D A N D E R

June 1992 * Issue Number 45

Journal of the Australian Veterinary Poultry Association

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Dander submissions on originals or 5 1/4 inch Wordperfect 5.1 formats please.

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In this issue a summary of the recent Sydney seminar on ascites will be presented. A list of keynote speakers at the August 1993 WVPA Sydney Convention will be published for the first time along with the usual juicy tid-bits. Thanks to all who submitted articles.

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Exotic Newcastle Disease in Water Birds

Deaths among double-crested cormorants (Phalacrocorax auritus) and white pelicans (Pelecanus erythrorhynchos) due to exotic viscerotropic velogenic Newcastle disease were diagnosed in northern Saskatchewan, Canada, in August and September 1990. Losses also were encountered in cormorant populations in the Provinces in Albert and Manitoba, but no virus was isolated. Mortality estimates included approximately 6,000 cormorants and 100 white pelicans. An undetermined number of ring-billed gulls (Larus delawarensis) also were involved.

Most of the cormorant losses were in full-sized young-of-the-year. A predominant clinical sign described in these birds was paralysis of one wing, with the wing held against the body. Less commonly, birds had paralysis of a leg or held their head in an unusual position. Because velogenic Newcastle disease is particularly dangerous to domestic poultry, anyone who encounters or handles sick cormorants, white pelicans, or gulls should avoid cross-contamination to chickens or turkeys.

The species of birds that were exposed to the virus on the breeding grounds in northern Saskatchewan move into the United States or farther south to winter. Wildlife authorities have been alerted concerning the disease outbreak. They have been encouraged to submit sick or dead birds promptly to a State or Federal diagnostic laboratory for appropriate testing. Foreign animal disease diagnosticians should be prepared to assist wildlife biologists with investigations of illness in these birds.

Myanmar reported **fowl plague** for July and August.

In Africa, 26 **Newcastle disease** (ND) outbreaks were reported from Algeria, Egypt, Kenya, Madagascar, Namibia and Zambia. (The virus was untyped and was assumed to be velogenic.) Cote-d'Ivoire noted disease activity but no outbreaks in July and August. In Europe, 4 outbreaks of ND were reported from Albania, 3 from the USSR, and 11 from Yugoslavia. An outbreak at Krasnodar, USSR during May resulted in the destruction of 20,200 birds. There were seven outbreaks of ND in Turkey during July-August. ND outbreaks in Haiti during February and March 1990 killed 67,000 of 89,000 affected birds. ND outbreaks were reported from Brazil (76) and Mexico (4). Asian outbreaks of ND were reported from Iran, Kuwait, Myanmar, Hong Kong and the Philippines. The disease was also reported from Myanmar, but no outbreaks were noted from there.

Three outbreaks of **velogenic viscerotropic Newcastle disease** (VVND) were reported in 1990: 1 from Botswana during July and August; 10 from peninsular Malaysia during January, March, April and June, killing 23,159 of 43,230 affected birds, and 4 from South Korea during June, 2 during July and 1 during August.

Hydropericardium Disease Update

Experiments carried out in Pakistan during the last 2 years have suggested that hydropericardium syndrome (HpS) in chickens is caused by the interaction of two agents: an avian adenovirus and a smaller, unidentified agent. The other agent, which is less than 25 nm in diameter, caused mortality without gross hemorrhages in 7- to 9-day embryonating chicken eggs when inoculated by yolk sac, chorioallantoic sac, or chorioallantoic membrane routes. The unidentified agent did not produce cytopathic effects in chicken embryo kidney cells, chicken embryo liver cells, or QT 35 cell line.

Adenovirus from HpS was successfully grown in chicken embryo kidney cells up to seven passages. It produced round cell cytopathic effects in chicken embryo kidney and chicken embryo liver cells but did not grow in QT 35 or Vero cell lines. This adenovirus also caused mortality in embryonating eggs of broiler breeders and specific pathogen-free white leghorn chickens. Dead embryos were grossly hemorrhagic.

A formalinized liver homogenate vaccine provided good protection in laboratory and field trials and is currently manufactured by a large number of private and public organisations. An improved vaccine for HpS was developed with higher infectious-agent contents, minimum cellular debris, and easy injectibility. In an experimental challenge-protection study, this vaccine provided good protection. Mortality in vaccinated birds was zero compared to 52.9 percent in the unvaccinated controls.

(from **Foreign Animal Disease Report, USDA Veterinary Services, Spring 1991**)

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LIFE MEMBERSHIP.....

The AVPA now has five life members: Len Hart, Beri Sonkovic, Hugh Bray, Dinah Fry-Smith and at the last meeting Rob Cumming.

It is interesting to note that four of them share a surprising characteristic. Len Hart told Paul at the meeting that he was not born in Australia but came here at four years fo age. That makes four of our five life members New Australians. "As far as I know, Hugh Bray is an Old Australian", Dr Gilchrist reports.

November AVPA Scientific Meeting

The meeting is scheduled for 11 and 12 November and will be held at Attwood. I have received some suggestions for the program already so make sure that you get your title in early to ensure a palce on the program.

Please note that Peter Young's address will change from 19th June to

Animal research Institute
Locked Bag No 4
Moorooka 4105
tel : 07-362-9400
fax : 07-892-5374

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...A group of vegetarians, vegans and animal rights activists have come together to form the Campaign against Omelettes. They have produced a hard hitting factsheet listing their grievances under pithy headlines like : "What's fat, boring and shapeless ? An omelette - the slime inside tells it all".

(from New Scientist, May 1992)

Next its Camapign against Custard...

AVPA SCIENTIFIC MEETING 6TH FEBRUARY 1992 SYDNEY UNIVERSITY

BROILER ASCITES

The following is a brief summary of the presentations at this seminar compiled from notes and tapes taken during the seminar. The interpretations reported here may reflect the recorder's own biases and understanding. Apologies are offered to the speakers if there are any misinterpretations, inaccuracies or omissions. Please write to the editor if any corrections are considered necessary.

SESSION 1 - ASCITES IN AUSTRALIA

A serviceman's view of ascites in broilers.

(a) Eric Bate, Baiada Poultry Pty Limited:

Ascites was first experienced in the Tamworth area in early 1980's. This involved the Hazlett bird and appeared to be feed related. The Sydney area was affected later. Over the next 4-5 years the level increased in both areas.

There are marked breed variations. Particular farms appear to experience more problems.

Ascites occurs mostly in the colder months with mortality rates of 2-4 birds per thousand per day over the last 3 weeks of life for about 4 months. It is tending to occur more frequently in summer this year.
Bird losses which begin after about 4 weeks of age continue at a similar rate till end of the batch - this is noticeably different from other diseases.

Factors thought to contribute:

Breed; stress (cold, large temperature variations, disease); faster growth rates (typical in winter); lighting (major influence, especially heavy restriction); ventilation and age (4-5 week old birds in good condition).

Approaches to control:

1. Lighting
2. Management awareness - brooding, variation, temperture control.

(b) Graeme Davey, Red Lea Poultry

Ascites has become much worse in the last 3 years. Red Lea has seen a 3% increase in total batch mortality due to this.

Red Lea have tried several control measures:

- minimising brooding temperature variations
- light control to slow growth rate
- feed: 3-4% lower energy level in grower & finisher phase - in hindsight, should have looked at starter.

Factors involved:

Genetic influence - more slowly growing birds are less affected.

Stress aggravates the condition - e.g catching for slaughter, walking through shed, weighing, etc.

Hatchery ventilation ??

Doesn't appear to be transmissible.

Doesn't respond to medication.

Good performing flocks have more problems.

Economic effects:

3% mortality is equivalent to $60,000 per year. This could be expanded to $90,000 if consideration given to lost sales. Also increases in plant downgrades occur from cases that survive to slaughter.

The Australian Poultry Limited experience with ascites.

Dr Rod Ryan, APL.

Believes that ascites is one manifestation of a broad disease syndrome encompassing late broiler mortality.

The Australian condition does not necessarily have similar aetiology to that of overseas reports and this could be misleading for Australia.

Breed susceptibility - within 7 pure lines grown on high energy feed, differences were seen in that one of these never experienced ascites, two rarely had the problem, three were occasionally troubled and one line had serious ascites. The condition was never seen when any line was fed on breeder rations.

The condition was first seen in pure lines in 1981 and nutrition has an influence.

KEY TO THE CONDITION: a row of lesions ("nodules") across the right atrio-ventricular (RAV) valve. It is believed that this lesion explains ascites.

The RAV lesions have been reproduced consistently by intramuscular injection of cultures of Salmonella sofia.

The history of salmonellae isolations at APL reveal that S.sofia was only first found in March 1981 and subsequently increased - the incidence of ascites and S.sofia followed each other. Late respiratory problems also correlated with S. sofia isolations. Every APL broiler flock is now S.sofia positive from tracheal isolation. It can also be isolated from lungs using enrichment culture methods.

Presence of the right strain of bird on the right feed is also necessary for the condition to be expressed.

In the overall late respiratory problem, there will be a percentage of birds with the RAV valve lesion. Studies in isolators using different ammonia levels (7-10ppm cf 2ppm) and reduced airflows have produced lesions in 9/12 and 1/12 "in contact" birds respectively.

The cause of these lesions??? - is it endocardosis, pulmonary hypertension or are they primary ?????

Under natural situations it is believed there is spread to in contacts from the percentage of affected birds in the flock.
Injection of S.sofia into birds will cause pericarditis. It is thought that S. sofia present in the trachea can develop into systemic infection. Increased respiratory effort by the bird is interpretted as an effort to lose body heat - lean lines do not lay down heat as fat so must increase respiration.

It is NOT believed that hypoxia is involved in the Australian ascites syndrome. APL birds actually show a decrease in PCV when affected which is contrary to overseas findings.

Field studies in broiler ascites.

Dr Garry Cross, University of Sydney.

John Matthiu, a masters student , conducted a mortality survey of four farms (total of 13 broiler sheds) during May - July 1990. Every bird which died on these farms was examined; a total of over 7700 birds. These flocks were of a "known" ascites susceptible breed.

Total mortality ranged from 2.19% to 4.36%.

Ascites mortality ranged from 0.77% to 1.90% (average 1.04%) of total birds placed. This was equivalent to about 30% of all mortalities. These levels were comparable to some overseas studies.

Ascites deaths were first seen between 2-14 days of age and increased with age. The time at which ascites mortality accounted for 50% of the mortalities in each shed varied between 4 and 6 weeks of age.

Over 1700 ascitic birds were necropsied between 21 and 42 days of age. Of these, 65% were male.

Although not disagreeing with Dr Ryan, of these 1700 ascitic birds, only 10-11 of them were seen to have A-V valve lesions compared to the higher incidence of these quoted for APL birds (note this study was not done in APL breeds).

There were no associations found between ascites incidence and mortalities due to other causes, nor with effects of farms or sheds within farms, nor with stocking density.

SESSION 2 - INTERNATIONAL SPEAKERS.

Pathophysiology of Ascites.

Dr Robert Wideman, Prof. of Avian Physiology, Pennsylvania State University.

Examination of ascites problems have been carried out in Mexico, Yemen, Columbia, Costa Rica, Brazil and USA.

Dilation of the right ventricle (RV) is the earliest gross lesion in problem flocks.

The earliest clinical sign is slight cyanosis. Examination of birds showing cyanosis reveals some RV dilation.

Early morning panting in cool conditions and cyanosis is indicative of overchilling during the night. This causes an increase in basal metabolic rate (BMR) and hence an increase in body temperature. As shed temperature rises in the morning, they feel hot. Can see up to 20% of the males with cyanosis in the mornings. These birds have been found to have arterial blood which is undersaturated with oxygen.

Suggestion - blood is being pumped too fast through the lung tp pick up sufficient oxygen, leading to systemic hypoxia due to an inappropriate perfusion of the lung.

Belief is that ascites should be renamed "Pulmonary Hypertension Syndrome" (PHS).

Features: dilated RV, thickened pulmonary artery, high RV:LV ratio and increased pulmonary arterial pressure.

Causes of PHS: really insufficient data but looks to be a "plumbing" problem.

The RV is a thin walled ventricle; blood is pumped at a low arterial pressure through a wide open pulmonary vasculature (not pre-contracted).

Circulatory principles * :

- flow rate is proportional to **Pressure** and **Resistance** of the vessel,
- volume of cardiac output (CO) of the left ventricle equals that of the right ventricle (i.e. same volume pumped by botrh ventricles).

Blood flow rates in chickens are low (e.g 7 week old male broiler = 200ml / kg bodyweight / minute).

Several factors will necessitate a higher CO:

1. **Fast growth:**

Broilers have been selected for body conformation: rapid increase in muscle mass and a slow increase in lung volume and capacity due to skeletal "restructuring" from this selection. Broilers are juvenile birds and have an immature pulmonary system.

Faster growth and a proportionately larger systemic circulation (due to increased muscle mass) requires a higher CO.

2 **Cold Temperatures:**

Feed is "burned" with oxygen to create heat. This entails a higher BMR and thus an increased blood flow. Also associated is a decreased peripheral resistance, thyroid hormone production and an increased oxygen demand (estimated at say 20%).

3. **High Altitude:**

Decreased oxygen partial pressure leeds to local hypoxia.

All the above result in a need for increased CO. As pulmonary arterial resistance is fixed, the increase in blood flow through the lungs can only be achieved by an increase in pulmonary artery pressure (see principles* above) -
the result is Pulmonary Hypertension (PH).

How does PH cause ascites?

PH develops leading to an increase in RV volume. This leads to dilation and thus to poor closure of the RAV valve. Regurgitation to the right atrium occurs and thence reflux to the vena cava. This increases pressure in the liver and causes transudation = ascites.

Early in the condition, the flow of blood through the lung is too fast for oxygenation, hence cyanosis.

Experimental reproduction of PHS:

Low temperature - gives the best model.
3000 m altitude
Low ventilation
Feed containing 1% NaCl
Feed containing thyroxin (T_3).

Control:

1. Anything which decreases growth rate.
2. Early feed restriction -skip-a-day tried in Mexico (days 5,7 &11).
3. Avoid cold temperatures (<18°C) in preference to increased ventilation.
4. Thiouracil (also STOPS growth).
5. Adequate ventilation (ammonia causes a drastic increase in pulmonary artery pressure !)
6. Keep dietary NaCl below 0.2%.
7. Avoid poor quality chicks (cull chicks will usually develop the runty type of ascites in the first 2-3 weeks).

Research:

Some birds are susceptible but others are not - why?
Perhaps selection could include measures of comb colour, PCV, ECG, etc - identify susceptibles and test their progeny.

Ascites in Broilers in Canada.

Dr Craig Riddell, Department of Veterinary Pathology, University of Saskatchewan, Canada

In Canada ascites accounts for about 0.21% of condemnations. There is a low incidence in all flocks. It was seen to be increasing by 1985.

If birds are held to 63 days, incidence increases to close to 2%. Information based on total mortality surveys.

Backyard poultry incidence often high (up to 20%) - due to temperature control, furazolidone overdose or unexplained reasons.

Many birds die from right heart failure without development of ascites - their viscera is often glistening and moist. A transverse section through the heart will show dilation of RV - this is the key feature for assessing ascites syndrome.

Pathogenesis:
Tumours of lymphatics - sporadic
Vascular injury - Vit E deficiency, dioxins.
Liver injury - crotalaria, amyloid
Hypoproteinaemia
Right Heart failure - the majority of cases.

Ascites can occurr without RH failure - needs close examination.

Causes of Right Heart Failure (RHF):

1.Pulmonary hypertension (increased blood flow or resistance)

2.Myocardial degeneration and inflammation.

(A) Pulomonary Hypertension:

Increased blood flow due to:

(i) Hypoxia - high altitude, poor conditions.

(ii) Increased oxygen demand -
Cold
Rapidgrowth
Thermogenic feeds

Barn conditions: (Julian & Wilson, 1991)

Looked at flocks with high and low ascites and air quality - measured $\%O_2$ and $\%CO_2$ - NO significant differences to explain high ascites barns.

Air quality may not be as improtant as was once thought.

Temperature: (Julian et al 1989) - consider this to be the main cause.

Rapid Growth Rate:

Birds with defective hearts were found to be heavier at 14 and 28 days.

Lighting programmes - continual light versus step-up programmes (e.g Classen's work) - step-up programmes are recommended - decreases leg problems, acute death syndrome and total mortality. Often there was not much ascites in these trials and no differences seen. Birds are much more active in these programmes, combs are redder.

This would seem an area to invest more research - cutting back light and/or growth. The compensatory period may put as much stress on the bird however.

Thermogenic Feeds:

Excess protein leads to increased BMR and thus increased body temperature and thus to an increased oxygen demand.

Poultry meal and tallow have been found to increase RV:total heart weight ratios - this mat have an influence.

Causes of PAH:

Resistance to blood flow in the lungs:

1. Lung pathology- Aspergillosis
Amiodarone (causes fibrosis)
-Ricketts
-Cartilaginous nodules ???
-Mineralisation

2. Anaemia

3.Hypovolaemia - NaCl

Mineralisation - excess dietary Ca (Bowes 1990) 3.22% Ca & 2.93% P lead to 50% morbidity with ascites and RHF and gout. Mineralisation of the lungs and kidneys were noted (also see Reece 1991 - ascites with high Ca ration).

(B) MYOCARDIAL DEGENERATION AND INFLAMMATION

Leucosis viruses - may produce myocarditis.
Furazolidone.
Rapeseed oil
Viral myocarditis
Chronic Circulatory Syndrome

Angara Disease in Pakistan - An Overview.

Mansur Ahmad.
(Currently at James Cook University, Townsville, Qld)

Synonyms for this disease: "Ascites syndrome", "Hydropericarium", "Myocarditis", "Hydropericarditis Syndrome", "Hepatonephritis".

Presently refered to as "Angara Disease".

First observed in October 1987 at Angara Goth, Karachi.
It appeared as high mortality in broilers between 3 to 5 weeks of age. The disease spread to other parts of Pakistan within 6 months.

A similar syndrome was produced by feeding broilers a high salt ration.

Attempts at isolation of viruses and bacteria failed (apart from E.coli in a few cases).

Possible adulteration of fish meal with salt was initially suspected.

Economic Effects:

Mortality usually exceeded 40%. resulted in the closure of 23% of the broiler farms.

Epidemiology:

Mostly affected commercial broilers. All genetic strains were affected. There were some unconfirmed reports in pigeons. Ducks, geese and indigenous birds were unaffected.

Appeared to affect healthy, fast growing birds most commonly. It was present in some sheds but not all.

There was no consistent correlation with Infectious Bronchitis, Infectious Bursal Disease, Mycoplasma, coccidiosis or high doses of coccidiostats.

Mostly affected birds aged 3 weeks but some cases occurred as early as the first week.

Disease Signs:

Varying degrees of depression, ruffled feather, anorexia and increased thirst. There could be sudden death without clinical symptoms.

There was gradual abdominal distension, dyspnoea, recumbency and eventual death.

Disease pattern suggested spread and transmission occurred.

Histological Findings:

Heart: massive interstitial oedema, intercellular haemorrhages, acute vesiculitis and myodegeneration.

Liver: multiple necrotic foci, mononuclear infiltration, interstitial inflammation. Basophilic/ acidophilic intranuclear inclusion bodies in hepatocytes.

Kidney: degenerative epithelial nephritis, intercellular haemorrhages, urate deposits in ureters.

Reproduction of the disease:

The disease was reproducible by injection of homogenates of liver, heart or bursa.

Inclusion Body Hepatitis (IBH) was diagnosed in April, 1988. The infective agent fulfilled Koch's postulates - an Avian Adenovirus. There is some evidence that CAA may have been involved (histopathology from Holland).

An inactivated vaccine prepared from infected liver homogenate produced encouraging protection.

SESSION 3 - EXPERIMENTAL STUDIES IN AUSTRALIA

Effects of lighting regime on ascites.

Peter Groves, Baiada Poultry Pty Limited.

A floorpen trial was conducted to evaluate differences in ascites incidence under either 23 hours continuous lighting throughout growout and a restricted lighting programme.

The restricted programme was: 23 hours light per day from days 1 to 3, then natural light only (about 12 hours) from days 4 to 28. From day 29, 6 hours of artificial light was added and at day 36 this was increased so that the birds received 23 hours of light per day.

Birds on continuous light grew faster up to day 21 than those on restriction, but thereafter the restricted birds caught and surpassed the growth rate of the full light group. feed conversion in resticted light birds was significantly better at all ages.

Ascites mortality was significantly greater in continuously lit birds from 21 days on. Overal ascites mortality was 6.13% for restricted birds and 15.2% for birds on continuous light.

Ascites mortality began to rise in the continuously lit birds from 21 days of age but this did not occur in the restrictively lit birds until after day 29.

Blood samples collected from a small sample of birds at 21 days in each group suggested that continuous lighting increased serum sodium and chloride and may have decreased serum inorganic phosphorus compared with birds on natural light periods. The significance or cause of this is unknown.

A field observation study was conducted over 280 broiler sheds during 1991 (Feb - Sept). There were three common lighting programmes in use in the operations studied during this time:

* Continuous light (≥ 20 hours per day throughout).
* Increasing (beginning on about 12 hours light in first week and then increasing by 2-3 hours per week until 23 hours maximum reached by 35 days).
* Restricted (12-18 hours light per day till 4-5 weeks, then increased to 23 hours by slaughter age)

Comparing ascites losses over these regimes for the entire period gave no advantage to the lower lighting programmes. However the distribution of these programmes was confounded by the month studied as most of the restricted programmes occurred in flocks placed only in August. August was also the month in which ascites losses were highest.

When August placements alone were considered, both the increasing and restricted regimes were significantly associated with lower ascites losses:

Lighting	Average ascites losses
Continuous	3.06%
Increasing	2.19%
Restricted	1.97%

The fraction of ascites losses which could be attributed to the lighting

programme in this study was 36%.

The lower light regimes were also associated with lower growth rate, especially between 21 and 35 days of age.

Conclusions:

Continuous lighting is associated with higher ascites mortalities and an earlier start to ascites losses.
Light restriction in the first few weeks decreases ascites mortality but ascites losses increase in the latter weeks as lighting levels also increase. Whether this is a direct effect is unknown.
Light restriction in the first weeks decreases early growth rate.
Varying the light regime can modify ascites risk but the effect is relative to the overall ascites risk as determined by other factors.

Experimental Studies on Ascites.

Garry Cross, University of Sydney.

A study was performed comparing organ:body weights in normal and ascitic birds sampled at 16, 18, 22, 25 29, 32, 36, 39 and 42 days of age.

The volume displacement of both lungs was measured, other organs were weighed and compared to overall liveweight.

Ascitic birds had smaller lung volume:liveweights than normals and this decreased with age for ascitic birds.

The GIT:liveweight ratio decreased with age for ascitic birds whereas in normals the relationship remained static.

The spleen:liveweight and liver:livweight ratios remained constant with age for ascitic birds but this increased in the normals.

Heart:liveweight for ascitic birds was always higher than normals.

Ascitic fluid:

There were no significant differences in most elctrolytes and proteins in ascitic fluid compared with blood plasma except that potassium and chloride in plasma exceeded that in ascitic fluid and calcium and albumin was greater in ascitic fluid.

Haematology:

Haemoglobin, PCV, WBC count, Heterophils, MCV and MHC for ascitic birds was higher than normal birds, while ESR and lymphocytes were higher in normals.

Serum Biochemistry:

The levels of total plasma protein, serum sodium, phosphorus, chloride, selenium, albumin and gamma-globulin for normal birds exceeded that of the ascitic birds, while serum potassium was higher in ascitic birds.

Effects of hypoxic incubation of eggs on ascites:

Selected eggs (on weight) were incubated with and without insulation tape applied around their equators until till day 18 of incubation. the chicks hatched from these eggs were kept under various temperature conditions during growout.

Birds from taped eggs which were brooded in relatively cold conditions had higher PCV's than those that were warm brooded.
The weights of birds from taped eggs exceeded that of untaped until 19 days.
Male birds which later developed ascites ahd significantly higher liveweights at 19 days than those that didn't.
Taping of eggs appeared to have no effects on ascites incidence.

(Summary prepared by P. Groves).
Thank you Peter : Terrific Job !
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KEYNOTE SPEAKERS FOR THE XTH WVPA CONGRESS SYDNEY 1993 :
(Kindly leaked to Dander by the scientific program organising committee as an exclusive....and free of charge !)

1) Environmental, husbandry, genetic and nutritional interactions in infectious diseases
Dr Egon Vielitz (Germany)

2) Recurrent and emerging diseases
Dr Stweart McNulty (Nthn Ireland)

3) Poultry production and public health
Dr Margaret MacKenzie (Australia)

4) Advances in diagnostic technology
Dr David Cavanagh (United Kingdom)

5) Education in avian medicine
Dr David Anderson (USA)

6) Diseases of the immune system
Dr Max Cooper (USA)

7) Metabolic doseases of poultry
Dr Craig Riddell (Canada)

8) Advances in the control of diseases by vaccines
Dr Mike Sheppard (Australia)

9) Non-specific enhancement of disease resistence
Dr Anne Kelso (Australia)

10) Diseases of village poultry
Dr Arnold Sinurat (Indonesia)

11) Diseases of cage and aviary birds
Dr Branson Ritchie (USA)

12) Welfare considerations in poultry production
Dr Paul Hemsworth (Australia)

13) Asia/Pacific Health Conference
Dr A Mustaffa-Babjee (Malaysia)

Preparations are well underway for the second mass mail out and the call for abstracts. If the response from the first mail out is anything to go by, a first class program will be assembled along with many good abstracts and posters.
Over the next few editions of Dander it is hoped the backgrounds of the key note speakers will be outlined.
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Preserving Eggs:

An English writer gives some valuable information concerning the preservation of eggs. HE says :-
"For several years I have watched the preserved egg class at Birmingham Show, and I have noticed that no one particular method has been uniformly successful in procuring winning samples. Sometimes a dozen eggs preserved in this way wins, sometimes in that, and from my observations of the class, I am unable to say which method has been the most successful. The object of the recipes for the various methods and preparations seems to be to prevent air permeating the pores of the shell, and this is attained, more or less successfully, in various ways...Here are a few methods as described in the Birmingham catalogues or derived from other sources...."

from **Poultry for Profit**, by Tho. H Young, Melbourne, 1904.
Next issue we will examine dry methods, wet methods and the water glass method of preserving eggs...

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EGGS ALL THE YEAR ROUND
LARGE EGGS in abundance, AND HEALTHY FOWLS by using the genuine
NORRIS' CONDITION SPICE
Positively a genuine article.
ALSO **NORRIS' ROUP PILLS**
sure cure 1/6° per box. From
Norris & Co, Chapel St, Prahran
^%^%^%^%^%^%^%^%^%^%^%^%^%^%^%^

Ninth International Symposium On Waterfowl, Pisa, Italy, 16-18 September, 1992.
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Asian Conference on Avian Coccidiosis, October 27-29, 1992
Biwako Hotel, Otsu, Japan
%^%^%^%^%^%^%^%^%^%^%^%^%^%^%^%

The 5th Conference of the Far East and South Pacific Federation of the WPSA, Seoul, Korea, Sept 23-25, 1993

Minutes of the AGM of the AVPA 6/2/92

Held at Holmes room, Sydney University after the scientific meeting with Faragher in the chair and Morrow taking minutes

1) Attendence
C. Morrow, T. Faragher, P. Gilchrist, MJ Lindsey, P. Young, JJ Swainston, L. Dowling, D. Marks, T. Ryan, P. Scott, Bruce Remington, Rami Cobb, Trevor Bagust, Tom Grimes, John Alexander, Barry Philps, Rod Jenner, R. B Cumming, Dinah Fry-Smith, Clive Jackson, Peter Groves.

Apologies.
I. Bell, H. Bray, K. Critchley, G. Firth, Beri Sinckovic, G & E Arzey, Websters.

3) Minutes of the last meeting
Motion: to accept the minutes from the last meeting that had been circulated in DANDER August 1991 (Morrow/Lindsey carried).

4.0 Business from the last meeting: none.

5) Reports
5.1 President's report (Trevor Faragher). Swan song
5.2 Treasurer's report (Paul Gilchrist). The report circulated with DANDER was withdrawn by the treasurer and a new report presented (attached).
Motion: that the new report be accepted (Gilchrist/Groves carried).
A debate ensured about the item Donation to EMAI $200
Motion: that the executive have a limit of $50 donation (except for organizing conferences) on expenditure to third parties (Lindsey/Cumming Lapsed motion). The treasurer agreed to provide more information about any donations made by the AVPA.
5.3 No reports from state corresponding secretaries
5.4 Report of the WPVA member (Roger Chubb).
Roger Chubb could not attend this meeting as he was on sabbatical in France.
5.5 Standing committee on Animal Welfare (Kim Critchley)
No report. Issues being considered include multiage housing.
5.6 Therapeutic subcommittee (Tom Grimes)
Report attached.
5.7 Standing committee on Exotic Diseases (Ian Bell)
Report attached
5.8 WVPA congress organising committee (Paul Gilchrist)
Report attached
4.9 Working party on constitutional ammendments (Ian Bell)
It was indicated that Ian Bell wished to stand down from this committee.
4.10 Working party on ILT eradication (G. Arzey)
No report recieved
4.11 Report of the editor of DANDER (Grant Richards)
The meeting expressed its appreciation of the way DANDER was being produced.

6.0 Elections

President	P. Gilchrist
Vice President	JT Faragher
President elect	P. Gilchrist
Secretary	C Morrow
Treasurer	P. Gilchrist
Scientific meeting Coordinator	P. Young
WVPA Bureau member	R. Chubb
AVA Policy councillor	C. Jackson
Alternate AVA Policy councillor	T. Grimes and T. Bagust

Convenor Standing Committee on Exotic disease C. Jackson
Convenor Standing Committee on Therapeutics T. Grimes (co-opting Ryan and Groves)

Committee on animal welfare	K Critchley
Honorary Auditor	S Taylor

7.0 General business
It was resolved
•that the AVPA provide assistance to the AVA in the preparation of an award to Dr Faragher.
•that the ILT working party be prompted for a report of the next business meeting detailing there meetings and progress
8.0 Next Meeting
The next Annual General Meeting will be held in February, 1993 to coincide with the Poultry Science Symposium.

The Ordinary General Meeting followed the AGM

1.0 Attendence and Apologies as for the AGM

2.0 Agenda
3.0 Minutes of the last meeting (Circulated with DANDER January 1992)
The minutes were accepted.

4.0 Business arising from the minutes: none

5.0 Reports

6.0 Correspondence
Dr Malcolm Smeal on the behalf of SCAHLs was organising a Avian Serology workshop with the aim of standardization of serological techniques within Australian and New Zealand government and industry laboratories. This workshop was held in February 1992.

7.0 General business
It was noted that all the life members of the AVPA were new Australians. The significance of this observation is still being contemplated by the executive.

8.0 Next meeting
The next scientific meeting will be held in Melbourne on November 11 and 12 (P Young to organize a program). An OGM will also be held on November 11 1992.

AUSTRALIAN VETERINARY POULTRY ASSOCIATION

STATEMENT OF ASSETS & LIABILITIES AS AT 31ST DECEMBER 1991

ASSETS:

Cash at bank:			
- NAB account		313.22	
- cash management a/c		6478.64	
- WVPA committee		232.40	
			7024.26
Fixed assets:			
recording equipment (fully depreciated)			
at cost	1342.50		
less depreciation	-1342.50		0
			7024.26

LIABILITIES:

Accumulated funds B/F:		11057.82	
Less: Deficit		-4033.56	7024.26
			7024.26

Bank Reconciliation:

NAB Statement 31/12/91	503.51
Less unpresented cheque 212	190.29
	313.22

All other accounts agree with Cash Book.

I have examined the books of account of the Australian Veterinary Poultry Association, for the period ended 31st December 1991, and have prepared statements to the best of my ability based on the information supplied.

.....
S.A. TAYLOR
(Accountant)

24/1/1991

AUSTRALIAN VETERINARY POULTRY ASSOCIATION

INCOME & EXPENDITURE STATEMENT FOR THE PERIOD ENDED 31ST DECEMBER 1991

INCOME:		
Subscriptions (ordinary)	3000.00	
Subscriptions (sustaining)	2080.00	
		5080.00
Interest received:		
- NAB cash management a/c		257.39
Conference income:		
- AVA contribution for speakers	1850.00	
- registration fees	2180.00	
		4030.00
Contribution from WVPA		3372.37
		12739.76
EXPENDITURE:		
Bank charges:		
- NAB account	59.82	
- cash management a/c	1.94	
- WVPA committee	18.87	
		80.63
Artwork & printing		7693.10
Postage & stationery		2931.99
Phones		51.25
Fares		60.70
Conference expenses:		
- Venue hire	2059.40	
- Speakers fees	1315.00	
- Speakers fares	636.00	
		4010.40
Accounting fees		150.00
Donation - Elizabeth Macarthur Institute		200.00
Subscriptions:		
- WVPA	664.85	
- Aust. fed'n animal welfare	135.40	
		800.25
AVA Conference expenses:		
- registration fees	150.00	
- fares	245.00	
		395.00
Sydney University - deposit on facilities		400.00
		16773.32
Deficit this year		-4033.56

AVPA STANDING COMMITTEE ON THERAPEUTIC AGENTS

REPORT TO AVPA AGM, 6/2/92

A. Purpose. Working Party was established by the Executive on 29/1/91 because "a number of important and controversial issues relating to the use of therapeutic agents in poultry had arisen". At the 1992 AGM, the WP was upgraded to a Standing Committee.

B. Terms of Reference. To handle any matters relating to the use of therapeutic agents which are referred to the AVPA by the AVA, Governments or members. Recommendations, policy statements and responses to be ratified by the Executive prior to release.

C. Membership. T. Grimes (Convenor), P. Groves, C. Jackson, B. Johnston, P. MacQueen, R. Cobb.

D. Conduct. WP has operated by meeting on 28/2/91, 2/5/91 and 26/9/91; and by participation in meetings of the NSW Poisons Advisory Committee on 6/2/91 (TG); Nitrofuran Task Force on 14/12/90, 22/2/91 and 1/5/91 (TG); AVA Therapeutics Committee on 5/4/91 (RC) and 12/6/91 (TG); NSW Stock Medicines Consultative Committee on 9/4/91 (PG) 13/8/91 (TG and PG) and 12/11/91 (TG and BJ); Veterinary Chemicals Advisory Committee (VCAC) Melbourne on 25/3/91 (TG); and the Sulphonamide Task Force on 5/2/91 and 16/7/91 (PG).

E. Reporting. Written and verbal reports have been given at AVPA meetings and circulated in Dander.

F. Business. Major topics have included input into proposed banning of nitrofurans, dimetridazole, sulphonamides and gentian violet for use in food-producing animals and veterinary servicing of the poultry industry. Current input is occurring into retention of sulphaquinoxaline and sulphadiazine for poultry, veterinary servicing of the layer industry, updating of the Code of Practice for Use of Antibiotics and Other Drugs in the Poultry Industry and NSW Branch AVA initiatives into increasing the level of residue testing of poultry meat to that of red meat.

Tom Grimes

TOM GRIMES
CONVENOR

Australian Veterinary Poultry Association

STANDING COMMITTEE ON EXOTIC DISEASES
Report to Annual General Meeting, 6 February 1992

The SCED addressed several important issues during the past year.

The major issue concerned the proposal to import fresh frozen and cooked chicken meats from the USA, Denmark, Thailand and New Zealand. The SCED prepared a detailed response which was submitted to AQIS through the AVPA and AVA Executives. We expressed strong concerns about the possible introduction of Newcastle disease, avian influenza, turkey rhinotracheitis and highly virulent Marek's disease and infectious bursal disease viruses, and *Salmonella Enteritidis*. We emphasised the risk posed by post-cook recontamination, and argued that imported product should comply with Australian standards for chemical residues, food additives and labelling. We recommended conditions under which importation could be allowed to proceed, and on these grounds opposed the importation of product from Thailand and the USA, and expressed reservations about Denmark.

The SCED also examined a proposal to close the Torrens Island quarantine station and relocate the hatching egg facility elsewhere. We contributed to a review of the importation protocols for hatching eggs and live birds. We reconfirmed the AVPA's policy of qualified support for the importation of Newcastle disease viruses into AAHL. We looked at proposed changes to procedures for the importation of biologicals, and at a proposal to import ostriches from Africa. Members also reviewed a video produced by AAHL on Newcastle disease and avian influenza.

I wish to thank the following members for their contributions to the SCED: Tom Grimes, Mark Lindsey, Paul Gilchrist, Clive Jackson, Chris Morrow, Kim Critchley, Keith Trinca, Ross Perry, Peter Groves, George Arzey, Graeme Burgess, Trevor Faragher, Patricia MacWhirter.

I Bell

Ian Bell
Convener

WVPA

SYDNEY 1993

REPORT OF CHAIRMAN OF WVPA CONGRESS ORGANISING COMMITTEE

5 FEBRUARY, 1992

The Preliminary Announcement of the Congress has been forwarded to 2,000 addresses including key individuals and Journals, Institutions, Universities, Government Departments, Associations and Branches of WVPA and WPSA.

200 responses have been received already.

$7,000 has been spent on Printing and mailing.

Applications have been prepared for funding by Industry Research Funds and by International Scientific Conference support by the Australian Government.

One commercial company has forwarded $1,500 as an advance payment towards their Major Sponsorship of $10,000 and two others have indicated their intention to do so.

Invitations have been sent to 6 overseas and 6 Australian Keynote Speakers and replies are being received.

Posters have been displayed and Leaflets distributed at seven International Conferences.

The Second Notice is being prepared at present.

Paul Gilchrist
Chairman

Call for papers

A two day Scientific meeting of the AVPA will be held in VIAS, Attwood, Melbourne on November 11 and 12. Please contact Peter Young to suggest topics or to profer papers. Topics already suggested included Exotic diseases, Chemical residues and farm chemicals, BLS (contact Peter Curtin), CAA and Animal Welfare (contact Chris Morrow). Some time will be available for papers outside the topics. An Ordinary General Meeting of the AVPA will be held on November 11. Registration and provisional program will be circulated in the next DANDER before the conference.

Contact: Dr Peter Young
Animal Research Institute
Locked Mail Bag
Moorooka, Q 4105

Phone 07 362 9400
Fax 07 892 5374

Membership renewal/application 92-93

NAME:..... Qualifications.....
Organization Site
Address ..... Work Phone ( ) ....
..... Home Phone ( ) ....
..... Postcode Fax ( ) ....

Membership Category [Ordinary $30/Sustaining $80 per unit/Life Nil]
Amount enclosed ..... MAKE CHEQUES PAYABLE TO THE AVPA.

Return to the Treasurer Dr Paul Gilchrist
Biological Technology Transfer
11 Gaza Road
Naremburn
NSW 2065

AVPA Sustaining members 1991 (♦ number of Units)

Arthur Webster Pty Ltd ♦♦♦♦♦
Marven Poultry Pty Ltd ♦♦♦
Linco Engineering Pty Ltd ♦♦
Smith Kline Beecham Pty Ltd ♦♦
Australian Poultry Ltd ♦
Golden Cockerel Pty Ltd ♦
Roche Products Pty Ltd ♦
Cyanamid Australia ♦♦
Elanco Products Company Pty Ltd ♦♦
Red Lea Poultry Pty Ltd ♦♦
AA Tegel Pty Ltd ♦
Bayer Australia Ltd ♦
Pfizer Agricare Pty Ltd ♦
Upjohn Australia Ltd ♦

FORTHCOMING CONFERENCES RELATED TO POULTRY

10-11 November (approx.) 1992.
Australian Association of Poultry Veterinarians, Scientific Meeting.

Contact:
Dr. Chris Morrow,
Victorian Inst. of Animal Science,
Mickleham Road,
Attwood, VIC., 3049.
ph: (3) 333 1200
fax: (3) 333 2932

9-10 February, 1993.
Australian Poultry Science Symposium,
University of Sydney,
Parramatta Road,
University of Sydney,
Camperdown.

Contact:
Professor D. Balnave,
Dept. of Animal Science,
University of Sydney,
Werombi Road,
Camden, N.S.W., 2570.
ph: (46) 550 277

18-21 April, 1993.
Recent Advances in Nutrition,
University of New England,
Armidale, N.S.W., 2351.

Contact:
Professor David Farrel,
Dept. of Microbiology & Nutrition,
University of New England,
Armidale, N.S.W., 2351.
ph: (67) 73-3333
fax: (67) 72-8235

16-19 August, 1993.
Xth Intl. Congress of the WVPA
Hilton Hotel, Sydney.

Contact:
Margaret Reid Pty. Ltd.,
19 Glenferrie Ave.,
Cremorne, N.S.W., 2090.
ph: (2) 908-4381
fax: (2) 908-1070

10-13 October, 1993.
Australian Poultry & Feed Convention
Jupiter's Casino, Gold Coast,
Queensland.

Contact:
Dr. Geoff Fairbrother,
P.O. Box 579,
North Sydney, N.S.W., 2059.
ph: (2) 929-4077

April 1994
Poultry Information Exchange
Gold Coast, Queensland.

Contact:
Paul Marion,
Dept. of Primary Industry,
G.P.O. Box 46,
Brisbane, QLD, 4001.
ph: (7) 239-3519
fax: (7) 239-3558

Thanks to HARVEY LANGFORD (Upjohn) for this list