



Intestinal Microbes: When does normality change into a health and performance insult? — David B. Anderson, Ph.D.

“Biography”

David B. Anderson was raised on a livestock and grain farm in Minnesota. He earned a Ph.D. from the University of Wisconsin majoring in Muscle Biology and Biochemistry. He served as an officer in the U.S. Army, as assistant professor in the Department of Animal Sciences at the University of Illinois, and is currently a Senior Research Scientist in Elanco Animal Health, Discovery Research and Development, Greenfield, Indiana.

Dr. Anderson's research career has been focused on meat animal growth and development, studying beta adrenergic agonists, estrogen/androgens, somatotropin, GHRH analogs, and nutritional effects. In 1998 he was a visiting scholar at the University of Illinois and a visiting scientist at Wageningen Agricultural University in the Netherlands studying alternatives to antibiotic growth promoters. He is author or co-author of 12 patents and 92 refereed publications/invited papers/book chapters/abstracts. He was the 2001 recipient of the Animal Growth and Development Award from the American Society of Animal Science. He has an adjunct appointment in the Department of Animal Science, Purdue University and is currently on the Board of Directors and Program Chairman for ASAS.

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Intestinal Microbes: When does normality change into a health and performance insult?

“Abstract”

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Introduction

The gastrointestinal tract harbors a metabolically active microbiota, primarily bacteria, which develop simultaneously a cooperative and a competitive relationship with the host animal. The epithelial lining of the GI tract is characterized by a high cell turnover and the constant production of a protective mucous coat. Together these two physiological processes provide effective innate defense against luminal threats. These innate defense functions of the gut epithelium, however, are provided at the expense of animal growth efficiency. Gut tissues represent approximately only 5% of body weight but account for 15 to 30% of whole body oxygen consumption and protein turnover because of the high rate of epithelial cell turnover and metabolism (Gaskins, 2001). Only 10% of the total protein synthesized by the GI tract is accumulated as new mass. Most proteins are lost sloughed epithelial cells or as secreted products such as mucous (Reeds *et al.*, 1993a). This paper describes how normal microbial populations can affect gut health and animal productivity.

Understanding Non-Specific Bacterial Enteritis

The mechanism of growth-promoting antibiotics can help to understand the role of gut microbial populations in gut health and productivity. Considerable evidence suggests that growth-promoting antibiotics modify gut microbial populations or activities (Visek, 1978). For example, feeding antibiotics does not induce a growth-response in germfree animals (Coates *et al.*, 1963), while infecting germfree animals with gastrointestinal bacteria from normal animals

results in growth depression (Coates, 1980). Further, the growth response to feeding antibiotics is enhanced in conventional animals raised under conditions with greater microbial load (Hays, 1969; Roura *et al.*, 1992). The most likely scenario for the mechanism of growth promotion by antibiotics is therefore that one or more of the organisms commonly inhabiting the animal gut, though not necessarily pathogenic, nevertheless cause a depression in growth which is reversed when the responsible organism(s) are metabolically inhibited or eliminated by inclusion of antibiotics in the diet (Coates, 1980). Supporting evidence for this hypothesis has been provided by studies in chickens demonstrating that monoassociation of germfree chicks with the Gram-positive, facultative anaerobe *Enterococcus (Streptococcus) faecium*, induced the growth depression obtained with a total gut inoculum (Fuller *et al.*, 1979). The growth-depressing effect of *E. faecium* was reversed by feeding penicillin. (Fuller *et al.*, 1983). These data, along with lack of effect in germfree animals, are consistent with the idea that feed-antibiotics are growth-permitting rather than growth-promoting.

This paper discusses the concept that the indigenous microbiota in the small intestine depresses growth by:

- 1) competing with the bird for nutrients, and
- 2) producing microbial metabolites that increase gut mucosa turnover and consequently reduce growth efficiency.

It is proposed that feeding antibiotics reverses microbial-induced growth depression by increasing the utilization of nutrients and by reducing maintenance costs of the gastrointestinal system.

Intestinal Bacteria Produce Growth-Depressing Metabolites

Phenolic/aromatic compounds. Highly toxic phenolic and aromatic compounds such as phenol, 4-methylphenol (*p*-cresol), 4-ethylphenol, indole, and 3-methylindole (skatole) are produced by bacterial degradation of tyrosine and tryptophan in the gut and excreted in the urine (Deichmann and Witherup, 1943). These phenolic compounds are not produced or excreted in the urine of germfree rats (Bakke and Midtvedt, 1970). It is generally accepted that, in humans, an increase in dietary protein results in increased production of fecal ammonia, fecal volatile sulfur substances, and urinary *p*-cresol (Geypens *et al.*, 1997). However, in fasting individuals, the amount of phenol and *p*-cresol excreted in the urine is unchanged, despite the complete absence of oral nutrition (Bures *et al.*, 1990). Thus, urinary phenol and *p*-cresol excretion does not depend solely on oral dietary protein intake, but rather may reflect metabolism of endogenous substrates by intestinal bacteria. A negative correlation ($r=-0.73$) between urinary *p*-cresol concentrations and body weight gain was observed in weanling pigs, suggesting that microbially-produced *p*-cresol may depress growth (Yokoyama *et al.*, 1982). Fecal and urinary excretion of phenolic and aromatic compounds, particularly *p*-cresol, were decreased in weanling pigs fed ASP-250 (Yokoyama, *et al.*, 1982). Further, the inverse relationship shown between volatile phenol excretion and weight gain in rats fed a sucrose diet containing 10% tyrosine was reversed when diets were supplemented with chlortetracycline (Bernhart and Zilliken, 1958). Therefore,

reduced bacterial production of phenolic compounds is a potential mechanism for gut microbially induced growth depression.

Ammonia. Ammonia is a toxic waste product of microbial amino acid deamination and urea hydrolysis mediated by the enzyme urease (Visek, 1981, 1984). Urease activity is ubiquitous among human intestinal bacteria (Suzuki *et al.* 1979). The concentration of ammonia found in the colon of conventional animals is several times that required for cell damage (reviewed by Visek, 1978), indicating that ammonia produced by gut microbial urease could have pronounced biological effects at concentrations occurring naturally. Several lines of evidence suggest that microbially-produced urease and the resulting high concentrations of ammonia are deleterious for growth. For example, urea hydrolysis does not occur in germfree animals (Levenson *et al.*, 1959) and portal ammonia is only 25% the concentration found in conventional controls (Warren and Newton, 1959). Rats and chicks immunized against urease have lower *in vivo* urease activity, lower ammonia concentrations, and faster growth than non-immunized controls (Dang and Visek, 1960). Pigs fed ion exchange resins capable of adsorbing ammonia also exhibited improved growth (Pond and Yen, 1987; Veldman and Van der Aar, 1997). Visek (1978) proposed that reduction of microbially-produced ammonia is a primary mechanism for the growth response induced by feed antibiotics.

Bile acid biotransformation. Feighner and Dashkevicz (1987, 1988) proposed that an important mechanism of growth-promoting antibiotics is the inhibition of microbial bile acid biotransformation in the gut. Microbial deconjugation and dehydroxylation of bile impairs lipid absorption by the host animal (DeSomer *et al.*, 1963; Eyssen, 1973) and produce toxic degradation products that can impair growth (Eyssen and DeSomer, 1963a). Bile acids are not deconjugated in the gut of germfree animals, demonstrating the important role of intestinal bacteria in this process (Madsen *et al.*, 1976). Although other bacteria, such as *Bacteroides*, *Bifidobacterium*, and *Clostridium* spp. (Kawamoto *et al.*, 1989; Stellwag and Hylemon, 1976; Grill *et al.*, 1995; and Gopal-Srivastava and Hylemon, 1988) possess bile-salt hydrolase activity, lactobacilli inhabiting the small intestine may be largely responsible for bile salt hydrolysis. For example, ileal bile salt hydrolase activity in conventional mice is reduced 86% by the elimination of lactobacilli from the microbiota, and by greater than 98% when both lactobacilli and enterococci are eliminated (Tannock *et al.*, 1989). These results indicate that lactobacilli are among the principal contributors to total bile salt hydrolase activity in the mouse intestinal tract. Using chicks, Eyssen and DeSomer (1963b) first suggested that bile acid transformation products might be responsible for the growth depression caused by intestinal bacteria. Additional evidence from chick studies showed that bile acid deconjugation by gut bacteria causes growth depression that is reversible by antibiotic supplementation (Fuller *et al.* 1984). Further, Feighner and Dashkevicz (1987, 1988) have shown an inverse relationship between the level of cholyltaurine hydrolase activity in the small intestine and growth rate in broiler chickens fed antibiotics.

Growth-depressing microbial metabolites. A summary of gut organisms responsible for the production of growth-depressing microbial metabolites discussed above is shown in Table B-1. It is interesting to note that although different types of bacteria may generate one or more of the metabolites mentioned, the Gram-positive facultative anaerobes, which are oxygen-tolerant and predominant in the small intestine, often produce all three toxins. This phenomenon may help explain why reducing these populations or modifying

their metabolism with antibiotics would enhance growth. The data implicating specific types of bacteria in the production of growth-depressing metabolites are based on cultivation techniques and therefore should be interpreted with caution.

It is curious that the class of organisms that appear to depress growth, namely Gram-positive facultative anaerobes including strains of *Lactobacillus* and *Enterococcus*, are also often used as probiotic organisms for enhancing health and promoting growth in livestock (reviewed in Jonsson and Conway, 1992). The growth-promoting effect of probiotics in livestock is less consistent than that observed with antibiotic supplementation (Jonsson and Conway, 1992). Supplementation of animals and humans with certain probiotic bacteria has been shown to provide protection against intestinal, diarrhea-producing pathogens (reviewed in McCracken and Gaskins, 1998). Therefore, probiotics may promote growth under situations in which certain pathogens are present; however, these same organisms in a different facility may suppress growth via the mechanisms discussed above.

Small Intestinal Microbiota Competitive With The Host

Culture-based studies have shown that microbial activity in the small intestine tends to be competitive with the host for energy and amino acids (Hedde and Lindsey, 1986). For example, bacterial utilization of glucose to produce lactic acid reduces the energy available to the host animal (Saunders and Sillery, 1982). Lactic acid also enhances peristalsis, thus increasing the rate of nutrient transit in the gut (Saunders and Sillery, 1982). As much as 6% of the net energy in pig diets can be lost due to bacterial utilization of glucose in the small intestine (Vervaeke *et al.*, 1979). Amino acids, which are also degraded by small intestinal bacteria, are made unavailable to the pig and produce toxic metabolites such as ammonia, cadaverine, and *p*-cresol. Although microbial activity in the cecum and colon tends to be cooperative with the host (Hedde and Lindsay, 1986), with estimates up to 5-20% of the pig's total energy being provided by fermentations of distal gut bacteria (Friend *et al.*, 1963), the small intestine is the principal site of nutrient and energy absorption. Further, bacterial populations in the small intestine are several orders of magnitude smaller than in the large intestine (Stewart, 1996). Therefore it is proposed that the benefits of growth-promoting antibiotics result from a substantial decrease in bacterial populations and consequent alterations in epithelial functions in the small intestine, whereas changes in large intestine microbial populations exert less impact on whole animal growth. In support of this hypothesis, most of the growth-promoting antibiotics target Gram-positive organisms (Table B-1), and the small intestinal microbiota consists predominantly of Gram-positive bacteria (Stewart, 1996). Available data on the spatial distribution of bacterial groups along the gastrointestinal tract were generated via culture-based techniques and are thus undoubtedly biased. Emerging molecular-based techniques as discussed by other speakers at this symposium will enable a more accurate evaluation of the concept that animal growth may be influenced by the spatial density and perhaps taxonomic composition of the microbiota along the gastrointestinal tract.

Gut Bacteria And Intestinal Inflammation

Intestinal bacteria play an important role in the development of the intestinal immune system (Gaskins, 1996). This immunogenic role of the gut microbiota is most clearly observed in the immaturity of the gut immune system in germfree animals, which have underdeveloped intestinal lymphoid tissues, substantially decreased numbers of lymphocytes (B and T-cells), and low antibody concentrations (Wostmann, 1996). These immune parameters convert to the normal state when germfree animals are associated with a full complement of intestinal bacteria (Carter and Pollard, 1971). Studies in which individual species or known groups of bacteria have been introduced into germfree animals have shown that different bacterial species may be very immunogenic, moderately immunogenic, or weakly/nonimmunogenic (McCracken and Gaskins, 1999). Obviously, bacterial stimulation of intestinal immune system development is crucial for protective immunity. However, one potential mechanism by which growth-promoting antibiotics may exert their effects is to decrease immunogenic bacteria inhabiting the small intestine. By limiting growth of small intestinal bacteria, growth-promoting antibiotics may decrease the energetic costs associated with the constitutive, low-level inflammation in the gut of conventional animals. Thus the trade-off between the costs of local inflammation versus the necessity of immune competence becomes an issue which will be influenced by the housing environment. Stahly and co-workers (1995) studied the impact of tylosin on rate, efficiency, and composition of growth in pigs subject to either a conventional or medicated early-weaning protocol. They determined that feeding tylosin improved weight gain and feed efficiency, increased body protein, and reduced body fat in both groups, but the magnitude of response was highest for the conventionally-weaned and perhaps more immunologically-challenged group. Roura and co-workers (1992) studied the relationship of the state of immune activation in broiler chickens to the growth-permitting ability of antibiotics. They also present data consistent with the postulate that feeding antibiotics may permit growth by preventing immunogenic stress and associated metabolic changes brought about by cytokines.

Bacteria Alter Gut Turnover And Maintenance Energy Requirements

The presence of normal gut bacteria contributes to a thicker gut wall, heavier intestinal weight, reduced absorptive capacity, and a more rapid mucosal cell replacement rate (Commission on Antimicrobial Feed Additives, 1997). The cause of these effects is unknown but may result from host responses to bacterial antigens or metabolic byproducts as discussed above. Based on data from germfree animals, it has been assumed that feeding antibiotics can reduce or prevent these negative effects. The most obvious difference between germfree and conventional animals is a thinner wall of the small intestine, with a reduction in connective tissue and lymphoid elements (reviewed by Coates, 1980). Microscopic evaluation of germfree intestine reveals a more regular and slender villus structure, with a thinner lamina propria. Further, the rate of renewal of epithelial cells is slower in germfree animals, which may have a beneficial effect on basal energy expenditure and energetic efficiency of nutrient utilization. These observations are consistent with the view of Reeds and coworkers (1993) that in rapidly growing young

animals, the gastrointestinal tract and the skeletal musculature draw from the same limited supply of nutrients and are, in effect, competitors for the deposition of nutrients.

Conclusions

It has been shown that antibiotics improve gut health and enhance productivity by the modification of intestinal microbial populations. Even though precise mechanisms underlying the beneficial effects of antibiotics remain unclear, these mechanisms help to understand and explain abnormalities of gut health in the absence of specific pathogens. An understanding of the inter-relationship of gut physiology, microbiology, and immunology to gut health will become increasingly important to critically evaluate the impact of the normal gut microbiota on animal growth. In that regard, it is particularly exciting that molecular techniques are now available that allow a better understanding of how the gut microbial profile is changing with various modifications to the external environment of the bird. These advances will allow the development of therapeutic treatments, novel technologies, management systems, and modified nutrition to optimize gut health and bird growth.

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Table B-1. Aromatic phenol production, bile salt hydrolase (BSH) and urease activities of intestinal bacteria 1.

Genera and species of Intestinal bacteria	Bile salt hydrolase activity	Urease activity	Aromatic phenol production
<i>Bacteroides vulgatus</i>	Kawamoto <i>et al.</i> , 1989		
<i>Bacteroides fragilis</i>	Stellwag and Hylemon, 1976		Bone <i>et al.</i> , 1976
<i>Bacteroides</i> sp.		Suzuki <i>et al.</i> , 1979	
<i>Bifidobacterium</i> sp.	Grill <i>et al.</i> , 1995	Crociani and Matteuzzi, 1982	Bone <i>et al.</i> , 1976
<i>Clostridium perfringens</i>	Gopal-Srivastava and Hylemon, 1988	Suzuki <i>et al.</i> , 1979	
<i>Enterococcus faecalis</i>			Bone <i>et al.</i> , 1976
<i>Escherichia coli</i>			Bone <i>et al.</i> , 1976
<i>Eubacterium</i> sp.		Suzuki <i>et al.</i> , 1979	
<i>Lactobacillus</i> sp.	Lundeen and Savage, 1990	Kakimoto <i>et al.</i> , 1990	Ward <i>et al.</i> , 1987; Yokoyama <i>et al.</i>
	Christiaens <i>et al.</i> , 1992	Suzuki <i>et al.</i> , 1979	1977; Yokoyama and Carlson, 1981
<i>Streptococcus</i> sp.		Varel <i>et al.</i> , 1987	

¹ Absence of reference does not indicate the lack of a given metabolic activity in that organism. In addition, it is likely that other, as yet unidentified, organisms possess these metabolic activities. Molecular techniques will provide a greater understanding of the extent of these metabolic activities in gut microbes.

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“Slide Presentation”

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Figure B-1.

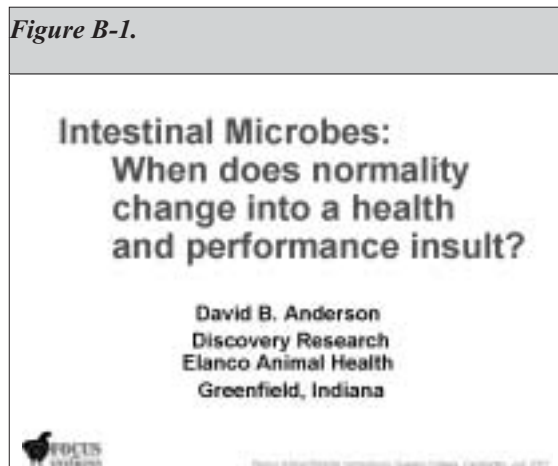


Figure B-2.

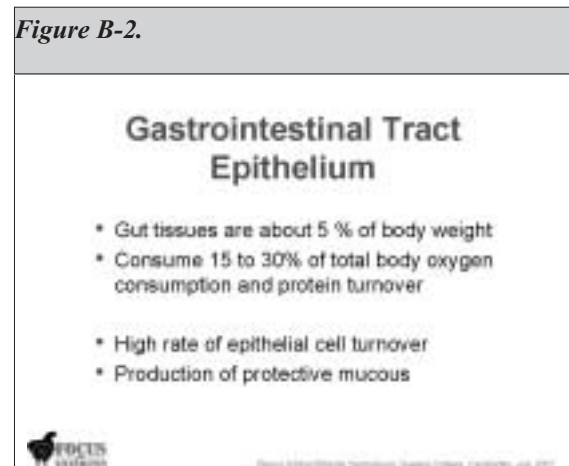


Figure B-3.

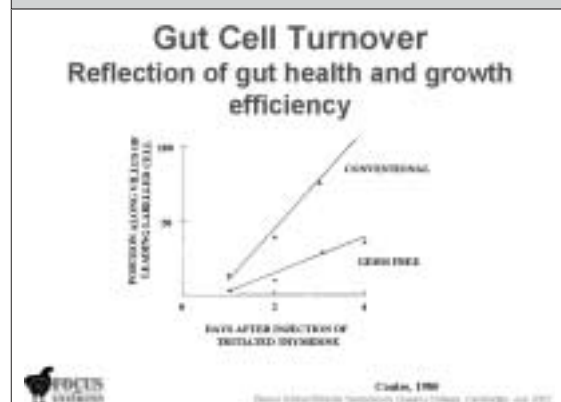


Figure B-4.

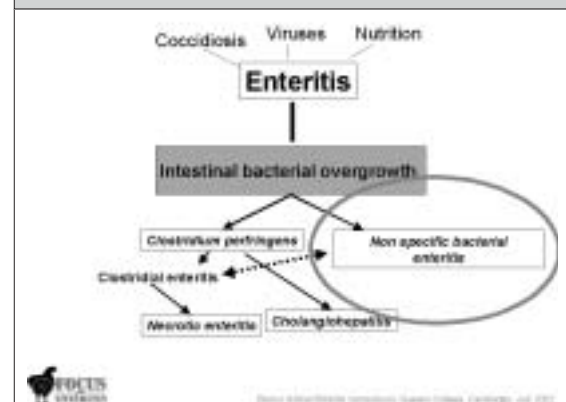


Figure B-5.

Non-specific bacterial enteritis:

What can be learned from studying how antibiotics improve gut health and enhance an animal's productivity?

Coates, 1963

Figure B-6.

- ### Mechanisms of Action by Growth-Promoting Antibiotics
- Increased feed intake and feed utilization
 - Improved protein metabolism
 - Modification gut microbial population
 - Inhibition of sub-clinical infections
 - Reduction microbial metabolites
 - Increase of available nutrients to host
 - Enhanced nutrient uptake by intestine
- Coates, 1963

Figure B-7.

- ### Evidence Suggesting Modification of Intestinal Microbial Populations
- Feeding antibiotics does not induce a growth-response in germfree animals (Coates, 1963)
 - Infecting germfree animals with intestinal bacteria from normal animals results in growth depression (Coates, 1963)
 - Growth response is enhanced by feeding antibiotics to conventional animals raised in unsanitary conditions (Hays, 1969; Roura, 1992)
- Coates, 1963

Figure B-8.

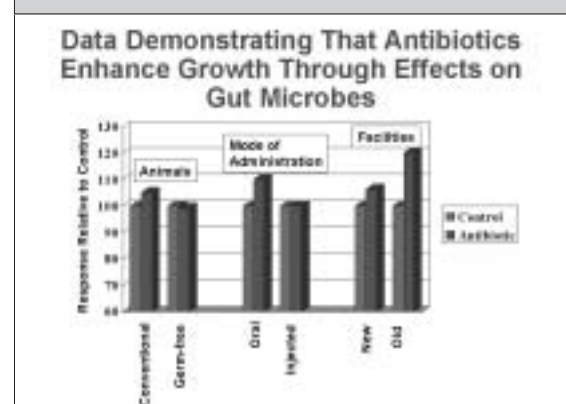


Figure B-9.

Evidence Suggesting Modification of Intestinal Microbial Populations cont.

- Germfree chicks given *Enterococcus faecium* show growth depression similar to those given a total gut inoculum (Fuller, 1979)
- Growth depression effects in germfree chicks with *Enterococcus faecium* was reversed by feeding penicillin (Fuller, 1983)



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Figure B-10.

Modification of Intestinal Microbial Populations

- Inhibition of sub-clinical infections
- Reduction growth-depressing microbial metabolites
- Reduction of microbial use of nutrients
- Enhanced nutrient uptake by intestine because of a thinner intestinal wall associated with animals fed growth-promoting antibiotics



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Figure B-11.

Reduction of Growth-depressing Metabolites

- Phenolic and aromatic compounds
 - p-cresol
 - indole
 - skatole
- Ammonia
- Bile acid transformation
 - deconjugation
 - dehydroxylation



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Figure B-12.

Reduction of Growth-depressing Metabolites

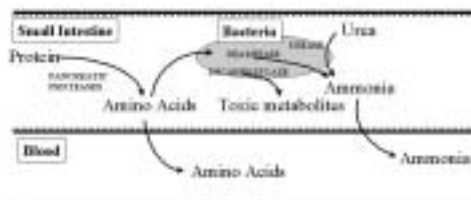
- Phenolic and aromatic compounds
 - p-cresol
 - indole
 - skatole
- Ammonia
- Bile acid transformation
 - deconjugation
 - dehydroxylation



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Figure B-13.

Protein Metabolism in the Small Intestine



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Figure B-14.

Reduction of Phenolic and Aromatic Compounds

- Bacterial degradation of amino acids i.e. tyrosine and tryptophan are excreted in feces and urine (Deichmann, 1995)
- Degradated AA products are not excreted in germ-free animals (Bakke and Midvedt, 1970)
- Negative correlation ($r=-0.73$) between urinary p-cresol concentrations and BW gain was observed in weaning pigs (Yokoyama, 1982)
- Excretion of these products is reversed in diets supplemented with chlortetracycline (Bernhart and Zilliken, 1958)



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Figure B-15.

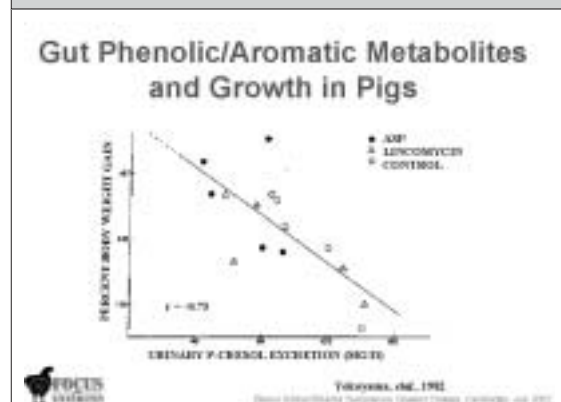


Figure B-16.

Reduction of Ammonia

- Toxic waste product of bacterial amino acids deamination and urea hydrolysis (Vissek, 1984)
- Ammonia concentrations in the colon of normal animals is several times the level necessary for cell damage (Vissek, 1973)
- Urea hydrolysis does not occur in germ-free animals (Levesson, 1958)
- Portal blood concentrations in germ-free is 25% of conventional animals (Wamer, 1959)
- Chicks immunized against urease to have decreased urease activity, lower ammonia concentrations and faster growth than non-immunized controls (Jiang, 1960)

Figure B-17.

Reduction of Growth-depressing Metabolites

- Phenolic and aromatic compounds
 - p-cresol
 - indole
 - skatole
- Ammonia
- Bile acid transformation
 - deconjugation
 - dehydroxylation

Figure B-18.

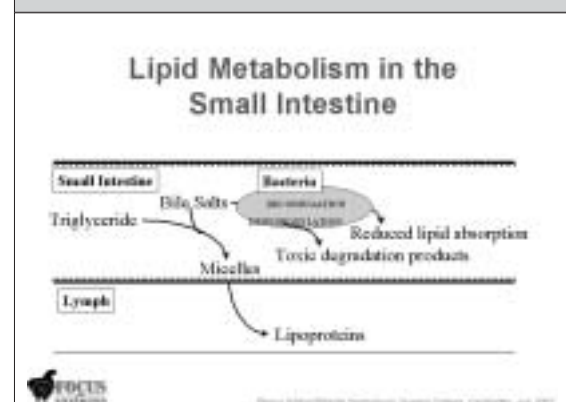


Figure B-19.

Reduced Bile Acid Transformation

- Microbial deconjugation and dehydroxylation of bile salts diminishes lipid absorption and reduces growth (Eysen, 1973)
- Bile acids do not undergo deconjugation in germ-free animals (Madsen, 1976)
- *Bacteroides*, *Bifidobacterium*, *Clostridium*, *Enterococcus* and *Lactobacillus* spp. all function in bile salt hydrolysis*

*Korolik, 1989; Ode, 1995; Erding, 1976; Opat-Somera, 1988; Tazaki, 1989

Figure B-20.

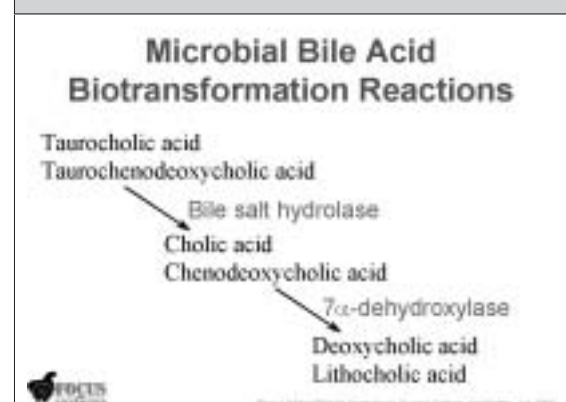


Figure B-21.

Influence of Diet¹ and Antibiotics² on Growth and Bile Salt Hydrolase Activity in Broilers

Diet	Wt. Gain	Bile salt hydrolase activity
Corn	152 g	1,270 ± 89
Rye	109 g	23,990 ± 1,518

Antibiotic	Wt. Gain	Bile salt hydrolase activity
Inactive	- 1.2 %	1,539
Active	+24.8 %	391

Feighner and Dushkevich, 1988(1), 1987(2)

Figure B-22.

Reduced Bile Acid Transformation, cont'd.

- Bile acid deconjugation and growth depression is reversed is reversed with antibiotic supplementation (Fuller, 1984)
- Antibiotic fed broilers demonstrated decreased cholytaurine hydrolase activity (bile salt enzyme) and increased growth rate (Feighner, 1987, 1988)
- Gram-positive facultative anaerobes which predominate the small intestine often produce all 3 toxins (Anderson, 1986)

Figure B-23.

Modification of Intestinal Microbial Populations

- Inhibition of sub-clinical infections
- Reduction growth-depressing microbial metabolites
- Reduction of microbial use of nutrients
- Enhanced nutrient uptake by intestine because of a thinner intestinal wall associated with animals fed growth-promoting antibiotics

Figure B-24.

Modification of Intestinal Microbial Populations

- Inhibition of sub-clinical infections
- Reduction growth-depressing microbial metabolites
- Reduction of microbial use of nutrients
- Enhanced nutrient uptake by intestine because of a thinner intestinal wall associated with animals fed growth-promoting antibiotics

Figure B-25.

Reduction of Competitive Microbial Populations Increasing the Availability of Nutrients to the Host

Figure B-26.

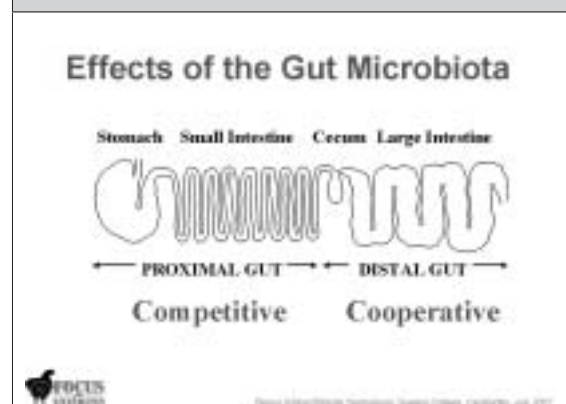


Figure B-27.

Reduction of Competitive Microbial Populations Increasing the Availability of Nutrients to the Host

- Culture based models have demonstrated that the microbial activity of the small intestine can compete with the host for energy and amino acids (Hedde and Lindsay, 1995)
- Lactic acid production from glucose reduces energy available to the host (Saunders, 1962)
- Lactic acid enhances peristalsis increasing the gut transit ingesta rate (Saunders, 1982)
- SI bacteria can result in a 6% net energy loss from glucose utilization (Vervaeke, 1976)



Figure B-28.

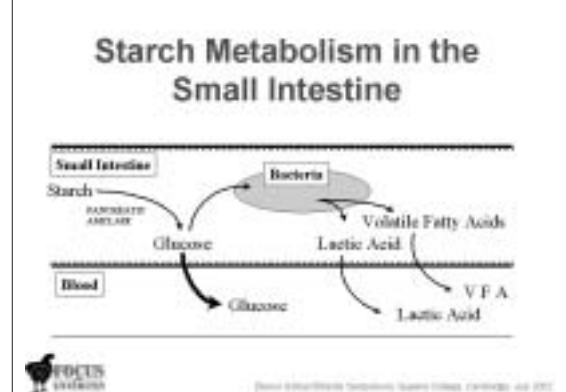


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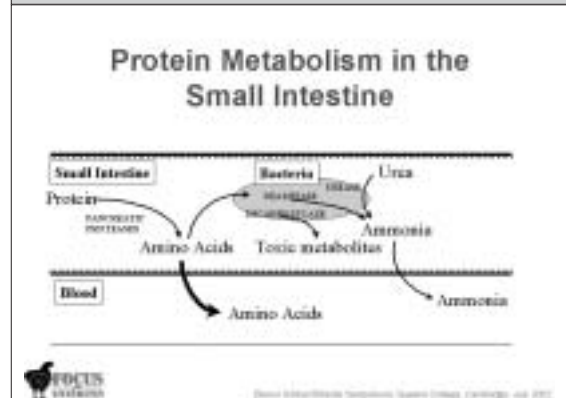


Figure B-30.

Microbial Effects on Bird Weight and Energy and Nitrogen Retention

Variable	Axenic	Lactobacillus	Mixed Culture
Body Weight (g)	359	347	313
Energy Retained (%)	38.7	31.9	34.0
Nitrogen Retained (%)	52.2	45.3	48.8

Kipfl and Clark, 1981




Figure B-31.

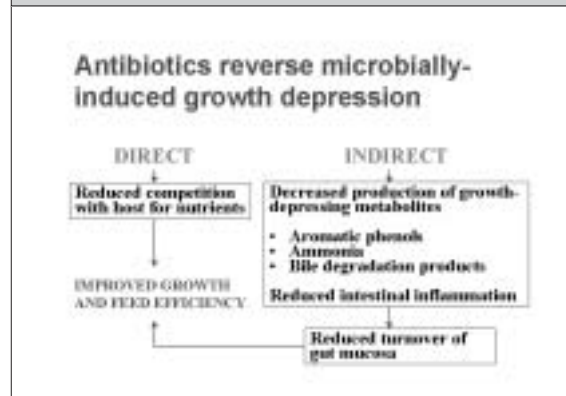


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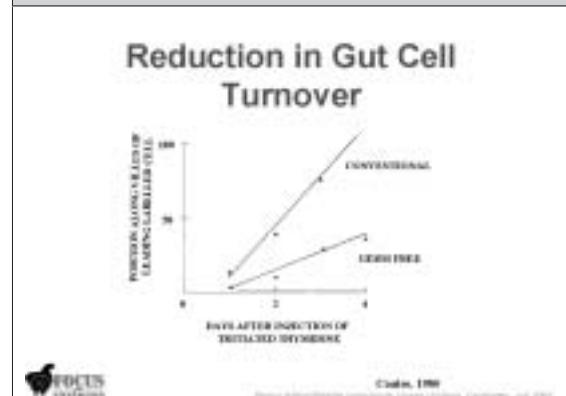


Figure B-33.

Fractional Protein Turnover				
Protein pool	Pool Size (kg)	FSR (Per day)	Synthesis (kg/day)	Distribution of synthesis (%)
Muscle tissue	3.7	0.1	0.37	35
Connective tissue	2.1	0.05	0.1	10
Liver	0.2	0.6	0.12	12
Plasma	0.15	1.5	0.22	21
GI tract	0.3	0.6	0.18	17
Other	0.85	0.05	0.05	5
Whole body	7.3	0.14	1.05	100

Figure B-34.

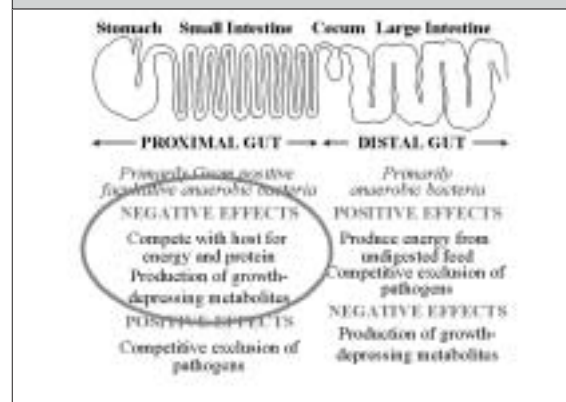



Figure B-35.

Summary

- It has been shown that antibiotics improve gut health and enhance productivity by the modification of intestinal microbial populations
- This mechanism of action helps to explain abnormalities of gut health in the absence of specific pathogens


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