Bird Words

The Monthly Newsletter of the Toowoomba Poultry Club

A WORD FROM THE PRESIDENT

Just a quick message to say hi and welcome to our first edition of our newsletter, put together by our publicity officer Graeme Thomas. We hope you enjoy reading it and if you have any suggestions or ideas for our next newsletter please email us.

Just a reminder the working bee for The Toowoomba Royal will now be held at the Toowoomba Showgrounds on Sunday 30th (not 23rd) January @ 10am. As a club we are the main volunteers at this show so any help would be appreciated. Schedules are also out for this event so get your entries in.

Until Next Edition

Steven Drysdale

The Future of the Fancy

It's a topic that everyone holds some sort of opinion on. We here of the decline of clubs, the reduction in breeders and the loss of any sought of interest in metropolitan areas due to such enlightened council regulations. The hobby is pushed further to the fringes of the major populated areas. We lose such a large base of potential members and future hobbyists by not getting access to the breeders of tomorrow. For many children the only contact they will ever have is looking at a show or sadly between two buns at McDonalds. But are we alone in this decline of interest and members? The answer is no. The battle for relevance and survival is not ours alone. Go to any club, sport and the woes that we so often bemoan are theirs as well. Society is changing for better or worse but nothing stands still. As custodians of this art it is up to us to show its relevance and the passion and skill that under pins what we do. Children look no different with a stare of pure amazement at chicks than they did fifty years ago. The variety and shear spectacle of some of our breeds still stops people in their tracks at the town shows. Our grandparents were in a society were access and the reality of keeping poultry was a daily necessity as well as a hobby. Unfortunately we are in a buyer's market so each and every fancier especially the young is gold. Whereas entertainment and hobbies were limit just twenty years ago now with the internet and migration you can be learning Brazilian Dance, Fighting in a country town or learning the art of speaking Klingon. The art of the fancy is not lost, we have just got to shout a bit louder to be heard.

WINNING EDGE POULTRY SEMINAR 2011

An Essential Seminar for All Poultry Breeders, Exhibitors and Enthusiasts

The next Winning Edge Seminar is on 05 February 2011, and will be held in Toowoomba, Queensland info@winningedgepoultry.com.au or Ph: 0407 494 350 Rod

http://www.winningedgepoultry.com.au/index.php

Email: twbapoultry@gmail.com

Health Check: This Issue **Marek's Disease**

Chickens are the most important natural host for Marek's disease virus, a highly cell-associated but readily transmitted alphaherpesvirus with lymphotropic properties of gammaherpesviruses. Quail can be naturally infected and turkeys can be infected experimentally. However, severe clinical outbreaks of Marek's disease in commercial turkey flocks, with mortality from tumors reaching 40-80% between 8-17 wk of age, were reported recently in France, Israel, and Germany. In some of these cases, the affected turkey flocks were raised in proximity to broilers. Turkeys are also commonly infected with turkey herpesvirus, an avirulent strain related to Marek's disease virus. Other birds and mammals appear to be refractory to the disease or infection.

Marek's disease is one of the most ubiquitous avian infections; it is identified in chicken flocks worldwide. Every flock, except for those maintained under strict pathogen-free conditions, may be presumed to be infected. Although clinical disease is not always apparent in infected flocks, a subclinical decrease in growth rate and egg production may be economically important.

Etiology

Three serotypes of the cell-associated herpesvirus are recognized. Serotypes 1 and 2 designate virulent and avirulent chicken isolates, respectively; serotype 3 designates the related avirulent turkey herpesvirus. Serotypes 2 and 3, as well as attenuated serotype 1 viruses, have been used as vaccines. Serotypes are identified by reaction with type-specific monoclonal antibodies or by biological characteristics such as host range, pathogenicity, growth rate, and plaque morphology. Currently, virulent serotype 1 strains are further divided into pathotypes, which are often referred to as mild (m), virulent (v), very virulent (vv), and very virulent plus (vv+) Marek's disease virus strains.

Transmission and Epidemiology

The disease is highly contagious and readily transmitted among chickens. The virus matures into a fully infective, enveloped form in the epithelium of the feather follicle, from which it is released into the environment. It may survive for months in poultry house litter or dust. Dust or dander from infected chickens is particularly effective in transmission. Once the virus is introduced into a chicken flock, regardless of vaccination status, infection spreads quickly from bird to bird. Infected chickens continue to be carriers for long periods and act as sources of infectious virus. Shedding of infectious virus can be reduced, but not prevented, by prior vaccination. Unlike serotypes 1 and 2, which are highly contagious, turkey herpesvirus is not readily transmissible among chickens (although it is easily transmitted among turkeys, its natural host). Attenuated serotype 1 strains vary greatly in their transmissibility among chickens; the most highly attenuated are not transmitted. Marek's disease virus is not vertically transmitted. The incidence of Marek's disease is quite variable in commercial flocks and depends on strain and dose of virus, age at exposure, maternal antibody, host gender and genetics, other concurrent diseases, and several environmental factors including stress.

PathogeneisCurrently, 4 arbitrary phases of infection in vivo are recognized: 1) early productive-restrictive virus infection causing primarily degenerative changes, 2) latent infection, 3) a second phase of cytolytic, productive-restrictive infection coincident with permanent immunosuppression, and 4) a proliferative phase involving nonproductively infected lymphoid cells that may or may not progress to the point of lymphoma formation. Productive infection may occur transiently in B lymphocytes within a few days after infection with virulent serotype 1 strains and is characterized by antigen production, which leads to cell death. Productive infection also occurs in the feather follicle epithelium, in which enveloped virions are produced. Latent infection of activated T cells is responsible for the longterm carrier state. No antigens are expressed, but virus can be recovered from lymphocytes by co-cultivation with susceptible cells in tissue cultures. Some T cells, latently infected with oncogenic serotype 1 strains, undergo neoplastic transformation. These transformed cells, provided they escape the immune system of the host, may multiply to form characteristic lymphoid neoplasms. Cell-mediated and humoral immune responses are both directed against viral antigens, with cell-mediated immunity probably being the most important.

Clinical Findings and Lesions

Typically, affected birds show only depression before death, although emaciation may be noted. A transient paralysis syndrome (unilateral leg paresis) has been associated with Marek's disease, causing a characteristic posture of one leg held forward and the other held backward as lesions progress. Chickens become ataxic for periods of several days and then recover. This syndrome is rare in immunized birds.

Enlarged nerves are one of the most consistent gross lesions in affected birds. Various peripheral nerves, but particularly the vagus, brachial, and sciatic, become enlarged and lose their striations. Diffuse or nodular lymphoid tumors may be seen in various organs, particularly the liver, spleen, gonads, heart, lung, kidney, muscle, and proventriculus. Lymphoid infiltrates may expand the iris muscle and distort the shape of the pupil. Enlarged feather follicles (commonly termed skin leukosis) may be noted in broilers after defeathering during processing and are a cause for condemnation. The bursa is only rarely tumorous and more frequently is atrophic. Histologically, the lesions consist of a mixed population of small, medium, and large lymphoid cells plus plasma cells and large anaplastic lymphoblasts. These cell populations undoubtedly include both tumor cells and reactive inflammatory cells. When the bursa is involved, the tumor cells typically appear in interfollicular areas.

Diagnosis

Usually, diagnosis is based on enlarged nerves and lymphoid tumors in various viscera. The rareness of bursal tumors helps distinguish this disease from lymphoid leukosis (*see* *Leukosis/sarcoma Group*); also, Marek's disease can develop in chickens as young as 3 wk of age, whereas lymphoid leukosis typically is seen in chickens >14 wk of age. Reticuloendotheliosis, although rare, can easily be confused with Marek's disease because both diseases feature enlarged nerves and T-cell lymphomas in visceral organs. A diagnosis based on typical gross lesions may be confirmed histologically, or better, by demonstration of predominant T-cell populations and Marek's viral DNA in lymphomas by histochemistry and PCR, respectively. Furthermore, Marek's disease lymphomas will usually lack evidence of clonally integrated avian retroviruses or alteration of the cellular oncogene *c-myc*.

Control

Vaccination is the central strategy for the prevention and control of Marek's disease. The efficacy of vaccines can be improved, however, by strict sanitation to reduce or delay exposure and by breeding for genetic resistance. Probably the most widely used vaccine consists of turkey herpesvirus. Bivalent vaccines consisting of turkey herpesvirus and either the SB-1 or 301B/1 strains of serotype 2 Marek's disease virus have been used to provide additional protection against challenge with virulent serotype 1 isolates. Several attenuated serotype 1 Marek's disease vaccines are also available; of these, the CV1988/Rispens strains appears particularly effective. A synergistic effect on protection, noted mainly between serotype 2 and 3 strains, has prompted the empirical use of other virus mixtures. Because vaccines are administered at hatching and require 1-2 wk to produce an effective immunity, exposure of chickens to virus should be minimized during the first few days after hatching. Vaccines are also effective when administered to embryos at the 18th day of incubation. In ovo vaccination is now performed by automated technology and is widely used for vaccination of commercial broiler chickens, mainly because of reduced labor costs and greater precision of vaccine administration. Proper handling of vaccine during thawing and reconstitution is crucial to ensure that adequate doses are administered. Cell-associated vaccines are generally more effective than cell-free vaccines because they are neutralized less by maternal antibodies. Under typical conditions, vaccine efficacy is usually >90%. Since the advent of vaccination, losses from Marek's disease have been reduced dramatically in broiler and layer flocks. However, disease may become a serious problem in individual flocks or in selected geographic areas.

Toowoomba Poultry Club Inc

Dedicated to keeping alive the values and heritage of fanciers in Queensland

Show Dates 2011

Stanthorpe 28-Jan - 29-Jan

Allora 4-Feb - 5-Feb

Clifton 11-Feb - 13-Feb

Killarney 18-Feb - 19-Feb

Cooyar 19-Feb

Bell 26-Feb - 27-Feb

Millmerran 26-Feb - 27-Feb

Pittsworth 4-Mar - 5-Mar

Tara 5-Mar

Inglewood 11-Mar - 12-Mar

Oakey 12-Mar

Warwick 25-Mar - 27-Mar

Toowoomba 31-Mar - 2-Apr

Dalby 8-Apr - 9-Apr

Goombungee-Haden 16-Mar

Goondiwindi 29-Apr - 30-Apr

Crow's Nest 6-May - 7-May

Ipswich 12-May - 14-May

Our Next Get Together

***6th February 2011 at 2pm,* 10 Cavell St East Toowoomba**

Info Bantam and Call Ducks

Black East Indian

First Standardized in Britain in 1865, the Black East Indian Duck shares its colour with the North American Cayuga. This bantam duck was alleged to have been imported to Britain by the Earl of Derby in about 1850. However, evidence suggests that it had been already in the possession of the London Zoological Society since 1831, the same year that the 13th Earl of Derby was elected President of the ZSL. At this time it was known as the "Buenos Ayres" duck, but there seems to be no evidence that South America or the East Indies were the places of origin. It has been known as "Labrador", "Brazilian", "Buenos Aires" and eventually "Black East Indie", the former being perhaps the most appropriate geographically. There is speculation that the black gene may have arrived via a close relative of the northern mallard, the American black duck (Anas rubripes). This is the bold assertion of early historians of the Cayuga, and it seems equally applicable to the Black East Indian. The drakes tend to retain their black plumage but the females develop patches of white as they get older. Impure black birds can show elements of brown pencilling, especially under the wings and throat.

Silver Appleyard Miniature

Developed in the 1980s and shown at the first BWA Championship Waterfowl Exhibition of 1987 by Tom Bartlett of Folly Farm, this Bantam Duck is a miniature version of the original Silver Appleyard produced by Reginald Appleyard in the mid twentieth century. The Miniature, first Standardized in 1997, is roughly a third of the weight of the original, large breed.

Silver Bantam

This bantam breed was formerly known as the Silver Appleyard Bantam. It was produced by Reginald Appleyard from a cross between a small Khaki Campbell duck and a white Call drake in the 1940s. The Silver Bantam does not have the same colour genes as the large Silver Appleyard, hence the change of name when the Miniature Appleyard was Standardized in 1997. The Bantam is very similar to the Abacot Ranger, which was also developed from Khaki Campbells and crossed to a white drake. In this way, the dusky mallard genes were retained and the hidden harlequin-phase genes