HISTORY OF AVIAN MYCOPLASMOSIS IN AUSTRALIA

KEVIN WHITHEAR

It is interesting to note that the occasion we are celebrating at this meeting, the formation of the AVPA, had its genesis back in 1961 at a meeting to discuss troublesome poultry respiratory diseases occurring at that time. The two diseases on the Agenda of the meeting were chronic respiratory disease (CRD) and ILT. The 1961 meeting resolved that the newly recommended name of *Mycoplasma gallisepticum* (MG) be adopted for the pathogen responsible for causing CRD in chickens and infectious sinusitus in turkeys. However, it took another decade before MG was definitively isolated and identified. Australian reports of the isolation and identification of the two other major poultry mycoplasma pathogens, *M. meleagridis* and *M. synoviae* (MS) quickly followed, as did a report that MG was egg transmitted.

In the 1970's some of the major poultry integrators in Australia decided to follow the lead of US poultry industry and attempt to eradicate MG and MS from breeder flocks. This required eliminating the cycle of egg transmission and using biosecurity to minimise the risk of external introduction of infection. This had some success down to grandparent level, but keeping parent flocks free of infection often seemed a step too far and these flocks regularly had breaks. These breaks often occurred when the flocks were in production resulting in high levels of egg transmission and mycoplasma disease in the progeny. To control this problem, antibiotics were extensively used; particularly tylosin, and not surprisingly strains of MG showing acquired resistance to tylosin and other macrolides emerged.

Eradication has rarely been achieved on multiple-age layer farm complexes. When MG-free pullets are introduced they invariably develop CRD and an associated decline in egg production. Overseas, vaccination with live F-strain MG vaccine was used to try and mitigate this problem. Work commenced in the early 1980's at the University of Melbourne to try and develop a vaccine for Australian egg producers. Initial attempts used killed vaccines, but with limited success, so attention was then directed at live vaccines. Strain 80083 was isolated from a parent broiler breeder flock. In the field this strain appeared to spread relatively slowly. Subsequent testing showed strain 80083 to be of moderate pathogenicity and strongly immunogenic, similar to F-strain, so it was decided to investigate whether strain 80083 could be developed as a potential vaccine candidate for Australian conditions. Ideally, a live MG vaccine should be less pathogenic than F-strain, so steps were taken to attenuate strain 80083. The

most productive approach proved to be the selection of temperature sensitive (ts^+) mutants following chemical mutagenesis of a culture the parent 80083 strain. The most promising of the ts^+ mutants was the ts-11 strain which has been used as a vaccine to control CRD and even eradicate MG in multiple-age layer flocks. Subsequently, the ts-11 strain was used to try and overcome the problem of MG breaks and egg transmission in parent breeder flocks.

Following the success of ts-11, a similar approach was used to develop a ts^+ mutant MS vaccine strain which was designated MS-H.

In the period since the development of the ts-11 and MS-H vaccine strains there have been steady advances in the field of avian mycoplasmology with scientists at the University of Melbourne at the forefront of this international research. Compared with the empirical approaches previously used, cutting-edge and rapidly developing molecular technologies are increasing our understanding of the genotypes and phenotypes of avian mycoplasmas. These advances have in turn led to faster and more accurate diagnostics, including the identification of strains; and improved control, including hopefully, rationally attenuated and efficacious live vaccines.