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President's Report

After our AGM discussions regarding the future structure of the AVPA, our small committee has been busy examining the actions required from the motion.

We have had a very fruitful meeting with the AVA and have hopefully identified a way to proceed. Secretary Ben Wells has circulated a summary of this meeting to the membership. The committee will continue to assess the process necessary and we should be able to bring a proposal to the membership shortly. Hopefully this will enable us to resolve the entire issue and allow us to move on with our core functions. This may involve a transition stage where we have essentially two groups operating but this should resolve successfully in due course.

On a sadder note, Dinah Fry-Smith, a life member, is seriously ill in Camden Hospital at the time of writing. We have sent flowers and fond wishes to Dinah on the AVPA's behalf. Dinah has been a strong long term supporter of and contributor to the AVPA and we hope for the best for her and her family in this current time of difficulty. I know she would appreciate all your thoughts.

Planning seems to be going well for the next scientific meeting in Adelaide (Glenelg) in October-November and we thank Kim Critchley and Margaret Sexton for their efforts in the task.

Peter Groves

Thanks to AVPA Sponsors

Belonging to AVPA has to be one of the most cost effective memberships of a professional organization available. Our sustaining members contribute funding which helps to keep AVPA membership fees to a minimum, while the generous sponsorship of our scientific meetings makes them the best value for money for attendees of any conference in Australia.

AVPA would like to acknowledge the sponsorship of the following organizations at our last two scientific meetings:

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Dander Celebrates 30 Years of Publication

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The Australian Veterinary Poultry Alliance is a Special Interest Group of the Australian Veterinary Association. Membership of the AVPA is available to individuals and groups working in, or interested in, any veterinary aspect of poultry.

Dander will be published quarterly (March, June, September and December). Contributions are welcome. Electronic copy is requested. Deadline for copy is by the end of the second week of the month of publication. Please send information on abstracts of interesting papers, summaries of reports, case histories, social news etc. to Kevin Whithear, School of Veterinary Science, The University of Melbourne, 250 Princes Highway, Werribee 3030, Victoria <kevingwt@unimelb.edu.au> fax 03 9731 2366.

Summary of Upcoming Scientific Meetings

| | |
|-----------------------|--|
| September 2007 | XV Congress of the WVPA. China International Conference Center for Science and Technology (CICCST), Beijing, P.R.China. September 13-16. Fax: +86 10 62174126. Email: llwang@wvpc2007.org. Web: www.wvpc2007.org . Abstracts must be submitted to the Congress Secretariat via the WVPC website no later than 31 May 2007. AVPA member Amir H, Noormohammadi is the Houghton Lecturer at the WVPA Congress. |
| October 2007 | AVPA Scientific Meeting Adelaide. Glenelg Marina Hotel. Date to be finalised between 29 th October 29 and November 2. Contact Dr Kim Critchley. Phone: 08 8272 4251; Email: critchley.kim@saugov.sa.gov.au |
| June-July 2008 | 23rd World's Poultry Conference and Sixth Asia Pacific Poultry Health Conference. Brisbane Conference and Exhibition Centre. June 29 - July 4. AVPA Contact Dr Kevin Whithear; Email: kevingw@unimelb.edu.au . |

MEMBERSHIP MATTERS

Membership List

Thanks to all members who have renewed their AVPA subscriptions for 2007.

New Members: AVPA welcomes the following new members: **Branco Karaconji, Brett Ruth** and **Shamon Shamon**.

Current members are asked to encourage potential new members to join the AVPA. There is always strength in numbers!

2007 Financial Members: George Arzey, Edla Arzey, Caroline Ash, Phillip Ashby, Trevor Bagust, John Barnett, Leone Basher, Carol Bates, Peter Beers, Susan Bibby, Doug Black, Pat Blackall, Wayne Bradshaw, Gabriel Brown, Glenn Browning, David Buckley, Graham Burgess, Brian Burke, Eleanor Chaine, Neil Christensen, Peter Claxton, Kim Critchley, Mike Cundy, Peter Curtin, Colm Culligan, Wieslaw Demkowicz, Kent Dietemeyer, Elizabeth Evans, Gordon Firth, Peter Gray, Tom Grimes, Peter Groves, David Hampson, Bob Hughes, Fakhrul Islam, Clive Jackson, Rod Jenner, Noel Johnston, Bob Johnston, Brian Jones, Wayne Jorgensen, Branco Karaconji, Vivien Kite, Azadeh Laghai, Phil Lehrbach, Mark Lindsey, Margaret MacKenzie, Michael McDermott, Paul McQueen, Con Malliadis, David Marks, Krystyna Minkiewicz, Linden Moffatt, Iain Mortimer, Kerry Mulqueen, Amir H. Noormohammadi, Frank Pace, Barry Philips, Ravi Ravindran, Rod Reece, Bruce Remington, Grant Richards, Julie Roberts, Simon Robinson, Ambrosio Rubite, Brett Ruth, Peter Scott, Margaret Sexton, Shamon Shamon, Wafi Shinwari, Jo Sillince, Peter Spradbrow, Jillian Templeton, Andrew Turner, Greg Underwood, Aileen Vanderfeen, Julie Wagner, Steve Walkden-Brown, John Walters, Ben Wells, Pam Whiteley, Bill Williams, Sarah Wiley.

Student Member: Gabriel Brown, Alireza Mahmoudian.

Life Members: Balkar Bains, Leon Barlow, Roger Chubb, Dinah Fry-Smith, Paul Gilchrist, Harvey Langford, Kevin Whithear.

Please see the AVPA website for information on sustaining members and links to websites

AVPA Sustaining Members 2007

Sustaining members contribute funds that help defray costs of services to members of the AVPA. We thank all sustaining members for their active interest and support.



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INCLUSION BODY HEPATITIS OF CHICKENS – OCCURRENCE AND CONTROL IN AUSTRALIA

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SUMMARY

Fowl adenoviruses (FadV) have been isolated from poultry in Australia since the 1960s, but inclusion body hepatitis (IBH) outbreaks from which FadV were isolated were not reported until 1974. Early outbreaks of IBH were thought to be precipitated by immunosuppression, particularly due to infectious bursal disease (IBD) virus. However in the early-1980s an acute form of IBH occurred in broilers throughout Australia, characterized by mortality rates as high as 40% over a 10-day period commencing at 7-14 days of age. Outbreaks occurred for 4-6 weeks in most progeny broiler flocks from specified breeding flocks. A serotype-8 (now 8b) FadV was isolated from livers of affected birds. Intranuclear basophilic inclusion bodies predominated in the livers of affected birds. IBH was reproduced with a serotype-8 FadV by inoculating day-old specific-pathogen-free (SPF) chickens by natural routes of infection and IBH occurred in in-contact chickens. Virological and serological evidence indicated that IBD virus or Chicken Anaemia virus (CAV) was not involved. A live vaccine, developed using one of the virulent serotype-8 FadV isolates and used to vaccinate breeders during rearing to ensure seroconversion prior to onset of lay, has been an integral part of a strategy to successfully control IBH in chickens in Australia in the last 15 years.

INTRODUCTION

FadV contain three subgroups - Group 1, Group 2 (hemorrhagic enteritis virus of turkeys, marble spleen disease virus of pheasants and avian adenovirus splenomegaly of chickens) and Group 3 (Egg Drop Syndrome virus). Group 1 AAV, which are in the genus Aviadenovirus, have been grouped into various serotype classifications in the past, but are currently classified by the International Committee on Taxonomy of Viruses into five aviadenovirus species, designated A-E based mainly on molecular criteria, and into 12 serotype subgroups, designated 1-11 (but includes 8a and 8b) based on cross neutralisation tests (Benko et. al. 2000).

The most important disease of chickens, in which FadV have been involved is IBH, with early reports in the USA (Helmboldt and Frazier 1963), Canada (Howell et. al. 1970; Pettit and Carlson 1972) and the United Kingdom (Young et. al. 1972; Macpherson et. al. 1974). A serotype-5 FadV, designated Tipton strain, was the first FadV incriminated in the aetiology of IBH (Bickford et. al. 1973; Fadly and Winterfield 1973; Rosenberger et. al. 1974). Type-8 (Grimes et. al. 1977a; Grimes et. al. 1978) and other serotypes of FadV (Gallina et. al. 1973; Cook 1974, McDougall and Peters 1974; McFerran et. al. 1976; McFerran and Adair 2003) have also been isolated from IBH outbreaks. The pathogenesis of IBH was considered to be primary immunosuppression caused by IBD virus followed by FadV infection (Rosenberger et. al. 1975; Fadly et. al. 1976). Aplastic anaemia was sometimes linked with FadV and IBH, but CAV may have been involved in these cases (Bulow et. al. 1986). Hydropericardium Syndrome or Angara Disease, that initially was reported in Pakistan (Anjum et. al. 1989) and

then in a number of other countries, is a variation of IBH occurring at 3-6 weeks of age and with serotype-4 AAV being involved (Cowen 1992; Hess 2000).

FadV OCCURRENCE IN AUSTRALIA

Early isolates of FadV in Australia from other than IBH were from infectious laryngotracheitis vaccine (Cox 1966), the bursa of Fabricius (Mustaffa-Babjee 1971; Spradbrow and Bains 1974), respiratory disease (Lim et. al. 1973; Mustaffa-Babjee and Spradbrow 1975a; Mustaffa-Babjee and Spradbrow 1975b; Van Kammen and Humphrey 1978; Hussein et. al. 1981), egg production problems (Boyle and McFerran 1976) and arthritis (Lim et. al. 1973; MacKenzie and Bains 1977; Hussein et. al. 1981). Serological surveys indicated that FadV were widespread in chicken flocks in Australia (Cox 1966; Mustaffa-Babjee 1971; Boyle 1973; Mustaffa-Babjee and Spradbrow 1975a). Serotypes identified included type 1 from cases of respiratory disease and arthritis (Hussein et. al. 1981) and types 1 and 4 from flocks with respiratory disease or egg production drops (Boyle and McFerran 1976). Kefford et. al. (1980) undertook the first substantial serotyping study in Australia of FadV isolated from IBH outbreaks using microneutralisation typing methods (Grimes 1976; Grimes and King 1977; Grimes et. al. 1977b).

IBH OUTBREAKS IN AUSTRALIA

The earliest reports of IBH in Australia were reported in 1974 based on histopathological lesions, the isolation of an FadV in the 1973 outbreak and reproduction of characteristic lesions by intravenous and intraperitoneal inoculation of 7-day old chickens (Wells and Harrigan 1974; Wells et. al. 1977). In the most severe outbreak mortality rates between 8.4% and 32.3%, commencing at 35-40 days and extending to 70 days, occurred on 5 broiler farms of one poultry company. Atrophy of the bursa of Fabricius, anaemia, cellulitis, eosinophilic Cowdry-type intranuclear inclusions and secondary bacterial infections occurred in these cases. There were other early reports of IBH in Australia (Bains 1977; Kefford et. al. 1980; Reece et. al. 1983; Reece et. al. 1985). Serotype 1 (Grimes 1979), serotype 8 (Kefford and Borland 1979) and serotypes 6, 7, 8, 6/7/8 and untypable (Kefford et. al. 1980; Reece et. al. 1986) FadV were isolated from affected livers containing either eosinophilic or basophilic intranuclear inclusions. Because there were various serotypes isolated, the outbreaks occurred in birds older than 21 days and there was atrophy of immune system organs it was considered at the time that some other agent, for example IBD virus or in retrospect CAV, was the primary cause of IBH in Australia with FadV being secondary opportunistic pathogens. Potent inactivated IBD vaccines only became available in Australia in 1987, mainly due to the awareness that intermediate-plus types of IBD viruses that occur in Australia can be immunosuppressive and could contribute to IBH outbreaks. It wasn't until 1995 that a live virulent CAV vaccine (Steggles Vaccine Laboratory), similar to that developed in Germany (Vielitz and Landgraf 1988) and still sold worldwide today, was available in Australia.

The possibility that FadV could be a primary pathogen in IBH outbreaks was postulated when a type 2/12 AAV was diagnosed in IBH in a 12-week old, specific-pathogen-free (SPF) cockerel (Reece et. al. 1986). While there were previous reports that FadV could be vertically transmitted (McFerran and Adair 2003), it was not considered that this led to IBH occurrence. IBH outbreaks causing up to 40% mortality over a 10-day period commencing in flocks at 7-14 days of age with a predominance of serotype-8 FadV and the finding that some affected flocks had no detectable maternal antibody to IBD virus (Reece et. al. 1986) provided circumstantial evidence that FadV could be the primary pathogen. Outbreaks occurred only in poultry companies that had superior biosecurity and sanitation programs for their breeder operations. Experimental reproduction of IBH in day-old SPF chickens dosed orally with a serotype-8 FadV and the occurrence of IBH in in-contact chicks provided further evidence that FadV could be a primary pathogen in IBH outbreaks (Reece et. al. 1987; Barr and Scott 1988; Erny et. al. 1991). A feature of these "acute" IBH outbreaks was that basophilic inclusions predominated over eosinophilic inclusions (Reece et. al. 1986) which may be the result of the rapid onset of acute IBH and death shortly after infection (Grimes et. al. 1978; Reece et. al. 1987). Involvement of IBD virus, CAV and reticuloendotheliosis virus seemed unlikely on the basis that convalescent sera were negative to these viruses (Barr and Scott 1987; Erny et. al. 1991).

FadV shown to have different degrees of pathogenicity were grouped into molecular Group E (contains serotypes 6, 7, 8a and 8b), but the FadV isolated from acute cases of IBH in flocks under 3 weeks of age were predominantly serotype-8 FadV, were the most virulent in challenge studies and could be differentiated from the remaining Group E FadV on pairwise comigrating restriction fragment (PCRFR) analysis (Erny et. al. 1991). Further studies in which recombinant FadV were developed indicated that the fiber was responsible for differences in virulence (Pallister et. al. 1996).

Epidemiologic evidence during an outbreak of IBH in New Zealand also suggested that IBH could result from vertical transmission of FadV (Christensen and Saifuddin 1989), although the outbreaks commenced between three and four weeks of age and thymic atrophy and anaemia occurred in affected broilers. Thymic and bursal atrophy occurred when a serotype-8 FadV isolated in NZ produced acute IBH when administered by natural routes of infection (Saifuddin and Wilks 1990; Saifuddin and Wilks 1992). IBD virus could not have been involved in the NZ outbreaks, as NZ chicken flocks were negative to this virus at the time. Epidemiological experience within an Australian national integrated chicken meat company also led to the conclusion that outbreaks of acute IBH due to serotype-8 FadV in flocks less than 14 days of age were vertically transmitted (E. Arzey, NSW Department of Primary Industries, *pers. com.*; Grimes 1992). There was a correlation between acute IBH outbreaks due to serotype-8 FadV in 52 flocks and specific breeder flocks. Seroconversion to serotype-8 FadV was demonstrated in two grandparent flocks which produced parent flocks that were placed over a six week period in different states and had outbreaks of acute IBH. Similar seroconversion correlations were found between parent flocks and broiler progeny with acute IBH. Clinical signs of runting and paleness, low blood packed cell volumes, autopsy lesions

of pale bone marrow and thymic atrophy and secondary infections with *Escherichia coli*, salmonella and aspergillosis, which are commonly seen in CAV-related disease, were not common features of acute IBH. IBH outbreaks have also been thought to have been due to a vertically transmitted serotype-3 FadV in the USA (Pilkington et. al. 1997) and serotypes 7, 8 and 11 in Canada (Gomis et. al. 2006). Maternal antibody was protective when day-old SPF chicks were injected intra-abdominally with a serotype-8 FadV (Grimes and King 1977). Protection studies in Australia raised the possibility that a serotype-8 FadV vaccine could be developed to prevent acute cases of IBH (Pallister et. al. 1993).

CONTROL OF IBH IN AUSTRALIA

Because serotype-8 FadV were isolated from the majority of outbreaks of acute IBH that occurred in chickens less than 14 days of age throughout Australia, there was evidence of vertical transmission, the disease had been reproduced in SPF chickens by natural routes of administration with plaque-purified serotype-8 FadV, the involvement of common immunosuppressant viruses had been eliminated and there was evidence that live serotype-8 FadV were protective against homologous challenge, a decision was made in 1989 to progress the development of a serotype-8 FadV vaccine for use in breeders to prevent "acute" IBH in progeny.

A virulent serotype-8 (now 8b) FadV isolated in chicken embryo liver cell cultures from the livers of 9-day old meat chickens with acute IBH was purified by limit-dilution technique, seedstocks produced in SPF cell cultures and an FadV vaccine was manufactured (G. Firth, Steggle's Vaccine Company now Intervet Australia, *pers.com.*). Replacement breeders vaccinated *per os* or by drinking water administration to infect at least 20% of the flock in mid rearing were bled at 16 weeks of age and tested for serum neutralising antibodies against serotype-8 FadV to confirm successful vaccination. This vaccination approach, administering a vaccine containing a virulent virus to a portion of a breeder flock during rearing, had been used successfully for many years to control avian encephalomyelitis in Australia and a similar approach had been used in Germany to control CAV disease (Vielitz and Landgraf 1988).

Vaccination of all meat and layer breeders of one national poultry company in Australia with this vaccine containing serotype 8 (now 8b) FadV commenced in 1990 and all other Australian chicken meat breeder companies adopted a similar vaccination program when the vaccine (Intervet Australia FAV) became available. Field experience over the last 15 years has indicated that the vaccine is safe, breeding flocks become serologically positive to serotype-8 FadV prior to onset of lay and acute IBH outbreaks have not occurred in progeny adequately immunised as judged from serological response. In fact, this vaccination approach has been regarded in Australia as a great "success story" for the control of an economically important poultry disease.

However there have been sporadic occurrences of IBH in Australian broiler flocks in the last year. Low mortality has occurred at 10-35 days of age. FadV isolated from only two outbreaks were typed (Intervet Boxmeer), being 2/12 in one outbreak and 9 with 8/6 cross reactions in the other. Investigations by technical staff of the poultry

companies involved indicated that immunosuppression may have been the primary cause of these outbreaks. Because FadV serotyping is not currently available in Australia, funding for a research project has been made available to the University of Melbourne by the Rural Industries Research and Development Corporation to facilitate appropriate investigations into any future IBH outbreaks. If there were widespread acute IBH outbreaks due to a specific FadV serotype or biotype other than that already included in the vaccine, then the need to include that FadV in the current live FadV vaccine would have to be considered. A similar approach has been reported with the use of inactivated adenovirus vaccines in some countries to prevent IBH (Toro et al. 2002; Alvarado et al. 2006) and hydropericardium syndrome (McFerran and Adair, 2003). Live vaccines are usually less expensive and administration costs are lower than for inactivated vaccines.

DISCUSSION

While it is accepted that immunosuppressive viruses can facilitate various FadV serotypes to cause IBH, there is also adequate evidence that some FadV can cause an acute form of IBH via vertical transmission. However it is sometimes difficult to determine the exact epidemiology of specific IBH outbreaks. Experimental reproduction of IBH using natural routes of infection usually results in IBH within 3-5 days and cannot usually be achieved in susceptible chicks older than about 10 of age (Reece et al. 1987), even when FadV are injected (Fadly and Winterfield 1973; Grimes et al. 1978), unless birds are immunocompromised (Reece et al. 1987). Hence it is difficult to explain how IBH/hydropericardium outbreaks that commence in flocks older than 4 weeks of age are due to primary FadV infection or how vaccination of breeders can modify such disease occurrences. It may be that some virulent FadV can be the "immunosuppressants", since FadV have been shown experimentally to cause atrophy of the bursa of Fabricius and the thymus (Saifuddin and Wilks 1990; Saifuddin and Wilks 1992). This could permit FadV to cause IBH in birds older than those in which IBH can be reproduced experimentally by natural routes of infection and could justify the use of vaccines, whether live or killed, in breeders to prevent IBH outbreaks in flocks as old as 3-5 weeks. Hence control of IBH in Australia has been by adopting appropriate vaccination programs for the immunosuppressants CAV, Marek's disease virus and IBD virus and by vaccinating breeding flocks with a live FadV vaccine which contains a serotype-8 (now 8b) FadV (Intervet Australia FAV vaccine).

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This is an expanded text of a presentation made by Tom Grimes at the 56th Western Poultry Disease Conference, Las Vegas, March 2007.

WVPA Bureau Member Report

15th CONGRESS OF THE WORLD VETERINARY POULTRY ASSOCIATION, BEIJING, 10-15 SEPTEMBER 2007

ALL THE INFORMATION IS NOW RIGHT AT YOUR FINGERTIPS < <http://www.wvpc2007.org/> >

Arrangements are proceeding well for the organization of this Congress.

Latest News – Deadline for Papers has just been Extended!

Right now, papers for either Oral or Poster presentation are being sent from numerous countries, including Australia. The details for the call for Papers is now showing that the deadline for sending abstracts has been extended a bit, by 2 months in fact, to now be 31 May 2007.

AVPA Members – As anyone who has ever been to China/Beijing will tell you, it's a terrific location. Both for a Scientific Congress, and for taking your family along.

Go on - Have a crack too at putting in a short paper on one of the interesting observations you have seen go by , since Christmas even? You won't miss out on getting a presentation, but the taxman might!. Guidelines below.

Any problems or information needed, just contact me at trevorjb@unimelb.edu.au

"Cheers M'Dears!" (as ABC "Collectors" Host Gordon is won't to say...)

Trevor Bagust

WVPA Bureau Member

GUIDELINES FOR SUBMITTING PAPERS

The paper should be prepared in English, using any common variant of Windows 95/98/2000/XP or above and Microsoft Word 97/2000/2003, single spaced (leave a space below the title and above the text) with the font type Times New Roman, and font size 12 for the title, 10.5 for the rest parts.

Title: Use boldface, with initial letter in capital.

Papers should contain the purpose of the introduction, materials and methods, results, discussions and references (no more than 5).

Paper text must not exceed 500 words or 3500 characters (excluding title and authors).

You must select your preferred method of presentation. After review, the scientific committee may adjust some papers for oral or poster presentation.

Candidates may submit more than one paper.

Authors may use their e-mail address and password to re-login the On-line Submission System and modify their paper until May 31, 2007

Papers will be reviewed according to scientific content, experimental design, relevance to the scientific program and adherence to paper guidelines. The authors will be informed of the acceptance or rejection of their submission by the end of June 30, 2007.

The presenters must complete and pay their registration fee for the Congress before May 31, 2007 to have their presentation permanently entered into the scientific program.

Submission deadline: May 31, 2007.

Draft Minutes of OGM, Sydney University, 14 February 2007

Welcome: The President welcomed members and opened the meeting at 1605

Present: Peter Gray, Bruce Remington, George Arzey, Mike McDermott, John Doyle, Paul MacQueen, Mir, Rod Jenner, Rod Reece, Pat Blackall, Bob Hughes, Julie Roberts, Neil Cooper, Clive Jackson, Raza Finaz, David Tinworth, Soy Rubite, Peter Claxton, Tom Grimes, Kevin Whitear, Branko Karaconti, John Reeves, Aileen Vanderfeen, Graham Burgess, Peter Scott, David Marks, Wayne Bradshaw, Kerry Mulqueen,

Apologies: Susan Bibby, Edla Arzey, Trevor Bagust, Simon Robinson, John Barnett, Wayne Jorgensen, Neil Christensen, Barry Philps, Dinah Fry-Smith, Mark Lindsay, Balkar Bains, Doug BlaxkPaul Gilchrist

Minutes of previous meeting: Accepted. David Tinworth/Bruce Remington

Matters Arising: Nil

Adelaide Meeting: It was resolved that the next scientific meeting would be held in Adelaide. Kim Critchley has taken the lead in organisation joined by Margaret Sexton. The meeting will be held at the Glenelg Marina Hotel on a date to be fixed between 29th October and 2nd November

Moved: D Tinworth/P Claxton that Kim, Margaret, Peter Scott and Amir be the scientific committee with power to co-opt. Passed.

It was suggested that the theme might be Food Safety utilising Dainne Davros from IMHS and the CRC resources based in Adelaide

World Poultry Congress: This will be held in Brisbane from 30 June 2008 to 4th July 2008. The 6th APPHC (Asia Pacific Poultry Health Conference) will provide the Poultry Health/Disease stream of the WPC. We have been invited to have a session on every day.

Kevin Whitear was appointed convenor of a scientific committee that includes Pat Blackall, Graham Burgess, Chris Morrow, Julie Roberts and Branko Karaconti, (Fort Dodge). The committee has power to co-opt

Meeting Closed at 1635

Ben Wells Hon Sec

Draft Minutes of AGM, Sydney University, 14 February 2007

Welcome: The President welcomed members and opened the meeting at 1635.

Present: Peter Gray, Bruce Remington, George Arzey, Mike McDermott, John Doyle, Paul MacQueen, Mir, Rod Jenner, Rod Reece, Pat Blackall, Bob Hughes, Julie Roberts, Neil Cooper, Clive Jackson, Raza Finaz, David Tinworth, Soy Rubite, Peter Claxton, Tom Grimes, Kevin Whitear, Branko Karaconti, John Reeves, Aileen Vanderfeen, Graham Burgess, Peter Scott, David Marks, Wayne Bradshaw, Kerry Mulqueen, Ben Wells. Peter Groves. Phil Ashby.

Apologies: Susan Bibby, Edla Arzey, Trevor Bagust, Simon Robinson, John Barnett, Wayne Jorgensen, Neil Christensen, Barry Philps, Dinah Fry-Smith, Mark Lindsay, Balkar Bains, Doug Black, Paul Gilchrist.

Minutes of Previous Meeting: Accepted. David Tinworth/Mike McDermott.

Business Arising: Nil.

President's Report: As circulated in Dander. Accepted. Peter Groves/Rod Jenner.

Treasurer's Report: Accepted. Peter Gray/David Tinworth. Passed with acclaim and thanks.

Exotic Diseases Committee Report: As circulated in Dander. George Arzey is now on the committee looking at SPF egg supply in Australia. Report accepted. George Arzey/ Bruce Remington.

Therapeutic Committee Report: As in Dander.

Web Site Report: The Web Site is still being supported by James Cook University. Graham Burgess asks that all members keep an eye on it and let him know of any omissions or errors. Graham will also look at the possibility of measuring the number of hits on the site but this is not simple as it is hosted within the James Cook University site

Dander Editor Report: Kevin Whithear commented Dander was becoming an historical document with the memoirs of Paul Gilchrist and another set of memoirs to come. The meeting expressed universal appreciation of the contribution of Paul Gilchrist

Amir suggested we publish case studies in Dander. The secretary is to write to the convenors of the Poultry Health Liaison Groups, George Arzey, Greg Parkinson and Rob Morton to explore the possibility of publishing case history elements of their meetings in Dander.

Kevin was thanked with acclaim

Future of the AVPA: Peter Claxton moved: *A working party be established to determine:*

the legal nature of the present relationship with AVA;
the actual nature of the relationship with AVA;
the legal owner of the finances and assets of AVPA;
the most appropriate structure (proprietary company or incorporation) to protect the rights of the present membership mix;
how to establish that structure in the most effective way;
how to retain control of our financial and other assets;
any other relevant matter.

Seconded by David Tinworth and passed unanimously

Discussion on the motion showed strong support for an independent AVPA which was also shown by the unanimous support of the 33 returned proxies for AVPA independence in the circulated motions. In essence this means *Option 3* from the letter from the AVA President. The view on *Option 1* in the AVA letter which asked for an ongoing relationship with AVA is the point of difficulty. The majority view was expressed by Bruce Remington in the phrase, "we should part friends." The key issue of our \$90,000 asset was addressed by the belief we could pick up the (verbal only) offer from AVA that our new independent body will set up accounts into which all income flows and all expenditure comes from the existing account over which AVA holds ownership. The effect of this would be to move all our money into our own hands over a period of a few years with the 2008 conference being suggested as providing a conduit of maximum diameter. Peter Groves warned this offer from AVA needed to be in a binding written form. Some Veterinarians, with both Kevin Whithear and Tom Grimes speaking, wanted our new structure to include an advisory relationship to AVA as requested by the AVA President in the letter. It was determined that it was the role of the working party to achieve this within the framework of a totally independent AVPA.

It was resolved that motions circulated would be laid on the table until the next meeting

A working Party was established consisting of Peter Groves, Peter Claxton, David Tinworth, Tom Grimes, and Ben Wells

Archives: Our only asset is a filing cabinet in Melbourne containing some of our old records. The Working Party on the future will also consider how to manage our archival records for both legal and historical perspectives

Election of Office Bearers: The existing Office Bearers were elected unopposed to provide continuity during this period.

| | | |
|-------------------------------------|----------------|----------------|
| President | Peter Groves | |
| Vice President | Peter Scott | |
| President Elect | Vacant | |
| Honorary Secretary | Ben Wells | |
| Honorary Treasurer | Peter Gray | |
| AVA Policy Councillor | Peter Groves | |
| Web Page Co-ordinator | Graham Burgess | |
| WVPA Bureau Member | Trevor Bagust | |
| Editor Dander | Kevin Whithear | |
| Scientific Program Co-ordinator AP6 | | Kevin Whithear |

Sub Committee Convenors

| | |
|--------------|--------------|
| Importation | George Arzey |
| Therapeutics | Susan Bibby |
| Welfare | John Barnett |

Life Member: It was moved (Rod Jenner/Peter Gray) that Kevin Whithear be offered *Life Membership* of AVPA in recognition of his lifetime of service to AVPA. This was passed with acclaim

Meeting closed 1748

Ben Wells

Secretary

APVMA Rationale for Banning DMZ in Breeder Poultry

In 2004 the APVMA released a draft review report in which it was proposed to limit the use of dimetridazole (DMZ) in breeder poultry for the treatment of histomoniasis (blackhead) in turkeys and chickens, with a withholding period of 28 days for meat and eggs from treated breeders. The rationale for allowing this use was that this withholding period would be sufficiently long to ensure that there were no measurable DMZ residues in excess eggs and meat from culled or spent breeder hens. Chicken layer hens and cockerels, broiler chickens and grower (meat) turkeys were to be excluded from treatment.

While the APVMA was considering assurances that the industry could manage these restrictions on the use of DMZ, the APVMA received submissions from State departments and queries from overseas regulatory authorities questioning the risk assessment methodology that was initially used by the APVMA. In the original assessment, it was assumed that the safe level (public health standard) for DMZ was at the Limit of Quantification (LOQ) of the available analytical method. However, as the LOQ is a function of the instrumentation used to measure residues, it has no correlation to the toxicological endpoints, which are biological measures. Therefore, it is invalid to use the method LOQ as a "pseudo" health standard.

The potential risk to human health is assessed by comparing the estimated consumption of residues in food commodities with the health standards (ADI¹ and ARfD² for that chemical. An intake less than 100% of the ADI/ARfD is generally considered to be acceptable. In contrast, an exposure level significantly greater than 100% is considered to present an unacceptable risk to human health. The Department of Health and Ageing has withdrawn the former ADI for DMZ on the grounds that there is an old and inadequate toxicology database and concerns that DMZ is a genotoxic carcinogen ie. may cause cancers by a direct damaging effect of DMZ or its metabolites on DNA. Furthermore, it has not established an ARfD. Therefore, the APVMA is not able to conduct an acute or chronic dietary intake risk assessment for DMZ residues in foodstuffs. In the absence of appropriate health standards, the APVMA is not able to establish a safe consumption level for DMZ.

Furthermore, as part of the DMZ Review, it was identified that:

- The existing residue definition for DMZ does not adequately cover all of the relevant residue components. In particular, the hydroxy metabolite of DMZ is toxicologically significant, and may be present in foods at twice the concentration of parent DMZ.
- Much of the available residues data were generated more than 30 years ago. The method LOQ in the studies was typically 0.1 mg/kg (100 ppb). However, contemporary methods for DMZ have an LOQ of 0.0001 mg/kg (0.1 ppb) ie. there has been a 1000-fold increase in method sensitivity.
- The APVMA sets withholding periods (WHPs) for drugs on the basis of the residue decline profile. For DMZ, the WHP needs to be long enough to allow residues to decline to below 0.0001 mg/kg (ie. the LOQ of contemporary methods). However, the available data only monitor the decline of DMZ residues down to 0.1 mg/kg, which necessitates extensive extrapolation beyond the actual data. The lack of empirical data for the residues decline profile between 0.1 and 0.0001 mg/kg means it is not possible to establish a suitable WHP to achieve 'nil' residues.

Thus, in view of the factors discussed above, the APVMA can no longer support the use of DMZ in breeder poultry.

Linden Moffatt

¹The ADI (Acceptable Daily Intake; units of mg/kg bodyweight/day) for humans is considered to be a level of intake of a chemical that can be ingested daily over an entire lifetime without any appreciable risk to health. It is calculated by dividing the overall No-Observed Effect Level (NOEL) from the laboratory animal toxicology studies by a safety factor. The magnitude of this factor (normally 100) is selected to account for uncertainties in extrapolation of animal data to humans, variation between humans, the completeness of the toxicological data base and the nature of the potential adverse effects. (See the Australian ADI table at <http://www.tga.gov.au/docs/html/adi.htm>).

²The ARfD (Acute Reference Dose) of a chemical is an estimate of the amount a substance in food and/or drinking water, normally expressed on a body weight basis, that can be ingested in a period of 24 h or less without appreciable health risk to the consumer on the basis of all known facts at the time of the evaluation. (See the Australian ARfD table at <http://www.tga.gov.au/docs/pdf/arfd.pdf>).

Experiences and Rewards in Pursuit of a Career in the Poultry Industry

Balkar S Bains

Introduction

In the early sixties the development of poultry industry attracted few veterinarians to seek a career path in the emerging and progressive commercial enterprises. In the beginning, the poultry industry was not aware of the potential roles a veterinarian could play within the enterprise. What attracted a few veterinarians to seek a career in the poultry industry and, especially the type of work that they passionately followed over so many years, is a curiosity for some. In response to suggestions from colleagues, the following is a brief insight of my pursuit of a career in the poultry industry. The contents are drawn from memory only and therefore mention of dates will be kept at a minimum.

Beginning in the Sub-Continent

My early growing up was in a rural community in the state of Punjab on an ancestral agricultural property. Individual education was always a priority that would lead to a career other than being engaged in the family farming. I matriculated from a private boy's high school that was regarded as being one of the best in the district. The learning of the English language commenced from 5th grade onwards. By the time of matriculation I was able to speak in four languages and able to read and write three of them. I was very much interested and actively participated in sport and intensely favoured soccer. To my delight, I was selected in the school soccer team that won all the district and state championships. This was the beginning of a road map to a future potential career in professional soccer.

For tertiary education I enrolled in a private college with selection of subjects leading to Bachelor of Arts degree, but this was a prelude to developing a career in sport, especially soccer. Over the ensuing years success followed and not only did I captain the victorious college team that won the state championship but I was also selected and participated in a professional soccer team that played at national levels. At the height of soccer season the soccer practice consumed about 5 hours every day and six days a week at the cost of study. My continued improvement and development as a future professional soccer player certainly met with many significant rewards on the way. Suddenly, an opportunity came my way to visit Australia that ultimately changed everything I had planned as a career.

Beginning in the Big Country

Upon arrival in Brisbane I was confronted with not only the different culture but also seriously handicapped in both written and spoken English. Four years of tertiary study for an arts degree was not relevant to qualify for entry for the veterinary science course. After a settling in period I ventured into running a layer farm that supplied eggs for four years to the then Egg Marketing Board of Queensland. Apart from the feed that was purchased from Barnes Milling, the rest of the activities I managed myself. It was a steep learning curve and I drew extensively on all relevant information in the form of brochures or pamphlets from the poultry section of the Department of Agriculture in Brisbane. This experience has many tales and perhaps

I could share one with you at this time. I noticed that some free-range pullets about 7-8 weeks old appeared depressed. It was a Saturday and I decided to take two pullets to the then Veterinary School located at Yeerongpilly for advice on what was wrong with them. The students on duty that morning quickly gathered around the pullets and asked relevant questions. The examination proceeded with first taking a temperature of both pullets but no one knew the normal temperature of the chicken. Post-mortem examination revealed something (*E. tenella*) infecting the intestines for which they prescribed to treat with potassium permanganate via drinking water for the next three days, which I followed diligently.



Balkar shortly after arrival in Australia

Undergraduate years

During undergraduate years I joined the University soccer team and actively participated in local competitions as well as interuniversity competitions. Queensland University soccer was well regarded at all levels of competition and I had the privilege and honour of being elected a captain of the team. A soccer commentator and coach Dick Tainton once wrote in a national magazine "An outstanding forward for Queensland University soccer team was B. Bains, who was the second top scorer of the tournament with seven goals. If I were selecting the Australian University soccer team to tour Asia, the top scorer G. Oey and Bains would be the first two I would choose". I appreciated receiving an award of Full Blue in Soccer from the University of Queensland.

During the holidays I worked three months on a broiler farm and another year three months on a broiler breeder farm owned by the poultry enterprising company Provincial Traders P/L (PTL). This collective experience of six months working under commercial conditions gave me a better understanding of the poultry industry of the future. The company was the largest broiler integrated enterprise

in Queensland including genetic breeding program as well a leader in commercial poultry feed production. The company also acquired a one third ownership in Hy-Line, the parent breeding company located in NSW. Hy-Line subsequently took over the genetic breeding program and supplied all PTL commercial breeder flock requirements.

Post graduation and career road map

Graduated in December 1964, but early in August the same year, I enquired from the head of the poultry division of PTL about the potential for full time employment. The enquiry met with a mixed response but they agreed to meet in person to discuss the potential role of a veterinarian in the poultry enterprise. The outcome of the meeting was positive and I was asked to report for duty in December 1964. From here on I was delegated the responsibility to establish a technical service team consistent with the poultry division objectives. What followed from here was a great experience for my personal and professional development which is briefly outlined below.

Diagnostic laboratory

A laboratory facility was established during 1966 mainly to support *Salmonella pullorum* eradication and virus isolation from chickens that had died of a disease later known to be infectious bronchitis uraemia. I was assisted for the first couple of years by a laboratory technician (Mrs. H. Adam), later by microbiologist (Ms. D. O'Boyle) for the next two years. The new microbiologist (Dr. M. MacKenzie) commenced in the beginning of seventies to head the laboratory facility. After a preliminary period of introduction I decided to take Margaret with me to visit field cases on a regular basis to give her a better understanding of the clinical disease and associated management factors. This simple and practical approach established a remarkable professional relationship between us that proved most successful in our endeavours for the next six years. The laboratory support for confirmation field diagnosis and investigative work became an important tool for future activity. The identification of viruses isolated at the PTL laboratory was often assisted by Professor Peter Spradborow. In all, the technical support group included a microbiologist, three laboratory technicians, 4 field service staff and me working as a team.

S. pullorum

The introduction of day old breeder chicks from Hy-Line came with vertically transmitted *S. pullorum*. The over all impact of this infection was overwhelming in both broiler and breeder flocks and required frequent farm visits. Furazolidone was the drug of choice for in-feed medication of broiler flocks with a good measure of success. The Poultry Industry Act required all breeder flocks to be blood tested and the results reported by a veterinarian. The eradication of *S. pullorum* from PTL flocks was directly linked with the *S. pullorum* status of Hy-Line flocks as a source of supply of day old breeder chickens. Frequent outbreaks of Pullorum in broiler flocks, hatchery issues and breeder flock blood testing and concurrent diseases was like managing multiple fire out breaks on hot summer day. The underlying cause for the failure to eradicate *S. pullorum* was determined to be the variant strain of *S. pullorum* that was not detected by the available antigens. Ultimately the eradication of *S.*

pullorum was achieved with the new antigen using variant strain *S. pullorum*. The disease was finally eradicated towards the end of 1968. This experience did lead us to take initiative to successfully change the Poultry Industry Act in a way that does not stipulate the blood test be carried out by only a veterinarian. It was a great step forward.

Infectious bronchitis (IB) uraemia

During mid sixties there was very little known about the aetiology and control of IB that caused significant morbidity and mortality in broiler flocks during winter months. The major sequel to IB was wet litter, dehydration, colibacillosis, coccidiosis and all palliative measures were directed towards minimising secondary infections to prevent economic losses. The wet litter in winter months was the most troublesome and on occasions catchers refuse to enter the sheds to pick chickens for the processing plant. To correct dehydration, frequent use of electrolyte via drinking water did not improve wet litter conditions. A feed specially formulated without animal protein was often used for about one week in an attempt to minimise further kidney damage. Once it was confirmed that the aetiological agent was a virus that could be isolated from kidneys of chickens dying of IB, I initiated virus attenuation in the limited facilities that were available at that time. It was a matter of continuing to look and learn as I became more and more involved in this initiative but not knowing the end point. Following repeated experiments it was established that that the 48th virus passage gave the best result. The testing facility consisted of cool room, wire floor brooding and rearing cages, and birds received a trial diet containing ten percent animal protein and were challenged at two weeks of age. This isolate, labelled G48, became the seed virus for vaccine production at PTL laboratory that was used to vaccinate commercial flocks owned and contracted by the company. This was achieved by the end of 1968 and was a major triumph, at least for me. The vaccine was most successful on all-in-all-out farms and less so in mixed age farms. The use of vaccine was limited to the winter months only, initially by drinking water method but that soon changed to hand held aerosol spray at the farm. The IB vaccine production continued in PTL laboratory for several years and finally gave way to commercial vaccine. The results of these studies were never published and often not acknowledged in any of the literature or the reports on this subject.

Coccidiosis

The prevalence of coccidiosis requiring treatment in broiler flocks was relatively common. The length of the broiler growing cycle, concurrent infections and sudden emergence of resistance to chemical anticoccidials were contributing factors. I considered it to be more important to observe coccidial lesions during routine autopsy as opposed to oocyst count or random check by lesion score in healthy chickens. To confirm the emerging resistance development to the currently-used anticoccidial, chickens from all flocks between 3 and 4 weeks of age during a one-week period were autopsied to observe the incidence and severity of lesions. The anticoccidial program for the breeder flocks was designed to provide protective immunity by 12-weeks of age and sporadic outbreaks of coccidiosis were often experienced. Among the anticoccidials, the introduction of clopidol (Coyden) was most memorable as it significantly improved the commercial situation beyond all expectations. After two

Years use of clopidol, suddenly *E. tenella* outbreaks became a problem and even double the recommended dosage failed to control coccidiosis. Attempts to use clopidol after a 2-year break were also unsuccessful.

Rapid development of resistance was also a problem for other coccidiostats available at the time. At the beginning of the seventies, the poultry industry was in serious need of an effective anticoccidial and welcomed the introduction of the ionophore coccidiostats. In preliminary local trials the recommended inclusion level of ionophore caused significant growth depression but was highly efficacious in coccidiosis control. These observations immediately changed the recommendations to be based upon feed intake influenced by the metabolisable energy in the feed. It was also observed that after withdrawal of the ionophore from feed, the chickens ate more feed and drank more water and the resulting additional growth came to be known as compensatory growth.



Balkar and staff at PTL Diagnostic laboratory. Who's the good-looking chick?

Readers will remember that *DANDER* has previously published memoirs of Paul Gilchrist and Rob Cumming that provided interesting and valuable insights into the development of the Australian poultry industry and particularly the control of various diseases that otherwise would have crippled industry growth. The above article is the first of three that chronicle the experiences of Balkar Bains and I am sure all will agree that it makes fascinating reading. In the next installment Balkar will describe other important disease problems that he confronted during his career.

Editor