D A N D E R

January 1993 Issue Number 47

Journal of the Australian Veterinary Poultry Association

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In this issue there is a series of papers which were presented at the November AVPA seminar in Melbourne. We continue to examine how to preserve eggs 1904 style and because of the school holidays I have included a "40 second Omelette" recipe so that readers can make a meal, keep the family happy and still have time to mow the lawns and read Dander before collapsing into bed.

The other significant piece of news is that your editor has upgraded his computer system. This happened after my computer consultant took one look at my old computer equipment, shook her head, and after a quick examination decided that not even face lift surgery would help! So, please submit all articles on micro floppies...and I can now probably operate all standard Word Packages, Extra points are awarded for Word Perfect with Windows. The computer has been named Sergyv, after namesake Rachmaninoff. They both have the attributes of brilliance!

WET METHODS OF PRESERVING EGGS

"Lime seems the basis of the various pickles, to which salt is at times added. A strong solution of boracic acid and water also produced a successful sample, but I could not obtain the exact proportions. This is the recipe of a pickle which will preserve eggs for several months in good condition for cooking, namely:- one gallon of soft cold water, 1ld of quicklime, 6ozs to 8ozs salt, 1oz of cream of tartar. Mix the lime and water in an earthenware jar; when cold, add the salt and cream of tartar. A thin film forms on the surface of the liquid, and the mixture should occasionally be stirred up, even while the eggs are in it..."

Reference: Poultry for Profit. (1904), by Tho H Young

TODAYS RECIPE

40 Second Omelette

SERVES ONE PERSON

2 eggs
2 tablespoons water
1 teaspoon butter
1/2 to 1 cup of filling
20cm non stick pan (not to be eaten)

a) Lightly beat eggs and water together with a fork.
b) Pre-Heat pan until hot. Melt butter and pour in egg mixture.
c) Using a spatula, draw cooked egg mixture to the centre of the pan. Tilt pan, allowing uncooked egg to flow to the edges. Repeat until mixture no longer flows.
d) Fill one side of omelette, fold and turn out onto a plate. Serve immediately.
e) If phone rings do not answer.
f) If you must answer phone, and your omelette burns, Mix with dog food and feed to cat. Start again.
Filling suggestions:
Savoury: Grated cheese, chopped ham, onions, chopped capsicum, herbs, corn kernels.
Sweet filling: Fresh or canned friut. Dust folded top with icing sugar and serve with cream/ice cream

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A V P A Meeting Feb 11-12, 1993

Venue: AVA Auditorium,
134 Hampden Road
Artarmon NSW 2064

Dinner: Wednesday Feb 10, 1993, 7pm
Genghis Khan Imperial Mongolian BBQ
77 Archer Street
Chatswood NSW 2067
Banquet menu: $19.00
Wine $5/carafe and bottled wine
Soft Drinks $3/carafe

Accomodation: Artarmon Inn
472 pacific Highway
Artarmon NSW 2064

phone: 02-412-1644
fax: 02-412-2112
special AVA rate $85 per night
contact: Cathie O'Connell
(600m walk from AVA)
Allow $10/head/day for lunches
There is scope for an informal dinner on Thursday 11th Feb at a local Artarmon hotel within 200m walk.
Suggested registration fee: $50.
See registration form enclosed with this issue.

Getting there: with the new harbour tunnel and new Gore Hill Freeway, which has an Artarmon exit (Reserve Road), the AVA is now extremely convienient for Sydney Airport.

Conference topics:
Day one: Coccidiosis :
Covering industry perspectives, research perspectives and coccidiostat company perspectives.

Day two: Case Studies and Fowl Cholera

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The 14th Annual Conference of the AAV (Association Avian Veterinarians) will be held in Nashville TN, August 31 to September 4, 1993. For further information contact the AAV Conference Office, 1625 S. Birch Street, Ste 106, Denver, CO 80222.
Phone 303-756-8380
fax 303-759-8861
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42nd Western Poultry Disease Conference -
To be held at the Capitol Plaza Holiday Inn, Sacramento, CA on February 28th, March 1-2, 1993.
Contact Dr Richard Yamamoto, Dept of EPM , School of Veterinary Medicine, Davis CA 95616. ph: 916-752-7719
fax: 916-752-5845

Infectious Bursal Disease (from "Foreign Animal Disease Report" 20-2 (Summer 1992)

Recent reports from Indonesia (Claude Nelson, International Services, APHIS) indicate a severe problem in that country's industry caused by a new strain of Infectious Bursal Disease virus (IBDV), which has yet to be reported in the United States. According to Officials in Singapore, the strain of IBDV presently moving throughout Asia is similar to that which affected Europe. A similar strain has also been observed and isolated from poultry in Japan and Peoples Republic of China in 1990, from Thailand in early 1991 and from Indonesia in the latter part of 1991.

The first outbreak of IBD in the US was observed on farms in the neighbourhood of Gumboro, DE, during 1962. Gumboro disease became a synonym for the condition. The causative agent of IBD has been classified as a diplornavirus, a solvent resistant arbovirus.

IBD has been reported from most major poultry producing areas of the world. Incidence is greatest in chicks 3-6 weeks of age. In fully susceptible flocks, the disease appears suddenly with morbidity approaching 100 percent. The earliest outbreaks are reported in 11-day-old chicks and mortality averages 4-8 percent. Incubation takes less than 24 hours, clinical signs develop in 2-3 days and last 5-8 days.

The first cases of IBD in Indonesia were reported in 1976. The disease is estimated to have existed in approximately 5 percent of poultry flocks. In September 1991, high mortality rates in broiler poultry farms were reported in West Java with the disease peaking in late October or early November. During the peak of the outbreak, mortality rates reached 40-70 percent from broiler flocks having no prior experience with IBDV and receiving no vaccination. In broiler and layer flocks previously infected with IBDV and vaccinated against it, mortality rates were reported to be 5-25 percent following recent exposure. It appears that a new virulent strain of IBDV is causing severe losses in Indonesias poultry industry.
To control IBD, Indonesian poultry premises established a good sanitation program that involved cleaning, detergent washing, disinfection and a 2-3 week hiatus between stocking. This control regimen was undertaken because water, feed and droppings are known sources of the virus. Farms established a vaccination program with emphasis on early vaccination (7 days of age or sooner). Layers are immunised at 6-7 day intervals at least three times. The theory is that a vaccination program and the passage of time will allow the highly virulent strain to become less virulent.
The disease outbreak has received considerable media attention in Indonesia. Indonesian Veterinary Services has initiated an intensive epidemiologic effort to determine how the new virus strain entered, where it came from and how it spread throughout the industry.

(Reference: Hofstat MS et al, Diseases of Poultry, 7th edition Iowa State University Press, 647,648)

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The 1993 Poultry Science Symposium Conference, Sydney 9-10 February:

Invited Authors are covering
1) Broiler Breeder Nutrition and Management (JT Brake). The same author has submitted a paper on Incubation and Egg Storage Research.
2) Use of Simulation Models in estimating the Nutritional requirements of Broilers (Rob Gous).
Rob Gous will also be touring Australia as guest of the WPSA.
The range of topics submitted cover behaviour, Ascites, nutrition, beak sensory nerves, feed enzymes, shell ultrastructure and heat stress.

Flock serological profiling made

easy

In a joint project with Zolosian Software, **TropBio** the biotechnology company which is owned by James Cook University of North Queensland is now marketing a plate reader interface program to complement the **Trop-ELISA** range of flock profiling serological kits.

The serological assays which are designed to test up to 80 serum samples on each plate are available as self contained kits containing five coated plates along with the appropriate diluents, wash buffers, conjugate and substrate solutions.

The kits can be used to detect immune responses to Newcastle disease, infectious bursal disease, infectious laryngotracheitis and the most recent addition to the list is infectious bronchitis virus. They have been designed for determining vaccine efficacy and for monitoring disease outbreaks and specific pathogen free flock status.

The plate reader interface program which can be loaded onto any IBM compatible computer can be configured to interface with the common Titertek (Labsystems) automatic plate readers as well as the BioRad range of readers and data is transferred using a serial cable. The range of plate readers which can be accessed will be extended in early 1993. The installation of the program onto the hard disk is simply carried out by inserting the program disk and turning on the computer. Upgrades will also be automatically installed.

Using the program is also very simple. The user is presented with a menu which can be selected by typing a letter, moving the curser with the arrow key or a mouse and selecting the appropriate menu item. One of the options in the program is Fill in positions on the plate. This displays a screen with a plate outlined at the top and on the bottom section of the screen there is a series of cells. The details of each flock are entered into the cells and the position on the plate allocated to each flock is progressively displayed in the top section of the screen. Up to 10 flocks can be tested on each plate. However, the confidence which can be applied to the data increases with the sample size. **TropBio** recommends a minimum of 16 samples per flock. The data relating to the flocks and the position on the plate can be printed and then stored as a data file.

When the test is completed the operator uses the program to read the optical densities from the plate reader which is under the control of the computer. The plate contains seven standard samples which are loaded in duplicate. The optical densities for the 80 test samples are each compared with the mean values of the seven control samples and allocated to one of eight titre groups.

The information on each flock can be displayed on the screen or a report can be printed. The first page of the report contains details on the full plate including date and type of test, details of the flocks tested, optical densities for all 96 wells, the groups to which each of the 80 samples were allocated, a graph showing the standard curve and a histogram of the distribution of titre groups for all 80 test samples.

The results for each flock are presented on separate pages. These pages contain details which identify the samples and the test plate. There is a histogram which shows the percentage of the flock samples which are allocated to each of the eight titre groups. Summary data includes the mean and standard error of the titres as well as the percentages which were allocated to low, medium and high titre groups. The first page is retained by the laboratory for quality assurance and as a record of the full test. The subsequent pages showing the flock details are intended for use by the epidemiologist to convey recommendations to the flock manager. The data is presented in a simple logical form which will allow decisions to be made on vaccine efficacy or disease status of the flocks.

The program can be purchased from **TropBio**. However, it is intended that complementary copies of the program will be made available for regular clients. Plate readers are relatively expensive items. However, as medical laboratories are upgrading their equipment second hand machines are being made available. **TropBio** will assist with the purchase and installation of an automatic plate reader, computer and printer for those labs which intend expanding their serological monitoring activities.

Please direct enquiries to:

TropBio
PO James Cook University
Qld 4811
Phone 077 814 328
Fax 077 791 526

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Editorial Note: The TropBio article was printed in recognition of the author complimenting Melbourne weather while standing in the middle of a cold rain squall.
Some folk will say anything to con an editor!

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Salmonella pullorum:

In the Australian Salmonella Reference Laboratory Monthly report for September, 1992 (p2) the following extract appears:
"...2 isolates from quail were received from Victoria. Follow up investigations are being carried out by agricultural authorities. Our last recorded isolate from poultry in Australia was in 1984. "

Third World Congress - Foodborne Infections and Intoxications: Berlin, 16-19 June 1992:

The Proceedings are held at South Australia Health Commission (Dr S Cameron) and AQIS/DPIE Canberra. It is 2 volumes and 1411 pages. A copy of the 46 page index is available from Hugh Bray No 10 Day Avenue, Rostrevor, South Australia 5073.

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VICTORIAN POULTRY ADVISORY COMMITTEE (VPAC)

The VPAC is a body set up to advise the Minister of Food and Agriculture on:-

1) Major issues influencing the performance of the Victorian Poultry Industry
2) Problems and opportunities for the poultry industry in both domestic and export markets
3) Government policy and regulatory issues which will improve the competitiveness of the poultry industry
4) Issues of industry development, training, promotion and public understanding of the poultry industry
5) Such matters as may be referred to it by the Minister.

The committee has 10 members and has a life of 5 years, although this may be extended at the ministers discretion.
At the first meeting in October. Anthony Ainsworth was elected as chairman and the Bendigo Avian Influenza was updated. The newly elected Minister will attend the second meeting scheduled for December 1992.

Reference Sera might get a new home: but are they safe?.....

Poultry Viruses and sera currently held as reference at NBSL are moving to Canberra to be looked after by TGAL. The feeling at the recent AVPA meeting in Melbourne was that this important resource must be kept in tact. There was doubt that this was going to be so. Contact Paul Gilchrist if you feel AVPA needs to seek assurance from TGAL that the reference biologicals will be maintained.

Snippets from "Aerosols" Newsletter of the World Veterinary Association October, 1991, Number 4:

UK news:
The virulent form of Gumboro disease is still causing mortality in many parts of the country. The two vaccines permitted under MAFF animal test certificates are Salsbury's Bursine II and Intervets D78. These are both intermediate strains and their use is definitely helping to reduce mortality. Various programs have been tried and there is now some success with spray vaccination at day old using 750-1000ml per 1000 birds. It is to be hoped these vaccines will soon receive full product licence status.

Israel : "...Until now, we in Israel have only immunised our breeding stock against AE, but we must now also consider recommending the immunisation of commercial replacement flocks."

from David Cavanagh, Houghton:
Molecular Analysis of IBV epidemiology :
"Strains of the Massachusetts serotype have been isolated in many parts of the world over a 50 year period. Sequencing has revealed that many of these isolates are not vaccine strains but are genuine Massachusetts serotype field isolates. Each of these differs from the classical M41 strain by only 2-3% of their S1 amino acids. The combination of serological and sequencing analysis has shown that despite extensive use of Massachusetts serotype vaccines, strains of this serotype still persist and, on occasion, cause disease and economic loss.....Additional studies have provided further circumstantial evidence that joint infection of chickens with two strains of IBV has led, on occasion, to recombination....Although different genotypes of IBV can co-exist, it would appear that the composition of the IBV population in an area is not constant..."

Newcastle Disease in Wild Birds in western Canada: (from C Riddell, Western College of Veterinary Medicine, Saskatchewan)
"In August-September 1990 significant mortality occurred in double-crested cormorants, white pelicans and ring billed, California and herring gulls in western Canada. The mortality occurred at lakes where major nesting colonies of cormorants and pelicans are found...Total mortality in Saskatchewan alone was estimated to include 7000 Cormorants, 100 pelicans and 2000 gulls....microscopic lesions were found in the central nervous system...one isolate from a cormorant was mesogenic while the other four isolates were velogenic...There is little commercial poultry production in the areas where the outbreaks occurred and there has been no evidence of spread of infection to commercial poultry.."

"Hepatitis-Splenomegaly" Syndrome"..a new condition in commercial egg laying flocks in Canada...low prevalence...there has been a sudden increase in mortality (2% per month) and a drop in egg production....livers were swollen and friable and were mottled tan and red with multifocal miliary pale areas and haemorrhage. Spleens were enlarged two or three times. The abdomens were full of a red fluid with the consistency of water...no significant histo lesions were noted in the pancreas, intestines, lungs sciatic nerve or kidneys. No significant pathogens on routine bacteriology..."
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On a personal note:
I remember teetering on the top of a ladder with Rob Shapcott at the bottom handing me tin foil light reflectors made from plates bought at a local supermarket. The aim was increased light intensity in the brooding area. It worked. Rob had a knack of making things simple. I'll never forget. (GR)

DID YOU FOLKS KNOW ABOUT THIS.....????????????

NATIONAL REGISTRY DOMESTIC ANIMAL PATHOLOGY
(submitted by R Reece)

Rod Reece, once Victorian based, and once Houghton based has returned to Sydney as the registrar of the National Registry of Domestic Animal Pathology, and of the Comparative Animal Pathology Registry. These are unique national resources and are available to YOU. The registries are basically libraries of histological slides and projection slides of normal and pathological conditions affecting animals in Australia and elsewhere.

Material from interesting cases are regularly added to the collections. The registrar provides a free second opinion service on histopathological slides. In addition, training is given on a one-to-one basis using a multi-header microscope, and more formal courses are held throughout Australia. Both Registries contain abundant avian material, and they can be supplemented from Rod's private collection. One or two days intensive tuition can be organised.

A histopathology training course will be held at the gorgeously beautiful NSW location of Camden in March/early April and will include a 1 to 1.5 day session : "A Guided Tour of Avian Histopathology".

Please contact Rod if you are interested in attending. This course will be given in regional centres in all states during 1993.

Contact Rod Reece:

Mon/Tues at Tooronga Zoo:
Phone 02-969-2777 ext 249
Thurs/Fri EMAI
Phone 046-293-314/309/361

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Hugh Bray sent this. Hugh is a member of the Food Borne Disease Group SA.

"I bring to your notice a recent publication by the SA health Commission. It is titled 'Food Borne Infection and Intoxication Outbreaks - An Investigation procedure'.

This publication is available from the SA Health Commission, attention Dr Scott Cameron. Price $10.00. "

A quote from the Preface is as follows: "This publication aims to assist all health professionals involved in the detection, prevention and evaluation of food-borne disease."ISBN 0 7243 4039 4

OF-COURSE YOU KNEW

...that Paul Gilchrist has moved, and that the new contacts are on page one of this issue of Dander.

...that all AVPA members are urged to register for the WVPA conference in Sydney Aug 1993

N O W

...that registering for the WVPA conference now will assist with organisational cash flow

....that Paul Gilchrist is now the AVA representative on EXANDIS and the Standing Committee on Exotic Diseases.

...that the next AVPA scientific meeting is on 11 and 12 February in Sydney, and features a one day session on Coccidiosis. Details elsewhere in this issue.

...that the Editor of Dander wishes you all Good Luck for 1993, and hopes that all your chickens return to the roost and rooster as applicable.

...that Peter Young can be contacted by fax at 07-892-5374 about AVPA Scientific Sessions.

BURSAL DISEASE IN INDONESIA

Paul Gilchrist attended a seminar on the "Epidemiology and Control of Gumboro Disease" sponsored jointly by the Indonesian Directorate General of Livestock Services and Romindo, the Indonesian subsidiary of Rhone Merieux. This company is the manufacturer of a number of bursal disease vaccines including a live virus vaccine, described as of intermediate pathogenicity, for use on day old chicks which have maternal immunity.

The Director of Livestock Services (Dr Sridadi) and Director of Animal health (Dr Suhadi) presented opening addresses. Next Dr Phil Lukert of the University of Georgia and Dr Daniel Gaudry of Rhone Merieux spoke.

Bursal Disease in Indonesia

The disease is now a serious economic problem in Indonesia. The very virulent pathotype of serotype 1 is responsible.
The Indonesian Authorities consider the pathogenic form of bursal disease (Gumboro) to be a major threat to the commercial chicken industry and a potential threat to native chickens.
Gumboro disease has been held responsible for a reduction of 75% in the number of broiler chickens produced. This occurred as a direct result of mortality and as a voluntary reduction in chick inputs due to fear of the disease.

DR LUKERTS PAPER:
Dr Lukert presented a brief historical review of the bursal disease from its origin in the USA in 1962 until the present time when it is recognised in every country except New Zealand. The first cases were seen in 5-6 week old chickens and showed 20-30% mortality; mucoid diarrhoea; heamorrhages in the leg, thigh and breast muscles; swollen oedematous and haemorrhagic bursae and necrosis of bursal follicles followed by complete atrophy of the follicles.
Subsequent cases occurred in younger birds as well. In birds from 1 to 21 days of age, the disease itself is mild but is followed by permanent, almost total immunosuppression leading to severe complicating disease such as gangrenous dermatitis. In birds over 21 days of age the disease itself is severe and tends to be followed by air sac infection, vaccine failures and decreased resistance to other infections.
The persistence of the virus is such that eradication or hygiene control are only partially successful and vaccination is necessary. Breeder flocks are uniformly antibody positive and thus chicks are expected to have maternal antibody.
Experimental results have shown that vaccine virus administered at 1 day of age to maternal antibody positive chicks can be seen by immunoflourescence to grow in tissue at the periphery of the bursa, but not in the medulla. it is thought that the virus was present in some of the more mature lymphocytes where it seems to have been held until maternal antibody faded and enabled it to multiply and stimulate active immunity.

Vaccination programs were devised, in the USA, to reduce damage from the disease, but some loss of production efficiency and bursal damage occurred. The usual vaccination methods until the end of the 70's was based on a live virus vaccination at 1 day of age and a second dose at 14 days of age.

The "European concept" of vaccination differed from the US system. It was based on oil adjuvanted, killed vaccine administered to breeders in hope of a uniformly high titre of maternal antibody in chicks which would protect them against early infection. This could then be followed by a dose of live virus vaccine as maternal antibody faded.
This was not uniformly successful due to lack of uniformity in maternal antibody in chicks which resulted in spread of pathogenic virus before the vaccine virus was introduced. When a very virulent infectious bursal disease virus occurred in Europe this vaccination method was ineffective apparently because the more pathogenic virus could more easily break through maternal immunity. This lead to a search for a vaccine virus of intermediate pathogenicity which would break through maternal immunity without causing disease. Experimental evidence showed that ":hotter" strains can break through higher maternal levels than milder strains. For example, one mild strain broke through an antibody titre of 500 while a hot strain broke through titres up to 5000. High titres are present in younger chicks and thus hot strains can break through earlier.
The flock situation is one in which some chickens have very low levels of maternal antibody and should be exposed a little later as the level of antibody fades. This has led to a concept of frequent vaccination with an "intermediate" strain.

There are four principles applied in newer concepts of vaccination against very virulent IBD:

*Hotter strains break through maternal antibody

*Higher doses are more effective. (Minimum titre = 10^4)

*Use an abnormal vaccination time (ie one day of age)
* Increase the frequency of administration (three times for severe challenge and twice for less severe)

VARIANT STRAIN
The serologically variant strain (serotype 2) is recognised only in the Americas. Most of the disease caused in chickens by bursal disease virus is caused by serotype 1. Serotype 2 virus is mainly found in turkeys, ducks and geese.
In chickens both serotypes suppress humoral immunity when administered a 1 day of age, but serotype 2 does not suppress it at 21 days of age.
Both serotypes suppress call mediated immunity when administered at 1 day of age, but serotype 2 causes only a transient suppression at 21 days of age.
The standard strain kills more lymphocytes and produces a stronger and more rapid antibody response.

In answer to questions Dr Lukert advised:

*IBD in layers does not have a lasting effect on production and birds eventually recover their immunity. It does have a lasting effect on production in broilers.

*The earlier the infection the worse the immunosuppression, but even in severe cases immunity is recovered by 12 - 14 weeks of age. (Note probably because some immune cells have migrated from the bursa even before hatching.)

*Eradication is not considered an option for any country where the very virulent virus is established as in Indonesia. It was attempted in the UK but was short lived. (NOTE It could be an option in Australia if a very localised outbreak occurred but as IBD is not a notifiable disease, and not considered exotic, it seems an unlikely candidate for eradication).

DR GAUDRY'S PAPER
A review of the world situation was again offered with some details of the forms present in various countries, ranging from the mild, immunosuppressive viruses typical of Australia through the chronic form with up to 20% mortality, to the very virulent form with 30-60% mortality.

The very virulent form was probably first seen in West Africa, then in Belgium in 1987, UK in 1988 and more recently through many parts of Europe, the middle east and South east Asia. It has not been reported in the Americas.
The occurrence of the very virulent virus has exposed the limits of protection from maternal antibody, the heterogenecity of the immune status of a flock and the need for different vaccination programs for broilers and layers.
Research shows that the level of maternal antibody in chick varies from one hen to another as would be expected, bit it also shows that the variation between chicks from the one hen also varies enormously. This means that even if you could select breeders with high antibody levels you could not anticipate high levels in their chicks. Flock antibody profiles are of limited value.
Evidence shows that field virus multiplies in the bursa even in the presence of high levels of maternal antibody.
Selection of strains for "hotness" showed a range of ability to break through maternal antibody. Severe strains can break through maternal antibody. Severe strains can break through some chicks as 12 days of age and by 21 days cab break through 50% of a flock. Intermediate strains such as the S706 vaccine can break through at 25 days with 50% by 25 days). Another strain broke through at 25 days with 50% at 35 days). Mild strains break through even later.
NOTE: This evidence of a correlation between the characteristics of "pathogenicity" and "invasiveness" (Break through ability) does not necessarily mean that there are not two separate characteristics which might be distinguishes eventually, leading to discovery of a low pathogenicity strain which is highly invasive.

Dr Gaudry recommends broilers be vaccinated three times at 1, 11 and 21 days of age. This has good results with only 2-3% mortalities.

Pullets have for some years been vaccinated at 1, 14 and 28 days but there have been some sever failures. he now recommends for pullets:-
One day of age: Live virus (strain 706) by eyedrop, coarse spray or beak drop.
7-10 days of age: inactivated vaccine, subcutaneously and strain 706 in the drinking water.
Some hatcheries are administering bursal disease vaccine together with HVT vaccine by injection and it seems OK but he stressed that it is unproven.

In answer to questions he advised:-
*Vaccination is not 100% effective but that the benefit of vaccination outweighs the cost of the disease and of the vaccine.

*The virus is very stable in the premises and clean up is never fully effective. Chlorine is probably most effective.

*Full benefits from the vaccination program come several batches along when the vaccine virus swamps the field virus. Vaccination needs to be continued however of the field strain will reassert itself.

*layer strains seem to be more susceptible to "Disease" than broiler strains but not to the immunosuppressive effect.
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At the November AVPA a case of triple addition of Coccidiostat due to computer stuff up (error) was reported. The feed caused severe mortalities in broiler breeders at a few weeks of age. Len Hart sent to Dander work done in 1966 with Dow Chemicals where incorporation of a blue dye enabled Mills to quantitatively test for coccidiostat rather than testing for active ingredients.
When dealing with expensive birds is not an on the spot test better than waiting for the coccidiostat company...even though the service offered is excellent? I think that we are sometimes conned into lower cost rather than efficient prevention.

AVIAN INFLUENZA OUTBREAK

ERADICATION PLANS AND OUTCOMES

1. INTRODUCTION

An Australian Veterinary Emergency Plan (AUSVETPLAN) for the management of an exotic animal health emergency in Australia was approved by Australian Agricultural Council in February, 1991. AUSVETPLAN includes strategies to be followed for the eradication of important exotic diseases. The National Disease Strategy or Eradication Plans for Virulent Avian Influenza are contained in Appendix L of Volume 2 of AUSVETPLAN.

This paper addresses the major points in the eradication plan for Virulent Avian Influenza, indicates where we complied and where we differed with the Plan.

2. DIAGNOSTIC CRITERIA

The diagnosis of Virulent Avian Influenza was made on 31/7/92 on the basis of—

history
mortality rate
clinical signs
positive immunofluorescence (AAHL) - within 5 hours

and, subsequently confirmed by VIAS (Attwood) and AAHL on the basis of virus isolation and identification as H7N3.

3. PATHOGENICITY

In this instance there was sufficient data to justify the declaration of the presence of Virulent Avian Influenza and commencement of eradication procedures without awaiting the results of pathogenicity tests. This was not an option open to the USDA during the Pennsylvania AI outbreak in 1983/84.

The daily doubling of mortalities in the 22/23 week old birds in 1 broiler shed was sufficient to indicate the immediate adoption if the stamping out policy was the most appropriate action.

4. SEROLOGY

Evidence of previous AI infection in the 2 sheds on the IP with 50 week old birds and in 11 week old ducks on DCP1 was extremely useful to me. From the first day I made the assumption that the introduction of the virus was due to contact between wild waterfowl and domestic ducks and from the ducks to the broiler breeders either by wild birds or faecal contamination of footwear worn by a person or persons moving from the duck farm DCP1 to the IP.

Whilst there is only circumstantial evidence of this route of transfer it is important to adopt a theoretical concept of the disease outbreak origin in order to adopt sufficient and adequate responses.

This "concept" approach is not in the manual. It is my personal method of creating order out of chaos and I recommend this or a similar approach to all veterinarians involved in control and eradication of disease outbreaks, endemic or exotic. If there is a rational explanation of where the disease has come from then you can make predictions about where it is going and take the necessary measures to prevent its spread. However, it is important to be flexible. If new information destroys your "concept" - admit the mistake and adjust your theories. It may eventuate that your theories were based on invalid assumptions but at least decisions have been made and action taken. If the disease is eradicated successfully then the fact that your assumptions were based on incorrect information is of little relevance.

5. RESISTANCE AND IMMUNITY

5.1 Resistance

The knowledge that waterfowl and other species of wild birds are innately resistant to disease but not infection and of previous survey results from various locations in Australia was critical to the establishment of an "outbreak hypothesis".

5.2 Immunity

The persistence of serological positive titres in chickens, ducks and turkeys is variable as are the absolute levels of the titres obtained in the various species. Collection of the published information on this topic would be useful to the disease eradication decision makers.

6. EPIDEMIOLOGY

6.1 Incubation Period

There is a great deal of variation quoted in the literature. The manual states "from a few hours to two to three days" and, then points out that "The OIE definition of a maximum incubation period is 21 days".

6.2 Modes of Transmission

The information that "AI virus from waterfowl can remain viable in faeces and water for up to 32 days" was the basis for many of the decisions made subsequent to the outbreak, including length of time sheds remained empty and duration of monitoring in the surrounding area.

The experience we have had with AI in Victoria confirms that contact is important in the spread of AI and airborne spread, whilst it occurs, in not a major method of spread of this virus.

The possibility of secondary spread by people and fomites has been pointed out to industry, and this particular owner, but the incentive to implement basic hygiene at entry/exit points appears to be directly related to lapse of time from the most recent outbreak.

Nevertheless, AI virus is not a highly infectious agent and treating an outbreak as if the virus is FMD will unnecessarily increase the cost of control measures.

6.3 Factors Influencing Transmission

The information that AIV is likely to survive only several days in carcase at ambient temperatures and up to 23 days at refrigeration temperatures is important to the decision making regarding disposal or otherwise of processed chickens.

My decision in this case was that processed frozen chickens were a relatively low risk and the best method of disposal was to put them into the wholesale/retail marketing chain leading to consumption of the product by humans.

7. PRINCIPLES OF CONTROL

There is no question that infection of commercial poultry flocks with highly pathogenic AI virus can be recognised quickly.

AUSVETPLAN states—

"The basis of eradication of AI in Australia will be:-

- the rapid imposition of effective quarantine on all birds on which any degree of suspicion may fall,
- the certain eliminations of the pathogen where it is known to have been present, and
- prevention of movements of contaminated materials".

"Key factors in achieving these objectives will be rapid reporting and diagnosis together with swift imposition of effective movement controls".

In my limited experience the keys to successful eradication are—

- prompt request for assistance when something appears to be out of the ordinary;
- rapid response by the appropriate authority; and,
- elimination of infected birds and dangerous contacts in the shortest possible time.

7.1 Quarantine and Movement

Quarantine and movement control is important in preventing spread of the disease.

In this section in the eradication strategy the manual states—

"Particular attention needs to be paid to workers on poultry farms who keep backyard poultry at home. It is advisable to destroy such birds as soon as possible, even though they may be ornamental or pet birds".

In my opinion this advice should be re-assessed or, at least, clarified. Destruction of cage birds which are normally kept indoors at home can create intense opposition to the control measures overall. I question whether the degree of risk involved justifies the antagonism engendered by this action of destroying pet birds.

7.2 Quarantine of Infected Premises

IP1 was placed in quarantine on 31/7/92. Imposition of quarantine requires the signing of a quarantine notice and delivery to the owner or person in charge of the enterprise.

Implementation of quarantine is only assured when you place 24 hour security on the entrance to the property and ensure that this is the only entrance. This did not happen until late on Friday 31/7/92 and failure by one of the part owners to accept the diagnosis may have led to a breach of quarantine on 1/8/92 if the actual presence of security had been neglected.

DCP1 and 2 were also placed in quarantine on 1/8/92.

7.3 Slaughtering Out

The IP was slaughtered out over 1/2 August. The 2 dangerous contact properties with poultry/ducks/pigeons were slaughtered out by 4/8/92.

7.4 Control Area

A "Control Area" with a 25k radius around IP1 was implemented on 31/7/92, although the formal paperwork was not completed until 4/8/92.

Pigeon racing was banned for the duration of the outbreak as were pet bird sales in Bendigo and sale of birds at the Sunday markets held at the Bendigo Showground.

7.5 Eradication Area

An "Eradication Area" with a 5k radius around the infected property was imposed on 3/8/92, although not officially legal until 7/8/92.

In the case of both the Control Area and the Eradication Area, the Shire, Parish, City, Borough boundaries were used rather than specifying the distance from the IP. Most people know the local government area in which they live, but have little concept of distances such as 5k or 25k.

A pull out draft or pro-forma for the conditions imposed in the "Control" and "Eradication" Areas would be a useful addition to the disease control strategy. It is difficult to concentrate on developing this type of document when there are constant interruptions.

7.6 Hatchery DCP3

The decision in this instance was to destroy the contents of the hatchery and institute cleaning an disinfection procedures. This decision was made on the basis of the level of potential contamination at the entrance/exit points. I was pleased to learn that the USDA had made identical decisions in their eradication program in 83/84.

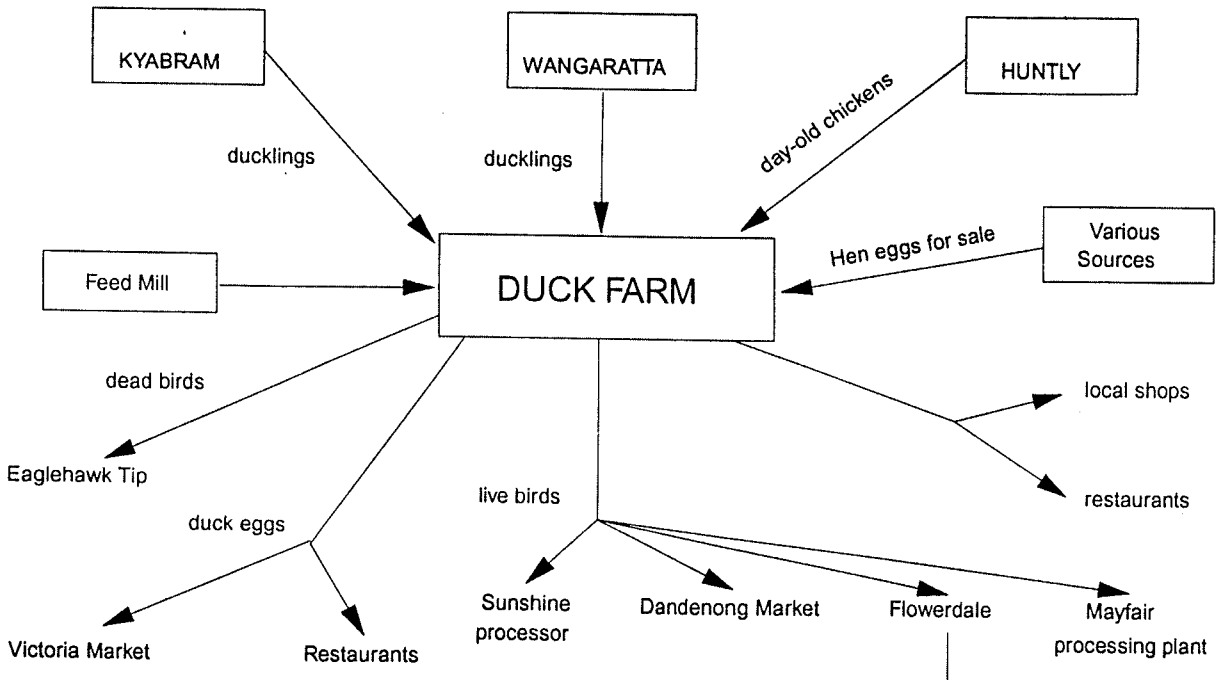
8 CONCLUSION

Successful eradication of exotic disease is dependent on having a set plan on which to base decision making, a good team (field and laboratory) to carry out the decisions and support from colleagues in other States and the Commonwealth, and from politicians.

Although this was a relatively small outbreak of a relatively benign and forgiving virus there were a total of 335 people involved in the eradication of this outbreak. AUSVETPLAN was the framework for the effective and efficient use of the personnel as well as the successful eradication.

Improvement of the current Plan requires only a few minor amendments and, perhaps, a user friendly presentation.

IT IS EASY TO MOVE EXOTIC VIRUSES AROUND.



This figure shows interactions between the Bendigo duck farm which had Avian Influenza and outside sources.
Most of Victoria was involved.
(source Mike Harrison, DARA, Vic)