.370	0.573	0.645
1	7	0.325	0.538	0.604
2	7	0.346	0.553	0.627
3	7	0.322	0.574	0.562
4	7	0.341	0.528	0.647
1	8	0.346	0.549	0.631
2	8	0.341	0.502	0.681
3	8	0.296	0.489	0.604
4	8	0.312	0.488	0.640

Appendix Table 5. Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period - Experiment 1.

Trt 1: negative control diet (0.40% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.15% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.20% cornstarch.

Trt 4: diet + 0.40% beta-glucan.

	_	Mean Squares				
			Overall			
Source	d.f.	ADG	ADFI	G:F		
Total	31					
Error	21	0.00067	0.00114	0.00107		
Repetition	7	0.00680	0.01410	0.00154		
Treatment	3	0.00107	0.00141	0.00096		
Coefficient of						
Variation, %		7.86	6.44	5.20		

Appendix Table 6. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period – Experiment 1.

			Phase 1		-		Phase 2	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG	(kg)	ADFI (kg)	G:F
1	1	0.157	0.222	0.707	0.29	96	0.448	0.661
2	1	0.148	0.216	0.686	0.34	16	0.515	0.671
3	1	0.201	0.254	0.791	0.29	97	0.459	0.649
4	1	0.192	0.239	0.805	0.35	58	0.510	0.702
1	2	0.153	0.227	0.673	0.28	37	0.447	0.643
2	2	0.178	0.272	0.655	0.33	30	0.512	0.644
3	2	0.132	0.191	0.689	0.32	24	0.452	0.718
4	2	0.187	0.253	0.740	0.38	39	0.518	0.752
1	3	0.163	0.227	0.719	0.28	33	0.415	0.683
2	3	0.168	0.230	0.729	0.27	72	0.401	0.680
3	3	0.154	0.200	0.771	0.31	4	0.429	0.730
4	3	0.153	0.215	0.709	0.32	26	0.438	0.746
1	4	0.138	0.207	0.670	0.45	57	0.630	0.726
2	4	0.149	0.242	0.617	0.52	20	0.701	0.741
3	4	0.146	0.221	0.660	0.44	19	0.649	0.693
4	4	0.186	0.241	0.772	0.44	14	0.641	0.693
1	5	0.123	0.202	0.610	0.38	33	0.585	0.655
2	5	0.133	0.216	0.617	0.45	51	0.616	0.732
3	5	0.133	0.245	0.544	0.39	98	0.603	0.659
4	5	0.142	0.227	0.625	0.41	3	0.578	0.714
1	6	0.136	0.224	0.609	0.35	53	0.526	0.670
2	6	0.152	0.220	0.690	0.38	39	0.545	0.714
3	6	0.127	0.215	0.591	0.38	34	0.573	0.670
4	6	0.153	0.221	0.692	0.39	92	0.497	0.789

Appendix Table 7. Means for average daily gain, average daily feed intake, and gain: feed for Nursery Phase 1 and Phase 2 - Experiment 2.

Trt 1: negative control diet (0.45% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.20% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.25% cornstarch.

Trt 4: diet + 0.20% beta-glucan + 0.25% carbadox.

		Mean Squares						
		Phase 1			Phase 2			
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	23							
Error	15	0.00027	0.00040	0.00188	0.00066	0.00072	0.00119	
Repetition	5	0.00089	0.00020	0.01203	0.01589	0.02914	0.00113	
Treatment	3	0.00066	0.00035	0.00475	0.00260	0.00160	0.00392	
No AB vs AB	1	0.00132	0.00103	0.00383	0.00683	0.00273	0.00739	
No BG vs BG	1	0.00049	0.00001	0.00690	0.00061	1.50E-06	0.00363	
AB x BG	1	0.00016	0.00001	0.00353	0.00036	0.00205	0.00074	
Coefficient of								
Variation, %		10.71	8.82	6.35	6.97	5.08	4.94	

Appendix Table 8. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Nursery Phase 1 and Phase 2 - Experiment 2.

0	Phases 1 & 2				······································	Phases 3				
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F			
1	1	0.227	0.335	0.677	0.488	0.806	0.605			
2	1	0.247	0.365	0.676	0.506	0.801	0.631			
3	1	0.249	0.356	0.699	0.480	0.784	0.612			
4	1	0.275	0.375	0.735	0.504	0.820	0.615			
1	2	0.220	0.337	0.654	0.430	0.735	0.586			
2	2	0.254	0.392	0.648	0.429	0.792	0.542			
3	2	0.228	0.321	0.709	0.449	0.684	0.657			
4	2	0.288	0.385	0.748	0.531	0.824	0.644			
1	3	0.223	0.321	0.695	0.444	0.720	0.617			
2	3	0.220	0.316	0.698	0.419	0.695	0.604			
3	3	0.234	0.315	0.744	0.425	0.684	0.622			
4	3	0.239	0.326	0.733	0.416	0.655	0.635			
1	4	0.298	0.418	0.712	0.546	0.863	0.633			
2	4	0.335	0.472	0.709	0.574	0.938	0.612			
3	4	0.298	0.434	0.685	0.553	0.913	0.605			
4	4	0.315	0.441	0.715	0.582	0.930	0.626			
1	5	0.253	0.393	0.643	0.562	0.863	0.652			
2	5	0.292	0.416	0.702	0.536	0.865	0.620			
3	5	0.266	0.424	0.626	0.562	0.954	0.589			
4	5	0.277	0.402	0.690	0.574	0.966	0.594			
1	6	0.245	0.375	0.653	0.537	0.828	0.649			
2	6	0.271	0.383	0.707	0.533	0.863	0.618			
3	6	0.256	0.395	0.648	0.550	0.892	0.617			
4	6	0.272	0.359	0.759	0.563	0.881	0.639			

Appendix Table 9. Means for average daily gain, average daily feed intake, and gain: feed for Nursery Phases 1 & 2 combined and Phase 3 - Experiment 2.

Trt 1: negative control diet (0.45% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.20% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.25% cornstarch.

Trt 4: diet + 0.20% beta-glucan + 0.25% carbadox.

		Mean Squares							
			Phases 1 & 2	2		Phase 3			
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F		
Total	23								
Error	15	0.00017	0.00033	0.00083	0.00046	0.00157	0.00077		
Repetition	5	0.00321	0.00732	0.00123	0.01305	0.03211	0.00024		
Treatment	3	0.00133	0.00081	0.00372	0.00111	0.00195	0.00054		
No AB vs AB	1	0.00346	0.00180	0.00586	0.00083	0.00385	0.00017		
No BG vs BG	1	0.00052	4.17E-06	0.00419	0.00143	0.00198	0.00031		
AB x BG	1	0.00001	0.00062	0.00111	0.00108	0.00003	0.00115		
Coefficient of									
Variation, %		4.92	4.85	4.15	4.20	4.81	4.48		

Appendix Table 10. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Nursery Phases 1 & 2 combined and Phase 3 - Experiment 2.

			Overall	
Trt	Rep	ADG	ADFI	GF
1	1	0.313	0.492	0.637
2	1	0.333	0.511	0.652
3	1	0.326	0.498	0.654
4	1	0.351	0.523	0.672
1	2	0.290	0.469	0.618
2	2	0.312	0.525	0.595
3	2	0.302	0.443	0.682
4	2	0.369	0.532	0.694
1	3	0.297	0.454	0.654
2	3	0.286	0.442	0.649
3	3	0.298	0.438	0.680
4	3	0.298	0.436	0.684
1	4	0.380	0.567	0.671
2	4	0.415	0.627	0.661
3	4	0.383	0.594	0.644
4	4	0.404	0.604	0.669
1	5	0.356	0.550	0.647
2	5	0.373	0.566	0.660
3	5	0.364	0.601	0.606
4	5	0.376	0.590	0.638
1	6	0.342	0.526	0.650
2	6	0.358	0.543	0.660
3	6	0.354	0.560	0.631
4	6	0.369	0.533	0.693

Appendix Table 11. Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d nursery period - Experiment 2.

Trt 1: negative control diet (0.45% cornstarch).

Trt 2: diet + 0.25% carbadox + 0.20% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.25% cornstarch.

Trt 4: diet + 0.20% beta-glucan + 0.25% carbadox.

		Mean Squares Overall				
	_					
Source	d.f.	ADG	ADFI	G:F		
Total	23					
Error	15	0.00018	0.00051	0.00061		
Repetition	5	0.00528	0.01318	0.00044		
Treatment	3	0.00109	0.00096	0.00117		
No AB vs AB	1	0.00238	0.00240	0.00098		
No BG vs BG	1	0.00081	0.00027	0.00155		
AB x BG	1	0.00007	0.00022	0.00098		
Coefficient of						
Variation, %		3.94	4.28	3.77		

Appendix Table 12. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period - Experiment 2.

			Phase 1		-	Phase 2			
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F		
1	1	0.704	1.458	0.483	0.686	1.818	0.378		
2	1	0.735	1.614	0.456	0.697	1.996	0.349		
3	1	0.701	1.532	0.457	0.742	2.118	0.35		
4	1	0.765	1.669	0.459	0.859	2.426	0.354		
1	2	0.641	1.358	0.471	0.645	1.944	0.331		
2	2	0.671	1.586	0.423	0.851	2.302	0.37		
3	2	0.735	1.592	0.462	0.754	2.265	0.333		
4	2	0.719	1.583	0.454	0.693	2.068	0.335		
1	3	0.693	1.501	0.462	0.706	2.082	0.339		
2	3	0.605	1.404	0.431	0.742	2.026	0.366		
3	3	0.712	1.418	0.502	0.746	2.211	0.337		
4	3	0.639	1.508	0.424	0.865	2.724	0.318		
1	4	0.717	1.674	0.429	0.835	2.249	0.371		
2	4	0.854	1.971	0.433	0.864	2.598	0.332		
3	4	0.745	1.554	0.479	0.649	1.799	0.361		
4	4	0.731	1.564	0.468	0.864	2.592	0.333		
1	5	0.757	1.687	0.449	0.832	2.348	0.355		
2	5	0.721	1.580	0.456	0.854	2.288	0.373		
3	5	0.701	1.379	0.508	1.088	2.508	0.434		
4	5	0.822	1.788	0.46	0.903	2.265	0.399		
1	6	0.831	1.759	0.472	0.746	2.245	0.332		
2	6	0.770	1.624	0.475	0.831	2.381	0.349		
3	6	0.764	1.599	0.478	0.838	2.370	0.353		
4	6	0.666	1.397	0.477	0.754	2.008	0.376		

Appendix Table 13. Means for average daily gain, average daily feed intake, and gain: feed for Finisher Phase 1 and Phase 2 - Experiment 2.

Trt 1: negative control diet (0.30% cornstarch).

Trt 2: diet + 0.10% chlortetracycline + 0.20% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.10% cornstarch.

Trt 4: diet + 0.20% beta-glucan + 0.10% chlortetracycline.

		Mean Squares						
		Phase 1				Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	23							
Error	15	0.00331	0.02028	0.00038	0.00760	0.06453	0.00054	
Repetition	5	0.00650	0.02460	0.00037	0.01786	0.03962	0.00134	
Treatment	3	0.00001	0.01407	0.00130	0.00763	0.05718	0.00013	
No AB vs AB	1	3.70E-07	0.02516	0.00232	0.01084	0.12284	0.00002	
No BG vs BG	1	4.00E-08	0.01670	0.00147	0.00905	0.04833	0.00006	
AB x BG	1	0.00004	0.00036	0.00011	0.00299	0.00036	0.00031	
Coefficient of								
Variation, %		7.94	9.04	4.25	10.98	11.37	6.53	

Appendix Table 14. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 1 and Phase 2 - Experiment 2.

			Phase 3			Overall				
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F			
1	1	0.632	2.403	0.263	0.666	1.979	0.337			
2	1	0.667	2.274	0.294	0.695	2.010	0.346			
3	1	0.740	2.624	0.282	0.729	2.177	0.335			
4	1	0.835	2.813	0.297	0.729	2.377	0.345			
1	2	0.742	2.377	0.312	0.682	1.909	0.358			
2	2	0.836	2.860	0.292	0.781	2.270	0.344			
3	2	0.754	2.640	0.286	0.747	2.176	0.344			
4	2	0.785	2.725	0.288	0.740	2.159	0.343			
1	3	0.757	2.612	0.29	0.722	2.087	0.346			
2	3	0.646	2.324	0.278	0.654	1.923	0.340			
3	3	0.701	2.801	0.25	0.716	2.162	0.331			
4	3	0.723	2.884	0.251	0.717	2.260	0.317			
1	4	0.696	2.633	0.264	0.740	2.262	0.327			
2	4	0.915	2.935	0.312	0.884	2.576	0.343			
3	4	0.966	2.840	0.34	0.816	2.190	0.372			
4	4	0.773	2.562	0.302	0.787	2.295	0.343			
1	5	0.808	2.635	0.307	0.796	2.249	0.354			
2	5	0.818	2.611	0.313	0.792	2.189	0.362			
3	5	0.956	2.874	0.333	0.894	2.276	0.393			
4	5	0.838	2.593	0.323	0.846	2.248	0.376			
1	6	0.873	2.633	0.332	0.824	2.229	0.370			
2	6	0.934	2.840	0.329	0.849	2.294	0.370			
3	6	0.977	2.998	0.326	0.864	2.335	0.370			
4	6	0.926	2.529	0.366	0.789	1.995	0.395			

Appendix Table 15. Means for average daily gain, average daily feed intake, and gain: feed for Finisher Phase 3 and the entire grow-finish stage - Experiment 2.

Trt 1: negative control diet (0.30% cornstarch).

Trt 2: diet + 0.10% chlortetracycline + 0.20% cornstarch.

Trt 3: diet + 0.20% beta-glucan + 0.10% cornstarch.

Trt 4: diet + 0.20% beta-glucan + 0.10% chlortetracycline.

		Mean Squares						
		Phase 3				Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	23							
Error	15	0.00486	0.03770	0.00043	0.00148	0.02318	0.00019	
Repetition	5	0.02908	0.02585	0.00256	0.01510	0.02904	0.00119	
Treatment	3	0.00978	0.06328	0.00012	0.00326	0.01461	0.00009	
No AB vs AB	1	0.00037	0.00060	0.00015	0.00019	0.01330	0.00001	
No BG vs BG	1	0.01760	0.12702	0.00014	0.00348	0.01887	0.00019	
AB x BG	1	0.01135	0.06222	0.00007	0.00611	0.01166	0.00006	
Coefficient of								
Variation, %		8.67	7.28	6.85	5.01	6.94	3.92	

Appendix Table 16. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Finisher Phase 3 and the entire grow-finish stage - Experiment 2.

		5	<u> </u>	
			Nursery-Finisher	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F
1	1	0.609	1.554	0.392
2	1	0.643	1.561	0.412
3	1	0.659	1.684	0.392
4	1	0.738	1.839	0.401
1	2	0.614	1.486	0.413
2	2	0.703	1.749	0.402
3	2	0.666	1.672	0.398
4	2	0.682	1.679	0.406
1	3	0.639	1.607	0.398
2	3	0.591	1.491	0.397
3	3	0.653	1.660	0.393
4	3	0.695	1.742	0.399
1	4	0.654	1.687	0.388
2	4	0.775	1.934	0.401
3	4	0.774	1.789	0.432
4	4	0.699	1.737	0.402
1	5	0.690	1.675	0.412
2	5	0.693	1.642	0.422
3	5	0.773	1.710	0.452
4	5	0.747	1.695	0.441
1	6	0.706	1.644	0.430
2	6	0.729	1.710	0.426
3	6	0.738	1.745	0.423
4	6	0.667	1.494	0.446

Appendix Table 17. Means for average daily gain, average daily feed intake, and gain:feed from nursery to finisher stage - Experiment 2.

Trt 1: negative control diet.

Trt 2: diet + antibiotic.

Trt 3: diet + beta-glucan.

Trt 4: diet + beta-glucan + antibiotic.

intuke, una guiniteea ny		ministier stug	e Experiment 2	•				
	_	Mean Squares						
		Nursery-Finisher						
Source	d.f.	ADG	ADFI	G:F				
Total	23							
Error	15	0.00167	0.01077	0.00016				
Repetition	5	0.00507	0.01343	0.00100				
Treatment	3	0.00415	0.01231	0.00014				
No AB vs AB	1	0.00146	0.00540	0.00004				
No BG vs BG	1	0.00825	0.02077	0.00035				
AB x BG	1	0.00275	0.01075	0.00002				
Coefficient of								
Variation, %		5.92	6.20	3.03				

Appendix Table 18. Analysis of variance for average daily gain, average daily feed intake, and gain:feed from nursery to finisher stage - Experiment 2.

		Carcass						
Trt	Rep	HCW	10th rib	LMA	FFLC			
1	1	180.00	1.91	44.58	53.88			
2	1	192.50	2.29	45.42	51.86			
3	1	195.00	2.29	48.52	52.44			
4	1	207.50	2.41	48.00	51.38			
1	2	179.00	2.67	42.52	49.81			
2	2	205.00	3.30	42.39	46.56			
3	2	191.50	2.54	44.71	50.43			
4	2	175.00	2.03	42.97	52.99			
1	3	181.00	1.91	41.94	52.95			
2	3	177.00	1.40	48.71	57.42			
3	3	178.50	2.16	43.35	52.08			
4	3	188.50	2.54	48.58	51.44			
1	4	196.00	1.91	49.81	54.34			
2	4	203.50	2.41	44.52	50.66			
3	4	176.50	2.16	45.81	52.97			
4	4	200.00	2.16	42.71	51.46			
1	5	186.50	2.29	43.23	51.42			
2	5	184.00	1.78	42.71	53.64			
3	5	192.50	2.29	39.68	50.33			
4	5	195.50	2.03	42.00	51.96			
1	6	171.50	2.29	36.97	50.09			
2	6	195.50	3.05	39.61	46.94			
3	6	191.50	3.94	39.68	43.14			
4	6	172.50	1.91	40.84	52.99			

Appendix Table 19. Means for hot carcass weight, 10th rib backfat, longissimus muscle area, and fat-free lean carcass of pigs - Experiment 2.

Trt 1: negative control diet.

Trt 2: diet + antibiotic.

Trt 3: diet + beta-glucan.

Trt 4: diet + beta-glucan + antibiotic.

	_		Mean Square	S			
		Carcass					
Source	d.f.	10th Rib	LMA	FFLC			
Total	23						
Error	14	0.12019	6.47772	3.05062			
Repetition	5	0.60646	29.25478	20.52457			
Treatment	3	0.23956	0.79721	5.91244			
No AB vs AB	1	0.51389	1.67620	14.21726			
No BG vs BG	1	0.02598	0.76544	0.51705			
AB x BG	1	0.12498	0.00637	1.86262			
HCW	1	2.04557	0.20374	57.40974			
Coefficient of							
Variation, %		14.95	5.82	3.40			

Appendix Table 20. Analysis of variance for 10th rib backfat, longissimus muscle area, and fat-free lean carcass of pigs - Experiment 2.

			Phase 1			Phase 2				
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F			
1	1	0.194	0.288	0.674	0.349	0.621	0.562			
2	1	0.195	0.260	0.750	0.403	0.638	0.632			
3	1	0.168	0.226	0.742	0.344	0.512	0.672			
4	1	0.152	0.214	0.711	0.372	0.579	0.642			
5	1	0.209	0.276	0.757	0.417	0.644	0.647			
1	2	0.183	0.216	0.849	0.346	0.531	0.653			
2	2	0.182	0.237	0.765	0.426	0.609	0.700			
3	2	0.207	0.249	0.832	0.374	0.544	0.687			
4	2	0.196	0.257	0.763	0.345	0.498	0.694			
5	2	0.194	0.228	0.849	0.353	0.537	0.658			
1	3	0.157	0.220	0.711	0.306	0.539	0.568			
2	3	0.149	0.188	0.797	0.404	0.579	0.697			
3	3	0.207	0.233	0.887	0.253	0.471	0.537			
4	3	0.146	0.192	0.759	0.306	0.477	0.642			
5	3	0.112	0.169	0.662	0.304	0.440	0.690			
1	4	0.193	0.245	0.790	0.313	0.538	0.582			
2	4	0.168	0.261	0.643	0.324	0.583	0.556			
3	4	0.188	0.255	0.738	0.350	0.550	0.636			
4	4	0.097	0.167	0.579	0.311	0.514	0.605			
5	4	0.191	0.258	0.741	0.358	0.559	0.640			
1	5	0.133	0.168	0.791	0.274	0.490	0.558			
2	5	0.135	0.163	0.830	0.303	0.471	0.644			
3	5	0.126	0.167	0.753	0.281	0.432	0.650			
4	5	0.122	0.153	0.795	0.264	0.464	0.569			
5	5	0.090	0.120	0.751	0.340	0.388	0.876			
1	6	0.147	0.173	0.850	0.299	0.469	0.638			
2	6	0.153	0.184	0.832	0.277	0.436	0.635			
3	6	0.192	0.202	0.955	0.278	0.446	0.623			
4	6	0.178	0.197	0.901	0.320	0.527	0.606			
5	6	0.156	0.195	0.802	0.346	0.508	0.680			
1	7	0.187	0.252	0.739	0.371	0.638	0.581			
2	7	0.135	0.192	0.702	0.333	0.575	0.579			
3	7	0.158	0.220	0.721	0.442	0.616	0.718			
4	7	0.140	0.188	0.748	0.336	0.582	0.577			
5	7	0.171	0.233	0.733	0.298	0.584	0.511			
1	8	0.165	0.239	0.689	0.298	0.581	0.514			
2	8	0.150	0.215	0.700	0.350	0.563	0.622			
3	8	0.133	0.194	0.689	0.323	0.499	0.647			
4	8	0.147	0.211	0.696	0.306	0.490	0.624			
5	8	0.230	0.310	0.741	0.304	0.574	0.529			

Appendix Table 21. Means for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 – Experiment 3.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + 0.20% acidifier.

Trt 5: BD + 0.20% beta-glucan + 0.20% acidifier.

			Mean Squares						
			Phase 1			Phase 2			
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F		
Total	39								
Error	28	0.00074	0.00084	0.00298	0.00133	0.00153	0.00412		
Repetition	7	0.00226	0.00522	0.01719	0.00490	0.01450	0.00504		
Treatment	4	0.00088	0.00101	0.00245	0.00157	0.00352	0.00641		
Coefficient of									
Variation, %		16.65	13.43	7.18	10.97	7.34	10.24		

Appendix Table 22. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 - Experiment 3.

		Р	hase 1 & 2			Phase 3	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F
1	1	0.271	0.454	0.597	0.336	0.890	0.378
2	1	0.299	0.449	0.665	0.258	0.537	0.481
3	1	0.256	0.369	0.693	0.317	0.636	0.499
4	1	0.253	0.382	0.663	0.297	0.568	0.524
5	1	0.313	0.460	0.680	0.279	0.756	0.369
1	2	0.265	0.373	0.710	0.223	0.464	0.481
2	2	0.304	0.423	0.718	0.319	0.566	0.563
3	2	0.291	0.397	0.733	0.311	0.600	0.519
4	2	0.271	0.377	0.717	0.256	0.463	0.553
5	2	0.273	0.383	0.714	0.274	0.567	0.484
1	3	0.232	0.380	0.610	0.264	0.568	0.464
2	3	0.277	0.384	0.722	0.252	0.505	0.499
3	3	0.230	0.352	0.652	0.334	0.597	0.560
4	3	0.226	0.334	0.677	0.308	0.587	0.526
5	3	0.203	0.298	0.683	0.319	0.572	0.557
1	4	0.253	0.392	0.647	0.191	0.424	0.451
2	4	0.246	0.422	0.583	0.266	0.492	0.541
3	4	0.269	0.402	0.669	0.271	0.618	0.439
4	4	0.199	0.332	0.598	0.281	0.755	0.373
5	4	0.275	0.408	0.673	0.128	0.347	0.368
1	5	0.203	0.329	0.617	0.301	0.618	0.488
2	5	0.219	0.317	0.692	0.306	0.605	0.505
3	5	0.203	0.299	0.678	0.233	0.532	0.439
4	5	0.188	0.298	0.630	0.207	0.430	0.482
5	5	0.156	0.245	0.635	0.200	0.464	0.431
1	6	0.223	0.321	0.694	0.188	0.414	0.454
2	6	0.215	0.310	0.693	0.314	0.642	0.488
3	6	0.235	0.324	0.727	0.258	0.511	0.505
4	6	0.248	0.362	0.686	0.191	0.384	0.496
5	6	0.251	0.351	0.714	0.229	0.553	0.414
1	7	0.279	0.445	0.626	0.383	0.775	0.494
2	7	0.234	0.384	0.611	0.313	0.520	0.601
3	7	0.300	0.418	0.718	0.388	0.887	0.437
4	7	0.238	0.385	0.618	0.261	0.552	0.472
5	7	0.235	0.386	0.608	0.256	0.581	0.440
1	8	0.232	0.410	0.565	0.253	0.487	0.519
2	8	0.250	0.389	0.644	0.345	0.666	0.518
3	8	0.228	0.346	0.659	0.274	0.499	0.548
4	8	0.227	0.350	0.646	0.206	0.444	0.464
5	8	0.266	0.442	0.603	0.401	0.775	0.517

Appendix Table 23. Means for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 – Experiment 3.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + 0.20% acidifier.

Trt 5: BD + 0.20% beta-glucan + 0.20% acidifier.

			Mean Squares						
		Р	Phases 1 & 2			Phase 3			
Source	d.f.	ADG	ADFI	G:F		ADG	ADFI	G:F	
Total	39								
Error	28	0.00056	0.00099	0.00091		0.00283	0.01542	0.00204	
Repetition	7	0.00388	0.00860	0.00569		0.00560	0.02135	0.00539	
Treatment	4	0.00068	0.00175	0.00350		0.00370	0.00794	0.00676	
Coefficient of									
Variation, %		9.64	8.46	4.56		19.35	21.74	9.34	

Appendix Table 24. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 - Experiment 3.

	cu for the	entite +2-a perioa - LA	perment 5.	
			Overall	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F
1	1	0.293	0.600	0.489
2	1	0.286	0.478	0.597
3	1	0.276	0.458	0.603
4	1	0.267	0.441	0.607
5	1	0.302	0.559	0.540
1	2	0.251	0.404	0.622
2	2	0.309	0.471	0.656
3	2	0.298	0.464	0.641
4	2	0.266	0.406	0.654
5	2	0.274	0.444	0.617
1	3	0.242	0.443	0.547
2	3	0.268	0.424	0.633
3	3	0.265	0.434	0.610
4	3	0.253	0.418	0.606
5	3	0.241	0.387	0.622
1	4	0.233	0.402	0.579
2	4	0.252	0.445	0.567
3	4	0.270	0.474	0.569
4	4	0.226	0.469	0.482
5	4	0.226	0.388	0.582
1	5	0.236	0.425	0.555
2	5	0.248	0.413	0.601
3	5	0.213	0.377	0.566
4	5	0.194	0.341	0.571
5	5	0.168	0.308	0.547
1	6	0.211	0.352	0.600
2	6	0.248	0.421	0.589
3	6	0.243	0.386	0.629
4	6	0.229	0.369	0.621
5	6	0.244	0.419	0.582
1	7	0.313	0.555	0.564
2	7	0.260	0.429	0.606
3	7	0.330	0.574	0.574
4	7	0.246	0.440	0.559
5	7	0.242	0.451	0.536
1	8	0.239	0.436	0.548
2	8	0.282	0.481	0.586
3	8	0.243	0.397	0.613
4	8	0.220	0.382	0.576
5	8	0.311	0.553	0.563

Appendix Table 25. Means for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period - Experiment 3.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + 0.20% acidifier.

Trt 5: BD + 0.20% beta-glucan + 0.20% acidifier.

		Mean Squares					
			Overall				
Source	d.f.	ADG	ADFI	G:F			
Total	39						
Error	28	0.00060	0.00260	0.00078			
Repetition	7	0.00321	0.01037	0.00381			
Treatment	4	0.00135	0.00239	0.00247			
Coefficient of							
Variation, %		9.58	11.64	4.77			

Appendix Table 26. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period - Experiment 3.

			Phase 1		Phase 2	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg) ADFI (kg) G:F	:F
1	1	0.184	0.311	0.592	0.386 0.635 0.60	508
2	1	0.209	0.310	0.673	0.390 0.671 0.58	581
3	1	0.182	0.285	0.638	0.427 0.684 0.62	524
4	1	0.240	0.319	0.752	0.419 0.648 0.64	548
5	1	0.182	0.300	0.605	0.373 0.616 0.60	505
1	2	0.173	0.287	0.602	0.350 0.612 0.57	571
2	2	0.157	0.280	0.562	0.480 0.785 0.61	511
3	2	0.192	0.299	0.643	0.396 0.637 0.62	522
4	2	0.169	0.304	0.558	0.401 0.663 0.60	505
5	2	0.200	0.331	0.604	0.433 0.676 0.64	540
1	3	0.167	0.252	0.662	0.377 0.634 0.59	595
2	3	0.159	0.308	0.515	0.381 0.607 0.62	529
3	3	0.188	0.284	0.660	0.420 0.628 0.66	569
4	3	0.189	0.346	0.547	0.383 0.669 0.57	573
5	3	0.187	0.304	0.613	0.372 0.619 0.60	502
1	4	0.128	0.222	0.575	0.441 0.712 0.61	519
2	4	0.178	0.277	0.641	0.370 0.551 0.67	571
3	4	0.150	0.245	0.611	0.360 0.572 0.62	529
4	4	0.152	0.237	0.639	0.410 0.644 0.63	536
5	4	0.178	0.235	0.760	0.326 0.452 0.72	722

Appendix Table 27. Means for average daily gain, average daily feed intake, and gain: feed for Phase 1 and Phase 2 - Experiment 4.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + probiotic.

			Mean Squares					
			Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	19							
Error	12	0.00040	0.00048	0.00433	0.00154	0.00414	0.00106	
Repetition	3	0.00148	0.00426	0.00457	0.00093	0.00702	0.00235	
Treatment	4	0.00040	0.00072	0.00160	0.00061	0.00294	0.00121	
Coefficient of								
Variation, %		11.23	7.67	10.56	9.93	10.12	5.23	

Appendix Table 28. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 - Experiment 4.

		Ph	nases 1 & 2			Phase 3			
Trt	Rep	ADG (kg)	ADFI (kg)	G:F	ADG (kg)	ADFI (kg)	G:F		
1	1	0.285	0.473	0.603	0.318	0.673	0.472		
2	1	0.300	0.491	0.611	0.363	0.621	0.584		
3	1	0.305	0.484	0.629	0.326	0.796	0.410		
4	1	0.330	0.484	0.682	0.367	0.715	0.514		
5	1	0.277	0.458	0.606	0.378	0.566	0.669		
1	2	0.262	0.450	0.581	0.333	0.855	0.390		
2	2	0.309	0.518	0.597	0.362	0.758	0.478		
3	2	0.295	0.468	0.629	0.368	0.882	0.417		
4	2	0.285	0.483	0.590	0.315	0.796	0.395		
5	2	0.317	0.504	0.629	0.317	0.697	0.455		
1	3	0.272	0.443	0.614	0.282	0.714	0.395		
2	3	0.270	0.457	0.591	0.309	0.732	0.422		
3	3	0.304	0.456	0.666	0.268	0.630	0.426		
4	3	0.286	0.508	0.564	0.263	0.644	0.409		
5	3	0.280	0.461	0.606	0.318	0.672	0.473		
1	4	0.267	0.439	0.607	0.205	0.717	0.286		
2	4	0.274	0.414	0.661	0.248	0.523	0.474		
3	4	0.255	0.409	0.623	0.222	0.510	0.436		
4	4	0.274	0.430	0.637	0.224	0.548	0.408		
5	4	0.252	0.343	0.735	0.227	0.466	0.488		

Appendix Table 29. Means for average daily gain, average daily feed intake, and gain: feed for Phases 1 & 2 combined and Phase 3 - Experiment 4.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + probiotic.

			Mean Squares					
		Р	hases 1 &	2		Phase 3		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	19							
Error	12	0.00029	0.00072	0.00144	0.00044	0.00453	0.00233	
Repetition	3	0.00119	0.00626	0.00237	0.01633	0.04996	0.01420	
Treatment	4	0.00030	0.00081	0.00119	0.00083	0.01092	0.01188	
Coefficient of								
Variation, %		5.99	5.84	6.08	7.00	9.96	10.73	

Appendix Table 30. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined and Phase 3 - Experiment 4.

U		1	Overall	
Trt	Rep	ADG (kg)	ADFI (kg)	G:F
1	1	0.296	0.540	0.548
2	1	0.321	0.534	0.600
3	1	0.312	0.588	0.530
4	1	0.342	0.561	0.611
5	1	0.311	0.494	0.630
1	2	0.286	0.585	0.488
2	2	0.326	0.595	0.548
3	2	0.319	0.606	0.527
4	2	0.295	0.587	0.502
5	2	0.317	0.568	0.558
1	3	0.276	0.533	0.517
2	3	0.283	0.549	0.515
3	3	0.292	0.514	0.568
4	3	0.279	0.553	0.504
5	3	0.292	0.532	0.550
1	4	0.248	0.525	0.472
2	4	0.265	0.450	0.589
3	4	0.244	0.443	0.552
4	4	0.259	0.465	0.557
5	4	0.244	0.385	0.635

Appendix Table 31. Means for average daily gain, average daily feed intake, and gain: feed for the entire 42-d period - Experiment 4.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + probiotic.

-		Mean Squares						
		Overall						
Source	d.f.	ADG	ADFI	G:F				
Total	19							
Error	12	0.00015	0.00083	0.00099				
Repetition	3	0.00420	0.01574	0.00380				
Treatment	4	0.00028	0.00169	0.00403				
Coefficient of								
Variation, %		4.21	5.42	5.73				

Appendix Table 32. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 42-d period - Experiment 4.

11			0 1	1
Trt	Rep	Day 0	Day 28	Day 42
1	1	8.91	34.49	59.23
2	1	7.78	27.41	77.68
3	1	6.55	44.28	47.54
4	1	11.92	33.86	51.02
5	1	7.03	45.15	74.89
1	2	12.31	31.10	34.35
2	2	10.15		
3	2	10.02	24.04	27.06
4	2	6.25	18.20	27.37
5	2	9.05	24.97	36.00
1	3	7.82	47.74	70.86
2	3	6.88	31.30	39.24
3	3	6.11	22.57	34.43
4	3	10.26	35.35	84.65
5	3	14.38	55.16	59.75
1	4	11.31	29.99	43.10
2	4	7.13	36.18	82.41
3	4	8.90	34.32	73.98
4	4		44.21	61.16
5	4	9.30	50.57	55.01

Appendix Table 33. Means for the IgA serum protein - Experiment 4.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + probiotic.

		Mean Squares				
		day of collection				
Source	d.f.	0	28	42		
Total	18					
Error	11	4.20854	71.49875	358.81996		
Repetition	3	8.61112	274.20250	310.81690		
Treatment	4	5.41946	60.56370	373.51230		
Coefficient of						
Variation, %		22.65	23.95	34.62		

Appendix Table 34. Analysis of variance for IgA serum protein - Experiment 4.

11			0 1	1
Trt	Rep	Day 0	Day 28	Day 42
1	1	521.03	447.00	654.97
2	1	549.60	372.80	491.22
3	1	254.06	328.17	395.57
4	1	437.72	236.72	399.22
5	1	594.30	359.03	590.63
1	2	653.30	354.63	424.93
2	2			
3	2	621.77	275.20	374.43
4	2	355.63	251.03	348.73
5	2	386.37	273.27	374.40
1	3	491.30	350.67	441.27
2	3	434.12	257.65	251.33
3	3	534.87	278.63	440.43
4	3	625.87	323.10	574.50
5	3	416.83	751.48	589.17
1	4		575.75	572.20
2	4	458.50	378.50	534.00
3	4	554.63	299.67	405.97
4	4	897.28	330.00	477.30
5	4		547.38	438.50

Appendix Table 35. Means for the IgG serum protein - Experiment 4.

Trt 2: BD + 0.25% carbadox.

Trt 3: BD + 0.20% beta-glucan.

Trt 4: BD + probiotic.

			Mean Squares	
	_		day of collection	n
Source	d.f.	0	14	28
Total	18			
Error	11	20412.46050	17147.86020	7496.23970
Repetition	3	3355.95950	10005.10717	9865.61724
Treatment	4	34938.57410	21300.71696	18981.95134
Coefficient of				
Variation, %		27.64	35.59	18.74

Appendix Table 36. Analysis of variance for IgG serum protein - Experiment 4.

				Phase 1		i	Phase 2	
Exp	Trt	Rep	ADG (kg)	ADFI (kg)	G:F	 ADG (kg)	ADFI (kg)	G:F
1	NC	1	0.144	0.262	0.550	0.355	0.548	0.649
1	AB	1	0.155	0.304	0.510	0.431	0.612	0.704
1	BG	1	0.194	0.300	0.646	0.445	0.681	0.654
1	NC	2	0.150	0.228	0.658	0.370	0.531	0.696
1	AB	2	0.118	0.183	0.641	0.342	0.498	0.687
1	BG	2	0.141	0.219	0.644	0.330	0.469	0.702
1	NC	3	0.166	0.291	0.572	0.257	0.411	0.625
1	AB	3	0.130	0.320	0.408	0.296	0.455	0.651
1	BG	3	0.116	0.292	0.398	0.303	0.450	0.673
1	NC	4	0.135	0.307	0.439	0.236	0.383	0.617
1	AB	4	0.168	0.240	0.698	0.328	0.492	0.667
1	BG	4	0.106	0.239	0.442	0.260	0.395	0.659
1	NC	5	0.207	0.302	0.687	0.408	0.565	0.723
1	AB	5	0.256	0.323	0.793	0.537	0.687	0.781
1	BG	5	0.296	0.370	0.800	0.504	0.666	0.757
1	NC	6	0.215	0.264	0.813	0.443	0.625	0.709
1	AB	6	0.213	0.286	0.743	0.400	0.602	0.664
1	BG	6	0.209	0.294	0.710	0.434	0.611	0.710
1	NC	7	0.179	0.253	0.707	0.415	0.542	0.766
1	AB	7	0.217	0.306	0.709	0.415	0.579	0.716
1	BG	7	0.183	0.271	0.674	0.379	0.612	0.619
1	NC	8	0.182	0.291	0.625	0.392	0.556	0.705
1	AB	8	0.162	0.226	0.715	0.387	0.522	0.741
1	BG	8	0.159	0.238	0.668	0.346	0.517	0.670
2	NC	1	0.157	0.222	0.707	0.296	0.448	0.661
2	AB	1	0.148	0.216	0.686	0.346	0.515	0.671
2	BG	1	0.201	0.254	0.791	0.297	0.459	0.649
2	NC	2	0.153	0.227	0.673	0.287	0.447	0.643
2	AB	2	0.178	0.272	0.655	0.330	0.512	0.644
2	BG	2	0.132	0.191	0.689	0.324	0.452	0.718
2	NC	3	0.163	0.227	0.719	0.283	0.415	0.683
2	AB	3	0.168	0.230	0.729	0.272	0.401	0.680
2	BG	3	0.154	0.200	0.771	0.314	0.429	0.730
2	NC	4	0.138	0.207	0.670	0.457	0.630	0.726
2	AB	4	0.149	0.242	0.617	0.520	0.701	0.741
2	BG	4	0.146	0.221	0.660	0.449	0.649	0.693
2	NC	5	0.123	0.202	0.610	0.383	0.585	0.655
2	AB	5	0.133	0.216	0.617	0.451	0.616	0.732
2	BG	5	0.133	0.245	0.544	0.398	0.603	0.659

Appendix Table 37. Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 from Experiment 1 to Experiment 4 (26 reps).

NC: negative control diet (BD).

AB: BD + 0.25% carbadox.

BG: BD + 0.20% beta-glucan.
		Phase 1					Phase 2			
Exp	Trt	Rep	ADG (kg)	ADFI (kg)	G:F	A	DG (kg)	ADFI (kg)	G:F	
2	NC	6	0.136	0.224	0.609		0.353	0.526	0.670	
2	AB	6	0.152	0.220	0.690		0.389	0.545	0.714	
2	BG	6	0.127	0.215	0.591		0.384	0.573	0.670	
3	NC	1	0.194	0.288	0.674		0.349	0.621	0.562	
3	AB	1	0.195	0.260	0.750		0.403	0.638	0.632	
3	BG	1	0.168	0.226	0.742		0.344	0.512	0.672	
3	NC	2	0.183	0.216	0.849		0.346	0.531	0.653	
3	AB	2	0.182	0.237	0.765		0.426	0.609	0.700	
3	BG	2	0.207	0.249	0.832		0.374	0.544	0.687	
3	NC	3	0.157	0.220	0.711		0.306	0.539	0.568	
3	AB	3	0.149	0.188	0.797		0.404	0.579	0.697	
3	BG	3	0.207	0.233	0.887		0.253	0.471	0.537	
3	NC	4	0.193	0.245	0.790		0.313	0.538	0.582	
3	AB	4	0.168	0.261	0.643		0.324	0.583	0.556	
3	BG	4	0.188	0.255	0.738		0.350	0.550	0.636	
3	NC	5	0.133	0.168	0.791		0.274	0.490	0.558	
3	AB	5	0.135	0.163	0.830		0.303	0.471	0.644	
3	BG	5	0.126	0.167	0.753		0.281	0.432	0.650	
3	NC	6	0.147	0.173	0.850		0.299	0.469	0.638	
3	AB	6	0.153	0.184	0.832		0.277	0.436	0.635	
3	BG	6	0.192	0.202	0.955		0.278	0.446	0.623	
3	NC	7	0.187	0.252	0.739		0.371	0.638	0.581	
3	AB	7	0.135	0.192	0.702		0.333	0.575	0.579	
3	BG	7	0.158	0.220	0.721		0.442	0.616	0.718	
3	NC	8	0.165	0.239	0.689		0.298	0.581	0.514	
3	AB	8	0.150	0.215	0.700		0.350	0.563	0.622	
3	BG	8	0.133	0.194	0.689		0.323	0.499	0.647	
4	NC	1	0.184	0.311	0.592		0.386	0.635	0.608	
4	AB	1	0.209	0.310	0.673		0.390	0.671	0.581	
4	BG	1	0.182	0.285	0.638		0.427	0.684	0.624	
4	NC	2	0.173	0.287	0.602		0.350	0.612	0.571	
4	AB	2	0.157	0.280	0.562		0.480	0.785	0.611	
4	BG	2	0.192	0.299	0.643		0.396	0.637	0.622	
4	NC	3	0.167	0.252	0.662		0.377	0.634	0.595	
4	AB	3	0.159	0.308	0.515		0.381	0.607	0.629	
4	BG	3	0.188	0.284	0.660		0.420	0.628	0.669	
4	NC	4	0.128	0.222	0.575		0.441	0.712	0.619	
4	AB	4	0.178	0.277	0.641		0.370	0.551	0.671	
4	BG	4	0.150	0.245	0.611		0.360	0.572	0.629	

Appendix Table 38. Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 from Experiment 1 to Experiment 4 (26 reps).

NC: negative control diet (BD).

AB: BD + 0.25% carbadox.

BG: BD + 0.20% beta-glucan.

		Mean Squares						
			Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F	
Total	77							
Error	44	0.00047	0.00061	0.00378	0.00136	0.00184	0.00155	
Experiment	3	0.00245	0.02043	0.09566	0.01283	0.03941	0.02968	
Rep(Exp.)	22	0.00233	0.00241	0.01739	0.00973	0.01566	0.00236	
Treatment	2	0.00023	0.00027	0.00169	0.00576	0.00430	0.00632	
Exp. x Trt.	6	0.00025	0.00044	0.00184	0.00015	0.00215	0.00183	
Coefficient of								
Variation, %		13.10	9.99	9.03	10.15	7.74	5.99	

Appendix Table 39. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 1 and Phase 2 - Experiment 1 to Experiment 4 (26 reps).

			Phases 1 & 2				
Exp	Trt	Rep	ADG (kg)	ADFI (kg)	G:F		
1	NC	1	0.250	0.405	0.617		
1	AB	1	0.293	0.458	0.640		
1	BG	1	0.320	0.491	0.651		
1	NC	2	0.260	0.380	0.685		
1	AB	2	0.230	0.341	0.675		
1	BG	2	0.236	0.345	0.684		
1	NC	3	0.212	0.339	0.625		
1	AB	3	0.213	0.350	0.610		
1	BG	3	0.209	0.330	0.635		
1	NC	4	0.183	0.316	0.578		
1	AB	4	0.248	0.373	0.664		
1	BG	4	0.183	0.298	0.613		
1	NC	5	0.308	0.434	0.710		
1	AB	5	0.396	0.505	0.784		
1	BG	5	0.400	0.518	0.772		
1	NC	6	0.329	0.444	0.740		
1	AB	6	0.306	0.444	0.689		
1	BG	6	0.321	0.453	0.710		
1	NC	7	0.297	0.398	0.748		
1	AB	7	0.316	0.442	0.714		
1	BG	7	0.281	0.442	0.635		
1	NC	8	0.287	0.424	0.677		
1	AB	8	0.274	0.374	0.733		
1	BG	8	0.253	0.377	0.670		
2	NC	1	0.227	0.335	0.677		
2	AB	1	0.247	0.365	0.676		
2	BG	1	0.249	0.356	0.699		
2	NC	2	0.220	0.337	0.654		
2	AB	2	0.254	0.392	0.648		
2	BG	2	0.228	0.321	0.709		
2	NC	3	0.223	0.321	0.695		
2	AB	3	0.220	0.316	0.698		
2	BG	3	0.234	0.315	0.744		
2	NC	4	0.298	0.418	0.712		
2	AB	4	0.335	0.472	0.709		
2	BG	4	0.298	0.434	0.685		
2	NC	5	0.253	0.393	0.643		
2	AB	5	0.292	0.416	0.702		
2	BG	5	0.266	0.424	0.626		

Appendix Table 40. Means of average daily gain, average daily feed intake, and gain: feed for Phases 1 & 2 combined from Experiment 1 to Experiment 4 (26 reps).

NC: negative control diet (BD).

AB: BD + 0.25% carbadox.

BG: BD + 0.20% beta-glucan.

~			Phases 1 & 2		
Exp	Trt	Rep	ADG (kg)	ADFI (kg)	G:F
2	NC	6	0.245	0.375	0.653
2	AB	6	0.271	0.383	0.707
2	BG	6	0.256	0.395	0.648
3	NC	1	0.271	0.454	0.597
3	AB	1	0.299	0.449	0.665
3	BG	1	0.256	0.369	0.693
3	NC	2	0.265	0.373	0.710
3	AB	2	0.304	0.423	0.718
3	BG	2	0.291	0.397	0.733
3	NC	3	0.232	0.380	0.610
3	AB	3	0.277	0.384	0.722
3	BG	3	0.230	0.352	0.652
3	NC	4	0.253	0.392	0.647
3	AB	4	0.246	0.422	0.583
3	BG	4	0.269	0.402	0.669
3	NC	5	0.203	0.329	0.617
3	AB	5	0.219	0.317	0.692
3	BG	5	0.203	0.299	0.678
3	NC	6	0.223	0.321	0.694
3	AB	6	0.215	0.310	0.693
3	BG	6	0.235	0.324	0.727
3	NC	7	0.279	0.445	0.626
3	AB	7	0.234	0.384	0.611
3	BG	7	0.300	0.418	0.718
3	NC	8	0.232	0.410	0.565
3	AB	8	0.250	0.389	0.644
3	BG	8	0.228	0.346	0.659
4	NC	1	0.285	0.473	0.603
4	AB	1	0.300	0.491	0.611
4	BG	1	0.305	0.484	0.629
4	NC	2	0.262	0.450	0.581
4	AB	2	0.309	0.518	0.597
4	BG	2	0.295	0.468	0.629
4	NC	3	0.272	0.443	0.614
4	AB	3	0.270	0.457	0.591
4	BG	3	0.304	0.456	0.666
4	NC	4	0.267	0.439	0.607
4	AB	4	0.274	0.414	0.661
4	BG	4	0.255	0.409	0.623

Appendix Table 41. Means of average daily gain, average daily feed intake, and gain:feed for Phase 1 & 2 combined from Experiment 1 to Experiment 4 (26 reps).

NC: negative control diet (BD).

AB: BD + 0.25% carbadox.

BG: BD + 0.20% beta-glucan.

	_	Mean Squares					
		Phase 1 & 2					
Source	d.f.	ADG	ADFI	G:F			
Total	77						
Error	44	0.00045	0.00063	0.00107			
Experiment	3	0.00425	0.02075	0.01206			
Rep(Exp.)	22	0.00432	0.00662	0.00364			
Treatment	2	0.00198	0.00178	0.00452			
Exp. x Trt.	6	0.00010	0.00068	0.00144			
Coefficient of							
Variation, %		8.01	6.31	4.91			

Appendix Table 42. Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 & 2 combined - Experiment 1 to Experiment 4 (26 reps).

VITA

Rodel Punzalan Cueno

Candidate for the Degree of

Master of Science

Thesis: EVALUATION OF BETA-GLUCAN, ANTIBIOTICS, AND ANTIMICROBIAL ALTERNATIVES ON GROWTH PERFORMANCE AND IMMUNOLOGICAL PARAMETERS IN WEANLING PIGS

Major Field: Animal Science

Biographical:

- Personal Data: Born in Laguna, Philippines, On April 1, 1979, the son of Vergilio and Arminda Cueno
- Education: Graduated from U.P. Rural High School in Laguna, Philippines in April 1996; received Bachelor of Science in Agriculture, major in Animal Science from University of the Philippines Los Baños, Laguna, Philippines in April 2000. Completed the requirements for the Master of Science degree in Animal Science at Oklahoma State University in July 2005.
- Experience: Employed as a Technical Assistant by Gladius Industries, Inc.; Employed as a Junior Animal Nutritionist by General Agri-Foods International, Inc.; Been a graduate research assistant at Oklahoma State University
- Professional Memberships: Gamma Sigma Delta Honor Society in Agriculture (U.P. System), member of Phi Sigma Biological Sciences Honor Society (Alpha Chi Chapter), Fulbright Association, and the American Society of Animal Science.

Name: Rodel Cueno

Date of Degree: July, 2005

Institution: Oklahoma State University

Location: Stillwater, Oklahoma

Title of Study: EVALUATION OF BETA-GLUCAN, ANTIBIOTICS, AND ANTIMICROBIAL ALTERNATIVES ON GROWTH PERFORMANCE AND IMMUNOLOGICAL PARAMETERS IN WEANLING PIGS

Pages in Study: 138

Candidate for the Degree of Master of Science

Major Field: Animal Science

Scope and Method of Study: Four experiments were conducted to evaluate the effects of beta-glucan (provided by Dong-Ahm BT, Seoul, South Korea), a product derived from yeast cell wall (*Saccharomyces cerevisiae*), along with a standard antibiotic and other antimicrobial alternatives (acidifier and a probiotic culture) on growth performance and immunological parameters in weanling pigs. Treatments were replicated with four to eight pens of five to eight pigs each. Initial BW ranged from 5.0 to 5.8 kg, and pigs were weaned at 19 to 21 d of age. In each experiment, pigs were fed in three dietary phases (1.6, 1.4 and 1.2% tLys) during the 42-d study. Pigs and feeders were weighed weekly to determine ADG, ADFI and G:F ratio. Blood samples were collected and analyzed in Exp. 1 and Exp. 4. Experiment 2 was continued to the growing-finishing phase for carcass traits measurements.

Findings and Conclusions: In Exp. 1, there were no differences (P > 0.10) in ADG, ADFI and G:F of pigs fed diets with either carbadox or 0.20% beta-glucan but inclusion of 0.40% beta-glucan reduced (P < 0.10) ADG. Addition of either 0.20% beta-glucan or carbadox lowered (P < 0.10) CRP level at d 14. In Exp. 2, there were no interactions (P > 0.10) between beta-glucan or carbadox inclusion though the addition of carbadox or beta-glucan increased (P < 0.05) ADG at the nursery phase. Carbadox lowered (P < 0.06) 10th rib fat and improved (P < 0.05) percent fat-free lean. In Exp. 3, supplementation of acidifier did not improve (P < P0.10) growth performance of pigs. Pigs fed beta-glucan had similar (P > 0.10) growth performance to pigs fed carbadox. However, the addition of beta-glucan improved (P < 0.10) growth performance as compared to inclusion of acidifier alone. Experiment 4 was similar to Exp. 3 except a probiotic culture was used. The addition of probiotic to diets with or without beta-glucan improved (P < 0.10) growth performance of pigs. Serum protein levels were variable across the dietary treatments. On the average across all experiments, the growth performance of pigs fed 0.20% beta-glucan was intermediate between carbadox and the negative control diet.

ADVISER'S APPROVAL: Dr. Scott D. Carter