Nutritional and Digestive Disorders of Poultry

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CONTEXT

Context Products Ltd
53 Mill Street, Packington
Leicestershire, LE65 1WN, United Kingdom

www.contextbookshop.co.uk

First published 2006
Reprinted 2013

British Library Cataloguing in Publication Data
Nutritional and Digestive Disorders of Poultry
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ISBN 978-1-899043-54-5

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THE DIGESTIVE TRACT

The digestive organs of chickens and turkeys comprise the beak, oral cavity, pharynx, anterior oesophagus, crop, posterior oesophagus, proventriculus, ventriculus, duodenum, ileum, jejunum, paired caecae, large intestine terminating at the cloaca in the coprodeum. Waterfowl do not have a crop but the oesophagus is capable of considerable distention. Accessory organs include the salivary glands, liver and gall bladder and the pancreas. The alimentary canal is provided with foci of gut-associated lymphoid tissue including caecal tonsils at the bifurcation of the caeca in addition to the bursa of Fabricius in immature birds.

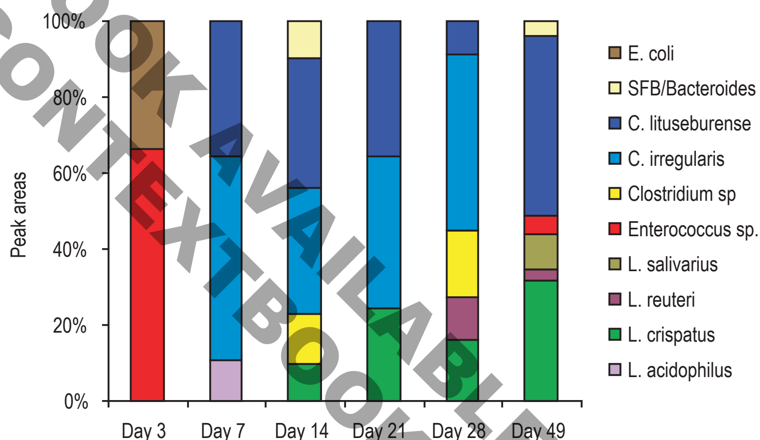
Oral cavity, crop and oesophagus

The oral cavity, tongue and pharynx are lined with squamous stratified epithelial tissue. The oesophagus, which is divided into anterior and posterior segments, extends from the pharynx to the stomach (proventriculus). Mucus membranes of the oesophagus are made up of stratified squamous epithelium interspersed with numerous mucous glands. The muscularis mucosa comprises longitudinal fibers and the muscularis is formed from an inner circular and an outer longitudinal layer of smooth muscle.

The crop, located to the right of the medial plane, is effectively a distention of the oesophagus, but mucous glands are more prominent in this structure.

coli, *Salmonella,* and *Pseudomonas* species possess fimbriae, which secrete proteinaceous adhesins which are used to attach the bacterium to the enterocytes. These structures have a specific affinity for the sugar residues of receptor sites on host cells, and are crucial to the successful colonization of the intestinal tract. Studies conducted on *E. coli* confirm a wide range of fimbria (also called pili) that vary in their numbers and characteristics according to the pathogenicity of the bacterial strain. The presence of specific enterocyte receptor molecules and the prevailing microenvironment of the intestinal tract alter gene expression in pathogens, stimulating the production of specific adhesins and toxins. Some strains of *E. coli* produce adhesins that can bind sialylated gangliosides, which predominate on the host cells of neonates. This property is known to be responsible for enteritis in piglets and may have a parallel in commercial avian species.

Figure 1.1 Evolution of bacterial profile in growing chickens



The intestinal microbial flora of adult chickens comprises over 40 species, predominantly located in the microenvironments of the jejunum and cecum. *Lactobacilli* predominate in the crop, duodenum, jejunum and ileum. *Streptococci*, *Clostridia* and other genera, including *Lactobacilli* comprise the major flora of the caecum. *Enterobacteriaceae* occur as both transitory and secondary components of the flora of the jejunum and ileum.

Characterization of bacterial species present in the gut has been subject to considerable revision in the past decade. Improvements in molecular analysis now allows previously non-culturable species to be characterized. This has led to an improvement in the understanding of the dynamics of bacterial populations in the intestinal tract of immature poultry.

Administration of acidifiers in drinking water and adding *Lactobacillus* probiotic cultures to feed promotes the development

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FEED CONSUMPTION AND DIGESTION

Feed intake

Appetite and repletion are under the control of the hypothalamus. Centers in the lateral hypothalamus are responsible for stimulating food intake. Satiety is mediated by centers in the ventromedial nuclei of the hypothalamus. Feed intake is stimulated by environmental factors including operation of mechanical feeding systems or recognition of feed. Low environmental temperature, absence of feed in the anterior digestive tract and possibly hypoglycemia stimulate intake of available feed.

Field observation suggests that broilers consume feed at 4-hour intervals. This has implications for dietary restriction programs and feed withdrawal prior to harvest of flocks required to prevent fecal contamination at the time of processing.

The beak is responsible for prehension of feed in either mash or pelleted form. Food particles are mixed with saliva and are transferred to the crop by gravity facilitated by extension of the neck. Food passes from the crop within 2 hours. Mash feed supplied to a bird with a empty crop will pass more rapidly to the proventriculus than whole grain in the form of wheat or corn which may remain in the crop for as long as 5 to 8 hours. Consistency, influenced by moisture content of ingesta, determines the passage rate, confirming the need for adequate availability of water to sustain feed intake at a level required for maintenance, in addition to egg production or growth. The crop undergoes peristaltic contractions transferring ingesta through the distal oesophagus to the proventriculus. The frequency of contractions and their duration increases proportionately with feed withdrawal. Peristaltic action in the crop is mediated by parasympathetic stimulation through the left and right vagal nerves.

The proventriculus undergoes regular peristaltic contractions, stimulated by the presence of ingesta. Contractions of the

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IMMUNITY

Poultry develop an active immune system following initial exposure to antigens such as pathogenic bacteria or toxins. The gut is invested with primary structures, secretions and mechanisms that act as a first line of defence against invading organisms. These include the secretion of acid in the proventriculus, grinding in the gizzard, enzyme secretions and protective layers of mucin. The development of protective mechanisms is dependent on the presence of food in the gastro-intestinal tract, and may take several days to activate, resulting in a period of susceptibility to pathogens in young chicks and poultry.

Immunosensitive structures within the gut act as primary sensors of antigens, most of which are ingested with feed or water or by the chicks interacting with their environment. Gut associated lymphatic tissue (GALT) is a component of the immune system responsible for initiation of the immune response against bacterial, viral and parasitic challenge in ingesta. The avian GALT includes the bursa of Fabricius, caecal tonsils (CT) and Meckel's diverticulum (MD) as well as Peyer's patches (PP), intraepithelial lymphocytes (IEL) and scattered immune cells located in the intestinal wall.

The immune system of poultry comprises the primary lymphoid organs, represented by the bursa of Fabricius and the thymus glands. The secondary lymphoid organs consist of the spleen, bone marrow and GALT, with lymphoid aggregators distributed through the subcutaneous region of the head (head-associated lymphoid tissue or HALT), in the upper respiratory tract (bronchial-associated lymphoid tissue or BALT) and the Harderian gland and conjunctiva (conjunctival-associated lymphoid tissue, or CALT).

The thymus and bursa are responsible for production of T- and B-cells respectively. These cells populate the secondary lymphoid organs involved in the initial defense against invading pathogens. As the chick ages and develops, these structures mature in response to exposure to antigens. This process is fundamental to immuno-competence in the adult bird.

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PRACTICAL NUTRITION OF COMMERCIAL POULTRY

Nutrition specifications

Modern broiler, breeder and egg production flocks require diets balanced in essential nutrients to achieve optimal reproductive efficiency, feed conversion, liveability, and immune response. Primary Breeders provide management guides incorporating nutrient specifications appropriate to the various ages and types of poultry. Nutritionists satisfy dietary requirements by blending available ingredients into diets on a least-cost basis. Generally, linear programming is used to develop formulations containing the most critical nutrients. These include:

- Metabolizable energy
- Crude protein
- Essential amino acids with specific concern to:
  - Methionine
  - Cystine
  - Lysine
  - Tryptophan
  - Threonine
- Fats and essential fatty acids (linoleic & linolenic acid)
- Macro minerals
  - Sodium
  - Calcium
  - Magnesium
  - Potassium
  - Chlorine as chloride
  - Phosphorus as phosphate
  - Sulphur as sulfate

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TECHNICAL FEED INGREDIENTS AND SUPPLEMENTS

Prophylactic antibiotics

Antibiotics stimulate growth and feed conversion efficiency and have been included in the diets of food animals for over 50 years. A review of available literature has confirmed that antibiotics have observable beneficial effects in 72% of approximately 12,000 trials conducted up to 1996.

During 1997, 15% of total antibiotic use in the EU, amounting to 1,600 million tons, was included in diets for monogastric animals. This compares with 33% of available compounds to treat food animals and 52% for human therapeutic applications.

The phenomenon of transferable antibiotic resistance became evident during the early 1960's. An epidemiologic association between administration of antibiotics to livestock and therapeutic failure of specific drugs in human populations led to scientific evaluation in the UK. In 1969 the Swann Commission recommended that any antibiotic with potential application in human medicine should not be routinely added to livestock diets. During the past decade, consumer and retail groups have convinced regulatory agencies, including the EU, to ban growth stimulating antibiotics (AGPs). This has resulted in research on alternative feed additives and a reappraisal of management practices to achieve optimal efficiency.

Mode of action of antibiotic growth promoters

Inclusion of sub-therapeutic levels of antibiotics in poultry diets influences growth rate by suppressing specific components of the intestinal flora. Microbial by-products, including ammonia and lactic

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NUTRIENT DEFICIENCIES

Causes of nutrient deficiencies

- Diets may be incorrectly formulated.
- Biological potency of vitamins or the availability of minerals may be sub-optimal.
- Deficiencies may occur due to removal of specified ingredients or supplements from rations.
- Destruction of fat-soluble nutrients occurs in feed due to oxidation.
- Loss of potency of heat-labile nutrients, such as vitamins, can occur if processing temperatures are too high or excessive shear forces are applied.
- Chemical antagonists in feed may increase the nutritional requirements of nutrients.
- The nutrient quality of ingredients may be depressed by excess moisture, mold contamination or inappropriate processing.

Under commercial conditions, multiple deficiencies can occur, and symptoms associated with suboptimal intake of a specific nutrient may not be clearly defined.

Pectoral musculature of a turkey showing experimentally-induced myodystrophy from a diet deficient in vitamin E, selenium and sulphur-containing amino acids

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FEED QUALITY AND DISEASE INTERACTIONS

Mycotoxicoses

Mycotoxicoses occur as a result of consumption of feed contaminated with toxic metabolites of fungi (Table 7.1). Over 200 mycotoxins have been described but aflatoxicosis, ochratoxicosis and fusarium toxicoses are the most commonly encountered problems in commercial poultry.

Mycotoxic fungi are responsible for significant financial loss to the poultry industry. Profitability is affected by decreased growth rate, feed conversion efficiency, livability and reproductive potential. Producers and consumers in tropical countries with high temperature and humidity are especially vulnerable to such toxicity and the immunosuppressive effects of mycotoxins, which exacerbate primary infections and interact with concurrent diseases.

Table 7.1 Principal toxic metabolites produced by common fungal contaminants of feed

Fungus	*Principal toxic metabolite*
Aspergillus flavus	Aflatoxins B, M, G; Kojic acid
A. parasiticus	Aflatoxins B, M
A. ochraceus	Ochratoxin A
A. fumigatus	Kojic acid, Aflatoxins
Fusarium tricinctum	T-2 toxin
F. sporotrichoides	Strachybotryotoxins
F. roseum	T-2 toxin
F. moniliforme	Zearalenone (F-2 toxin)
Gibberella zea	2-Deoxynivalenol (DON)
Penicillium viridicatum	Ochratoxin A
P. cyclopium	Ochratoxin A
Claviceps purpurea	Sclerotoxins

Mycotoxicoses in poultry

Mycotoxicoses affect poultry by lowering growth rate, feed conversion efficiency and reproductive potential (Table 7.2).

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ABNORMAL CONDITIONS

Gizzard erosion

Synonym: Black vomit

Occurrence and economic significance

Gizzard erosion is a sporadic problem in the broiler industries of countries that incorporate significant quantities of fishmeal in diets. Depending on the prevalence rate in a flock, and the severity of the condition, mortality may attain 10% through the first 14 days. Growth rate in survivors will be suppressed and gizzards condemned at processing reduce plant yield.

Etiology

Gizzard erosion is due to the presence of a specific biogenic amine, identified as 'gizzerosine' in 1983. The compound [2-amino-9-(4-imidazolyl)-7-azanonanoic acid] is formed by a reaction between the epsilon amino radical of lysine with the imidazolylethyl radical of histamine during processing of fishmeal. Gizzerosine is formed when fishmeal containing high levels of biogenic amines is heated to temperatures exceeding 130°C for three hours.

Clinical signs

Mild gizzerosine toxicity in broilers results in depressed growth rate and moderately elevated mortality. With severe toxicity, as observed in South Africa, Peru and Chile, broilers will regurgitate blood-stained ingesta termed 'black vomit.'

Pathology and pathogenesis

Gizzerosine has been shown to be over 100 times more active than histamine in stimulating secretion of hydrochloric acid in the proventriculus. The action of gizzerosine administered under experimental conditions can be reversed by pretreatment with

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INFECTIONS OF THE DIGESTIVE SYSTEM: STRATEGIES TO PREVENT DISEASE IN FLOCKS

Introduction

Prevention of disease in commercial poultry operations requires the application of a coordinated program of biosecurity, vaccination and hygiene. In order to develop control procedures it is important to understand the mechanisms by which pathogens are introduced into commercial poultry farms and how disease agents are disseminated among units.

Biological transmission occurs when the pathogen multiplies in the infected host, which then transmits the agent when placed in contact with susceptible flocks. Mechanical transmission involves transfer of a pathogen from an infected source or reservoir host to a susceptible flock by contaminated personnel, equipment, insect vectors, rodents, wild birds, or dust carried by wind.

High density production of broilers requires close attention to ventilation, operation of feeding systems and providing adquate feed and drinking space

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APPLIED IMMUNOLOGY AND THE CONTROL OF DISEASE

Introduction

Maintaining the health and productivity of flocks is dependent on an intact immune system. Exposure to a disease agent or administration of a vaccine should elicit both protective tissue and humoral antibody responses. Appropriate function of the immune system depends on adequate nutrition, available potable water, adequate ventilation and an ambient temperature within the thermoneutral range and an environment conducive to homeostasis. A comprehensive biosecurity program is required to protect young flocks against exposure to disease-causing agents before flocks can be immunized by vaccination. The integrity of the immune system can be seriously impaired by early exposure to viruses responsible for Marek's disease, infectious bursal disease and chicken anemia, in addition to ingesting feed contaminated with mycotoxins.

Vaccination

Vaccination involves the administration of a specific antigen to stimulate the immune system to produce homologous antibodies. The system should function against viral, bacterial and protozoal infections. Different types of vaccine are available to the poultry industry.

Live attenuated vaccines

These products incorporate a modified pathogen capable of stimulating immunity but produce no apparent clinical infection. Attenuation is achieved by growing the pathogen for a series of reproductive cycles ('passage') in a laboratory host system, which reduces pathogenicity for the target species. Attenuated vaccines are widely used to protect flocks against viral and bacterial diseases,

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DIGESTIVE DISEASES IN POULTRY

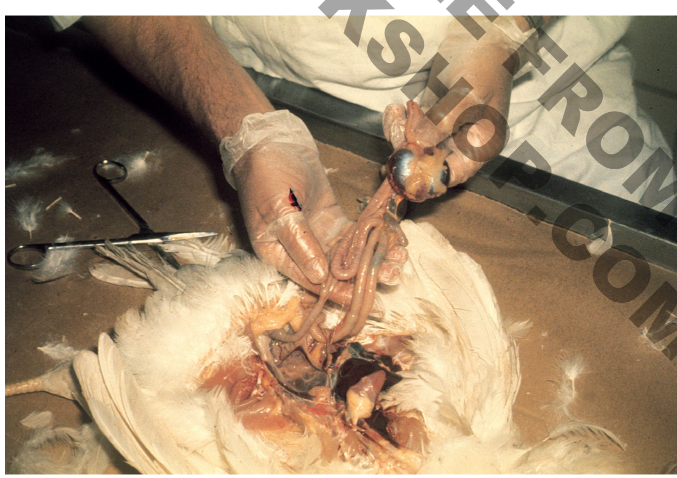
COCCIDIOSIS

Coccidiosis in broiler chickens

Occurrence and economic significance

Coccidiosis occurs worldwide, affecting all commercial species of poultry. It is estimated that producers spend in excess of $400 million annually to prevent and treat coccidiosis. In the absence of effective preventive measures, including anticoccidial feed additives or vaccination and supporting management procedures, immature flocks are susceptible to outbreaks which result in mortality and predispose to clostridial enterotoxemia. Mild clinical coccidiosis detracts from growth rate, feed conversion efficiency and pigmentation, which lower the quality of carcasses.

Diligent examination of the intestinal tract is required as a component of a thorough post-mortem examination to detect gross lesions



Clostridial enterotoxemia is an economically significant sequel of mild coccidiosis in broilers and replacement breeding stock, causing necrotic enteritis.

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DISEASES OF PUBLIC HEALTH SIGNIFICANCE

Food-borne diseases associated with poultry have assumed greater significance as consumption of poultry meat and eggs has increased. In addition, world trade in poultry products is subject to compliance with predetermined levels of safety as imposed by importing nations. The significant diseases of consumers associated with consumption of poultry products include:

- Paratyphoid *Salmonella* infection resulting from consumption of contaminated poultry meat.
- *Salmonella* Enteritidis infection acquired from undercooked eggs and egg products.
- Campylobacteriosis from undercooked poultry meat and products.
- Listeriosis from processed chicken and turkey products including sausages and salamis.

In addition to primary infection, prolonged or inappropriate administration of antibiotics to flocks results in the emergence of drug-resistant human pathogens.

The poultry industry worldwide is under increased scrutiny from regulatory officials and food industry associations to reduce the level of *Salmonella* spp. on poultry products.

Paratyphoid salmonellosis

Etiology

Salmonella serovars other than Group D may be associated with food-borne infection on meat and on egg shells. *Salmonella Enteritidis* is responsible for a specific egg-borne infection.

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ECONOMICS APPLIED TO POULTRY PRODUCTION[1]

Long-term profitability in a competitive market requires optimization of resources, control of costs and innovative marketing. Nutrition represents the largest proportion of costs, attaining 70% of pre-harvest broiler, turkey and egg production. Diseases of the gastro-intestinal tract are frequently significant detractors from least-cost production under commercial conditions. This is especially the case involving erosive diseases which reoccur due to inadequate preventive measures.

Long-term profitability requires that the incremental return in the form of enhanced egg production, hatchability, livability, growth rate and feed conversion efficiency should exceed expenditure on nutritional supplements and investment in biosecurity and disease control measures.

Analyzing costs of poultry production

Costs relating to live bird production can be classified into fixed and variable components. Fixed costs do not change as a result of an increase in the volume of production and include depreciation, interest on fixed capital, salaries, overhead, and lease payments. Variable costs are proportional to the volume of production such as the number of broilers per flock or cycles per year. Feed, labor, packaging material, fuel, vaccines and medication, purchase of day-old chicks and breeding stock, are examples in this category. Figure 13.1 shows the relationship between total cost, volume of production and profit. The concept of apportioning expenditure is important in projecting the effects of disease on total production cost. A decrease in broiler weight delivered to a plant due to increased mortality or

[1] This section includes text and diagrams prepared by the author and published in the 'Poultry Disease Manual' issued by the American Soybean Association. Reproduced by permission.

depressed growth rate will adversely affect production cost and efficiency. Processing plants, hatcheries, and feed mills operate at a break-even cost approximating 70% to 80% of design capacity due to their relatively high proportion of fixed costs.

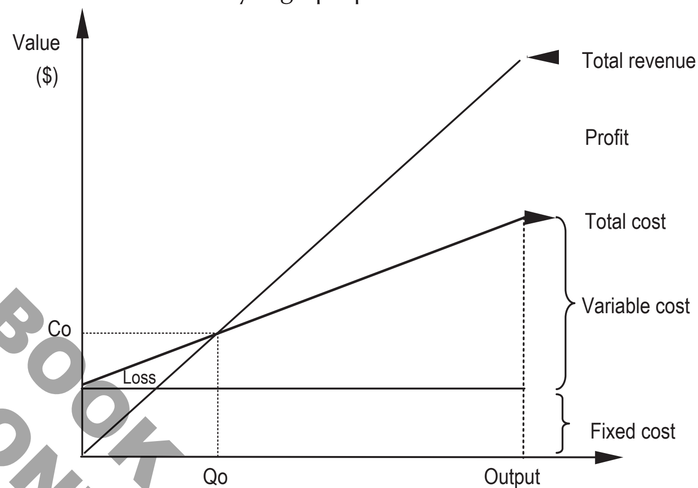

Figure 13.1 Conceptual relationship between cost and revenue

Fixed costs which are constant are not dependent on the number of flocks raised per year or the output of eggs. Fixed costs are illustrated by the line parallel to the horizontal (quantity) axis. Total costs are represented by the area which encompasses both fixed and variable costs. In this example, unit selling price is considered constant over volume of throughput and accordingly revenue is linear and proportional to the quantity produced. At the break-even point (quantity Qo) total revenue is numerically equal to total costs. At this level of production, fixed costs represent approximately half of the total cost. At a higher throughput, variable cost assumes a greater proportion of total cost. Offsetting fixed costs by increasing production level is the basis of efficiency through economy of scale, which benefits progressive integrations and cooperatives in mature industries. In the context of individual farms, there are limits to increasing production volume. Altering stocking density from 20 to 25 birds/m^2 increases throughput by 25%. Delaying slaughter of a broiler flock to attain a higher live mass (1.75 to 1.95 kg) may increase biomass by 11%. Reducing intercrop interval from 10 to 5 days may result in an 8% increase in broiler live mass over a year. Implementing these management changes introduce an increased risk of disease and the financial impact of infections will be intensified.

The severity of viral respiratory diseases such as bronchitis or laryngotracheitis is influenced by environmental and clinical stress. The effect of intercurrent low-grade conditions such as pasteurellosis, mycoplasmosis or coccidiosis may be exacerbated by increased biodensity. Secondary infections such as *E. coli* septicemia will

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