

## EVOLUTION OF DISEASES IN THE INTENSIVE LIVESTOCK INDUSTRIES. IS THE POULTRY INDUSTRY A VIABLE MODEL?

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### SUMMARY

The occurrence of disease is a direct manifestation of the failure of a production system to provide adequate compensation for increased physiological demands on the stock in that system.

The long term control of disease requires an holistic approach to the “ecopathology” of the biosystem. The development of this approach typically follows an evolutionary process including a number of phases that are conceptually distinct. This paper contends that the evolutionary process is sufficiently generic that empirical evidence from the poultry industry may be used as a model for other intensive industries.

### INTRODUCTION

The concepts advanced in this paper are derived from observations on the incidence and initiation of disease syndromes as they occur in various animal production systems. The chronological sequence of disease syndromes in intensively reared broiler poultry is presented as a model for other intensively reared animal species.

#### Central concepts

The central concepts of this model are:-

1. The emergence of new disease syndromes is simply a manifestation of a failure to adequately compensate for changes to the total animal production system.
2. To achieve long term control of disease syndromes in intensive animal systems an holistic approach to the entire biosystem is essential. This is conceptually distinct from traditional veterinary medicine where the primary focus is on the individual animal and the pathogen(s).

#### Investigative tools

The relevance of the symptoms of clinical disease observed in individual animals is directly related to the degree to which those individuals reflect the herd/flock/system status. Further to this point, clinical and pathological evidence of disease obtained from individual birds/animals should be reconciled with the data from the flock/herd history before making a diagnosis.

The flock/herd history is the primary diagnostic tool for the investigation of disease syndromes in intensive animal production systems. As the complexity of the syndrome increases there is a greater need to augment the primary information with additional data (microbiology, gross/histo/clinical pathology), however, the clinical history remains the basic focus.

## TRADITIONAL PRODUCTION SYSTEMS

### Environmental agents of change

Prior to domestication by man, changes to animal and their associated ecosystem occurred under a self regulating process of biological optimisation that Charles Darwin referred to as natural selection.

The pressure of natural selection ensured that following a structural disruption to the ecosystem, only those individuals capable of compensating for the change were able to thrive. This process was all encompassing covering diverse inter-relationships between the animal and its natural food sources, physical environment, internal and external parasites, environmental pathogens and microflora. For all these inter-relationships the rate of the occurrence of the **catalysing** changes was slow and resulted in selection/adaption at all nodes of the biological matrix.

### Modified environments and domestication

In the most basic form the “farming” of animals for food may be little more than shepherding, that is, protecting from predators and preventing escapes.

Even at this unsophisticated level, farmers were aware that the selection of breeding animals on the basis of particular desirable traits would result in progeny which accentuated these selected traits. Operating with small populations of animals, and in the absence of pharmacological aids, the selective breeding process slowly resulted in the evolution of many regional breeds of livestock. These animals, while purposefully selected for particular morphological traits, also benefited from the processes of natural selection with respect to adaption to the presence of biological stressors in their environment.

As was the case under natural selection, the rate and quantitative increment of change to the population means and production systems was low, thus providing the livestock with ample potential for natural adaption.

### Uncompensated change: early intensification

By the middle of the twentieth century the rising cost of land, **labour** and energy, combined with the emergence of the more aggressive business philosophies of a post-war industrialised world, the result was a generalised drive towards improving economic efficiencies in all fields including livestock production, in essence more output from the same (or fewer) resources.

Given that the generic cost equation for animal products consists of a numerator composed of the costs (livestock value, land cost and area, **labour**, feed, overheads and energy) and a denominator composed of the outputs (weight, number of animals) and the multipliers (dressing yield, batch rate), it is implicitly obvious that to reduce the cost of production one has to either:-

- i) decrease the costs, or
- ii) increase the outputs/multipliers.

Equally obvious is that the variables most amenable to rapid and independent manipulation are the land area and the number of animals, ergo: the birth of the intensive animal industry!

The chicken meat industry is demonstrably the most intensified of the three major intensive meat industries (chicken, pigs, feedlot cattle). This is illustrated by the comparative industry statistics in the following table. (Table 1).

## Intensive Animal Industry - Selected Statistics (1991-92)\*

	Chicken	Pigs	Feedlot
farmgate production (kilotonnes)	600	374	464
live value (\$M)	840	711	603
Concentration #	91%	19%	6%
Proportion of largest five enterprises			
Employing staff			
veterinarian	5/5	3/5	0/5
nutritionist	3/5	1/5	0/5
geneticist	3/5	1/5	0/5
integrated feed production	5/5	5/5	5/5
primary breeding	4/5	2/5	0/5
commercial breeding	5/5	5/5	0/5
abattoirs	5/5	2/5	3/5
wholesale marketing	5/5	5/5	5/5
value added processing	3/5	2/5	1/5
retailing	0/5	0/5	0/5

AN Internal market Research data (unpublished)

Percentage of total annual production of meat type controlled by the five largest enterprises

## ANIMAL HEALTH IN THE INTENSIVE INDUSTRIES

Intensive animal health programmes should all have the following core objectives:-

1. To achieve complete and continuous freedom from both subclinical and clinical disease. This will require control of disease syndromes involving both primary and secondary pathogens.
  - a) Primary pathogens must be controlled by either:-
    - i genetic selection (eg Lymphoid leucosis)
    - ii eradication (eg Pullorum disease)
    - iii vaccination (eg Avian encephalomyelitis)
  - b) The control of disease syndromes involving ~~opportunistic or secondary~~ pathogens must focus principally on the environmental factors. In these situations a narrow approach based on chasing pathogens rather than ameliorating the environmental stressors will only achieve disease suppression at best.
2. To always keep long and short term strategies in their correct perspective the use of antimicrobial therapy should be regarded as an interim measure to effect some control over a dysfunctional production system while the appropriate long term control strategies based on a) or b) are formulated and enacted. Examples of where this approach may be required could include outbreaks of Fowl Cholera (Pasteurella multocida) or Infections Coryza (Haemophilus paragallinarum). Swine dysentery (Serpulina hvodvsenteriae) would be a typical porcine example.

### The poultry industry

At the outset, intensive poultry rearing was approached in a similar fashion to traditional farming, that is, the rearing of 5,000 chickens was regarded in the same way as the rearing of one chicken multiplied 5,000 times. This approach is seriously flawed from a disease pathogenesis perspective. The placement of one chicken in a small barn does little to change the biological environment of the barn, however, the placement of 5,000 chickens in the same barn will effect considerable changes. For example, in the absence of modifications to the ventilation system there will be a considerable increase in the concentration of:

- airborne bacteria
- viruses,
- desquamated epithelial cells,
- dust from dried faeces,
- ammonia gas from the breakdown of uric acid,
- vaporised moisture.

Also oxygen tension could theoretically decrease, CO<sub>2</sub> may rise and barn temperature may become elevated.

Most of the aforementioned changes are manifestations of a substantial reduction in the **dilution effect** of the “natural” air environment of extensive agriculture.

In addition to these changes, close inter-bird contact increases the potential for the spread of fragile pathogens such as Mycoplasma gallisepticum, while the increased number of birds in close proximity to each other provide a substantially enhanced “pathogen catchment and excretion system” for agents such as Infectious Laryngotracheitis (ILT) virus. High density rearing also leads to spectacular increases in the availability of the infectious stages of the high fecundity internal parasites (particularly coccidia) and facilitates the transmission of ectoparasites.

Intuitively one can see the inherent danger in assuming intensive animal husbandry is the same as extensive husbandry with the excess space removed. The new environment of the intensive system clearly has the potential to produce uncompensated change and, therefore, the emergence of an array of new disease syndromes. History tells us that is precisely what has occurred and that typically it was the bacterial agents, both primary and opportunist pathogens that rose to early prominence.

#### THE PHASES OF DISEASE CONTROL IN INTENSIVELY REARED POULTRY

The following empirical model divides the disease control history of intensive poultry rearing into a sequence of phases. The accompanying analysis attempts to determine why the sequence flows the way it does by describing the driving forces of each phase. Throughout the analysis attention is repeatedly drawn to the use of antimicrobial therapy as an indicator of the conceptual approach to disease control during each phase. The use of **antimicrobials** to control disease reflects a “killing pathogens” approach which is generally more consistent with traditional single animal medicine with treatment directed at primary diseases, then it is for flock/herd medicine and diseases of complex aetiology. In addition, the use of **antimicrobials** in itself is not a permanent solution, in that it does not provide a long term correction for uncompensated changes to the production system.

Antimicrobial therapy may be contrasted with more long lived disease control methods including:-

- i) Environmental engineering
- ii) Eradication of primary pathogens
- iii) Vaccination (potentially)

The processes of each phase of intensification are sufficiently generic that the model may be used as a predictive tool across other species, principally intensively reared pigs and cattle. A diagrammatic representation of the model is presented in Figure 1. This figure depicts relative changes, in some key indicators, of the evolution and control of diseases in an intensive animal production system.

This model uses existing diseases/industry practices/control procedures as practical examples of each stage of the model. This is not done in an attempt to suggest sub-standard practices in any industries, merely to demonstrate that greater intensification and economies of scale tend to drive the evolution of control to greater efficiency. It is hoped that these processes will provide an insight into the future for the newer industries.

#### Phase 0: Sporadic disease in extensive systems

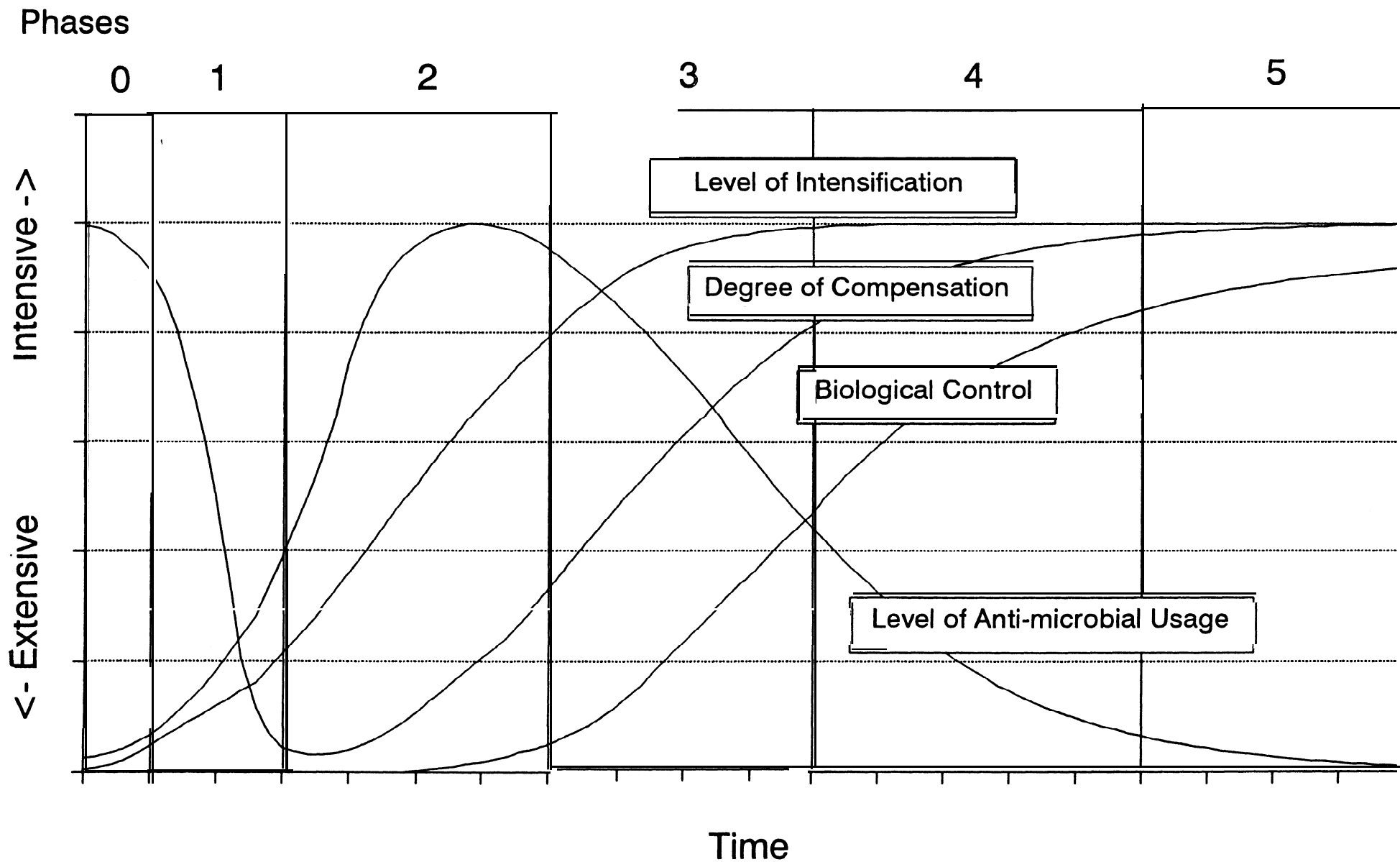
The cycle begins with the production system balanced and “disease” occurring as single animal infections with a primary pathogen as the case. Common examples are seen today in the extensive dairy industry and could include Staphylococcal mastitis and Bloat. While hygiene management may play a role in these conditions they are not classic communicable diseases and are largely controlled by individual animal therapy.

#### Phase 1: Primary pathogens aided by intensification

As greater numbers of animals are brought closer together the potential for larger and more rapidly spreading epizootics increases, as does the potential for the transmission of primary pathogens that are sensitive to inactivation in the environment.

A typical example is the poultry disease Infections Coryza caused by the bacterium Haemophilus paragallinarum. First isolated in 1932 by de Blicke (1933) the causal agent is quite delicate and suffers rapid inactivation outside the host, surviving only 4 hours in tap water at ambient temperature (Page 1962). The main reservoir of infection is the asymptomatic carrier occurring as a result of a previous outbreak. Clinical disease usually follows environmental stress (eg. increased humidity, ammonia and cold stress), the introduction of young susceptible stock or both of these factors.

The control of Infections Coryza (IC) as it occurs in “evolutionary Phase 1” is of interest as it highlights the impact of the short to medium term focus on control that is typically seen in newly intensified industries. Eradication of the disease is simply a matter of logistics, that is depopulation and effective quarantine. In Australia, all primary breeders are free of I.C., in addition the disease is not vertically transmitted. Depopulation and basic disinfection followed by restocking with “clean” stock will successfully remove the disease. Quarantine will keep a site clean. Despite these facts the modern egg laying industry continues to incur losses due to IC primarily because of the entrenched multi-age flock structure encouraged by the legislative supply regulation mechanisms and the carry over systems that still exist. Contrasting this situation is the broiler breeder industry in which the disease has been eradicated by following suitable epizootiological control procedures. Occasional outbreaks do occur, these will be discussed under Phase 3.



**Figure 1: The Phases of Disease Control in the Intensive Animal Industries**

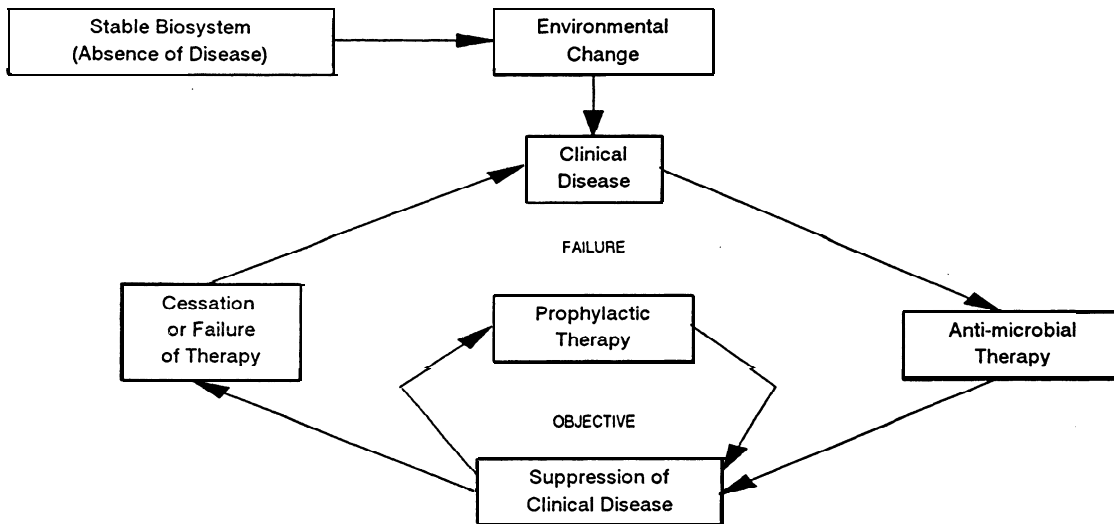


Figure 2: The Use of Prophylactic Therapy to Control Disease

At the same conference Janzen presented a paper (Van Donkersgoed, 1990) in which he cited the following reason for treatment failure with respect to Bovine Respiratory Disease -

1. 'Wrong diagnosis
2. Pathology too advanced
3. Insufficient duration of treatment
4. Inadequate dose rate
5. Drug insensitivity (occasional)

The combination of the above presentations suggests that, at least in part, the North American feedlot industry is operating in Phase 2.

Ironically, by the time a production system reaches Phase 2, many of the primary pathogens of Phase 1 may have been eradicated through good epidemiological practise. Further to this, the obvious preclusions to eradicating opportunist pathogens from the normal flora may be used to rationalise the use of prophylactic treatment.

### Phase 3: The environment as a factor in disease control

As the financial analyses of intensive animal production become more sophisticated, the full cost of disease (losses, treatment and prevention) become identified in a more formal fashion and invariably the conclusion is drawn that it is more economical to eradicate than live with a disease. The limiting factor is then available technology. Although there is no comparable reference for the poultry industry, Cutler and Gardner (1988) carried out a detailed review of the Australian Pig Industry.

This paper identifies the high cost of "living with" diseases such as Swine Dysentery (\$99/sow/year) and colibacillosis (\$75-\$85/sow/year). Costs expressed per sow actually relate to the entire piggery but are calculated per breeding sow to allow easy interpretation.

Financial pressures such as these have encouraged producers to re-assess their philosophies of disease control to include many environmental and husbandry factors as part of the process. Research on this subject is plentiful as exemplified by a recent local paper (Skirrow et al 1991) in which the authors assess the risk factors associated with post-

ning diarrhoea in pigs using a French “Ecopathology” model in which the only disease not listed as risk factor is “a recent episode of Transmissible Gastroenteritis (TGE)”, an exotic disease to Australia. All other risk factors relate to the physiological status of the piglet and environmental stressors.

Not surprisingly the sequelae to this approach tends to be a focus on correcting “ecopathological” imbalances, that is, attempting to re-stabilise the complete biosystem.

During Phase 3 it is not uncommon for some diseases caused by primary pathogens to re-occur. It is also possible that these diseases may be treated using antimicrobial therapy in Phase 1 (eg Fowl, Cholera, Infections Coryza). The essential difference between Phases 1 and 3 is the recognition of the role environmental stressors in the pathogenesis of disease.

In Phase 3 the rational use of medicants as temporary suppressants to control zoonotic spread prior to eradication is regarded as a compromise, rather than the primary focus of the control strategy as it was in earlier Phases. Figure 3 is an expanded version of Figure 2 and depicts the evolution of the control strategy.

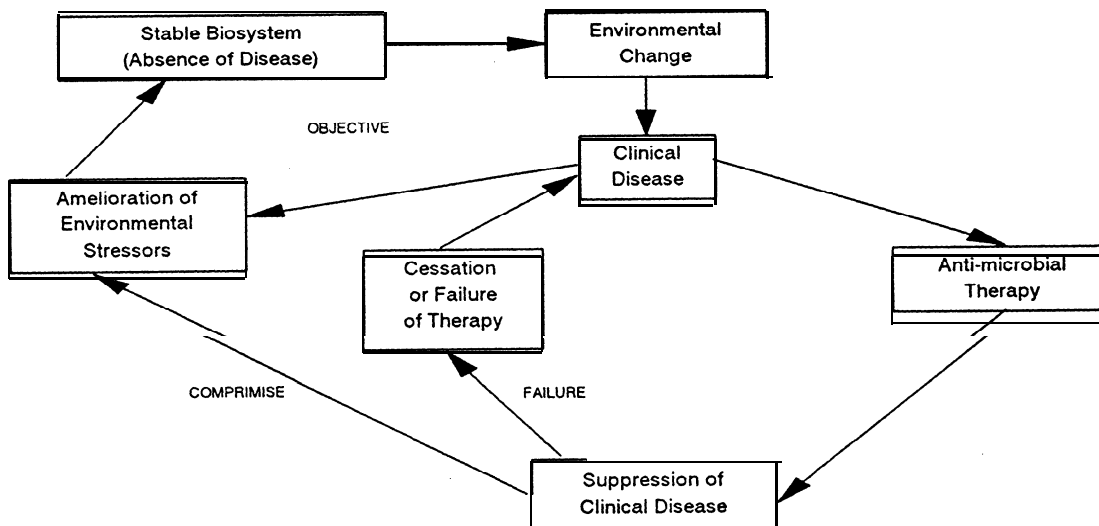


Figure 3: Correcting Environmental Instability to Control Disease

In the more advanced form (Figure 3) of the model the objective is to remain out of the original (Figure 2) loop, although partial entry for a single pass may occur as the result of an economic or biological compromise.

The “Modified Medicated Early Weaning and Off-Site Production” system described by Conner (1992) is a good example of a rational compromise strategy designed to gain control over internal and external parasites and as many as ten infectious disease agents:-

- *Mycoplasma hypneumoniae*
- *Actinobacillus pleuropneumoniae*
- *Haemophilus perasuis*
- *Streptococcus suis*
- *Treponema (Serpuline) hyodysentariae*
- *Pasteurella Multocida*
- *Bordetella bronchiseptica*
- *Leptospira spp*
- T.G.E. virus
- Pseudorabies



It is important to note that in Phase 3 the perpetual prophylaxis loop has been deleted. This was the primary focus in Phase 2.

#### Phase 4: Highly developed systems - complex syndromes

This stage is essentially a late - Phase 3, the major difference occurring not in the approach to control, but the type of breakdown.

Disease syndromes become increasingly more complex with immunosuppressive viruses playing a major role in de-stabilising the physiological balance of the system. Commonly, long term control strategies involving extremely detailed knowledge of the pathogenesis of the syndrome are required. Runting Stunting Syndrome (RSS) of poultry is an example, as possibly is Porcine Reproductive and Respiratory Syndrome (PRRS).

Because of the multifactorial nature of the disease agents involved and the pathophysiological processes that occur, the control of diseases typified by this phase requires the use of increasingly complex diagnostic aids. The procedures include the traditional histopathology and virology, progressing to immunological diagnostic aids such as the ELISA and Immunohistology through to gene probes and the polymerase chain reaction (PCR) multiplication of nuclear material.

In Phase 4 the use of advanced diagnostic tools to identify specific pathogens tends not to be driven by the desire for a narrowly focussed quick-fix treatment programme, but rather to elucidate a complex pathogenesis. This allows for the implementation of a multifactorial control programme commensurate with the complexity of the disease syndrome.

Ultimately, it is hoped that projects of this type will lead to a conquering of sophisticated pathogens such as the coccidia and complex disease entities involving immunosuppressive agents and a multitude of physical and biological stressors.

### CONCLUSIONS

This paper has presented a sequential view of the disease challenges that have arisen in the broiler poultry industry. The industry responses to these challenges have been discussed in an attempt to analyse the reasoning processes that have produced this evolutionary cycle. The author believes that both the environmental trigger factors leading to new disease syndromes and the industry responses are not species dependent and may, therefore, serve as a useful insight to other intensive livestock industries.

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