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RELATIONSHIPS OF ENZOOTIC PNEUMONIA, ATROPHIC RHINITIS, AND ANTIBIOTIC FEED MEDICATIONS TO GROWTH PERFORMANCE IN PIGS

By

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ABSTRACT

RELATIONSHIPS OF ENZOOTIC PNEUMONIA, ATROPHIC RHINITIS, AND ANTIBIOTIC MEDICATIONS TO GROWTH PERFORMANCE IN PIGS

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The relationships of enzootic pneumonia (EP) and atrophic rhinitis (AR) lesions at slaughter, sequential periods of pig growth from weaning to slaughter, use of feed additive medications, and season were studied in four trials conducted over two and one half years in a farrow-to-finish swine production unit.

Conclusions made from this study included: 1) severity of EP and AR lesions were not significantly related to each other; 2) EP and AR lesions were most severe in pigs with lower daily gain during the growth periods just prior to slaughter; 3) less than 9% of the variation in days to 230 was explained by EP and AR lesions; 4) feed medication had no effect on days to 230 or the severity of EP and AR lesions.

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INTRODUCTION

Respiratory diseases can have a significant, negative impact on the efficiency and profitability of pig production as a result of depressed feed intake, less efficient feed conversion, reduced daily gain, and increased death loss. Among the numerous respiratory diseases of swine, enzootic pneumonia (EP), and atrophic rhinitis (AR) are of particular interest because of their high prevalence among and within swine herds worldwide, their perceived effects on growth performance, and their role as initiators of other respiratory diseases. Both EP and AR induce distinctive and easily measured lesions resulting in the perceived need of producers and their veterinarians to treat these problems, typically with feed grade antimicrobial compounds. However, the relationships of EP and AR to growth performance (GP), the interrelatedness of EP and AR, and the effectiveness of feed medications in controlling EP and AR are not well documented in the scientific literature and anecdotal experiences of producers and veterinarians tend to be equivocal.

Developing an improved understanding of these relationships is an important issue within the swine industry. The continually increasing scrutiny of using feed grade medications with respect to violative meat residues and consumers' desire for residue free pork may eventually result in the ban of most feed medications for disease control and growth performance enhancement.

The use of computer simulated growth models for designing and implementing nutrition programs is becoming more commonplace. Because most of these growth models are feed consumption driven, and both EP and AR may inhibit feed intake, the incorporation of disease factors into these models is apparent. Crenshaw (1986) stated that existing models are variable and incomplete because they either consider only environmental or nutritional inputs, or oversimplify the growth process in trying to simulate a total production unit. The ultimate growth model must include all of the major inputs that impact pig growth including nutritional, environmental, genetic, disease, and management factors.

The overall objective of the following study was to identify relationships between EP and AR measured at slaughter, and growth performance by phase of production. In addition, the effect of feed grade antibiotic medication on EP, AR, and GP was evaluated.

LITERATURE REVIEW

The following review summarizes the scientific literature pertinent to the objectives of the experimental study. Topics include the diagnosis of enzootic pneumonia and atrophic rhinitis with respect to their presence, severity and lesion measurement, the relationships of EP and AR on the growth performance of pigs, and the effect of feed grade antibiotic medications on growth performance and the severity of EP and AR.

Enzootic Pneumonia

Enzootic pneumonia is considered to be the world's most prevalent swine disease (Underdahl et al., 1980). By definition, enzootic pneumonia is a pneumonia of animals indigenous to a certain locality, analogous to an endemic disease in man. As used in swine medicine, the term describes a disease entity and provides a descriptive basis for the macroscopic lesions indicative of the disease. Enzootic pneumonia refers to a pneumonic condition consistently present in a population of swine and the presence of macroscopic lesions primarily characterized by antero-ventral consolidation of lung parenchyma. Affected areas are darker in color and firmer in palpable consistency compared to normal lung tissue. These same lesions have been given other descriptive terms as summarized by Jericho (1968). Over time and by further study, the term enzootic pneumonia has become the most accepted.

The lesions described above also are suggestive of Mycoplasma hyopneumoniae infection. However, the lesions themselves do not conclusively indicate their cause. McKean et al. (1979) investigated the diagnostic significance of macroscopic lung lesions in response to concerns of specific-pathogen-free (SPF) standards that classify swine herds as being free of M. hyopneumoniae. Macroscopic lesions were compared to serological tests (complement fixation and latex agglutination), organism isolation, and microscopic lesion evaluation. They found that evaluation of macroscopic lesions alone was not sufficient to accurately determine a herd's M. hyopneumoniae infection status. Armstrong et al. (1984) confirmed these findings and recommended using at least a combination of macroscopic and microscopic lesions to evaluate a swine herd's status for M. hyopneumoniae. However, the use of both macroscopic and microscopic lesion criteria incorrectly classified 32% of infected animals as negative.

In spite of the false negative diagnoses that can occur using only macroscopic lung lesions for diagnosis, further studies have been conducted to quantify the association between such lesions and the presence and extent of M. hyopneumoniae infection. Morrison et al. (1985a) found a positive correlation ($r=0.46$, $p<0.001$) between the extent of macroscopic pneumonia lesions and the extent of M. hyopneumoniae infection evaluated by fluorescent antibody testing. However, Pasteurella multocida and Haemophilus sp. infections significantly contributed to the severity of lesions. Additionally, the cause and extent of such lesions can be complicated by differences in evaluation methods, seasonal variations, management and environmental factors, and pig age at lung evaluation.

Enzootic pneumonia appears to be the best term for this discussion because EP describes the epidemiological pattern as opposed to being lesion or cause specific. Although this terminology also creates a lot of ambiguity in the study of pneumonia lesions in swine, the term EP provides an accurate, medically correct, and pattern specific term that can be applied to all swine pneumonia studies with some degree of mutual understanding and accuracy.

Many thorough and very detailed reviews of enzootic pneumonia exist. Pullar (1948) described the "catarrhal pneumonia" (red hepatization) especially affecting the cardiac and apical lung lobes. That description was applied in a discussion of infectious pneumonia of unknown etiology. Jericho (1968) provided a firm foundation for further study through his discussion of the pathogenesis of swine pneumonia. His discussion of the multifactorial nature of swine pneumonia includes a summary table of studies dating from 1931-1966. This summary pointedly illustrates that despite the specific agents studied, their epidemiological, anatomical, and histological manifestations were not specific for any particular etiology. That is perhaps the best information base for the use of the term enzootic pneumonia. It avoids any misuse of macroscopic lesions for specific diagnoses and it forewarns of the complexity of trying to delineate the specifics of any association between enzootic pneumonia and growth performance of pigs.

Evaluation for any association between EP and growth performance (GP) began with investigations of the incidence of EP within and among swine herds. Slaughter prevalence of pneumonia lesions was reported as far back as the 1930's (Lamont, 1938). In the mid 1950's, MacPherson and Shanks (1955) reported a prevalence of 55% for market hogs. Their results were comparable to earlier studies. In addition, they found

the prevalence of EP in slaughtered sows was only 6%. This large difference between market age pigs and sows initiated questions of age differences and indicated that lesion regression may affect the prevalence and severity of pneumonia at slaughter.

Bertschinger (1972) and Livingston (1972) performed studies that suggested lesions of enzootic pneumonia naturally regressed within two months after exposure to M. hyopneumoniae. However, Underdahl (1980) conducted a similarly designed study and found no evidence of lesion regression or recovery. Backstrom and Bremer (1976) and Flesja et al. (1980) reported a decrease in prevalence of pneumonia with increasing age and weight. They reported a peak prevalence from 25-65 kg (55-143 lb) body weight.

Table 1 summarizes reports on the relationship of enzootic pneumonia to growth performance (Morrison, 1985). Ten of the studies reported a decrease in average daily gain, six reported a decrease in feed efficiency, and eleven reported insignificant or inconsistent effects of pneumonia on growth performance.

Table 1. Summary of Reports: Association of Enzootic and Growth Performance in Swine

Author	Year	Study Method	Results
Betts et al.	1953	Exp. inoculation	↓ADG 25%; ↓FE 25%
Betts et al.	1955	Exp. inoculation	↓ADG 14%; ↓FE 17%
Shuman et al.	1956	Before & After One Herd	No Signif. Effect
Young et al.	1959	Before & After One Herd	↓ADG
Goodwin	1963	Before & After One Herd	↓ADG 5%
Englert et al.	1964	Exp. inoculation	No Signif. Effect
Bjorklund et al.	1965	Test Station	No Signif. Effect
Eikmeier et al.	1965	Observation One Herd	No Signif. Effect
Truijen	1967		↓FE 8.6%
Huhn	1970	Observation Test Station	↓ADG 14% (moderate-severe pneum.)
Schroder et al.	1971[a]		No Signif. Effect
Zimmerman et al.	1973	Exp. inoculation	ADG Insignif. ↓FE 3.0%
Lindqvist	1974	99 Herds	↓ADG (moderate-severe pneum.)
Backstrom et al.	1975	One Herd	No Signif. Effect
Braude et al.	1975	Before & After One Herd	↓ADG 5.6%; ↓FE 4.6%
Jericho et al.	1975	Test Station	No Signif. Effect
Lundeheim et al.	1979	Test Station	↓ADG with ↑ Pneum.
Muirhead	1979	Five Herds	↓ADG & FE
Zimmerman et al.	1982	Exp. inoculation	No Signif. Effect
Straw et al.	1983	Test Station	↓ADG with ↑ Pneum.
Takov et al.	1984	27 Herds	Inconsistent Effect
Morrison et al.	1985	4 Herds	No Signif. Effect

ADG = average daily gain; FE = feed efficiency; Before & After = measurements before and after the introduction of respiratory infectious agents.

[a]quoted by Plonait (1978)

(adapted from Morrison, 1985)

In addition to the studies listed in Table 1, others have investigated the relationship of enzootic pneumonia and growth. Willeberg et al. (1978) reported a slight and varying tendency of depressed gain in those pigs with pneumonia lesions at slaughter versus those with no lesions. They reported a "high" correlation between production parameters such as growth rate and the "severe" category of respiratory lesions at slaughter, but these correlations were not nearly as apparent when mild lesions were included. Unfortunately, no specific correlation figures or any growth performance data were provided in this report. Their overall conclusion was that productivity was affected more by clinical episodes of pneumonia than by subclinical respiratory disease assessed at slaughter.

Madsen (1982) used SPF pigs to study experimental Mycoplasma infections and found infected pigs had higher average daily gains than did uninfected controls. The conclusion was that the role of Mycoplasma as a pathogen, and thus enzootic pneumonia as an affecter of growth, had been overestimated.

Burch (1982) studied 30-70 kg. pigs from two production units and concluded that the major effect of EP was reduction in average daily gain (ADG). "Strong" negative correlation was noted between the high range of lung scores (40-55% involvement) and growth rate over the last month before slaughter. No correlation figures were reported but the decreased gain was statistically significant.

Pointon et al. (1985) reported two studies on enzootic pneumonia and growth. In the first study, naturally infected pigs had a 12.7% ($p<0.01$) decrease in growth rate from 50-85 kg (110-187 lb). In a second study, pigs from inoculated gilts had a 15.9% ($p<0.001$) decrease in growth rate from 18-85 kg (40-187 lb).

Goodwin (1971) addressed the economic aspects of EP in the British pork industry. He discussed three main effects whereby enzootic pneumonia could exert an economic impact on production: 1) depressed feed efficiency; 2) variable growth rates; 3) general debilitating effect. Even though actual monetary values related to prevalence and lesion severity were estimated, Goodwin acknowledged the limitations of these estimates due to inconsistent correlation between lesions at slaughter and growth performance, and between herd differences.

Pijoan et al. (1985) discussed the economics of EP and strongly suggested the need for considering all contributing production factors and detailed records before assigning any economic losses to EP. The need for detailed production records, complete diagnostic and epidemiological workup, and a standardized method of evaluating pneumonic lesions was addressed.

Straw et al. (1989) discussed an estimation of EP costs, and presented formulas and regression equations for calculating losses on an individual herd basis. These estimates were questionable in that they were developed from only a few of the studies reviewed in Table 1 and several other studies that evaluated antibacterial medications. Hence, study design and data collection variability limit the application of these equations to other production units. This concern was openly stated in their presentation and should warn of the direct extrapolation of economic losses, such as those presented in many trade journals, from one production unit to another.

This variation and lack of uniform applicability to the pork industry is in large part due to the variability in the design and execution of the studies that generated the information. All of the studies mentioned were retrospective in nature, and varied in

their design and method of pneumonia lesion evaluation. The most significant variation occurred with lung evaluation techniques. For example, among the studies reported in Table 1, Huhn (1970) used a six point scale, Lindqvist et al. (1974) used a two category method, Backstrom et al. (1975) used a three category system, Jericho et al. (1975) used four categories, and Straw et al. (1983) divided the total lung among the seven lobes (25% per diaphragmatic lobe and 10% for each of the other 5 lobes), evaluated the percent involvement in each individual lobe, and then calculated a total percent involvement.

Morrison et al. (1985) evaluated four different methods of analyzing lung scores by examining 560 pigs from 41 different herds. They evaluated; 1) assessment of individual lung percentage involvement with calculation of mean and standard deviation (S.D.) for each herd; 2) counting only those lungs with greater than a predetermined level of pneumonia and using that figure to calculate prevalence; 3) scoring only the worst, "maximally affected", lung in the herd sample; and 4) allocating lungs to categories of the extent of pneumonia. They concluded that the most informative method was assessing the percentage of lung involved and calculating a mean for the herd sample. And, further, "the more detailed the scoring system and the larger the sample size, the greater will be the degree of confidence in the interpretation."

Evolution of study design to a common, detailed evaluation method would make the data generated more meaningful and supportive to epidemiological efforts to specify disease patterns and correlate lesion severity to economic losses.

Atrophic Rhinitis

Atrophic rhinitis (AR) is an infectious disease of swine that results in varying degrees of nasal turbinate atrophy. The condition varies in severity from mild, internal turbinate atrophy to severe alteration of surrounding structures of the nasal, premaxillary or maxillary bones. The presence of AR in swine herds has been reported since 1830. A very complete review of the historical progression of AR was presented by Switzer and Farrington (1975).

Early etiological studies first reported Bordetella bronchiseptica as the causative agent (Switzer, 1956). At the same time, through the 1950's and into the early 1970's, there were multiple studies that found pure cultures of Pasteurella multocida caused similar turbinate atrophy. De Jong et al. (1980) discovered that certain strains of P. multocida produced a thermolabile toxin that caused severe turbinate atrophy. The interrelationship of B. bronchiseptica and P. multocida has been closely studied since that discovery.

Pedersen and Barford (1981) noted that challenging pigs with a combination of B. bronchiseptica and toxin producing P. multocida produced clinical AR that was much more severe compared to challenge with B. bronchiseptica alone. Further work by Elling and Pedersen (1983, 1984, 1985) investigated the link between toxigenic P. multocida and the severity of AR. They concluded that the P. multocida toxin enhances osteoclast activity and impairs osteoblast activity resulting in increased severity and persistence of turbinate atrophy.

Evaluation of the severity of AR has always involved methods to quantify the degree of turbinate atrophy. Clinical evaluations have included external signs such as

sneezing, tearing, and snout deformity. These clinical signs have been further evaluated by examining the turbinates using rhinoscopy in the live pig and snout cross sections at post mortem or slaughter. Such studies have led to the description of various scoring techniques and development of increasingly sophisticated methods of evaluation (Done, 1979, Done and Upcott, 1982, Done et al., 1984). Methods range from simple estimates by rhinoscopy (Shuman et al., 1956) to detailed post mortem analysis using computerized morphometry measurements (Done et al., 1984). The most commonly accepted method has been post mortem evaluation of nasal cross section at the level of the first upper premolar using measurement of the space between the floor of the nasal cavity and the ventral scroll of the ventral nasal turbinate on each side of the nasal septum (Runnels, 1982). These measurements are taken on a minimum of ten randomly selected animals per herd. Depending on herd size and frequency of evaluation, an even larger sample may be necessary (Pointon et al., 1990) to accurately estimate herd prevalence and severity. The measurements are then transformed into a scoring system of 0 (normal) to 5 (severe turbinate atrophy). This system was best publicized in the United States by the Elanco TRAC system (Elanco, 1985) but has existed as the Weybridge system in other countries for thirty years (Done et al., 1984).

A review of studies that investigated the relationship of AR and growth performance (ADG) was presented by Morrison (1985) (Table 2). Of the seventeen studies reviewed, nine noted decreased ADG related to the presence of AR. Seven studies reported no effect. One study (Giles et al., 1980) reported no significant effect on individual animals but an overall decrease in ADG in affected herds when compared with nonaffected herds. These studies all utilized at least a beginning and ending weight

for analysis, with no consistent pattern to weights taken between the beginning and end. Therefore, nothing can be stated about possible associations of AR and growth performance to particular phases of pig growth.

Table 2. Summary of Reports: Association of Atrophic Rhinitis and Growth Performance in Swine

Author	Year	Study Method	Results
Kristjansson, et al.	1955	One Herd	↓ ADG
Shuman et al.	1956	One Herd (284 pigs, 2 yrs)	↓ ADG
Young et al.	1959	One Herd	No Signif.
Earl et al.	1962	Test Station (1099 pigs, slaughter only 127)	↓ ADG
Bjorklund et al.	1965	Test Station	No Signif.
Pearce et al.	1967	Three Herds (875 pigs)	No Signif.
Fredeen et al.	1967	Two Herds	No Signif.
Backstrom et al.	1975	One Herd	↓ ADG 5%
Goodnow et al.	1979	Two Herds (Vaccine Trial)	↓ ADG
Arthur et al.	1980	One Herd	No Signif.
Jackson et al.	1982	Test Station	↓ ADG (0.02 kg/day)
Giles et al.	1980	Twelve Herds (Only 12-24 pigs/herd were used)	No Pig Effect ↓ ADG by herd
Pedersen et al.	1981	Vaccine Trial	↓ ADG with severe AR
Backstrom et al	1982	Five Herds	No Signif.
Pedersen et al.	1982	Vaccine Trial	↓ ADG
Straw et al.	1983	Test Station	No Signif.
Takov et al.	1984	Two Herds	↓ ADG in one herd

ADG = Average Daily Gain

(adapted from Morrison, 1985)

More recent studies also have reported variable effects on performance. Backstrom et al. (1985) studied seven farrow-to-finish herds and concluded that only severe AR adversely affected ADG and the magnitude of this effect varied between herds. Baalsrud (1987) studied nine herds and found AR affected pigs with moderate or severe lesions had significantly reduced growth rates compared to nonaffected pigs. Genetic factors may influence the severity of AR. Kennedy and Moxley (1980) reported increasing heterosis significantly decreased AR. Others have also reported that genetics influences AR (Smith, 1983; Popescu-Vifor and Militaru, 1986). However, differences between breeds generally were inconclusive, and heritabilities were low and variable (Jubb and Kennedy, 1970; Bendixen, 1971; Kennedy and Moxley, 1980).

In summary, AR is: 1) a multifactorial disease; 2) not etiologically specific; 3) not an all-or-nothing phenomenon. All considered, there is no simple, consistent, numerical relationship between AR and growth performance at this time. Consequently, determining AR's effects on growth performance and production economics will depend on structuring studies to extract the pertinent and significant information from the "comparison of the variably affected with variably normal populations" (Done, 1985).

Enzootic Pneumonia and Atrophic Rhinitis

If both enzootic pneumonia (EP) and atrophic rhinitis (AR) are multifactorial in nature, what is the possibility of quantifying any consistent relationships between the two diseases and the effect of either or both on the growth performance of pigs?

Empirical extrapolation of anatomical and physiological functions of the nasal turbinates suggests the possibility of a direct relationship between AR and EP. The

function of nasal turbinates is to prewarm and filter inspired air before it enters the lungs. Turbinates damaged by AR should be less effective in performing these functions and therefore allow more irritating air to reach the lungs, which in turn could create a better environment for EP or exacerbate preexisting pneumonic conditions. As logical as this association might appear, actual study of this association over the years has produced variable results. The results of eighteen studies dealing with the association of AR and EP are presented in Table 3.

Table 3 - Summary of Reports: Association of Enzootic Pneumonia, Atrophic Rhinitis and Growth Performance in Swine

Author	Year	Study Method	Results
Young et al	1959	One Herd (N=213)	EP = ↓ADG AR = NE EP:AR = NR
Bjorklund & Henrickson	1965	One Facility (2.5 yr) (N=320)	EP = NE AR = NE EP:AR = NE
Backstrom & Bremer	1978	10-15 Herds (2 groups/herd)	NA
Muirhead	1979	General Discussion	NA
Lundeheim	1979	Test Station (N=10,000)	NE
Flesja et al	1979	3 years; N = 33,000	EP freq. = 20-95% AR freq. = 1.5-45%
Flesja et al	1980	N = 350,000	EP:AR = + Cor. (no correlation numbers)
Flesja et al	1981	N = 350,000	Herd size R^2 20-40%
Backstrom et al	1982	Six Herds (N = 180)	EP (Only Y/N) AR = NE (3) ↓ADG (1) EP:AR No Cor.
Straw et al	1983	Test Station (N = 686)	EP ↓ADG, r = -0.26 AR = NE EP:AR = No Cor.
Flesja et al	1984	12 Herds; 3 years (N = 9800)	EP ↓ADG AR ↓ADG EP:AR = None Given
Takov et al	1984	27 Herds; 3 years (25-30/herd)	EP = NE r = .034 AR = NE r = .089 EP:AR r = .164 Individ. r = .492 Herd

Table 3 continued

Author	Year	Study Method	Results
Straw et al	1984	Test Station (N=831)	EP ↓ADG r = -0.25 AR = NE EP:AR = No Cor.
Morrison et al	1985	37 Herds (N=462)	EP Not Given AR Not Given EP:AR r = 0.177 Individ. r = 0.515 Herd
		(Age at slaughter on only 95 pigs.)	
Backstrom et al	1985	7 Herds (N=210)	EP Not Given AR ↓ADG EP:AR No Cor.
Nascimento et al	1986	Random Slaughter (N=1259)	EP Not Given AR Not Given EP:AR Not Given
		(AR presence = ↑ risk of bronchopneumonia 1.4 x)	
Turlington et al	1986	9 Herds (N=392)	EP ↓ADG AR ↓ADG EP:AR Not Given
		(Lung & Snout Score vs. Performance = R^2 = 0.2)	
Scheidt et al	1990	3 Herds (N=516)	EP = NE AR = NE EP:AR Not Given
		(ADG Finish vs Snout Score r = 0.17) (ADG Total vs Snout Score r = 0.16)	

EP = enzootic pneumonia; AR = atrophic rhinitis
freq. = frequency of occurrence (incidence)
ADG = average daily gain
N = number of animals studied
NE = no effect
NR = not reported NA = no analysis
Cor. = correlation
r = correlation coefficient ($p \leq 0.05$)
R^2 = r * r

The studies summarized varied with respect to experimental design, method of evaluating EP and AR lesions, statistical analysis, and reporting of results. Study designs included evaluation of single herds, multiple herds, and totally random data collection at slaughter facilities. The evaluation methods for EP varied from a simple present or absent score to a specific percentage of lung tissue involved. Likewise, AR evaluation went from a simple present or absent score to the complete 0 to 5 scoring system previously mentioned. Analysis and reporting of results ranged from no analysis at all to a presentation of correlation coefficients with their associated level of statistical significance.

Other than the associations mentioned in Table 3, the most common respiratory disease correlation reported was the positive correlation between pneumonia and herd size as studies in the mid 1970s when confinement production was expanding (Larson and Backstrom, 1974; Lindqvist, 1974; Backstrom and Bremer, 1976; Aalund et al., 1976). However, only a few of the studies summarized in Table 3 provided herd size information.

The most complete data set regarding evidence of diseases at slaughter and their interrelationships was generated by Norwegian researchers who evaluated more than 300,000 slaughtered pigs over approximately four years (Flesja et al., 1979; 1980; 1981; 1982; 1984). In these studies, all post-mortem lesion data were reported as frequency or incidence and were related to each other on that basis. Consequently, correlation coefficients could not be generated and no disease/growth relationships were studied. Such a large database could have provided sufficient observations for developing specific

inferences about disease and growth performance had individual pig performance data been available.

In the 1980 article of Flesja et al., the strongest disease/disease associations were:

1. Atrophic rhinitis is positively associated ($p < 0.001$) to all other recorded thoracic lesions (pneumonia, pleurisy, abscesses, pericarditis, etc.) and liver lesions ("white spots", perihepatitis), other than ascarid scars.
2. Moderate and severe pneumonia are associated with other thoracic lesions and with ascarid scars ($p < 0.001$), but not associated with AR.
3. All of the commonly occurring lesions decreased in frequency as the slaughter weight of the hogs increased.

In the 1981 article of Flesja et al., they collected lesion data from more than 90 individual herds and again found no association between EP and AR.

Only in very recent years have studies been undertaken to evaluate specific statistical relationships of EP, AR and growth performance. Straw et al. (1983, 1984) evaluated these relationships in a test station setting with multiple source pigs and one pig per source. Their 1983 data found EP correlated with decreased ADG ($r = -0.26$, $p=0.001$), AR had no effect on ADG ($r=0.026$, $p=0.54$), and no correlation between EP and AR ($r=-0.005$, $p=0.99$). Their 1984 study repeated the trial of 1983 and the results and conclusions were similar.

In contrast, Takov et al. (1984) studied 25-30 herds by evaluating 25-30 animals per herd. They found no association between EP, AR, or growth rate by individual

animal. The same results were obtained in 18 of the herds during the next year. However, on a herd basis EP and AR were positively correlated ($r=0.492$, $p<0.001$).

In 1985, Morrison et al. studied 37 herds, approximately 13 animals per herd, and found the same association between EP and AR as reported by Takov et al. (1984). There was weak association between AR and EP on an individual animal basis ($r=0.177$, $p<0.001$), but the association was fairly strong on a herd basis ($r=0.515$, $p<0.001$). There were no growth performance comparisons evaluated because accurate ages were available on only 95 of the 462 head studied.

Backstrom et al. (1985) studied seven herds, evaluating 30 animals per herd. They reported an association of AR with decreased ADG but saw no association between EP and growth performance, or EP and AR. However, the animals with moderate and severe EP lesions were eliminated from the analysis to "avoid the AR:EP combined effect".

Turlington et al. (1986) reported decreased ADG with "severe" cases of EP or AR in a study of 44 animals from each of nine farms. They also reported that snout and lung scores together explained 20% of the performance variation between pigs ($R^2=0.2$) and explained 40% of the performance differences between farms ($R^2=0.4$).

In the most current study, Scheidt et al. (1990) reported correlations of EP and AR to growth rate (days to market) at $r=0.15$ and $r=0.14$ respectively ($p<0.001$). Both were statistically significant in their level of association but may be of debatable biological significance because of the small r values. This study involved three herds and evaluated 117 to 213 animals per herd.

Considering all of these studies, the question arises, why is the expected biological and physiological relationship between EP and AR not statistically significant in all studies? Furthermore, why is the relationship of either or both to growth performance so variable and inconsistent when it is known that such entities compromise normal biological function and should therefore interfere with optimum growth?

What other variables contribute to the situation? How do pigs compensate for such biological trauma without adverse effects on growth? If pigs can truly compensate up to a certain level of disease severity, how can we identify those critical levels such that pigs can be specifically managed to avoid exceeding these thresholds so that growth performance is not adversely affected?

Growth and Performance

Growth performance of pigs is the basic denominator in establishing and evaluating the profitability of pork production units. Standards for expected daily gains and feed efficiencies have been established, continually revaluated, and changed/updated over time. Some of the currently accepted standards for growth performance are excerpted from Mayrose et al. (1985) and presented in Table 4.

Table 4. Rating Growth Performance of Pigs

	Rating		
	Excellent	Average	Poor
Production Parameter:			
Average Daily Gain (lb) 40 lb to market	> 1.4	1.2 - 1.4	> 1.2
Feed Efficiency 40 lb to market	< 3.4	3.4 - 3.8	> 3.8
Days to 230 Birth to market	< 182	182 - 227	> 227

(adapted from Mayrose et al., 1985)

Most of the past research on growth performance has focused on a wide range of nutritional and environmental factors and the resulting physiological impact on the biological responses of the pig, primarily growth rate. Investigations have explored various affecters of feed efficiency (FE) and average daily gain (ADG) in all phases of the pork production cycle. Scientific reports on these studies abound and are too numerous and varied in scope to specifically discuss here. In addition, the information is constantly being updated as specific details about the many factors that affect growth, namely genetics, nutrition, environment, management, and health, are being more specifically evaluated for their individual impact.

Genetic factors have been studied to support the continued improvement of seedstock for the subsequent improvement of pig performance in both reproductive and growth performance potentials. With recent changes in the pork industry structure, consumer demand to decrease animal fat consumption, and the development of major advances in lean growth biotechnologies, swine genetics is being studied with renewed intensity. Some studies, such as McLaren et al. (1985), continue to show that overall there are no highly significant differences in growth rates between purebred and crossbred sired pigs farrowed from crossbred F1 generation females.

Environmental studies continue as different types of confinement production facilities are evaluated and as different geographic locations are investigated as potential production areas. The impact of consumer concern over animal welfare will also exert increasing control on the type of housing systems used.

Nutritional studies will always be generated because of the varied alternative feedstuffs available for consideration as ingredients in swine diets. The basic

corn/soybean meal diet still is predominant, but changes in genetics, environment, and available feed ingredients provide more than ample opportunity and justification for continual study. In addition, the development of new feed additives requires additional studies as to how they can be used effectively in swine production.

Factors that have been evaluated in the least detail for their relationship to growth performance are management and health. Realizing the intangible intricacies and the extreme variability of these two factors, it is easy to understand that in the past the "proper management" of genetics, nutrition, and environment have been generally accepted as the method to optimize health and minimize any detrimental effect that disease might have on growth. But with the increasing sophistication of information management systems, the intricacies of management/health relationship to production, especially the economic relatedness of health to production efficiency, is of more critical interest. Developing a data base of sufficient size and detail for investigating the relationships of specific disease/management aspects to growth performance would be an expensive and formidable task given the number of other factors and potential interactions that impact growth performance on an individual pig and herd basis.

With sufficient design, detailed information, and proper analysis, the variables affecting growth performance can be evaluated and placed in proper perspective to each other in developing a more complete understanding of swine growth performance. As the data becomes more complete and accurate, the information can then be incorporated into mathematical models of growth that can be used to evaluate and predict the impact of any change in one or more factors on overall growth performance.

Feed Additives for Growth Enhancement

There exists numerous research reports in the scientific literature documenting the efficacy of feed additive antibacterials in pig diets. These reports cover the entire range of approved feed additives. Reported studies range from simple additive vs. no additive trials to more complex questions of feed additive interaction with various disease or environmental factors.

In general, antibacterial feed additives have been used extensively for thirty years or more and have played a major role in the pork production industry. Their primary indication for use has been improvement of average daily gain and feed efficiency.

Edmonds et al. (1985) briefly reviewed and further studied several commonly used antibacterials in the diets of weaned pigs. Their study addressed the question of whether feed additive antibacterials could effectively reduce or eliminate the post weaning "slump" that had been firmly established in other studies. Their studies found variable responses. However, starter diets continue to be the focal point of feed additive use due to the high stress of weaning and the generalized belief that antibiotic feeding has its greatest benefit under periods of stress or adverse production conditions.

Hays (1979) produced a technical report that gave an overview of antibacterial feed additives (AFA) used in the diets of grow/finish hogs (>40 lb. bodyweight). He reported that AFA often, but not always, significantly improved the rate and efficiency of gain depending on the conditions of the trial and the background of the pigs.

Cromwell et al. (1984) confirmed the variability caused by the previous background of the pigs. Studying a single antibacterial feed additive, they concluded that

failure to include an additive in finisher (> 120# bodyweight) diets following medication of grower phase (40-120# bodyweight) diets may result in the loss of growth enhancement realized during the grower phase. Nickelson (1985) reviewed this same issue and raised more questions about compensatory gains and the offsetting of performance enhancement derived from feed additives.

A major use of feed additive medications has been for treating or controlling overt disease situations. Several studies have demonstrated improved growth performance of pigs when AFA were used in relatively disease contaminated environments versus more sanitary ones. Most of these studies, as indicated in the review by Moser et al. (1985), were used as background information for further studies that were not related to a specific disease entity. No studies were found that attempted to address the complex situation of a combination of disease entities versus feed additive response.

Specific disease-related studies of AFA are most likely found among the documentation used by pharmaceutical manufacturers to gain regulatory approval for marketing the additives. These studies are too numerous and too specific to document and review and are often unavailable for independent review. Also, most of these studies were performed in a research setting that was not reflective of the production environment typical of commercial pork production. Simply reviewing the Feed Additive Compendium and understanding FDA regulations would give some idea of the vast amount of such information that exists.

Finally, a point to consider in any review of existing disease/feed additive studies is that diseases are not expressed clinically as all or nothing phenomena, and they rarely occur caused by a single entity. Disease severity is situation dependent and can

be affected by genetics, environment, nutrition, and management practices. The question remains, how do these production factors affect response to antibacterial feed additives?

STUDY OBJECTIVES

Enzootic pneumonia and atrophic rhinitis are commonly diagnosed, but what significant impacts do these two diseases have on the growth performance of pigs? What severity is necessary to justify disease control efforts? What beneficial effects are realized by using currently approved feed additive medications for prevention and control of these two diseases? What other production factors might be involved?

These questions arise out of all the topics reviewed in the literature for this study. The most common is the question of interrelationships between EP, AR, and GP. These relationships were not consistently defined, investigated, or quantified in the literature reviewed. Also, many of the reports developed associations of EP, AR, and GP by using small numbers of pigs from multiple herd sources or a moderate number of pigs from a single production unit. Few studies used proper or comparable statistical analysis.

Therefore, the following study was designed to develop more detailed information with respect to the relationships between EP, AR, feed additive medications, and GP. The specific aims of this study were to:

1. Evaluate the relationship of the prevalence of slaughter lesions of enzootic pneumonia and atrophic rhinitis with each other and with sequential periods/phases of pig growth from birth to market.

2. Evaluate the effect of feed additive medication on the prevalence and severity of enzootic pneumonia and atrophic rhinitis at slaughter.
3. Evaluate the effect of feed additive medication on sequential periods of growth performance.
4. Evaluate the relationship of individual pig preselection (preweaning) data to growth performance and to the prevalence and severity of EP and AR lesions at slaughter.

MATERIALS AND METHODS

Study Herd

General information

The Michigan State University Swine Research Center was used. This farrow-to-finish herd maintained approximately 150-180 sows, farrowing 17 groups of 20 sows per year (1 group every 3 weeks).

Genetics

The dams were a mixture of purebred (Hamp, York, Duroc, Landrace), F1, and 3-way cross females resulting in the production of purebred and crossbred pigs. Hand mating was used to provide maximum control of matings and maintenance of genetic records of all offspring produced.

Environment/Facilities

The herd was housed in total confinement facilities. The farrowing facility contained two rooms, each containing twenty crates. Each room was mechanically ventilated. The nursery facilities were narrow buildings with 14 pens along an outside wall. The pens were 6' x 8' and intended to house up to 12 pigs per pen. A self feeder provided for ad libitum feed intake and water was provided by nipple drinkers in each

pen. Pens were partially slatted over a "Y" gutter with a drain plug and the plug was pulled every 10-14 days or as needed. The grow/finish building was divided into two separate rooms, one utilized as a grower (50-125 lbs.) and the other as a finisher (125 lbs. to market). There were 16 pens per room evenly divided on either side of a center alley. The dimensions of the pens were 4.5'x 14' in the grower and 6'x 14' in the finisher and were intended to house up to 12 pigs per pen.

Pen dividers were mounted such that they could be removed allowing 24 pigs per pen. There were individual, two hole, wooden feeders and a nipple drinker for each pen. Pen floors were total cement slats over a flush gutter. Minimum ventilation was mechanically provided during cool weather. Natural ventilation was used during warmer weather by opening large panels in both sidewalls of the building.

Pig Flow and Routine Management Practices

Females were bred and gestated in a breeding/gestation facility. They were moved into the farrowing facility approximately seven days prior to parturition. The only vaccines used on adults were Lepto-Parvo-Erysipelas given prebreeding to females and twice per year to boars.

All newborn pigs were processed within the first 24 hours after birth. The following procedures were included:

1. Ear notching with individual pig identification
2. Needle teeth clipping
3. Tail docking
4. Clipping of umbilical cord
5. Birth weight measurement

6. Iron injection - 200 mg of gleptoferron iron

7. Penicillin injection - 150,000 IU each of procaine and benzathine penicillin

Some litter transferring occurred, but the individual pig notches maintained identity to the original litter.

In addition to these procedures, pigs were castrated at three weeks of age, weaned at 4 weeks, and received an erysipelas bacterin at 8 weeks of age. Internal parasite control was accomplished with the use of Atgard^{R} or Ivomec^{R} on the breeding herd and Banminth^{R} (Pyrantel Tartrate) at 96 grams/Ton during the entire starter period (5-6 weeks) after weaning.

Pigs were weaned into one of two nurseries at approximately four weeks of age. Pigs remained in the nursery for 6 weeks before moving to the grow/finish building.

Normal pig flow through the grow/finish facility involved using the first (North) room as a grower facility where pigs stayed approximately 6 weeks. Pigs were then moved to the second (South) room for the finish period. Pigs remained in this grow/finish facility until removed as breeding stock replacements, culled, or sent to slaughter.

Cull or removal criteria included death, severe and non-improving illness or injury, and extremely poor growth, usually related to illness or injury.

Nutrition

All feed used was in meal form and produced by a stationary milling system at the production unit. Starter, grower, and finisher phase diets were identical for each treatment except for the medication type. The basic diet formulas were as follows;

Pig Diets

Ingredient	Starter	Grower	Finisher
Ground Corn	1104	1448	1610
SBM 44	500	470	330
Dried Whey	300	0	0
Limestone	20	22	20
MonoDical Phos	30	30	15
MSU VTM Premix	15	10	10
Sel-Vit E Premix	20	10	10
Salt	5	10	5
Lysine	3	0	0
Copper Sulfate	1	0	0
Banminth 48	2	0	0
*Lincomycin 20	(10/1)	(1)	(1)
*NeoTerramycin 50/50	(0.75)	0	0
*Aureomycin 50	0	(1)	(1)
	2000 lb	2000 lb	2000 lb

* Addition of these items were as a direct substitute for an equal weight of corn to create the required diet medication levels.

Health/Disease Status

The herd was validated and qualified for brucellosis and pseudorabies, respectively.

The production unit had a well documented prevalence and severity of both EP and AR. Most of the documentation was obtained slaughter health check data generated by Dr. David Ellis, Swine Extension Veterinarian. Dr. Ellis' data indicated that more than 50% of the pigs had enzootic pneumonia lesions of $\geq$ 5% total lung involvement with an average severity of almost 9%. With respect to AR severity, using a 0-3 scale (none, mild, moderate, severe), nearly 50% of the animals showed some degree of turbinate atrophy and the average score was 0.62. Data compiled just prior to starting this study is presented in Table 5.

In addition, post mortem and diagnostic laboratory submissions confirmed the presence of both diseases at numerous times prior to and over the course of the study.

Table 5. MSU Swine Unit Slaughter Check Analysis

Parameter	Source of Data: Meats Lab (n=98)	Trial Pigs (n=67)	All Pigs (n=165)
Lung Scores (% involvement)			
Avg.	8.14	9.73	8.79
SD	10.24	9.94	10.11
n≥5%	45	40	80
%≥5%	49.9%	59.7%	51.5%
Rhinitis			
Avg. Space (in MM)	4.78	NA	NA
SD	1.37	NA	NA
Avg. Score	.46	.85	.62
0=None n(%)	61 (62)	28 (42)	89 (54)
1=Slight	20 (21)	24 (36)	44 (27)
2=Moderate	17 (17)	12 (18)	29 (17)
3=Severe	0 (0)	3 (4)	3 (2)
Mange Score			
Avg.	.81	.72	.77
0=None n(%)	35 (36)	34 (51)	69 (42)
1=Mild	47 (48)	18 (27)	65 (39)
2=Moderate	16 (16)	15 (22)	31 (19)
3=Severe	0 (0)	0 (0)	0 (0)
Liver Score			
1=Pos.n(%	46 (47)	12 (18)	58 (35)
2=Neg.	52 (53)	55 (82)	107 (65)
Growth Data			
ADG (40#-Mkt)	1.48	1.48	1.48
SD	.34	.21	.29

n = number of animals; Avg. = Average; SD = Standard Deviation; Pos. = positive; Neg. = negative; ADG = average daily gain.

Study Design

Animal Selection Procedure

The pigs utilized for each of four trials in the study, except Trial 4, were selected out of a single farrowing group of 20 sows with a goal of selecting 160 pigs per study. Trial 4 utilized pigs from two consecutive farrowing groups farrowed three weeks apart in order to obtain sufficient pig numbers. Groups were selected in the fall or spring months to evaluate seasonal effects.

Pigs within the selected farrowing groups were weighed, individually identified by ear notch at birth. Additionally, a 21 day weight was obtained for each pig. Weaning was done as close to 28 days of age as possible with no more that 5 days difference in weaning time between the oldest and youngest litters.

Pigs were selected at weaning and randomly assigned to one of 16 nursery pens with a maximum of 10 pigs/pen. Pigs were blocked according to weaning weight, sex, and litter. Blocking by litter was performed in an attempt to spread the genetic variation within the production unit as evenly as possible across all treatments within each trial. When total litter stratification was not possible, weaning weight and sex were given priority. Pens were then randomly assigned to a treatment within each trial.

After 6 weeks in the nursery, pigs were moved to the grower where 2 nursery pens were combined to create one grower pen with 20 pigs/pen. Pigs spent approximately 6 weeks in the grower facility and were then moved to the finish room for the remainder of the study. Pigs were weighed at 3 week intervals until market weight was reached. The common endpoint (trial end), used to calculate days to 230 lb was, the

weight measured after the pigs had been in the finishing facility for 6 weeks.the study was set at the time of marketing of the first pigs.

Trial Organization

Data were generated by four (4) trials performed over a period of two and a half years. Trial I involved 157 pigs farrowed in October of 1984 and slaughtered in April and May of 1985 (Spring). Trial II included 145 pigs farrowed in January and slaughtered in July and August of 1985 (Summer). Trial III included 126 pigs farrowed in May and slaughtered in November and December of 1985 (Fall). Trial IV included 160 pigs farrowed in December 1985 and January 1986 and slaughtered in June and July of 1986 (Summer). A tabular summary of pig numbers per trial and the seasons represented in each trial is presented in Table 6.

Table 6. General Trial Information: Trial Pig Numbers and Season

Trl	Trt	Number Started	Season Started	Season Completed
1	LI	79	Fall	Spring
	CO	78		
		157		
2	LI	73	Winter	Summer
	NT	36		
	CO	36		
		145		
3	LI	63	Spring	Fall
	CO	63		
		126		
4	LI	80	Winter	Summer
	NT	40		
	CO	40		
		160		

LI = Lincomycin; NT = Neo-Terramycin; CO = Control

Feed Medications Levels/Treatments

The trials were organized such that only two treatments were used in Trials I and III while three treatments were used in Trials II and IV.

The two treatments in Trials I and III were:

1. Negative control (no feed medication)
2. Lincomix at 200 g/ton for 3 weeks followed by 20 g/ton until the start of the last finish period.

The three treatments for Trials II and IV were:

1. Negative control (no feed medication)
2. Lincomix at 200 g/ton for 3 weeks followed by 20 g/ton until the start of the last finish period.
3. Neo-terramycin (75 g/ton of each component) for 6 weeks followed by 50 g/ton of chlortetracycline until the start of the last finish period.

The number of pigs started in each trial, by each treatment, is listed in Table 6. The medications were used at approved levels as described in the Feed Additive Compendium. Usage claims, as stated in the Compendium, were:

1. Lincomycin 200 g/ton

   For reduction in the severity of swine pneumonia caused by Mycoplasma hyopneumoniae. Feed as sole ration for 21 days.

   (Accomplished by adding 10 lb. of Lincomycin 20 per ton of feed.)

2. Lincomycin 20 g/ton

   For increased rate of weight gain in growing/finishing swine. Feed as sole ration from weaning to market weight.

   (Accomplished by adding 1 lb. of Lincomycin 20 per ton of feed.)

3. NeoTerramycin 75 g/ton of each

   Neomycin (70-140 g/ton) as an aid in the treatment of bacterial enteritis.

   Oxytetracycline (50-150 g/ton) as an aid in the maintenance of weight gain and feed consumption in the presence of atrophic rhinitis.

   (Accomplished by adding 1.5 lb. of NeoTerra 50/50 per ton of feed.)

4. Aureomycin 50 g/ton

   Chlortetracycline (50-100 g/ton) for prevention of bacterial enteritis. Maintenance of weight gain in the presence of atrophic rhinitis; reduction of incidence of cervical abscesses.

   (Accomplished by adding 1 lb. of Aureomycin 50 per ton of feed.)

Data Collection

Although data collection for this study centered on growth performance and enzootic pneumonia/atrophic rhinitis evaluation at slaughter, other data were collected to allow for the analysis of additional factors. Data were collected on an individual animal or pen basis.

Live-Pig Data

Preselection data included individual pig identification by ear notch, date of birth, and birth weight.

The individual identification, date of birth, and birth weight were the basic starting data for tracking growth performance and calculating the figures for average daily gain (ADG) and days to 230 lbs (DYS230).

The growth data included the initial weight or weaning weight of each pig at selection and individual weights taken twice in the nursery phase (at 3 weeks and 5 1/2 weeks post weaning) and twice in each of the grower and finisher phases (at 3 week intervals). Although the growth study ended officially at the time the first pigs were marketed out of a group, weights were measured during the follow-up period until all pigs were removed.

Table 7 explains the pig weighing data points and provides an abbreviated title for each point. All weights were recorded in pounds.

Table 7. Individual Pig Weights: Abbreviation, Definition, and Timing

BWT = birthweight
AJWT = 21 day adjusted weight
AGS = starting age (in days)
STWT = starting weight (at beginning of trial in the nursery)
N1WT = weight at the end of the first nursery period (3 weeks)
N2WT = weight at the end of the nursery phase (2.5-3 weeks after N1WT)
G1WT = weight at the end of the first grower period (3 weeks after N2WT)
G2WT = weight at the end of the grower phase (3 weeks after G1WT)
F1WT = weight at the end of the first finish period (3 weeks after G2WT)
EWT = weight at the end of the trial (3 weeks after F1WT)

Feed Usage

Disappearance of feed from the self feeders was recorded by pen. Empty feeder weights were always obtained before pigs were placed in a particular pen. Feed was delivered in 50 lb. bags. Weights of the bagged feed were taken occasionally to monitor consistency of feed production and delivery. Bags of feed were added to feeders at regular intervals to keep feed fresh and maintain continual ad libitum feed intake. All feed additions were dated and recorded as they occurred. Each time pigs were weighed, feeders were also weighed to determine residual feed left in the feeder. The residual feed figure was then subtracted from the total feed usage over the particular growth period to determine feed utilization by pen. Feed usage, combined with pig weight gains during the same period, was used to calculate feed efficiency (gain/feed) by pen.

General Observations

Clinical observations were made for coughing, sneezing, tear stained eyes, diarrhea, or other clinically evident abnormalities of the pigs. Such observations were made at least twice per week and were used only as a monitor of the general health status of the pigs. In some instances, pigs were removed from the trial based on the severity of signs.

Slaughter Data/Disease Severity Scoring

Complete slaughter checks were performed as described by the TRAC program (Elanco, 1985). Slaughter evaluation began after the carcasses were dehaired. Carcasses were examined externally for evidence of structural abnormalities involving feet and legs,

any swellings or lesions of the skin and joints, and the small, red, papular lesions indicative of mange.

Of the external observations made, only mange (MNG) lesions were actually included in the data analysis because of the general acceptance of mange as a cause of poor performance in growing pigs. Mange lesions were scored on a scale of 0 to 3 (0=none, 1=mild, 2=moderate, 3=severe) as described in the TRAC protocol.

Internal examination of each carcass included a cursory examination for general normality of organ systems. Specific data were recorded for liver, lung, and snout lesions as follows:

Livers

Livers were visually examined for lesions indicative of ascarid larval migration (milk spots). Livers were scored on a scale of 0 to 3 [0=none, 1=mild (<5 lesions), 2=moderate (5-10 lesions), 3=severe (>10 lesions)].

Lungs

Each lung lobe was visually examined and palpated. Normal lung tissue was identified as being a light pink or "salmon" color with a "spongy" texture on palpation. Enzootic pneumonia lesions were identified as consolidated areas of red to gray hepatization that were firm to palpation. Notation of other specific abnormalities such as pleuritis, pericarditis, abscesses, etc. were made.

Figure 1 illustrates the typical lung configuration from both a dorsal and lateral view, identifying all seven (7) lung lobes, and shading in areas typical of the location of EP lesions. The areas assessed do not necessarily align themselves with the anatomic division of the lung lobes. For ease of visualization, the lobes were divided as drawn in Figure 1. Essentially, the cranial and middle, and middle and causal lobes were divided by a line that extends perpendicular from the dorsum of the lung down to where the lobes form an acute angle ventrally.

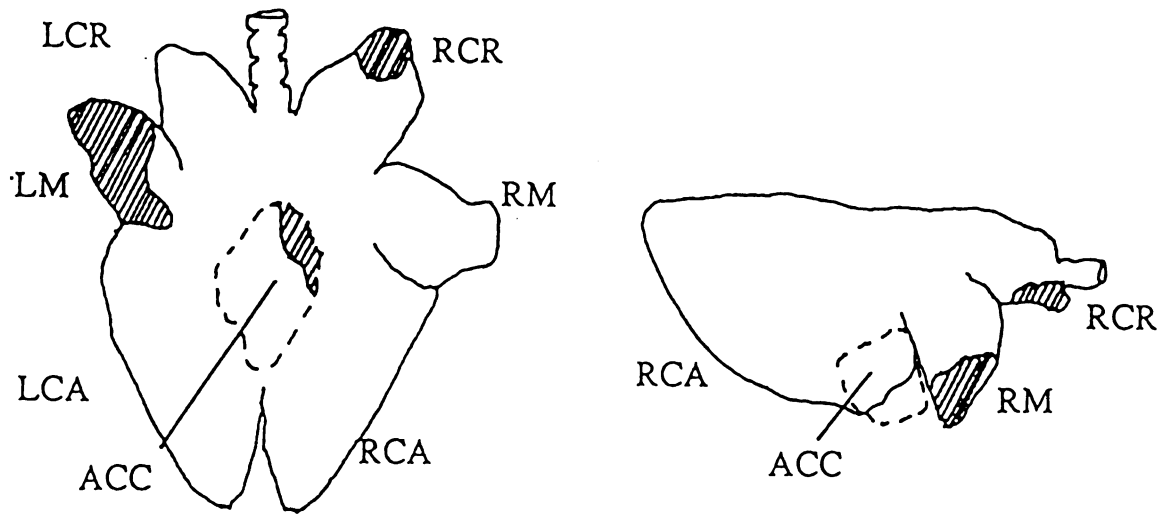

Figure 1. Schematic of Lung Structure - Individual Lobes and Enzootic Pneumonia Lesion Location. LCR = left cranial; LM = left medial; LCA = left caudal; RCR = Right cranial; RM = Right medial; RCA = Right caudal; ACC = accessory. Shaded areas indicate typical enzootic pneumonia lesion locations.

All seven lung lobes were evaluated individually and an estimate of the percentage of pneumonic involvement was recorded for each lobe. For consistency, individual lobes were evaluated and recorded in the same order of left cranial (LCR), left medial (LM), left caudal (LCA), accessory (ACC), right cranial (RCR), right medial (RM), and right caudal (RCA). In addition, the number of lobes having pneumonia lesions was recorded as well as an estimation of the total pneumonic involvement of the entire lung field.

Snouts

Atrophic rhinitis lesions were evaluated by examining a cross section of the snout. The cross section was made at the level of the first upper premolar. Snouts were then evaluated for evidence of AR by examining turbinate atrophy and deviation of the nasal septum.

Turbinate atrophy was evaluated by several methods:

1) The space from the floor of the nasal cavity to the most ventral nasal turbinate was measured in millimeters and recorded for each side of the nasal cavity (right and left).
2) Each quadrant of the nasal cavity (left dorsal and left ventral, right dorsal and right ventral) was evaluated for turbinate atrophy and scored on a scale of 0 to 3 (none, mild, moderate, severe).
3) An average turbinate space was calculated by adding both ventral turbinate atrophy measurements together and dividing by two.

4) A total rhinitis score was calculated by averaging the quadrant scores previously taken.

In addition, nasal septum deviation was visually evaluated and scored on a system of 0 to 3 (none, mild, moderate, severe).

Figure 2 illustrates the snout cross sections with normal nasal turbinate configuration and with some degree of turbinate atrophy.

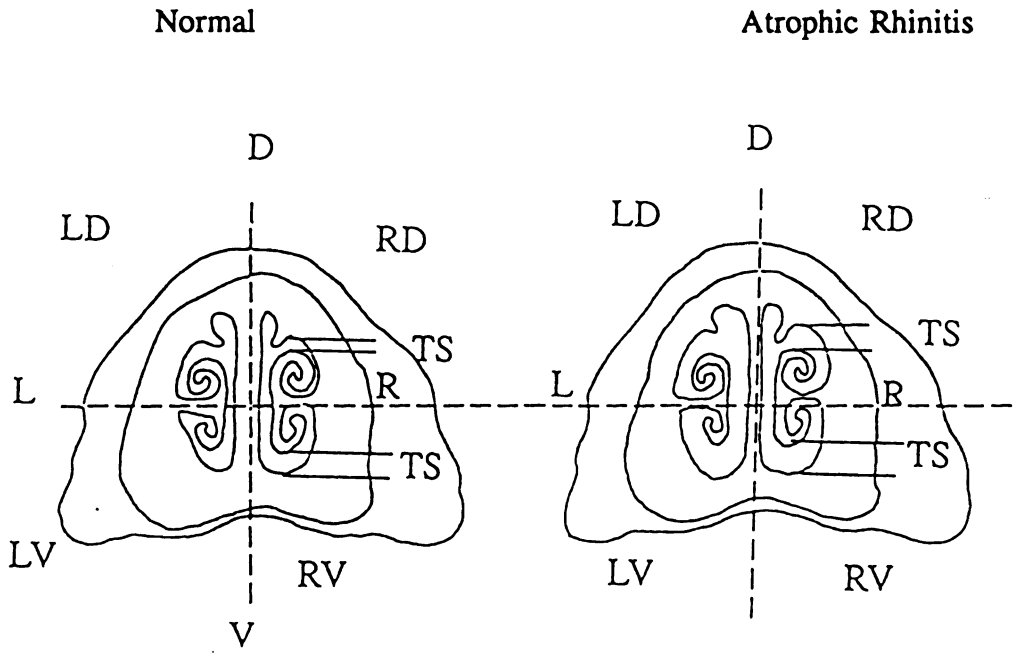

Figure 2. Schematic of Nasal Turbinate Structure - Normal Turbinates and Atrophic Rhinitis Lesion Measurement. R = Right; L = Left; NS = nasal septum; V = ventral; D = Dorsal; TS = turbinate space measured in millimeters.

Calculated Values

Growth Data Calculations

Average daily gain (ADG) was calculated for each pig for each time period between weighings. The values were calculated by subtracting the pig weight at the beginning of each period from the ending weight of that same period and dividing the result by the number of days the individual pig spent in that period. Pigs that were removed for any reason in between scheduled weighings were weighed at removal and their ADG was calculated in the same manner using removal weight as the ending weight and actual days spent in the period as the denominator.

Since each phase of production (nursery, grower, finisher) contained two weigh periods, an ADG was calculated for each pig for each growth phase and a cumulative ADG was calculated for each pig through the end of each subsequent growth phase.

Table 8 lists the various calculated ADG figures.

Table 8. Calculated Average Daily Gain Figures - Abbreviation and Definition

PADGN1	= pig ADG for first nursery period
	= (N1WT-STWT)/days in N1
PADGN2	= pig ADG for second nursery period
	= (N2WT-N1WT)/days in N2
PADGNT	= pig ADG for total nursery phase
	= (N2WT-STWT)/days in N1+N2
PADGG1	= pig ADG for first grower period
	= (G1WT-N2WT)/days in G1
PADGG2	= pig ADG for second grower period
	= (G2WT-G1WT)/days in G2
PADGGT	= pig ADG for total grower phase
	= (G2WT-N2WT)/days in G1+G2
PADGGC	= cumulative pig ADG through grower phase
	= (G2WT-STWT)/days in N1+N2+G1+G2
PADGF1	= pig ADG for first finish period
	= (F1WT-G2WT)/days in F1
PADGF2	= pig ADG for second finish period
	= (EWT-F1WT)/days in F2
PADGFT	= pig ADG for total finish phase
	= (EWT-G2WT)/days in F1+F2
PADGFC	= cumulative pig ADG through end of trial
	= (EWT-STWT)/days in N1+N2+G1+G2+F1+F2

ADG = average daily gain in lb./day
See Table 7 for explanation of weight abbreviations.

Days to 230 lbs. (DYS230) was calculated for each pig. The following formula was used:

$$DYS230 = (\text{Actual age}) + \frac{(230 - \text{Actual wt.}) * (\text{Actual age} - 38)}{\text{Actual wt.}}$$

[Where **Actual age** is given in days and **Actual wt.** is given in pounds.]

The age and weight at trial end were used to calculate DYS230.

Feed Conversion Calculations

Feed conversion data were calculated by pen. Feed conversion data was calculated for each growth period, for each total growth phase, and cumulative through each successive growth phase.

Feed conversion was reported as pounds of gain per pound of feed used (Gain/Feed = GNFD). Table 9 lists the various calculated GNFD values generated.

Table 9. Calculated Gain to Feed - Abbreviations and Definitions

GNFDN1	= gain per feed fed for first nursery period
GNFDN2	= gain per feed fed for second nursery period
GNFDNT	= gain per feed fed for total nursery phase
GNFDG1	= gain per feed fed for first grower period
GNFDG2	= gain per feed fed for second grower period
GNFDGT	= gain per feed fed for total grower phase
GNFDGC	= cumulative GNFD through grower phase
GNFDF1	= gain per feed fed for first finish period
GNFDF2	= gain per feed fed for second finish period
GNFDFT	= gain per feed fed for total finish phase
GNFDFC	= cumulative GNFD through end of the trial
GNFD	= pig weight gain per feed fed (lb/lb)

Percentage Pneumonia Calculations

The total percentage of pneumonic involvement was calculated using the estimated pneumonic percentages of each lobe in conjunction with an estimate of the percentage of total lung weight that each lobe contributed. The latter estimate was generated by sharp dissection of 12 normal lungs and weighing individual lung lobes/areas as described previously. The estimates for each lobe as a percentage of total lung weight derived from these dissections were as follows:

Lobe	Percent of Total Lung
LCR	4
LM	9
LCA	25
ACC	5
RCR	7
RM	15
RCA	35
Total	100

These percentages were comparable with those used by Morrison et al. (1985a) as presented in Table 10.

Table 10. Percentage of Total Lung Contributed by Each Lobe

Lobes	Percent of Total Lung	S.E.
LCR	7.1%	0.3
LM	6.9%	0.4
LCA	31.6%	0.6
ACC	4.6%	0.2
RCR	11.9%	0.5
RM	7.5%	0.2
RCA	30.0%	0.7

Morrison et al. (1985a)

The percent pneumonic involvement for the entire lung was calculated using the following formula:

(percent involvement of LCR)* 0.04
+(percent involvement of LM) * 0.09
+(percent involvement of LCA)* 0.25
+(percent involvement of ACC)* 0.05
+(percent involvement of RCR)* 0.07
+(percent involvement of RM) * 0.15
+(percent involvement of RCA)* 0.35
=Total Percent Pneumonic Involvement

Parameter Abbreviations

Table 11 lists all abbreviations of data analyzed in the study.

TABLE 11. Abbreviations for Data Points

BWT	= birthweight (lb)
AJWT	= 21 day adjusted weight (lb)
AGS	= starting age (days)
STWT	= starting weight (lb)
N1WT	= pig wt. at end of first nursery period (lb)
N2WT	= pig wt. at end of second nursery period (lb)
G1WT	= pig wt. at end of first grower period (lb)
G2WT	= pig wt. at end of second grower period (lb)
F1WT	= pig wt. at end of first finish period (lb)
EWT	= pig wt. at end of the trial (lb)
DYS230	= days from birth to 230 lb
CLNG	= calculated percentage volume of lung involved with pneumonia
ELNG	= estimated percentage volume of lung involved with pneumonia
ARN	= average rhinitis (turbinate) space (mm)
TRN	= total rhinitis (turbinate) space (mm)
SDEV	= septal deviation score
LIV	= liver score for ascarid scars
MNG	= mange score
PADG	= pig average daily gain (lb/day)
GNFD	= pen gain per feed fed (lb/lb)
N1	= first nursery period
N2	= second nursery period
NT	= total nursery phase
G1	= first grower period
G2	= second grower period
GT	= total grower phase
GC	= cumulative through grower phase
F1	= first finish period
F2	= second finish period
FT	= total finish phase
FC	= cumulative through end of trial

Abbreviations may be combined to indicate data points.

Statistical Analysis

General Information

Initial data handling involved double checking all data cells on all pigs involved for accuracy and completeness and then generating any calculated data points required (eg. feed efficiency, average daily gain, days to 230, calculated lung percentage pneumonia, average rhinitis). All statistical analyses were then performed using SAS (1982).

Descriptive Statistics

Descriptive statistics generated included means, standard deviations, minimum and maximum values, standard errors, variances and coefficients of variation. Data summaries by treatment and within trial, and by treatment across all trials are presented in Appendix I, Tables 1-7.

Associations

Data were evaluated for association utilizing correlation statistics. Correlation figures were used as a "measure of the degree of association or interdependence of two variables." (Gill, 1978) The statistical program (SAS, 1982) generated Pearson correlation coefficients (r), the number of data points utilized in generating the coefficients (n), and the significance level of the resulting correlation (p). As explained by Gill (1978), the Pearson (product-moment) correlation is a "unitless measure of the joint distribution of two random variables." It is a measure of linear relationship whose values range from -1 to +1. The upper limit (+1) implies a perfect linear relationship.

The lower limit (-1) implies a perfect inverse linear relationship. Zero implies no linear relationship or interdependence, but does not eliminate the possibility of a curvilinear relationship. Correlations were conducted by treatment within trial and across trials and treatments.

Analysis of Variance (ANOVA)

The most in-depth statistical analysis was done using the SAS (1982) General Linear Model (GLM) program for ANOVA. A significance level of $p<0.05$ was designated. The significance of the relationships of specific parameters was tested using Scheffe's test for post-hoc data comparisons (Gill, 1978). For the variables within the three main categories identified previously (preweaning, growth, and health data) the means were compared by trial, by treatment, and for trial/treatment interactions.

R Square Analysis

The RSQUARE (SAS, 1982) procedure was performed to evaluate the usefulness of certain variables for model testing with the goal of determining mathematical relationships between disease (EP and AR) and GP. The procedure gives variables selected in the models, along with the associated R^2 of the model, to aid in determination of which variables to use in the MODEL statements for further linear model analysis.

An example of the general form of an RSquare model statement would be:

$$Y = X_1, X_2, X_3, \text{ etc.}$$

where Y equaled DYS230 as an overall representative of pig growth, and the X values represent variables measured in the study and of interest in modeling pig growth.

RSquare values (R^2) are mathematical estimations of the proportion (percentage) of variation that occurs with the dependent variable (Y) that can be explained by the linear regression of the dependent variable on the independent variable(s) (X) in the model. (Snedecor & Cochran, 1980) RSquare values are used to judge the structure and completeness of linear models. The higher the R^2 value, the more complete the mathematical model is judged to be. Low R^2 values indicate the absence of one or more significant variables in the model. Whether high or low, the R^2 values generated are always open to further assessment based on the biological concepts that may or may not coincide with the mathematical results.

Discriminant Analysis/Logistic Regression

The techniques of Discriminant Analysis and Logistic Regression (SAS, 1982) were attempted to further analyze the relationships of EP and AR to pig growth data generated over time and delineate more precisely the relationship(s) of each three week growth phase to the end measurements of DYS230, CLNG, and ARN. This required mathematically isolating each phase to remove, or at least minimize, the confounding of serial correlations of weight measurements over time.

The potential benefit of such analysis was to help identify a growth period earlier than the finish phase that might be a strong predictor of DYS230 and/or be of importance in estimating or predicting the prevalence of EP or AR and their impact on performance.

RESULTS

Trial Completion and Pig Removal Data

Table 12 summarizes data relating to the number of pigs that finished the study and indicates the reasons why pigs were removed prior to completion. The completion success rates for Trials I, II, III, and IV were 78.34%, 76.55%, 90.48%, and 83.75%, respectively. Removal reasons included death, severe and progressive illness or injury, and extremely poor growth related to illness or injury. These reasons would normally result in losses of about 6-8% from weaning to market; 3-4% as deaths and 3-4% as culls or underweight marketings. Additionally, some animals were removed for use as replacement breeding animals. Non-castrated boars were removed early in the grower phase. Gilts continued in the trials until late in the finish period.

Only a few pigs were removed due to death. Most sick pigs were identified early and were removed when their condition was judged to be irreversible with respect to further growth and survival. The most common removal reason was for use as breeding animals in all four trials and within each treatment group except with CO and NT treatments in Trials I and II where removal due to terminal ileitis was at least as frequent.

Table 12 - Trial Completion Success

Trl	Trt	Number Started	Number Completed	Percentage Completed	Removal Reasons			
					Breeding	Ileitis	Slow Growth	Other
1	LI	79	63	79.75%	8	1	5	3
	CO	78	60	76.92%	4	7	3	3
		157	123	78.34%	12	8	8	6
					(35.3%)	(23.5%)	(23.5%)	(17.6%)
2	LI	73	58	79.45%	7	1	1	6
	NT	36	27	75.0%	3	3	2	1
	CO	36	26	72.22%	2	2	3	3
		145	111	76.55%	12	6	6	10
					(35.3%)	(17.6%)	(17.6%)	(29.4%)
3	LI	63	63	95.24%	2	1	0	0
	CO	63	54	85.71%	5	0	2	2
		126	114	90.48%	7	1	2	2
					(58.3%)	(8.3%)	(16.7%)	(16.7%)
4	LI	80	74	92.5%	2	0	1	3
	NT	40	35	87.5%	4	0	1	0
	CO	40	25	62.5%	4	0	1	10
		160	134	83.75%	10	0	3	13
					(38.5%)		(11.5%)	(50.0%)
Total	LI	295	255	86.44%	19	3	7	12
	NT	76	62	81.58%	7	3	3	1
	CO	217	165	76.04%	15	9	9	18
		588	482	81.97%	41	15	19	31
					(38.7%)	(14.2%)	(17.9%)	(29.2%)

LI = Lincomycin; NT = NeoTerramycin; CO = Control

Descriptive Statistics

All variables measured are presented in abbreviated form as described in Table 11. Descriptive statistics (mean, standard deviation, and coefficient of variation) were calculated for each variable measured and are presented in Tables 1-7 of Appendix I.

Preweaning Data

Preweaning variables included BWT, AJWT, AGS, and STWT. Across all 4 trials, these variables had a coefficient of variation of 0.25 or less except for the actual starting age of the pigs in Trial IV. The SD of starting age in Trial IV was more than 7 days, versus just over 3 days in the other trials.

Growth Data

Variables used as indicators of individual pig growth were pig weights by periods N1, N2, G1, G2, F1, F2, and EWT and average daily gains by periods and phases PADG-N1, N2, NT, G1, G2, GT, GC, F1, F2, FT, and FC. Individual pig weights tended to be more variable in Trials I and II compared to Trials III and IV. The variation in weights was very comparable between the CO and LI treatment groups in all four trials. The variation in weights of the NT groups generally was higher than either the CO or LI groups, especially during the nursery phase. For the average daily gain data, larger variations occurred within the nursery periods (PADGN1, N2, & NT) in each of the four trials compared to either the grower or finisher phases. In Trial II, during the first nursery period (PADGN1), the C.V. for all three treatments were greater than 0.5 (CO=2.02, LI=.5, NT=.53).

In Trial I, the C.V. for ADG for the total grower phase (PADGGT) of the CO pigs was greater than LI pigs (0.44 vs. 0.15).

In Trial IV, there was a wider variation in ADG in the first finish period (PADGF1) for the CO group (0.43) than for either the LI (0.27) or NT (0.10) treated groups.

Health Data

Variables used as indicators of enzootic pneumonia and atrophic rhinitis include CLNG, ELNG, ARN, TRN, and SDEV. Their variation was relatively high with C.V.'s > 0.50, and many > 1.00.

Associations Between Variable Groups

An example of the generated correlation data is presented in Table IV in Appendix II. Variables were divided into three major groups: preweaning, health, and growth. Probability figures are given for all correlation coefficients calculated. For evaluation in this study, correlation coefficients with $p \leq 0.05$ were considered to be significant.

Preweaning Data

Preweaning data (BWT, AJWT, AGS, and STWT) had minimal or no significant correlation to any of the health data (CLNG, ELNG, ARN, TRN, SDEV, LIV) collected at slaughter. One exception was the relationship of BWT to both atrophic rhinitis variables (ARN and TRN). The correlation was both negative and significant for the CO

and NT groups (CO: -0.161 and -0.178; NT: -0.287 and -0.24 respectively), but was positive and not significant for the LI group.

The statistical significance of associations between preweaning data and subsequent growth data (weights, average daily gains, and DYS230) was somewhat variable. In general, the preweaning data was more strongly and significantly associated with the early phase (nursery) gains compared to later periods (grow/finish). When associations were significant, the correlation coefficients were generally 0.25 or larger and all were positive in value. Preweaning and growth data associations tended to be more pronounced in LI and NT groups compared to CO. And the associations were more consistent in the LI groups compared to the NT groups.

Growth Data

Growth data correlations comparing the various periods, though included in the example table in Appendix II, are not reported because the serial correlations negate any meaningful interpretation.

Health Data

The association of health data to growth data yielded a mixture of both positive and negative correlations. In general, those associations that were statistically significant were negative such that increased severity of disease was associated with decreased growth. The health data collected at slaughter was most consistently associated with the last phases of growth. The most consistent among these relationships were the CLNG, ELNG, and ARN related to F1WT and EWT for the LI and NT treatments. The

correlations were negative and ranged from -0.1 to -0.25. For CO pigs, only ARN was significantly correlated (r= -0.2).

Mange score (MNG) was negatively correlated with late grower and both finish periods and positively correlated with days to 230. Correlations between SDEV, TRN and LIV, and growth and preweaning data were rarely, if ever, statistically significant. They tended to approach significance with later growth periods and r values were always negative. Their correlation with other health data was more significant and always positive.

The relationship of DYS230 to CLNG and ELNG was only significant across the trials in the LI treated group (0.277 and 0.24, respectively). For the CO and NT groups the relationship was much less and not significant.

Since this study was conducted to evaluate health and performance, a summary of only the most consistent and significant correlations ($p \leq 0.05$) between these health and growth variables are presented in Table 13.

TABLE 13 - Health Data Correlation Summary

	CLNG			ELNG			AVGRN			TRN			SDEV			LIV			MNG			DYS230		
	C	L	NT	C	L	NT	C	L	NT	C	L	NT	C	L	NT	C	L	NT	C	L	NT	C	L	NT
BWT							-0.16		-0.29	-0.18			-0.17									-0.26	-0.25	-0.25
AJWT			0.33			0.27				-0.23			-0.23	-0.19						0.19		-0.28	-0.31	-0.52
AGS												0.14		0.23			-0.12			-0.15				
STWT							-0.18						-0.17									-0.34	-0.38	-0.58
N1WT																						-0.48	-0.43	-0.53
N2WT							-0.16	-0.15													-0.27	-0.6	-0.52	-0.57
G1WT																						-0.63	-0.54	-0.51
G2WT																			-0.29		-0.41	-0.7	-0.63	-0.65
F1WT		-0.24			-0.13		-0.2	-0.21									-0.13		-0.33	-0.23	-0.37	-0.83	-0.73	-0.79
EWT		-0.23			-0.16		-0.21	-0.16											-0.29			-0.88	-0.82	-0.85
CLNG	---	---	---	0.94	0.93	0.93	0.31	0.12		0.26			0.18										0.28	
DYS230					0.24		0.24	0.21	0.26					0.14					0.32	0.13	0.44	---	---	---
ELNG				---	---	---	0.32			0.27			0.19											
ARN							---	---	---	0.73	0.79	0.77	0.23	0.24		0.29	0.2							
TRN										---	---	---	0.43	0.34		0.26								
SDEV													---	---	---		-0.14							
LIV																---	---	---						
MNG																			---	---	---			

All numbers printed are Pearson Correlation Coefficients
statistically significant at $p < 0.05$.

AJWT = Adjusted Weaning Weight
BWT = Birthweight
C = control treatment
STWT = Starting Weight
AGS = Starting Age
L = Lincomycin treatment
N1WT = Pig Weight at end of first Nursery period
NT = Neo-Terramycin treatment
N2WT = Pig Weight at end of second Nursery period
G1WT = Pig Weight at end of first Grower period
G2WT = Pig Weight at end of second Grower period
F1WT = Pig Weight at end of first Finish period
EWT = Pig Weight at end of Trial
CLNG = Calculated Pneumonia Percentage
DYS230 = Days to 230
ELNG = Estimated Pneumonia Percentage
ARN = Average Rhinitis Space
TRN = Total Rhinitis Space
SDEV = Septal Deviation
LIV = Liver (Ascarid) Score
MNG = Mange Score

Associations Within Variable Groups

In addition to the relationships between each of the three major groups of variables (preweaning, health, and growth), each group contained sufficient variables to look at relationships within each group.

Preweaning Data

Within the preweaning variables, BWT was strongly and positively correlated with AJWT. Starting weight (STWT) was closely and positively associated with AGS, BWT, and AJWT. AGS was negatively correlated with BWT and AJWT. No other significant associations within the preweaning data variables were apparent.

Growth Data

Within the growth variables, relationships were positive, significant, and generally smaller in magnitude as the time between measurements increased. The general nature of these associations was expected considering that growth data are simply repeated measurements of the same variable over a continuum of time and were therefore serially correlated.

Health Data

Within the health variables, associations were not consistent. The most consistent and significant associations were within the CO groups where CLNG and ELNG were positively associated with all the atrophic rhinitis values (ARN, TRN, and SDEV). Similar data from LI and NT groups were not statistically significant nor consistent.

Liver and mange scores were both inconsistent and insignificant in their relationship to other health data variables.

Analysis of Variance (ANOVA)

Scheffe's test for post data comparisons (Gill, 1978) was indicated because the data contained missing cells, were unbalanced, and because many of the comparisons were performed either as the trials progressed or after the data were collected. For tests defined in the original protocol and specifically described for comparison using Duncan's multiple range test, the ANOVA was completed as described in addition to using Scheffe's test. As performed, results were identical for both methods.

Results of these analyses are presented in Tables 14-16. In addition, feed efficiency data by pen were compared and the results are presented in Table 17.

Preweaning Data

The differences between trials among the preweaning data points were minimal (see Table 14). BWT was somewhat greater in pigs farrowed in winter or spring (Trials II, III, and IV) compared to pigs farrowed in late summer or fall (Trial I). However, the difference was not statistically significant and these differences in BWT tended to disappear as the pigs progressed in age (ie. AJWT and STWT). Starting ages (AGS) were somewhat different between trials but generally insignificant across all four trials.

Table 14 - Analysis of Preweaning Data

		Slaughter Season / Trial No.				
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall
BWT	CO	3.45	3.40	3.55	3.79	3.53[a]
	LI	3.48	3.59	3.64	3.67	3.59[a]
	NT		3.49		3.86	3.69[b]
AJWT	CO	12.57	12.38	13.52	15.70	13.39[a]
	LI	12.59[e]	13.16[e]	13.54[e]	15.56[f]	13.75[a]
	NT		12.96		15.95	14.53[b]
AGS	CO	29.39	27.56	32.79	23.50	28.99[a]
	LI	29.47[e]	27.34[e]	32.83[f]	23.24[g]	27.92[a]
	NT		27.53		23.40	25.36[b]
STWT	CO	17.50	15.72	19.55	18.40	17.97
	LI	17.46[e]	16.43[f]	19.48[g]	18.12[g]	17.81
	NT		16.41		18.60	17.57

Treatments: CO = Control; LI = Lincomycin; NT = NeoTerramycin
BWT = Birthweight; AJWT = 21 Day Adjusted Weaning Weight
AGS = Starting Age; STWT = Starting Weight

ab = for each preweaning variable, treatments with different superscripts were significantly different for overall (ie. across all trials) ($p<0.05$).

efg = for each preweaning variable, columns with different superscripts were significantly different for trial:treatment interaction ($p<0.05$).

Growth Data

Growth data, as evaluated by ADG calculations, are summarized in Table 15. For the first nursery period, the average daily gain (PADGN1) was significantly greater in Trial III compared to other trials. The gain for N1 in Trial IV was also greater than that in Trials I and II. However, across all four trials there was no significant difference in PADGN1 between treatments and there were no significant trial:treatment interactions.

For the second nursery phase (PADGN2) only Trial IV showed significantly higher ADG than the other trials. Across all 4 Trials, the LI and NT groups significantly outgained the control groups, but were not significantly different from each other. There was no significant interaction between trial and treatments.

For the entire nursery period (PADGNT) pigs in Trials III and IV gained better than either Trials I or II and pigs in Trial III gained more than Trial IV pigs. Across all four trials, both the LI and NT groups significantly outgained the CO group and there was no significant difference between LI and NT. There was no significant trial:treatment interaction for the nursery phase.

For the first grower period (PADGG1), there was no significant difference in gains between trials. Across all four trials, the NT group outgained both the LI and CO groups and there was no significant trial:treatment interaction.

For the second grower period (PADGG2) the gains were significantly better in Trial II than in either Trial III or IV. However, across all four trials, there was no significant difference in gain between LI, NT and CO, and there was no significant trial:treatment interaction.

For the total grower phase (PADGGT) the gains in Trial II were significantly greater than all other trials except Trial IV, and Trial I had the least gains of all. Across all four trials, gains in LI and NT groups were similar and continued to exceed CO gains. There was a significant trial:treatment interaction with the main difference being reduced gains in Trial I.

For the cumulative phases of grower and nursery (PADGGC), Trial III gained significantly better than all trials except Trial II. Trial I gains were much less than any of the other three trials. Across all four trials, LI and NT significantly outgained the CO. For the first finish period (PADGF1), Trial III daily gains were significantly greater than any other trial. Trial IV daily gains were the lowest, although not significantly different from Trial II. Across all four trials, there were no significant differences between the daily gains within any of the three treatments. There was some significant trial:treatment interaction with higher gains in Trials I and III.

For the second finish period (PADGF2), Trial II gains were the greatest, but were statistically different only from Trials I and IV. Across all four trials, CO had significantly better daily gains than either LI or NT. In addition, the ADG for LI was significantly greater than NT. There were no significant trial:treatment interactions.

For the total finish phase (PADGFT), Trial III ADG was significantly greater compared to the other trials. Trial IV ADG was the lowest. Across all four trials, CO had significantly better ADG than either LI or NT. In addition, as with PADGF2, LI treatment resulted in significantly better ADG compared to NT treatment. There were no significant trial:treatment interactions. For the entire study, cumulative across all growth periods (PADGFC), Trial III daily gains were significantly greater than for any

other trial while the other three trials did not significantly differ from each other. Across all four trials, there was no significant difference in ADG between any of the three treatments and there were no significant trial:treatment interactions.

For DYS230, there were no significant differences between treatments. With respect to trial:treatment interactions, DYS230 in Trial III were significantly less than Trials I and II, but similar to Trial IV.

Table 15 - Analysis of Growth Data

		Slaughter Season / Trial No.				
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall
	CO	0.52	0.59	0.99	0.67	0.69
PADGN1	LI	0.58[e]	0.63[ef]	1.23[g]	0.68[fh]	0.76
	NT		0.56		0.72	0.65
	CO	1.06	0.99	1.04	1.19	1.07[a]
PADGN2	LI	1.20[e]	1.15[e]	1.16[e]	1.28[h]	1.20[b]
	NT		1.16		1.35	1.26[b]
	CO	0.77	0.74	1.03	0.91	0.87[a]
PADGNT	LI	0.88[e]	0.88[e]	1.21[g]	0.95[h]	0.97[b]
	NT		0.88		1.00	0.95[b]
	CO	—	1.44	1.54	1.47	1.50[a]
PADGG1	LI	—	1.57	1.49	1.52	1.53[ba]
	NT		1.56		1.67	1.62[c]
	CO	—	1.83	1.55	1.62	1.64
PADGG2	LI	—	1.76[f]	1.57[g]	1.65[g]	1.66
	NT		1.73		1.61	1.66
	CO	1.19	1.16	1.55	1.52	1.36[a]
PADGGT	LI	1.48[e]	1.66[f]	1.53[g]	1.54[fgh]	1.55[b]
	NT		1.64		1.62	1.63[b]
	CO	0.99	1.22	1.30	1.23	1.16[a]
PADGGC	LI	1.18[e]	1.29[f]	1.38[fg]	1.26[fh]	1.27[b]
	NT		1.28		1.33	1.31[b]
	CO	1.79	1.66	1.89	1.36	1.72
PADGF1	LI	1.78[e]	1.58[f]	1.98[g]	1.54[fh]	1.71
	NT		1.63		1.80	1.72
	CO	1.64	1.92	1.81	1.57	1.72[a]
PADGF2	LI	1.45[e]	1.77[f]	1.68[fg]	1.50[e]	1.59[b]
	NT		1.57		1.38	1.47[a]

Table 15 continued

		Slaughter Season / Trial No.				
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall
PADGFT	CO	1.74	1.79	1.85	1.50	1.74[a]
	LI	1.67[e]	1.68[e]	1.81[g]	1.52[h]	1.66[b]
	NT		1.60		1.55	1.57[c]
PADGFC	CO	1.34	1.41	1.52	1.35	1.41
	LI	1.38[e]	1.42[e]	1.54[g]	1.36[e]	1.42
	NT		1.39		1.41	1.40
DYS230	CO	182.69	180.67	173.73	178.43	178.85
	LI	182.02[e]	181.57[e]	172.04[g]	179.39[eg]	178.98
	NT		184.47		175.06	179.36

Treatments: CO = Control; LI = Lincomycin; NT = NeoTerramycin

PADG = Pig Average Daily Gain

N1 & N2 = First & Second Nursery Periods respectively

NT = Total Nursery Phase

G1 & G2 = First & Second Grower Periods respectively

GT = Total Grower Phase

GC = Cummulative through end of grower phase (NT + GT)

F1 & F2 = First & Second Finish Periods respectively

FT = Total Finish Phase

FC = Cumulative through end of the trial (NT + GT + FT)

DYS230 = Days to 230

abc = for each preweaning variable, treatments with different superscripts were significantly different overall (ie. across all trials) ($p < 0.05$).

efgh = for each preweaning variable, columns with different superscripts were significantly different trial:treatment interaction ($p < 0.05$).

Health Data

Table 16 summarizes health data results. For CLNG and ELNG, there were no significant differences between trials or between treatments across all four trials and there were no significant trial:treatment interactions.

For ARN, TRN, and SDEV there were no significant differences for any of the comparisons made. The ARN scores tended to be more severe in pigs farrowed in winter months (Trials II and IV), but these differences were not significantly different.

Liver scores were significantly higher in Trials II and IV compared to Trials I and III. Across all four trials, NT pigs had significantly higher LIV compared to either CO or LI. There were no significant trial:treatment interactions.

Mange scores in Trial IV were significantly greater than the other trials. Across all four trials, there were no significant differences between treatments for MNG. There was some significant trial:treatment interaction in that MNG of LI and CO pigs in Trials II and IV were significantly increased.

Table 16 - Analysis of Health Data

		Slaughter Season / Trial No.				
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall
CLNG	CO	6.62	7.82	8.44	6.65	7.38
	LI	4.84	9.13	8.07	7.60	7.37
	NT		5.02		7.74	6.54
ELNG	CO	6.47	6.81	7.66	5.84	6.80
	LI	5.52	7.95	7.38	6.38	6.76
	NT		4.59		6.68	5.75
AVGRN	CO	5.45	6.48	5.19	6.00	5.63
	LI	4.93^{e}	5.97^{f}	5.22^{e}	5.79^{ef}	5.49
	NT		6.54		5.91	6.19
TRN	CO	2.0	2.27	1.90	2.04	2.02
	LI	1.58	1.64	2.03	1.58	1.70
	NT		1.85		1.71	1.77
SDEV	CO	0.5	0.31	0.35	0.08	0.36
	LI	0.36^{e}	0.26^{eh}	0.38^{e}	0.10^{h}	0.26
	NT		0.3		0.09	0.18
LIV	CO	0.72	1.35	0.94	1.68	1.04^{a}
	LI	1.76^{e}	1.53^{f}	0.93^{e}	1.54^{fh}	1.45^{ba}
	NT		1.52		1.56	1.54^{a}
MNG	CO	0.37	0.39	0.06	0.56	0.25
	LI	0.13^{e}	0.22^{ef}	0.03^{fg}	0.74^{h}	0.31
	NT		0.04		0.29	0.18

Treatments: CO = Control; LI = Lincomycin; NT = NeoTerramycin

CLNG = Calculated Percentage Pneumonia
ELNG = Estimated Percentage Pneumonia
ARN = Average Rhinitis Space (Turbinate Atrophy)
TRN = Total Rhinitis Space (Right side + Left side)
SDEV = Septal Deviation Score
LIV = Liver (Ascarid) Score; MNG = Mange Score

abc = for each preweaning variable, treatments with different superscripts were significantly different overall (ie. across all trials) ($p < 0.05$).

efgh = for each preweaning variable, columns with different superscripts were significantly different trial:treatment interaction ($p < 0.05$).

Feed Efficiency Data

Feed efficiency data were calculated as gain/feed and are summarized in Table 17. Statistical analysis by pen was performed on all nursery phases, but only on the total and cumulative phases of the grower and finisher because of missing cells present in Trial I. For the growth periods analyzed, there were significant differences between treatments and across all four trials for GNFDN2 and GNFDNT only. In these two periods, LI pens were significantly more efficient in their gain than CO pens. There were no differences between NT and either LI or CO. All other phases analyzed showed no significant differences between treatments.

For all phases, there were significant trial:treatment interactions although these interactions varied by growth phase. Trial III had significantly better feed efficiency (FE) in periods N1 and N2. Trial II had significantly better FE for the cumulative grower phase (GNFDGC). Both Trials II and III had better FE than Trial IV for the cumulative data through the end of the trial (GNFDFC).

Table 17 - Analysis of Feed Efficiency Data (Gain/Feed)

		Slaughter Season / Trial No.					
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall	Overall Feed/Gain
	CO	0.54	0.522	0.713	0.522	0.586	1.71
GNFDN1	LI	0.564[a]	0.603[a]	0.777[g]	0.543[a]	0.617	1.62
	NT		0.617		0.542	0.58	1.72
	CO	0.476	0.405	0.348	0.458	0.422[a]	2.37
GNFDN2	LI	0.489[a]	0.466[a]	0.361[g]	0.477[a]	0.451[b]	2.22
	NT		0.448		0.479	0.464[ab]	2.16
	CO	0.498	0.436	0.478	0.480	0.478[a]	2.09
GNFDNT	LI	0.513[a]	0.509	0.513	0.499	0.508[b]	1.97
	NT		0.495		0.498	0.497[ab]	2.01
	CO	—	0.411	0.393	0.431	0.409	2.44
GNFDG1	LI	—	0.411	0.372	0.43	0.407	2.46
	NT		0.406		0.46	0.433	2.31
	CO	—	0.355	0.307	0.292	0.316	3.16
GNFDG2	LI	—	0.332	0.298	0.3	0.311	3.22
	NT		0.32		0.302	0.311	3.22
	CO	0.348	0.38	0.344	0.323	0.348	2.87
GNFDGT	LI	0.371[efg]	0.368[ef]	0.33[cde]	0.328[cde]	0.35	2.86
	NT		0.358		0.339	0.349	
	CO		0.402	0.363	0.343	0.368	2.87
GNFDGC	LI		0.392[f]	0.36[g]	0.349[g]	0.368	2.72
	NT		0.381		0.359	0.37	2.70
	CO	0.31	0.284	0.282	0.224	0.282	3.55
GNFDF1	LI	0.309	0.287	0.296	0.262	0.288	3.47
	NT		0.272		0.253	0.263	3.80
	CO	0.265	0.257	0.245	0.22	0.25	4.00
GNFDF2	LI	0.212	0.239	0.229	0.234	0.234	4.27
	NT		0.221		0.207	0.214	4.67

Table 17 continued

		Slaughter Season / Trial No.					
		Sp Trial 1	Su Trial 2	F Trial 3	Su Trial 4	Overall	Overall Feed/Gain
	CO	0.293	0.269	0.262	0.222	0.267	3.74
GNFDFT	LI	0.272[e]	0.26[ef]	0.262[ef]	0.247[f]	0.26	3.85
	NT		0.244		0.23	0.237	4.20
	CO		0.328	0.306	0.288	0.307	3.25
GNFDFC	LI		0.322[f]	0.306[f]	0.302[g]	0.311	3.22
	NT		0.307		0.3	0.304	3.29

Treatments: CO = Control; LI = Lincomycin; NT = NeoTerramycin

GNFD = Gain to Feed Ratio

N1 & N2 = First & Second Nursery Periods respectively

NT = Total Nursery Phase

G1 & G2 = First & Second Grower Periods respectively

GT = Total Grower Phase

GC = Cummulative through end of grower phase (NT + GT)

F1 & F2 = First & Second Finish Periods respectively

FT = Total Finish Phase

FC = Cumulative through end of the trial (NT + GT + FT)

abc = for each preweaning variable, treatments with different superscripts were significantly different overall (ie. across all trials) ($p < 0.05$).

efgh = for each preweaning variable, columns with different superscripts were significantly different trial:treatment interaction ($p < 0.05$).

Additional Statistical Analysis

R Square Analysis

The analytical model used DYS230 as the dependent variable versus a selection of both preweaning and health data as independent variables. The model statement was as follows:

DYS230 = BWT,AJWT,ELNG,ARN,TRN,SDEV,LIV,MNG

Pig weights and calculated ADG data collected during the study were not used in this analysis due to their serial correlation with DYS230. Feed efficiency data was not used because it is a growth performance parameter and because its measurement was done by pen rather than by individual pig resulting in numbers of observations too small to be of significant use in selecting variables for modeling.

Modeling results presented in Table 18 (by trial and treatment) indicate the best linear model using from 2-8 of the selected independent variables. Values shown are both the calculated R^2 (RSQ) and the R^2 adjusted for the degrees of freedom of the model (ADJRSQ).

For the smallest model (2 independent variables) the highest ADJRSQ was 49%, attained in the control pigs in Trials II and IV. Although the R^2 values were similar, the exact sequence of independent variables used to obtain them were not. Adding the rest of the 6 independent variables raised the ADJRSQ to a high of nearly 55%, again for one of the CO groups (Trial IV, CO = 55.1%) and also for one NT group (Trial IV, NT = 53.4%).

Through all of the different model sizes, the highest ADJRSQ was just over 59% and occurred in the 5 and 6 variable models of the CO group in Trial IV. The lowest ADJRSQ was 7.8% for the all 8 variable model of the NT group in Trial II.

Table 18 - RSquare Modeling

Model: DYS230 = BWT, AJWT, ELNG, ARN, TRN, SDEV, LIV, MNG

	Trt/Trt	RSQ	ADJRSQ	BWT	AJWT	ELNG	ARN	TRN	SDEV	LIV	MNG
Best 2	1CO	37.9	35.7		X						X
	2CO	53.7	49						X	X	
	3CO	16.9	13				X	X			
	4CO	53.5	49.1		X						X
	1LI	37.7	35.4				X				X
	2LI	28.9	26.1			X			X		
	3LI	32	30				X			X	
	4LI	15.4	12.9		X					X	
	2NT	30.2	23.2		X					X	
	4NT	49.9	46.6		X						X
Best 3	1CO	45.4	42.4	X						X	X
	2CO	54.9	47.7					X	X	X	
	3CO	24.8	19.4	X			X	X			
	4CO	60.8	55		X	X				X	
	1LI	47	44	X			X				X
	2LI	38.4	34.8			X			X	X	
	3LI	37.4	34		X		X			X	
	4LI	19.1	15.5		X	X			X		
	2NT	36.6	26.6		X			X		X	
	4NT	56.9	52.5	X	X						X
Best 4	1CO	48.1	44.3		X		X	X			X
	2CO	61.4	52.8				X	X	X	X	
	3CO	31.1	24.4	X		X	X	X			
	4CO	63.7	56		X	X		X			X
	1LI	51	47.3		X					X	X
	2LI	44.2	39.8			X			X	X	X
	3LI	41.8	37.6		X		X	X		X	
	4LI	22.3	17.6		X	X				X	
	2NT	39.5	26		X		X			X	X
	4NT	60.4	54.9	X	X		X				X
Best 5	1CO	52.4	48		X		X	X		X	X
	2CO	65.5	55.4		X		X	X	X	X	
	3CO	31.7	23.1	X	X	X	X	X			
	4CO	68.5	59.7	X	X	X				X	X
	1LI	54.7	50.3		X		X			X	X
	2LI	47.1	41.6			X			X	X	X
	3LI	44.4	39.2		X	X	X	X		X	
	4LI	23.6	17.8		X	X	X			X	
	2NT	42.7	25.8		X		X			X	X
	4NT	62.5	55.8	X	X			X			X
Best 6	1CO	55.3	50.2	X	X		X	X		X	X
	2CO	66.3	53.6		X		X	X	X	X	
	3CO	32.7	22.4	X	X	X	X	X			X
	4CO	70	59.4	X	X	X				X	X

Table 18 continued

	Trl/Trt	RSQ	ADJRSQ	BWT	AJWT	ELNG	ARN	TRN	SDEV	LIV	MNG
	1LI	56.1	50.9	X	X		X			X	X
	2LI	49.9	43.7			X		X	X	X	X
	3LI	44.5	38.2		X	X	X	X		X	
	4LI	23.9	16.8		X	X	X			X	X
	2NT	44	22.9		X	X	X			X	X
	4NT	63.6	55.5	X	X			X	X		X
Best 7	1CO	55.6	49.6	X	X		X	X		X	X
	2CO	66.6	51		X	X	X	X	X	X	
	3CO	32.8	20.4	X	X	X	X	X	X		X
	4CO	70.6	57.7	X	X	X		X		X	X
	1LI	56.3	50.1	X	X	X	X			X	X
	2LI	51.3	44		X	X		X	X	X	X
	3LI	44.5	37.1		X	X	X	X	X	X	
	4LI	23.9	15.5		X	X	X		X	X	X
	2NT	44.9	19.2		X	X	X		X	X	X
	4NT	64.6	55.1	X	X			X	X	X	X
Best 8	1CO	55.7	48.7	X	X	X	X	X		X	X
	2CO	66.6	47.6	X	X	X	X	X	X	X	X
	3CO	32.8	18.2	X	X	X	X	X	X		X
	4CO	70.7	55.1	X	X	X	X	X		X	X
	1LI	56.5	49.4	X	X	X	X		X	X	X
	2LI	51.4	42.4		X	X	X	X	X	X	X
	3LI	44.5	35.8	X	X	X	X	X	X	X	
	4LI	23.9	14.1	X	X	X	X		X	X	X
	2NT	45.2	13.9	X	X	X	X		X	X	X
	4NT	64.7	53.4	X	X	X		X	X	X	X
All 8	1CO	55.7	47.7						*		
	2CO	66.6	43.5								
	3CO	32.8	16							*	
	4CO	70.8	52						*		
	1LI	56.5	48.3					*			
	2LI	51.4	41.7	*							
	3LI	44.6	34.6								*
	4LI	24	12.7					*			
	2NT	45.5	7.8					*			
	4NT	64.8	51.6				*				

(* = variable that contributed least to the RSQ value)

Trl/Trt = Trial number and Treatment (CO = Control: LI = Lincomycin; NT = NeoTerramycin)
RSQ = R^2 value for the model indicated
ADJRSQ = R^2 value adjusted for variation and unequal number of observations
BWT = Birthweight; ADJWT = 21 day adjusted weight
ELNG = Estimated percentage pneumonia; ARN = Average rhinitis space
TRN = Total Rhinitis space; SDEV = Septal deviateion score; LIV = Liver ascarid score
MNG = Mange score
X = variable(s) included in the model

Discriminant Analysis and Logistic Regression

Procedures of Discriminant Analysis and Logistic Regression were attempted with SAS. Results of these analyses provided no additional meaningful interpretations of the data.

DISCUSSION

Trial Completion and Pig Removal

The number of pigs exhibiting clinical signs indicative of terminal ileitis (TI) and confirmed by the Michigan Animal Health Diagnostic Laboratory was problematic in this herd, especially during Trials I and II. Also, the often vague but yet significant clinical manifestation of TI resulting in slow growth also leads to concerns that many of the animals removed for slow growth may have been affected by TI. These findings are in direct disagreement with the report of Straw (1990) who suggested that TI does not affect growth rate.

One further note of explanation is needed with regard to the number of animals removed in Trial IV CO treatment, listed as "Other" reason. Six of the ten animals essentially disappeared. They were most likely mistakenly removed for sale or use for other educational purposes without adequate notification to allow for collection of final weight and slaughter check data.

Descriptive Statistics

Gill states that coefficients of variation (CV) "smaller that 0.01 are rare in biological sciences, and values larger that 3 or 4 are uncommon in most areas of research. For many biological traits, sample coefficients tend to be in the range of 0.05

to 0.5." (Gill, 1978) The variation between sample populations chosen to begin each trial was minimal as indicated by the low CV of BWT, AJWT, AGS, and STWT across treatment groups within each trial. As stated in the materials and methods, pigs were blocked by weight, sex and litter. Across all four trials, the CV for each of these four variables was less than 0.23, which indicated the success of the allotment procedure.

The variation in means for growth data were generally larger in magnitude but smaller in percentage as the pigs became older, as expected.

Associations Between Variable Groups

General

In evaluating associations, calculated correlation coefficients should be viewed with caution. Correlation coefficients (r) were considered weak if less than 0.25, moderate if between 0.25 and 0.5, and strong if greater than 0.5. At the upper extreme, correlation coefficients in biological measurements are somewhat suspect if they are larger than 0.9. No matter what level of correlation is attained, an additional caution in interpreting their significance is that simple correlation does not imply any causative relationship between the two associated variables.

The primary concerns of this study were to evaluate the relationships between indicators of enzootic pneumonia (CLNG and ELNG), atrophic rhinitis (TRN, ARN, SDEV) and growth performance. Correlation coefficients provided one general measure of these relationships.

Preweaning Data

The significance of the stronger preweaning:growth associations within treated pigs compared to untreated pigs was of questionable importance since no treatments were administered until after the preweaning period. Also, the differences in consistency between the LI and NT groups may have been directly related to the lesser numbers involved in the NT group (295 vs. 76) and fewer repetitions with the NT treatment (only two trials).

Health Data

The consistent association of the health data with the last phases of the growth data could be expected given the proximity in time that the measurements were taken. The correlation of mange (MNG) to daily gain was negative in the grower and finish phases, and was positive with days to 230. Both correlations could be an indication of a detrimental effect of mange on growth performance.

Associations Within Variable Groups

Preweaning Data

One would generally not expect BWT to have any particular association with AGS since AGS is more affected by actual weaning age which was set consistently by the planned pig flow in this production unit. The negative association between AGS and AJWT is a result of the adjustment calculation and not pertinent to this study.

Health Data

Liver scarring due to ascarid larval migration and mange scores were not correlated with AR or EP lesions. Perhaps the expected links between different disease entities generally do not exist, were of a nature other than the linear one examined by the correlation procedures, or were not detectable in this study.

One interesting aspect of associations between health data variables was the very nearly perfect positive correlation between CLNG and ELNG. In all three treatment groups, the correlation coefficient was greater than 0.90. This indicated that either measurement could be used to estimate the severity of pneumonic lesions. Also, the positive correlation between rhinitis measurements (ARN, TRN, SDEV) were consistent.

Analysis of Variance (ANOVA)

Preweaning Data

The lack of major, significant differences among preweaning data (see Table 14, page 97) between treatments within any given trial was another indication of the success in blocking pigs within the trials to provide an evenly based study group for each treatment. This minimization of bias regarding sex, genetics, weight, and age was the basis for stronger confidence in the statistical comparison of data collected throughout the remainder of the trials.

The slight advantage in BWT for Trials II, III, and IV compared to Trial I was somewhat expected due to historic evidence and records of the production unit studied. The disappearance of weight differences as the pigs progressed in age might be expected since 3-4 weeks of lactation and environmental influences can exert very

significant effects on pig weight. However, a specific explanation could not be discerned.

Growth Data

The isolated significant differences documented in the early growth periods (see Table 15, page 97), such as the NT treated pigs having a higher PADGG1 than either the CO or LI groups across all trials, and Trial I exhibiting poorer weight gains than any of the other three trials, had no firm logical or supportable explanation.

The trial:treatment interaction with higher ADG in Trials I and III coincided with the lower finish phase gains expected in the warmer summer weather associated with Trials II and IV. The cooler weather during the finish phase in Trial III pigs which were slaughtered in the fall season may explain the lower DYS230 compared to the other three trials. However, the seasonal comparison was not repeated to allow for more stringent statistical evaluation.

In summary, ADG differences by treatment were generally as expected up through the grower phase. By individual growth period from the second nursery period (PADGN2) to the second grower period (PADGG2), and for the total and cumulative nursery and grower phases, the LI and NT treatments had better average daily gains than CO. Zimmerman's (1986) review of the literature on the use of antimicrobials in pig production supports these observed differences.

From the statistical evaluation of trial:treatment interactions among these same nursery and grower periods (significance only for PADGG1, PADGGT, & PADGGC), it appeared that gains were better during the late winter and spring (Trials II and IV).

However for PADGGC, the improved gains occurred during the late spring and summer months (Trial III). These differences coincide with the spring months which biologically could have supported better feed intake and growth due to more favorable environmental temperatures and ventilation rates. The findings of Straw et al. (1985) suggested that performance of pigs in a test station were best when the pigs entered the station during the spring and summer months. More extensive support for this conclusion was not found in the literature reviewed.

Zimmerman's (1986) review questioned the value of antimicrobials for growth performance enhancement in the finish periods (125-220 lbs.) and suggested that the response to antimicrobial feed additives decreases with increasing age. These concepts supported the differences in gain patterns observed in this study in the finish periods compared to the nursery and grower periods.

Finish phase data (PADGF1, PADGF2, & PADGFT) showed a reversal of the performance differences between CO, LI, and NT groups that were established in the nursery and grower phases. The gains in the second and total finish periods (PADGF2 & PADGFT) were greater for CO pigs than for either LI or NT. In addition, the LI group showed significantly better gains than the NT groups.

These reversals in gain are often described as "compensatory gains." However, this concept lacks any sound biological explanation and is not consistently observed. Unsubstantiated claims suggest that decreasing the level or complete removal of feed additive antimicrobials during the finish period results in drastic changes in the microbial gut flora and environment of previously medicated pigs. As the digestive system adapts to this change, gains are slowed to the point where the nonmedicated pigs, while not

experiencing such changes in the intestinal environment, continue to gain at the same rate and actually surpass the previously medicated groups.

The lack of significant trial:treatment interactions in the finish periods or phases of the study also adds some doubt to the significance of such interactions noted in the grower.

For the cumulative aspect of all growth periods, across all four trials, the lack of statistically significant differences in ADG between any of the three treatments leads to several considerations. First, it might have been that the levels of treatment used, though FDA approved for such use, were not high enough to yield a significant effect. Second, there may not have been enough pigs studied to detect any differences. Third, other factors not accounted for may have significantly interfered with treatment effect. Among the variables that might be included here are genetics, season, nutrition, and environment. However, in the study design, the factors were applied evenly across all treatment groups. There also may not have been a difference.

Health Data

It was expected that the health data (see Table 16, page 100) would yield some significant differences between medicated and nonmedicated groups, especially those parameters related to respiratory disease. However, there were no significant differences between treatments across all four trials for any of the variables measured.

Numerous studies in support of FDA approved claims for both medications used have indicated significant, positive benefits. But, as Zimmerman's (1986) review stated, the benefits described were in growth performance and made no mention of a

corresponding differences in disease lesions as measured in this study. Apparently, disease treatment or control by feed medication resulted in improved performance without appreciable alteration of disease lesion severity.

Other considerations as to why such benefits or differences were not evident in this study were much the same as those discussed for growth data. In addition, it must be considered that the levels of EP and AR in the unit studied were not high enough to demonstrate significant improvement or control in response to the treatments used. There may be a threshold of both disease entities beyond which response is measurable and significant, but below which differences can not be detected.

Feed Efficiency Data

As with the pig growth data, it was expected that feed efficiency data (see page 86) would show significant improvement among treated groups over controls. Such expectations were also supported by Zimmerman's review (1986).

The significant treatment differences in the nursery phases were expected, but the magnitude of difference was somewhat less than expected. The literature reviewed by Zimmerman (1986) covering studies from 1950-1985, found an average of 6.5% better feed efficiency for treated versus control groups during the nursery period over all studies conducted. Within those studies, trials using lincomycin averaged 6.7% improvement in feed efficiency. No Neo-Terramycin trials were reported.

After converting the gain/feed data from Table 11 into feed/gain for comparison with Zimmerman's review (1986), the magnitude of difference between treated groups

and controls during the starter period (GNFDNT) was 5.7% and 3.8% for the LI and NT treatments, respectively.

Zimmerman's review stated that, for the grow/finish period, the average improvement in feed efficiency of treated versus control pigs was 2.4%. Therefore, the lack of significant differences through grower and finisher in this study was not expected. In fact, the LI pigs were only 0.3% more efficient than both CO and NT pigs in the grower period. In the finish period, the CO pigs were actually more efficient than either the LI or NT groups. By comparison, the lincomycin studies reported in Zimmerman's review averaged 1.7% better FE than controls during the grow/finish period overall. Again, there were no Neo-Terramycin studies reported.

Zimmerman's review also stated that the magnitude of difference in feed efficiency through grow/finish is only about 30% of that obtained in the nursery phase.

Several possible reasons for differences between the feed efficiency results of this study and the expectations provided by past studies include genetics, type of production facilities, seasonal variation, dietary differences, and differences in health status. In particular, the age and function problems of the feeders used in the grower and finish phases in this study varied somewhat by pen and could have contributed to variation in feed availability and wastage, and consequently impacted the accuracy of FE measurement.

Additional Statistical Analysis

R Square Analysis

Modeling DYS230 by RSquare analysis (see Table 18, page 90) using preweaning and health data provided many interesting and diverse results:

1) There were no distinguishable or consistent patterns as to when variables entered the "best" models nor if they remained a part of successively larger models. This questions the variables selected for the model. Perhaps they were not the best variables to choose or perhaps their variation within the number of pigs studied was too large to give the significant, consistent relationships needed for accurate, consistent modeling.
2) The most significant independent variables were not the same within a given treatment group across all four trials.
3) The AJWT, LIV, and MNG variables were found to be more significant than expected as they were selected in many of the smaller models. For AJWT and MNG, this was reflective of their consistently significant correlation to DYS230 across nearly all treatments and trials.
4) As model sizes became progressively larger, the adjusted R^2 value often decreased. One would expect R^2 to increase as more known significant variables were added to the model. However, this expectation can only be realized if the independent variables have significant relationship to the dependent one and are likewise significantly related among themselves. This confirms the correlation data previously presented which also indicated

that the variables selected for this modeling exercise were not well correlated.

5) The fact that the model statement included two weight (BWT and AJWT) and three atrophic rhinitis (ARN, TRN, and SDEV) measurements makes the exercise of caution in interpretation important because of the colinearity within each group of variables.

Discriminant Analysis

The discriminant procedures of SAS were designed for revealing differences among classes of observations that in the case of continuous variables such as DYS230, CLNG, and ARN, appear to require much larger observation numbers to delineate any added significance.

The lack of results with the discriminant analysis procedures could be somewhat expected. The complex and unbalanced nature of the data set and the complexity of the manipulations account for most of the expected failure.

Also to be considered are the basic statistical assumptions and purposes of discriminant analysis. They include the assumptions that the independent variables have equal variance within each group and that correlations among the variables within each group are the same (Montgomery et al., 1986), neither of which were true in this study.

SAS procedures (1985) also states the purpose of Discriminant Analysis is to predict classes or differences between classes of variables. The continuous nature of the growth and disease measurement variables of this study would therefore nullify any expectation of success using this procedure unless the measurements were grouped in

categories or classes by range or other transformation method. Such categories would be applicable to studying the significance of data such as the charts published by the TRAC study (1985), but would not be specific enough to be useful in actual mathematical modeling of disease:growth interactions.

Logistic Regression

The results of the Logistic Regression differ only slightly from the regression model using actual data where DYS230 in Trial IV was not significantly different from any other trial.

The results of the Logistic Regression ultimately differ so very little from those of the initial regression model that it provides no better understanding of any time specific relationship of growth phases to actual DYS230.

CONCLUSIONS

Several statistically significant relationships between enzootic pneumonia, atrophic rhinitis, and growth performance were evident. However, the relationships were inconsistent in magnitude and significance across trials and treatment groups.

Specifically, the following relationships between growth performance, enzootic pneumonia lesions, atrophic rhinitis lesions and feed additive medications were observed:

1) Enzootic pneumonia lesions were most severe in pigs with lower rate of gain in the last 3-12 weeks prior to slaughter. This relationship was not affected by season of the year.

2) Atrophic rhinitis lesions were most prevalent and severe in pigs with lower rate of gain primarily in the last 6 weeks, but in some cases as much as 15 weeks, before slaughter. The relationship between atrophic rhinitis and growth was also not affected by season.

3) As the prevalence of atrophic rhinitis and enzootic pneumonia lesions increased, it took longer for pigs to reach 230 pounds. However, less than 9% of the variation in DYS230 could be explained by variation in the levels of either of these diseases.

4) Because DYS230 was not reduced by feed medication, the possibility of "compensatory gain" occurring in CO pigs after medication was removed from treated pigs during the finish phase was apparent.

5) The prevalence and severity of enzootic pneumonia and atrophic rhinitis lesions were not significantly related to each other.

6) There was no relationship between feed additive medication and the prevalence and severity of lesions of enzootic pneumonia and atrophic rhinitis at slaughter.

7) Regarding the effect of feed additive medication on sequential periods of growth performance and feed efficiency: a) There was a significant benefit to pig growth rate in the nursery and grower phases with the feed additive medication programs used. b) This benefit also was evident in the feed efficiency data in the nursery phase only. c) The consistency of the benefit for Neo-Terramycin was not adequately tested. d) In the finish phase, the feed additive medication program utilized in this study had no beneficial effect on growth rate. In fact, the removal of feed additive medication in the finish phase appeared to be detrimental to the growth rate of those pigs who receive feed medication in the nursery and grower phases.

8) Regarding the evaluation of the relationship of pig preselection data and feed additive medication to enzootic pneumonia and atrophic rhinitis lesions: There were a few negative correlations between preselection data points, and the pneumonia and rhinitis lesions. Because preselection data

was used to block the pigs utilized in the individual trials, and pig weights were serially correlated, any direct relationship of these data to slaughter lesions or medication treatments was either irrelevant or impossible to quantify.

9) The calculated method for assessing the severity of lung lesions of enzootic pneumonia, used in this study and derived from dissection studies, can be accurately duplicated using estimation techniques that are more compatible with the time efficiencies required in performing slaughter checks.

In addition to these somewhat limited, although statistically supported conclusions generated from this study, there were many questions left unanswered and several new ones generated. For the correlations of variables measured, there were general questions of why most of them were so variable in their statistical significance and how significant were their magnitudes? For instance, why were the growth phases not more strongly correlated?

The lack of strong, consistent, mathematically defined relationships in this study was in opposition to the perception that anything that adversely affects the form and function of the respiratory tract must have a negative effect on the pigs' growth performance. Perhaps, there is a critical threshold of enzootic pneumonia and atrophic rhinitis that must be present before performance is impaired. Seemingly, the level of biological insult to the respiratory tract that results in decreased growth performance has not been defined.

The possibility also must be considered that either the variables measured in this study were inadequate in number to allow significant delineation of the relationships evaluated, were poorly measured, or simply were not the correct ones to account for a significant amount of variation in the dependent variables studied. Perhaps not enough animals were observed to allow detection of the level of change desired. Pointon et al. (1990) published guidelines for determining sample size for a given expected prevalence, desired confidence level in the results, and desired accuracy of the estimates. Since the disease lesions studied, especially enzootic pneumonia, were capable of at least some resolution over time, it was stated that lesions recorded at slaughter should only be related to pigs within eight weeks of market weight. However, the use of these tables is related to prevalence estimates within a population and may not be applicable to studies such as the present one where concern is focused on disease severity in the individual pig and the subsequent growth performance of that same individual.

Some question could be given to the disease lesion measurement techniques utilized. Morrison (1985) reviewed the various methods for pneumonia lesion measurement. In addition, some reports have utilized computer topography methods to further refine the accuracy of measurement (Done and Upcott, 1982). As long as the measurements are taken in a consistent manner by a single technique and individual observer, study results should be accurate and suitable for statistical analysis.

As related to variables not measured or to improper measurement of included variables, the question of study design arises. One consideration was that the study of seasonal effects should include repeated studies in similar time periods. Two summer

slaughter studies, one fall, and one spring did not provide data for valid statistical comparisons of seasonal differences.

A second consideration was the genetic variation involved. Although the pigs were blocked by litter, which would balance genetics across treatments in the study, the number of genotypes might still have significantly affected the relationships studied. For the mathematical study and delineation of relationships as attempted in this study, a single genetic base with larger numbers per group would be more desirable.

Post trial discussions with statisticians, other than those involved in designing the study, suggested that analysis by trial and treatment was faulty because only the treatment variable was fixed while the trial variable was not. Also, the number of pen replications with each trial and treatment were insufficient to facilitate the best analysis.

For the linear models evaluated using the ANOVA procedures in SAS, the primary question was how significant were the resulting R^2 values and what other independent variables could have been added to the models to improve the resulting R^2? It could very well be that the R^2 values from this study were lower than expected because expectations were too high. There were no values reported elsewhere in the literature to compare or support such expectations. Expectations exist from intuition or perceptions regarding the biological nature and the known variables affecting disease and performance. These perceptions and intuitions may not be very accurate.

Potentially important variables that may not have been adequately considered in this study include both sex and genetics. Even though the study design included blocking for sex and litter to minimize the effect of these variables, the fact that the production unit studied contained a mixture of purebred and crossbred pigs may have diluted or

simply adversely affected the magnitude and significance of not only the R^2 values obtained but also the correlations between variables.

Additionally, simple alterations/variations in execution of the study could have significantly altered results. Such variations include the use of data from an MOF facility in Trial I, incomplete weight data (grower phases) in Trial I, and not scheduling the trials to repeat, as nearly as possible, the calendar time frame of a previous trial.

In a broader perspective, questions arise as to the applicability of results of this study to other studies and/or other swine production units. It is generally accepted that extrapolation of results from one production unit to another regarding performance and disease control is dangerous at best. So how do the results from the study of this production unit compare to others? Can the variations noted in this study be considered "normal" variations for pork production in any type of production unit?

No extensive literature values were found to make firm comparisons. Most likely that is due in part to the lack of well defined protocols for general use, the cost of doing such extensive studies, and the interest of commercial producers in putting their units through such scrutiny.

It does appear that if the primary variables of interest measured in this study (DYS230, CLNG, ARN) were made more discrete (by category or range), rather than continuous, the statistical evaluation of their relationships might be enhanced by use of discriminant analysis and logistic regression. But, such a shift in focus would not be as specific in generating information for models capable of predicting the small increments of performance change that result in economically significant changes.

Modeling of pork production must become more refined and detailed to allow more accurate decision making based on smaller increments of change. For example, there is important value to increments of only one day of the total days to 230 pounds. If the average days to 230 is approximately 180 days, then any one day change is only a 0.55% change in the total average. That is a very small increment of change to measure accurately and with confidence in experimental studies. However, this one day difference is significant to managers of pork production, especially when it is applied to several thousand animals as exist in many production units today.

The weak points in this study's design included factors such as facilities, season, and genetics that affect the relationships evaluated and the planned statistical analysis.

Therefore, to build upon the strengths of this study and to overcome it's weaknesses, further research efforts to study the relationships between enzootic pneumonia, atrophic rhinitis, and growth performance should include:

1. Fix the variables of environment and management. This would include limiting all repetitions to the same buildings and pens used previously and repeating studies in more closely related calendar time periods to more accurately capture seasonal differences.
2. Further study of genetic effects on the relationship of disease and growth performance. The genotype of study animals should be well defined and limited to one genetic base within a study unless actual genetic comparisons were being made.
3. Increasing the number of study animals would improve the opportunity for accuracy, significance, and confidence in results.

4. Studies should be set to collect serially correlated data more frequently and in larger numbers for specifically evaluating temporal relationships of endpoint data, such as pneumonia and rhinitis scores, to growth data points occurring at variable time lengths prior to the endpoint data collection. More specifically, studies should be designed to take advantage of the mathematical capabilities of discriminant analysis and logistic regression.

5. Further exploration of the concept of days to 40 pounds as a predictor of subsequent performance to market might be of benefit to decisions made in currently evolving production schemes. With the advent of multiple site production and options of feeder pig or market hog sales, establishing an accurate predictor of DYS230 at the 40 pound stage of pig growth could be a very beneficial tool for making economic decisions.

6. There is a need to expand the information based record collection systems in active production units and use them to establish methods of on-farm studies to accurately track disease, medication, environment, nutrition and management related impacts on performance.

7. Epidemiologically based studies, designed specifically for the quantification of the multitude of determinants of disease, should be coupled with development of on-farm methods to institute optimal and economically sound management schemes.

8. Activities of meat inspection personnel should be expanded to include data collection relevant to on-farm records and epidemiological research studies as mentioned above.

In general, future research should continue to be of a very specific nature. More effort should be put into standardization of study design, variable measurement and statistical analysis so that resulting data may be combined with and compared to other studies to allow for the development and testing of detailed mathematical models needed for future expert analysis systems and more critical decision making.

Statistical significance needs to be producible for the changes that are economically significant to production. However, current methods of study and analysis do not yet produce the results necessary to meet that need. Until such a situation is realized, the ultimate use of detailed, expert analysis of cost:benefit scenarios in the control of enzootic pneumonia and atrophic rhinitis to optimize performance of pork production will fall far short of ideal.

APPENDIX I

Table 1 - MEANS - Treatment within Trial - Trial 1

	Control					Lincomycin			
Item	Mean	STD	C.V.	N	:	Mean	STD	C.V.	N
BWT	3.445	0.852	0.24	78	:	3.475	0.905	0.26	79
AJWT	12.571	2.796	0.22	78	:	12.592	3.02	0.23	77
AGS	29.385	3.264	0.11	78	:	29.468	3.23	0.10	79
STWT	17.5	4.206	0.24	78	:	17.456	4.023	0.23	79
N1WT	28.513	7.471	0.26	78	:	29.728	7.142	0.24	79
N2WT	48.122	12.164	0.25	78	:	52.276	12.024	0.23	78
G1WT					:				
G2WT	92.801	24.699	0.26	73	:	106.743	16.66	0.15	74
F1WT	158.97	22.559	0.14	66	:	167.403	22.485	0.13	72
EWT	188.39	24.76	0.13	65	:	194.593	21.761	0.11	70
CLNG	6.62	8.383	1.26	60	:	4.839	5.977	1.23	63
DYS230	182.69	16.52	0.09	65	:	182.02	22.053	0.12	70
ELNG	6.467	7.31	1.13	60	:	5.524	6.36	1.15	63
ARN	5.45	3.28	0.60	60	:	4.927	1.888	0.38	62
TRN	2	2.408	1.20	60	:	1.581	1.807	1.14	62
SDEV	0.5	0.873	1.74	60	:	0.355	0.655	1.84	62
LIV	0.717	0.865	1.20	60	:	1.758	0.918	0.52	62
MNG	0.367	0.823	2.24	60	:	0.129	0.338	2.62	62
PADGN1	0.524	0.195	0.37	78	:	0.583	0.223	0.38	79
PADGN2	1.06	0.408	0.38	78	:	1.2	0.314	0.26	78
PADGNT	0.773	0.23	0.29	78	:	0.877	0.224	0.25	78
PADGG1					:				
PADGG2					:				
PADGGT	1.192	0.532	0.44	73	:	1.477	0.236	0.15	74
PADGGC	0.995	0.3	0.30	73	:	1.178	0.171	0.14	74
PADGF1	1.788	0.254	0.14	66	:	1.779	0.298	0.16	72
PADGF2	1.635	0.354	0.21	65	:	1.447	0.314	0.21	70
PADGFT	1.737	0.231	0.13	65	:	1.671	0.238	0.14	70
PADGFC	1.339	0.172	0.12	65	:	1.384	0.151	0.10	70
GNFDN1	0.54	0.041	0.08	8	:	0.564	0.031	0.06	8
GNFDN2	0.476	0.043	0.09	8	:	0.489	0.02	0.04	8
GNFDNT	0.498	0.029	0.06	8	:	0.513	0.015	0.03	8
GNFDG1					:				
GNFDG2					:				
GNFDGT	0.348	0.025	0.07	4	:	0.371	0.008	0.02	4
GNFDGC					:				
GNFDF1	0.31	0.026	0.08	4	:	0.309	0.043	0.14	4
GNFDF2	0.265	0.017	0.06	4	:	0.212	0.025	0.12	4
GNFDFT	0.293	0.02	0.07	4	:	0.272	0.033	0.12	4
GNFDFC					:				

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 2 - MEANS - Treatment within Trial - Trial 2

Item	Control Mean	STD	C.V.	N	Lincomycin Mean	STD	C.V.	N	NeoTerramycin Mean	STD	C.V.	N
BWT	3.403	0.768	0.22	36	3.592	0.808	0.22	73	3.497	0.839	0.23	36
AJWT	12.381	2.18	0.17	36	13.164	2.102	0.15	73	12.964	2.625	0.20	36
AGS	27.556	3.791	0.13	36	27.343	3.564	0.13	73	27.528	3.715	0.13	36
STWT	15.717	3.614	0.22	36	16.426	3.226	0.19	73	16.414	3.87	0.23	36
N1WT	24.708	6.89	0.27	36	29.623	8.06	0.27	73	28.208	8.805	0.31	36
N2WT	46.048	10.193	0.22	31	52.806	11.9	0.22	72	52.788	12.691	0.24	33
G1WT	81.25	11.224	0.13	28	88.437	16.887	0.19	71	89.438	16.226	0.18	32
G2WT	117.85	13.305	0.11	28	123.214	21.886	0.17	70	124.125	18.854	0.15	32
F1WT	152.75	14.948	0.09	28	158.174	19.343	0.12	69	158.344	22.701	0.14	32
EWT	193.10	18.161	0.09	28	195.879	19.351	0.09	66	191.406	26.847	0.14	32
CLNG	7.822	7.132	0.91	26	9.132	10.433	1.14	58	5.02	4.18	0.83	27
DYS230	180.67	11.966	0.06	28	181.569	17.965	0.09	66	184.465	22.586	0.12	32
ELNG	6.808	5.123	0.75	26	7.948	8.769	1.10	58	4.593	3.846	0.83	27
ARN	6.481	1.905	0.29	26	5.974	1.58	0.26	58	6.537	1.737	0.26	27
TRN	2.269	1.687	0.74	26	1.638	1.385	0.84	58	1.852	1.748	0.94	27
SDEV	0.308	0.471	1.52	26	0.259	0.48	1.85	58	0.296	0.465	1.57	27
LIV	1.346	0.892	0.66	26	1.534	1.03	0.67	58	1.519	0.975	0.64	27
MNG	0.385	0.196	0.50	26	0.224	0.421	1.87	58	0.037	0.192	5.18	27
PADGN1	0.595	1.203	2.02	36	0.628	0.315	0.50	73	0.562	0.299	0.53	36
PADGN2	0.994	0.416	0.41	31	1.145	0.264	0.23	72	1.155	0.337	0.29	33
PADGNT	0.738	0.239	0.32	31	0.887	0.252	0.28	72	0.882	0.246	0.27	33
PADGG1	1.442	0.254	0.17	28	1.573	0.204	0.12	70	1.556	0.245	0.15	32
PADGG2	1.83	0.183	0.1	28	1.756	0.271	0.15	69	1.734	0.276	0.15	32
PADGGT	1.163	0.189	0.16	28	1.66	0.173	0.10	69	1.639	0.227	0.13	32
PADGGC	1.216	0.151	0.12	28	1.292	0.173	0.13	69	1.28	0.193	0.15	32
PADGF1	1.662	0.29	0.17	28	1.58	0.323	0.20	69	1.629	0.417	0.25	32
PADGF2	1.922	0.325	0.16	28	1.773	0.394	0.22	66	1.574	0.458	0.29	32
PADGFT	1.792	0.26	0.14	28	1.684	0.261	0.15	66	1.602	0.353	0.22	32
PADGFC	1.408	0.141	0.10	28	1.424	0.146	0.10	66	1.387	0.193	0.13	32
GNFDN1	0.522	0.192	0.37	4	0.603	0.06	0.1	8	0.617	0.156	0.25	4
GNFDN2	0.405	0.116	0.29	4	0.466	0.02	0.04	8	0.448	0.033	0.07	4
GNFDNT	0.436	0.105	0.24	4	0.509	0.018	0.04	8	0.495	0.051	0.1	4
GNFDG1	0.411	0.015	0.04	2	0.411	0.037	0.09	4	0.406	0.017	0.04	2
GNFDG2	0.355	0.001	0.00	2	0.332	0.011	0.03	4	0.32	0.005	0.02	2
GNFDGT	0.38	0.006	0.02	2	0.368	0.019	0.05	4	0.358	0.002	0.00	2
GNFDFC	0.402	0.022	0.05	2	0.392	0.024	0.06	4	0.381	0.017	0.05	2
GNFDF1	0.284	0.015	0.05	2	0.287	0.012	0.04	4	0.272	0.011	0.04	2
GNFDF2	0.257	0.001	0.00	2	0.239	0.022	0.09	4	0.221	0.017	0.07	2
GNFDFT	0.269	0.006	0.02	2	0.26	0.017	0.07	4	0.244	0.005	0.02	2
GNFDFC	0.328	0.007	0.02	2	0.322	0.021	0.07	4	0.307	0.011	0.04	2

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 3 - MEANS - Treatment within Trial - Trial 3

Item	Control Mean	STD	C.V.	N	:	Lincomycin Mean	STD	C.V.	N
BWT	3.551	0.683	0.19	63	:	3.644	0.663	0.18	63
AJWT	13.522	2.367	0.17	63	:	13.538	2.774	0.20	63
AGS	32.794	3.199	0.09	63	:	32.825	3.353	0.10	63
STWT	19.548	3.264	0.16	63	:	19.484	3.337	0.17	63
N1WT	40.373	8.63	0.21	63	:	45.286	8.964	0.19	63
N2WT	59.548	10.459	0.17	62	:	66.557	9.031	0.13	62
G1WT	92.397	13.513	0.14	58	:	97.817	11.97	0.12	60
G2WT	124.93	17.518	0.14	58	:	130.8	15.027	0.11	60
F1WT	165.66	18.297	0.11	57	:	172.367	20.108	0.11	60
EWT	215	21.045	0.09	56	:	217.767	24.674	0.11	60
CLNG	8.44	7.871	0.93	50	:	8.065	8.704	1.07	60
DYS230	173.73	15.52	0.08	56	:	172.038	16.406	0.09	60
ELNG	7.66	7.061	0.92	50	:	7.383	7.618	1.03	60
ARN	5.188	1.542	0.29	48	:	5.217	1.86	0.35	60
TRN	1.896	1.547	0.81	48	:	2.033	1.365	0.67	60
SDEV	0.354	0.483	1.36	48	:	0.383	0.613	1.60	60
LIV	0.94	0.843	0.89	50	:	0.933	0.778	0.83	60
MNG	0.06	0.24	4	50	:	0.033	0.181	5.48	60
PADGN1	0.986	0.386	0.39	63	:	1.229	0.34	0.27	63
PADGN2	1.036	0.261	0.25	62	:	1.159	0.14	0.12	62
PADGNT	1.027	0.228	0.22	62	:	1.208	0.175	0.14	62
PADGG1	1.541	0.266	0.17	58	:	1.489	0.23	0.15	60
PADGG2	1.549	0.327	0.21	58	:	1.571	0.374	0.23	60
PADGGT	1.545	0.263	0.17	58	:	1.53	0.226	0.14	60
PADGGC	1.302	0.2	0.15	58	:	1.375	0.162	0.11	60
PADGF1	1.887	0.283	0.14	57	:	1.979	0.501	0.25	60
PADGF2	1.811	0.295	0.16	56	:	1.681	0.374	0.22	60
PADGFT	1.846	0.222	0.12	56	:	1.812	0.332	0.18	60
PADGFC	1.515	0.159	0.10	56	:	1.538	0.179	0.11	60
GNFDN1	0.713	0.058	0.08	7	:	0.777	0.042	0.05	7
GNFDN2	0.348	0.019	0.06	7	:	0.361	0.033	0.09	7
GNFDNT	0.478	0.029	0.06	7	:	0.513	0.023	0.04	7
GNFDG1	0.393	0.029	0.07	3	:	0.372	0.019	0.05	3
GNFDG2	0.307	0.018	0.06	3	:	0.298	0.01	0.03	3
GNFDGT	0.344	0.021	0.06	3	:	0.33	0.009	0.03	3
GNFDGC	0.363	0.014	0.04	3	:	0.36	0.008	0.02	3
GNFDF1	0.282	0.013	0.04	3	:	0.296	0.014	0.05	3
GNFDF2	0.245	0.009	0.04	3	:	0.229	0.008	0.03	3
GNFDFT	0.262	0.011	0.04	3	:	0.262	0.011	0.04	3
GNFDFC	0.306	0.003	0.01	3	:	0.306	0.006	0.02	3

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 4 - MEANS - Treatment within Trial - Trial 4

	Control				Lincomycin				NeoTerramycin			
Item	Mean	STD	C.V.	N	Mean	STD	C.V.	N	Mean	STD	C.V.	N
BWT	3.785	0.684	0.18	40	3.668	0.698	0.19	80	3.863	0.651	0.16	40
AJWT	15.704	2.19	0.13	40	15.559	2.155	0.13	80	15.947	2.462	0.15	40
AGS	23.5	7.254	0.30	40	23.238	7.266	0.31	80	23.4	7.441	0.31	40
STWT	18.4	3.247	0.17	40	18.119	3.223	0.17	80	18.6	3.934	0.21	40
N1WT	32.4	6.566	0.20	40	32.463	6.304	0.19	80	33.75	7.312	0.21	40
N2WT	53.275	9.84	0.18	40	54.738	8.999	0.16	80	57.25	9.909	0.17	40
G1WT	78.85	14.726	0.18	40	81.788	12.859	0.15	80	87.025	14.355	0.16	40
G2WT	118.34	18.407	0.15	38	119.722	16.012	0.13	79	125.9	17.113	0.13	40
F1WT	147.82	21.806	0.14	34	151.885	18.955	0.12	78	163.526	19.335	0.11	38
EWT	198.51	25.82	0.13	33	199.636	24.602	0.12	77	207.237	26.252	0.12	38
CLNG	6.649	6.807	1.02	25	7.601	6.722	0.88	71	7.741	7.842	1.01	34
DYS230	178.43	16.481	0.09	33	179.39	16.663	0.09	77	175.062	20.048	0.11	38
ELNG	5.84	4.913	0.84	25	6.375	5.33	0.83	72	6.676	6.034	0.90	34
ARN	6	2.72	0.45	25	5.791	2.216	0.38	74	5.912	1.998	0.33	34
TRN	2.04	1.837	0.90	25	1.581	1.588	1.00	74	1.706	1.567	0.91	34
SDEV	0.0833	0.282	3.38	24	0.095	0.295	3.10	74	0.088	0.288	3.27	34
LIV	1.68	0.557	0.33	25	1.541	0.847	0.54	74	1.559	0.824	0.52	34
MNG	0.56	0.583	1.04	25	0.743	0.76	1.02	74	0.294	0.629	2.13	34
PADGN1	0.667	0.204	0.30	40	0.683	0.202	0.29	80	0.721	0.208	0.28	40
PADGN2	1.195	0.255	0.21	40	1.275	0.22	0.17	80	1.345	0.207	0.15	40
PADGNT	0.906	0.203	0.22	40	0.952	0.18	0.18	80	1.004	0.181	0.18	40
PADGG1	1.472	0.387	0.26	40	1.524	0.24	0.15	80	1.67	0.253	0.15	40
PADGG2	1.622	0.384	0.23	38	1.654	0.377	0.22	79	1.608	0.327	0.20	40
PADGGT	1.519	0.217	0.14	38	1.539	0.217	0.14	79	1.616	0.186	0.11	40
PADGGC	1.234	0.168	0.13	38	1.257	0.165	0.13	79	1.325	0.158	0.11	40
PADGF1	1.36	0.589	0.43	34	1.538	0.429	0.27	78	1.802	0.34	0.18	38
PADGF2	1.566	0.273	0.17	33	1.5	0.301	0.20	77	1.383	0.332	0.24	38
PADGFT	1.495	0.251	0.16	33	1.524	0.233	0.15	77	1.546	0.251	0.16	38
PADGFC	1.35	0.172	0.12	33	1.363	0.156	0.11	77	1.414	0.165	0.11	38
GNFDN1	0.522	0.045	0.09	4	0.543	0.043	0.08	8	0.542	0.049	0.09	4
GNFDN2	0.458	0.041	0.09	4	0.477	0.036	0.08	8	0.479	0.046	0.1	4
GNFDNT	0.48	0.019	0.04	4	0.499	0.016	0.03	8	0.498	0.019	0.04	4
GNFDG1	0.431	0.105	0.24	2	0.43	0.104	0.24	4	0.46	0.082	0.18	2
GNFDG2	0.292	0.02	0.07	2	0.3	0.034	0.11	4	0.302	0.001	0.00	2
GNFDGT	0.323	0.001	0.00	2	0.328	0.017	0.05	4	0.339	0.01	0.03	2
GNFDGC	0.343	0.001	0.00	2	0.349	0.014	0.04	4	0.359	0.01	0.03	2
GNFDF1	0.224	0.001	0.00	2	0.262	0.035	0.13	4	0.253	0.043	0.17	2
GNFDF2	0.22	0.004	0.02	2	0.234	0.01	0.04	4	0.207	0.021	0.1	2
GNFDFT	0.222	0.002	0.00	2	0.247	0.021	0.08	4	0.23	0.031	0.14	2
GNFDFC	0.288	0.001	0.00	2	0.302	0.003	0.01	4	0.3	0.018	0.06	2

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 5 - MEANS - Treatment within and across Trials - Control Treatment

Item	Trial 1 Mean	Trial 1 STD	Trial 1 C.V.	Trial 1 N	Trial 2 Mean	Trial 2 STD	Trial 2 C.V.	Trial 2 N	Trial 3 Mean	Trial 3 STD	Trial 3 C.V.	Trial 3 N	Trial 4 Mean	Trial 4 STD	Trial 4 C.V.	Trial 4 N	All 4 Trials Mean	All 4 Trials STD	All 4 Trials C.V.	All 4 Trials N
BWT	3.445	0.852	0.24	78	3.403	0.768	0.22	36	3.551	0.683	0.19	63	3.785	0.684	0.18	40	3.531	0.756	0.21	217
AJWT	12.571	2.796	0.22	78	12.381	2.18	0.17	36	13.522	2.367	0.17	63	15.704	2.19	0.13	40	13.393	2.456	0.18	217
AGS	29.385	3.264	0.11	78	27.556	3.791	0.13	36	32.794	3.199	0.09	63	23.5	7.254	0.30	40	28.986	4.41	0.15	217
STWT	17.5	4.206	0.24	78	15.717	3.614	0.22	36	19.548	3.264	0.16	63	18.4	3.247	0.17	40	17.965	3.654	0.20	217
N1WT	28.513	7.471	0.26	78	24.708	6.89	0.27	36	40.373	8.63	0.21	63	32.4	6.566	0.20	40	32.041	7.533	0.23	217
N2WT	48.122	12.164	0.25	78	46.048	10.193	0.22	31	59.548	10.459	0.17	62	53.275	9.84	0.18	40	52.152	10.902	0.20	211
G1WT					81.25	11.224	0.13	28	92.397	13.513	0.14	58	78.85	14.726	0.18	40	85.619	13.347	0.15	126
G2WT	92.801	24.699	0.26	73	117.857	13.305	0.11	28	124.931	17.518	0.14	58	118.342	18.407	0.15	38	165.51	20.042	0.12	197
F1WT	158.97	22.559	0.14	66	152.75	14.948	0.09	28	165.667	18.297	0.11	57	147.824	21.806	0.14	34	158.044	19.993	0.12	185
EWT	188.39	24.76	0.13	65	193.107	18.161	0.09	28	215	21.045	0.09	56	198.515	25.82	0.13	33	199.139	22.769	0.11	182
CLNG	6.62	8.383	1.26	60	7.822	7.132	0.91	26	8.44	7.871	0.93	50	6.649	6.807	1.02	25	7.384	7.776	1.05	161
DYS230	182.69	16.52	0.09	65	180.674	11.966	0.06	28	173.732	15.52	0.08	56	178.431	16.481	0.09	33	178.85	15.462	0.08	182
ELNG	6.467	7.31	1.13	60	6.808	5.123	0.75	26	7.66	7.061	0.92	50	5.84	4.913	0.84	25	6.795	6.566	0.96	161
ARN	5.45	3.28	0.60	60	6.481	1.905	0.29	26	5.188	1.542	0.29	48	6	2.72	0.45	25	5.626	2.576	0.45	159
TRN	2	2.408	1.20	60	2.269	1.687	0.74	26	1.896	1.547	0.81	48	2.04	1.837	0.90	25	2.019	1.96	0.97	159
SDEV	0.5	0.873	1.74	60	0.308	0.471	1.52	26	0.354	0.483	1.36	48	0.0833	0.282	3.38	24	0.361	0.635	1.75	158
LIV	0.717	0.865	1.20	60	1.346	0.892	0.66	26	0.94	0.843	0.89	50	1.68	0.557	0.33	25	1.037	0.815	0.78	161
MNG	0.367	0.823	2.24	60	0.385	0.196	0.50	26	0.06	0.24	4	50	0.56	0.583	1.04	25	0.248	0.602	2.42	161
PADGN1	0.524	0.195	0.37	78	0.595	1.203	2.02	36	0.986	0.386	0.39	63	0.667	0.204	0.30	40	0.696	0.579	0.83	217
PADGN2	1.06	0.408	0.38	78	0.994	0.416	0.41	31	1.036	0.261	0.25	62	1.195	0.255	0.21	40	1.069	0.349	0.32	211
PADGNT	0.773	0.23	0.29	78	0.738	0.239	0.32	31	1.027	0.228	0.22	62	0.906	0.203	0.22	40	0.868	0.224	0.25	211
PADGG1					1.442	0.254	0.17	28	1.541	0.266	0.17	58	1.472	0.387	0.26	40	1.497	0.305	0.20	126
PADGG2					1.83	0.183	0.1	28	1.549	0.327	0.21	58	1.622	0.384	0.23	38	1.635	0.318	0.19	124
PADGGT	1.192	0.532	0.44	73	1.163	0.189	0.16	28	1.545	0.263	0.17	58	1.519	0.217	0.14	38	1.355	0.371	0.27	197
PADGGC	0.995	0.3	0.30	73	1.216	0.151	0.12	28	1.302	0.2	0.15	58	1.234	0.168	0.13	38	1.163	0.266	0.22	197
PADGF1	1.788	0.254	0.14	66	1.662	0.29	0.17	28	1.887	0.283	0.14	57	1.36	0.589	0.43	34	1.721	0.349	0.20	185
PADGF2	1.635	0.354	0.21	65	1.922	0.325	0.16	28	1.811	0.295	0.16	56	1.566	0.273	0.17	33	1.721	0.316	0.18	182
PADGFT	1.737	0.231	0.13	65	1.792	0.26	0.14	28	1.846	0.222	0.12	56	1.495	0.251	0.16	33	1.735	0.264	0.15	182
PADGFC	1.339	0.172	0.12	65	1.408	0.141	0.10	28	1.515	0.159	0.10	56	1.35	0.172	0.12	33	1.406	0.162	0.11	182
GNFDN1	0.54	0.041	0.08	8	0.522	0.192	0.37	4	0.713	0.058	0.08	7	0.522	0.045	0.09	4	0.586	0.119	0.2	23
GNFDN2	0.476	0.043	0.09	8	0.405	0.116	0.29	4	0.348	0.019	0.06	7	0.458	0.041	0.09	4	0.422	0.077	0.18	23
GNFDNT	0.498	0.029	0.06	8	0.436	0.105	0.24	4	0.478	0.029	0.06	7	0.48	0.019	0.04	4	0.478	0.05	0.1	23
GNFDG1					0.411	0.015	0.04	2	0.393	0.029	0.07	3	0.431	0.105	0.24	2	0.409	0.05	0.12	7
GNFDG2					0.355	0.001	0.00	2	0.307	0.018	0.06	3	0.292	0.02	0.07	2	0.316	0.03	0.1	7
GNFDGT	0.348	0.025	0.07	4	0.38	0.006	0.02	2	0.344	0.021	0.06	3	0.323	0.001	0.00	2	0.348	0.025	0.07	11
GNFDGC					0.402	0.022	0.05	2	0.363	0.014	0.04	3	0.343	0.001	0.00	2	0.368	0.027	0.07	7
GNFDF1	0.31	0.026	0.08	4	0.284	0.015	0.05	2	0.282	0.013	0.04	3	0.224	0.001	0.00	2	0.282	0.035	0.13	11
GNFDF2	0.265	0.017	0.06	4	0.257	0.001	0.00	2	0.245	0.009	0.04	3	0.22	0.004	0.02	2	0.25	0.02	0.08	11
GNFDFT	0.293	0.02	0.07	4	0.269	0.006	0.02	2	0.262	0.011	0.04	3	0.222	0.002	0.00	2	0.267	0.029	0.11	11
GNFDFC					0.328	0.007	0.02	2	0.306	0.003	0.01	3	0.288	0.001	0.00	2	0.307	0.017	0.06	7

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 6 - MEANS - Treatment within and across Trials - Lincomycin Treatment

Item	Trial 1				Trial 2				Trial 3				Trial 4				All 4 Trials			
	Mean	STD	C.V.	N	Mean	STD	C.V.	N	Mean	STD	C.V.	N	Mean	STD	C.V.	N	Mean	STD	C.V.	N
BWT	3.475	0.905	0.26	79	3.592	0.808	0.22	73	3.644	0.663	0.18	63	3.668	0.698	0.19	80	3.592	0.775	0.21	295
AJWT	12.592	3.02	0.23	77	13.164	2.102	0.15	73	13.538	2.774	0.20	63	15.559	2.155	0.13	80	13.748	2.521	0.18	293
AGS	29.468	3.23	0.10	79	27.343	3.564	0.13	73	32.825	3.353	0.10	63	23.238	7.266	0.31	80	27.969	4.737	0.16	295
STWT	17.456	4.023	0.23	79	16.426	3.226	0.19	73	19.484	3.337	0.17	63	18.119	3.223	0.17	80	17.814	3.462	0.19	295
N1WT	29.728	7.142	0.24	79	29.623	8.06	0.27	73	45.286	8.964	0.19	63	32.463	6.304	0.19	80	33.766	7.554	0.22	295
N2WT	52.276	12.024	0.23	78	52.806	11.9	0.22	72	66.557	9.031	0.13	62	54.738	8.999	0.16	80	56.113	10.578	0.18	292
G1WT					88.437	16.887	0.19	71	97.817	11.97	0.12	60	81.788	12.859	0.15	80	88.583	14.045	0.15	211
G2WT	106.74	16.66	0.15	74	123.214	21.886	0.17	70	130.8	15.027	0.11	60	119.722	16.012	0.13	79	119.54	17.527	0.14	283
F1WT	167.40	22.485	0.13	72	158.174	19.343	0.12	69	172.367	20.108	0.11	60	151.885	18.955	0.12	78	161.849	20.149	0.12	279
EWT	194.59	21.761	0.11	70	195.879	19.351	0.09	66	217.767	24.674	0.11	60	199.636	24.602	0.12	77	201.419	22.601	0.11	273
CLNG	4.839	5.977	1.23	63	9.132	10.433	1.14	58	8.065	8.704	1.07	60	7.601	6.722	0.88	71	7.374	8.149	1.10	252
DYS230	182.02	22.053	0.12	70	181.569	17.965	0.09	66	172.038	16.406	0.09	60	179.39	16.663	0.09	77	178.975	18.343	0.10	273
ELNG	5.524	6.36	1.15	63	7.948	8.769	1.10	58	7.383	7.618	1.03	60	6.375	5.33	0.83	72	6.763	7.055	1.04	253
ARN	4.927	1.888	0.38	62	5.974	1.58	0.26	58	5.217	1.86	0.35	60	5.791	2.216	0.38	74	5.486	1.955	0.35	254
TRN	1.581	1.807	1.14	62	1.638	1.385	0.84	58	2.033	1.365	0.67	60	1.581	1.588	1.00	74	1.701	1.543	0.90	254
SDEV	0.355	0.655	1.84	62	0.259	0.48	1.85	58	0.383	0.613	1.60	60	0.095	0.295	3.10	74	0.264	0.518	1.96	254
LIV	1.758	0.918	0.52	62	1.534	1.03	0.67	58	0.933	0.778	0.83	60	1.541	0.847	0.54	74	1.449	0.889	0.61	254
MNG	0.129	0.338	2.62	62	0.224	0.421	1.87	58	0.033	0.181	5.48	60	0.743	0.76	1.02	74	0.307	0.569	1.85	254
PADGN1	0.583	0.223	0.38	79	0.628	0.315	0.50	73	1.229	0.34	0.27	63	0.683	0.202	0.29	80	0.759	0.366	0.48	295
PADGN2	1.2	0.314	0.26	78	1.145	0.264	0.23	72	1.159	0.14	0.12	62	1.275	0.22	0.17	80	1.198	0.251	0.20	292
PADGNT	0.877	0.224	0.25	78	0.887	0.252	0.28	72	1.208	0.175	0.14	62	0.952	0.18	0.18	80	0.97	0.245	0.25	292
PADGG1					1.573	0.204	0.12	70	1.489	0.23	0.15	60	1.524	0.24	0.15	80	1.53	0.225	0.14	210
PADGG2					1.756	0.271	0.15	69	1.571	0.374	0.23	60	1.654	0.377	0.22	79	1.664	0.343	0.20	208
PADGGT	1.477	0.236	0.15	74	1.66	0.173	0.10	69	1.53	0.226	0.14	60	1.539	0.217	0.14	79	1.55	0.224	0.14	282
PADGGC	1.178	0.171	0.14	74	1.292	0.173	0.13	69	1.375	0.162	0.11	60	1.257	0.165	0.13	79	1.27	0.181	0.14	282
PADGF1	1.779	0.298	0.16	72	1.58	0.323	0.20	69	1.979	0.501	0.25	60	1.538	0.429	0.27	78	1.705	0.39	0.22	279
PADGF2	1.447	0.314	0.21	70	1.773	0.394	0.22	66	1.681	0.374	0.22	60	1.5	0.301	0.20	77	1.592	0.343	0.21	273
PADGFT	1.671	0.238	0.14	70	1.684	0.261	0.15	66	1.812	0.332	0.18	60	1.524	0.233	0.15	77	1.664	0.283	0.17	273
PADGFC	1.384	0.151	0.10	70	1.424	0.146	0.10	66	1.538	0.179	0.11	60	1.363	0.156	0.11	77	1.422	0.157	0.11	273
GNFDN1	0.564	0.031	0.06	8	0.603	0.06	0.1	8	0.777	0.042	0.05	7	0.543	0.043	0.08	8	0.617	0.1	0.16	31
GNFDN2	0.489	0.02	0.04	8	0.466	0.02	0.04	8	0.361	0.033	0.09	7	0.477	0.036	0.08	8	0.451	0.057	0.13	31
GNFDNT	0.513	0.015	0.03	8	0.509	0.018	0.04	8	0.513	0.023	0.04	7	0.499	0.016	0.03	8	0.508	0.018	0.04	31
GNFDG1					0.411	0.037	0.09	4	0.372	0.019	0.05	3	0.43	0.104	0.24	4	0.407	0.065	0.16	11
GNFDG2					0.332	0.011	0.03	4	0.298	0.01	0.03	3	0.3	0.034	0.11	4	0.311	0.026	0.08	11
GNFDGT	0.371	0.008	0.02	4	0.368	0.019	0.05	4	0.33	0.009	0.03	3	0.328	0.017	0.05	4	0.35	0.025	0.07	15
GNFDGC					0.392	0.024	0.06	4	0.36	0.008	0.02	3	0.349	0.014	0.04	4	0.368	0.026	0.07	11
GNFDF1	0.309	0.043	0.14	4	0.287	0.012	0.04	4	0.296	0.014	0.05	3	0.262	0.035	0.13	4	0.288	0.032	0.11	15
GNFDF2	0.212	0.025	0.12	4	0.239	0.022	0.09	4	0.229	0.008	0.03	3	0.234	0.01	0.04	4	0.228	0.02	0.09	15
GNFDFT	0.272	0.033	0.12	4	0.26	0.017	0.07	4	0.262	0.011	0.04	3	0.247	0.021	0.08	4	0.26	0.022	0.09	15
GNFDFC					0.322	0.021	0.07	4	0.306	0.006	0.02	3	0.302	0.003	0.01	4	0.311	0.015	0.05	11

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

Table 7 - MEANS - Treatment within and across Trials - NeoTerramycin Treatment

Item	Trial 1 Mean	Trial 1 STD	Trial 1 N	Trial 2 Mean	Trial 2 STD	Trial 2 C.V.	Trial 2 N	Trial 3 Mean	Trial 3 STD	Trial 3 N	Trial 4 Mean	Trial 4 STD	Trial 4 C.V.	Trial 4 N	All 4 Trials Mean	All 4 Trials STD	All 4 Trials C.V.	All 4 Trials N
BWT				3.497	0.839	0.23	36				3.863	0.651	0.16	40	3.6896	0.741	0.20	76
AJWT				12.964	2.625	0.20	36				15.947	2.462	0.15	40	14.534	2.523	0.17	76
AGS				27.528	3.715	0.13	36				23.4	7.441	0.31	40	25.355	5.896	0.23	76
STWT				16.414	3.87	0.23	36				18.6	3.934	0.21	40	17.565	3.852	0.21	76
N1WT				28.208	8.805	0.31	36				33.75	7.312	0.21	40	31.125	7.999	0.25	76
N2WT				52.788	12.691	0.24	33				57.25	9.909	0.17	40	55.233	11.17	0.20	73
G1WT				89.438	16.226	0.18	32				87.025	14.355	0.16	40	88.097	15.104	0.17	72
G2WT				124.125	18.854	0.15	32				125.9	17.113	0.13	40	125.111	17.778	0.14	72
F1WT				158.344	22.701	0.14	32				163.526	19.335	0.11	38	161.157	20.784	0.12	70
EWT				191.406	26.847	0.14	32				207.237	26.252	0.12	38	199.999	26.332	0.13	70
CLNG				5.02	4.18	0.83	27				7.741	7.842	1.01	34	6.536	6.576	1.00	61
DYS230				184.465	22.586	0.12	32				175.062	20.048	0.11	38	179.361	14.575	0.08	70
ELNG				4.593	3.846	0.83	27				6.676	6.034	0.90	34	5.754	5.246	0.91	61
ARN				6.537	1.737	0.26	27				5.912	1.998	0.33	34	6.189	1.9	0.30	61
TRN				1.852	1.748	0.94	27				1.706	1.567	0.91	34	1.771	1.635	0.92	61
SDEV				0.296	0.465	1.57	27				0.088	0.288	3.27	34	0.18	0.373	2.07	61
LIV				1.519	0.975	0.64	27				1.559	0.824	0.52	34	1.541	0.886	0.57	61
MNG				0.037	0.192	5.18	27				0.294	0.629	2.13	34	0.18	0.5	2.77	61
PADGN1				0.562	0.299	0.53	36				0.721	0.208	0.28	40	0.646	0.265	0.41	76
PADGN2				1.155	0.337	0.29	33				1.345	0.207	0.15	40	1.259	0.288	0.22	73
PADGNT				0.882	0.246	0.27	33				1.004	0.181	0.18	40	0.949	0.22	0.23	73
PADGG1				1.556	0.245	0.15	32				1.67	0.253	0.15	40	1.619	0.248	0.15	72
PADGG2				1.734	0.276	0.15	32				1.608	0.327	0.20	40	1.664	0.303	0.18	72
PADGGT				1.639	0.227	0.13	32				1.616	0.186	0.11	40	1.626	0.204	0.12	72
PADGGC				1.28	0.193	0.15	32				1.325	0.158	0.11	40	1.305	0.175	0.13	72
PADGF1				1.629	0.417	0.25	32				1.802	0.34	0.18	38	1.723	0.374	0.21	70
PADGF2				1.574	0.458	0.29	32				1.383	0.332	0.24	38	1.47	0.392	0.26	70
PADGFT				1.602	0.353	0.22	32				1.546	0.251	0.16	38	1.571	0.301	0.19	70
PADGFC				1.387	0.193	0.13	32				1.414	0.165	0.11	38	1.402	0.177	0.12	70
GNFDN1				0.617	0.156	0.25	4				0.542	0.049	0.09	4	0.58	0.114	0.2	8
GNFDN2				0.448	0.033	0.07	4				0.479	0.046	0.1	4	0.464	0.041	0.09	8
GNFDNT				0.495	0.051	0.1	4				0.498	0.019	0.04	4	0.497	0.036	0.07	8
GNFDG1				0.406	0.017	0.04	2				0.46	0.082	0.18	2	0.433	0.057	0.13	4
GNFDG2				0.32	0.005	0.02	2				0.302	0.001	0.00	2	0.311	0.011	0.04	4
GNFDGT				0.358	0.002	0.00	2				0.339	0.01	0.03	2	0.349	0.013	0.04	4
GNFDGC				0.381	0.017	0.05	2				0.359	0.01	0.03	2	0.37	0.017	0.05	4
GNFDF1				0.272	0.011	0.04	2				0.253	0.043	0.17	2	0.263	0.028	0.11	4
GNFDF2				0.221	0.017	0.07	2				0.207	0.021	0.1	2	0.214	0.018	0.08	4
GNFDFT				0.244	0.005	0.02	2				0.23	0.031	0.14	2	0.237	0.02	0.08	4
GNFDFC				0.307	0.011	0.04	2				0.3	0.018	0.06	2	0.304	0.013	0.04	4

STD = standard deviation
C.V. = coefficient of variation
N = number of observations used

APPENDIX II

Table IV - GAIN CORRELATION - Control Diet Over All Four Trials

	BWT	AJWT	STWT	AGS	CLNG	ARN	MNG	DYS230	PADGN1	PADGN2	PADGNT	PADGG1	PADGG2	PADGGT	PADGGC	PADGF1	PADGF2	PADGFT	PADGFC
BWT		0.5303	0.2857	-0.098	0.0074	-0.160	0.0005	-0.256	0.0796	0.2869	0.2698	0.0232	0.0376	0.1282	0.1708	0.0569	-0.025	0.0059	0.1513
		0.0001	0.0001	0.1475	0.9252	0.043	0.9946	0.0005	0.2429	0.0001	0.0001	0.7958	0.6779	0.0725	0.0164	0.4415	0.7309	0.9363	0.0414
		217	217	217	161	159	161	182	217	211	211	126	124	197	197	185	182	182	182
AJWT			0.6141	-0.232	0.0559	-0.144	-0.001	-0.284	0.1483	0.2164	0.2923	0.1028	-0.068	0.0856	0.1709	-0.087	-0.090	-0.129	0.1131
			0.0001	0.0005	0.4812	0.0684	0.9845	0.0001	0.0289	0.0016	0.0001	0.2519	0.4477	0.2316	0.0163	0.2343	0.2267	0.0805	0.1284
			217	217	161	159	161	182	217	211	211	126	124	197	197	185	182	182	182
STWT				0.4143	-0.063	-0.178	0.0265	-0.344	0.0012	0.1924	0.4635	0.2389	0.0878	0.1544	0.2934	0.0964	0.0301	0.1114	0.3456
				0.0001	0.421	0.0242	0.7382	0.0001	0.9852	0.005	0.0001	0.007	0.3317	0.0302	0.0001	0.1914	0.6866	0.1343	0.0001
				217	161	159	161	182	217	211	211	126	124	197	197	185	182	182	182
AGS					-0.122	-0.043	0.0265	-0.054	0.2235	-0.008	0.2821	0.0916	0.1527	0.0855	0.1723	0.2871	0.1173	0.3287	0.3433
					0.1205	0.589	0.7382	0.4686	0.0009	0.907	0.0001	0.3073	0.0903	0.232	0.0155	0.0001	0.1147	0.0001	0.0001
					161	159	161	182	217	211	211	126	124	197	197	185	182	182	182
CLNG						0.3083	0.0473	0.1083	-0.032	0.0142	-0.019	0.1979	-0.151	0.0703	0.0477	-0.109	-0.110	-0.158	-0.058
						0.0001	0.5508	0.1713	0.6811	0.8574	0.809	0.0472	0.1292	0.3752	0.548	0.1679	0.1622	0.0441	0.4611
						159	161	161	161	161	161	101	101	161	161	161	161	161	161
ARN							0.1144	0.2406	-0.075	-0.067	-0.104	-0.039	0.0584	-0.026	-0.068	-0.157	-0.197	-0.244	-0.186
							0.1507	0.0022	0.3462	0.3975	0.188	0.6988	0.5656	0.7384	0.3929	0.0475	0.0124	0.0019	0.0185
							159	159	159	159	159	99	99	159	159	159	159	159	159
MNG								0.3164	-0.134	0.0187	-0.108	-0.158	0.1442	-0.326	-0.272	-0.260	-0.281	-0.348	-0.354
								0.0001	0.0894	0.8136	0.1693	0.114	0.1501	0.0001	0.0005	0.0008	0.0003	0.0001	0.0001
								161	161	161	161	101	101	161	161	161	161	161	161
DYS230									-0.443	-0.424	-0.584	-0.534	-0.354	-0.654	-0.733	-0.509	-0.540	-0.673	-0.889
									0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
									182	182	182	117	117	182	182	182	182	182	182
PADGN1										0.1487	0.7636	0.3203	-0.042	0.2392	0.5280	0.1481	0.1704	0.2123	0.5905
										0.0308	0.0001	0.0003	0.6356	0.0007	0.0001	0.0442	0.0214	0.004	0.0001
										211	211	126	124	197	197	185	182	182	182
PADGN2											0.7506	0.3097	0.2147	0.4117	0.5766	0.0624	-0.113	0.0312	0.3916
											0.0001	0.0004	0.0166	0.0001	0.0001	0.3987	0.1272	0.675	0.0001
											211	126	124	197	197	185	182	182	182
PADGNT												0.4039	0.0804	0.4338	0.7452	0.1541	0.0750	0.1889	0.6761
												0.0001	0.3744	0.0001	0.0001	0.0362	0.314	0.0106	0.0001
												126	124	197	197	185	182	182	182
PADGG1													0.1668	0.7108	0.6970	0.0426	0.2448	0.1671	0.5212
													0.064	0.0001	0.0001	0.6451	0.0078	0.0716	0.0001
													124	124	124	119	117	117	117
PADGG2														0.7903	0.5571	0.1049	0.2604	0.2066	0.4016
														0.0001	0.0001	0.2561	0.0046	0.0254	0.0001
														124	124	119	117	117	117
PADGGT															0.9149	0.1456	0.3466	0.3165	0.7520
															0.0001	0.0479	0.0001	0.0001	0.0001
															197	185	182	182	182
PADGGC																0.1612	0.2622	0.2898	0.8404
																0.0284	0.0003	0.0001	0.0001
																185	182	182	182
PADGF1																	0.2369	0.7923	0.5777
																	0.0013	0.0001	0.0001
																	182	182	182
PADGF2																		0.7571	0.5806
																		0.0001	0.0001
																		182	182
PADGFT																			0.7525
																			0.0001
																			182

Pearson Correlation Coefficient
Prob > |R| Under HO:RHO=0
Number of Observations

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