American Association of Avian Pathologists
Biographies of Professionals in Poultry Health

Walter S. Staples

1913 - 2004

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Date: 2007
Revised:

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Poultry Health Research at Cobb's

A personal narrative by Walter Staples on his research accomplishments

I was hired by Robert Cobb Sr., owner of Cobb's Pedigreed Chicks, as a broiler service man in May of 1951. At the time CRD (Chronic Respiratory Disease) was causing high mortality, poor feed conversion and high processing plant condemnation; and expensive antibiotic applications were of little help in controlling the disease. My degree in poultry husbandry from the University of Maine was of little if any help to solve the problem and broiler service men turned to the veterinary staffs at the local state Universities for help. Dr. Frank Witter at the University of Maine, Dr. Robert Dunlop at the University of New Hampshire and Dr. Henry Van Roekel at the University of Mass, at Amherst and their staffs diagnosed the disease in flock after flock and recommended drug applications available at the time for treatments. Within a few years the causative agent was isolated as a PPLO (Pleuro-Pneumonia-like Organism) and then specifically as *Mycoplasma gallisepticum*, shortened to MG.

Because most of the flocks that I serviced were in NH or southern Maine, I took birds for autopsy mostly to Dr. Dunlop and his staff at the University of N.H. at Durham. As soon

as an antigen was available, Dr. Dunlop prepared quantities and tested flocks when I requested.

It promptly became obvious that it was a flock rather than an individual bird problem because if one bird in a flock tested positive, all were positive or soon would be. On the other hand, if a flock tested negative, and no positive birds were moved onto the same farm, the flock remained negative. This suggested that the disease was spread from bird to bird in a flock but that it did not "live over" in a poultry house unless infected birds came in contact.

Therefore, if flocks occurred negative and remained negative during their life-times by "accident", then it was obvious that if one knew why and how it happened, it could be repeated by design. I was determined to discover the "how and why".

Shortly after the antigen for testing became available, Dr. Van Roekel reported proof of embryo transmission of MG after having found the agent in infected air sacs of unhatched - pipped embryos. Dr, Dunlop demonstrated the autopsy procedure for me. Now I had two 'tools', the blood test (for detecting maternal antibodies in day old chicks and for testing flocks at any age) and the identification of air sac lesions in unhatched-pipped embryos which would provide the rate of transmission from the breeder flock to its progeny. And perhaps even more important, I had access to many breeder flocks in the New England area and to chicks and unhatched-pipped embryos at the Cobb hatchery in Littleton, Mass.

There were very few individuals of decision making level in the company who had faith in or supported my proposal that if free flocks occurred by accident, they could all be raised free, that is, the disease eradicated, by design; and the genetic division considered my research as competition and interference and fought it to the end. Fortunately, Mr. Cobb, whose decision was final, did see the possibility and allowed me to continue my research ON MY OWN TIME.

During the next three years I opened more than 300,000 unhatched-pipped embryos (nights and weekends) and tested thousands of chicks and laying breeder hens. I set up a negative flock of 500 breeders, let them become naturally infected during production and trap-nested and opened the unhatched-pipped embryos and took thousands of blood samples for testing at the University. I also graphed the occurrence of lesions in the pipped embryos from hundreds of flocks and from the data was able to correlate the lesion occurrence with the actual number of chicks that survived to hatch with the infection. From the information provided on the graphs it was possible to select flocks that were not passing on MG through the eggs and to reproduce MG negative chicks from breeder flocks that tested positive for the disease.

There appeared to be two strains of MG, one which was highly pathogenic and embryo lesions ran as high as 60% and never stopped transmitting. The most common strain if on an isolated farm, that is, if no other poultry on the farm and no bronchitis or Newcastle outbreaks, or other stress problems, would peak at 25% more or less and decrease to zero before the end of the production life of the flock.

A glance at the graph record of the occurrence of unhatched-pipped embryos would provide accurate information from which to determine what size flock could be hatched without any transmission. Most single age breeder flocks, even though testing positive and having experienced the disease during growth would hatch chicks with no embryo transmission and if not exposed to infected birds would remain negative. In one test, we split a flock of 25000 chicks from a breeder flock that indicated transmission of 1 positive in 25000 and both 12500 flocks were free of MG when hatched and at production.

Now it was obvious why and how negative flocks had occurred by accident; and we knew how to make it happen by design. When Mr. Cobb gave the word to "go negative" we placed replacement flocks based on the information obtained from the embryo examination graphs and never had one of the replacement flocks test positive or have the disease.

I am still amazed that Mr. Cobb's decision to lead the way to eradicate MG in the broiler industry was successful with so much effort made to delay or prevent it. First, Dr. Van Roekel had successfully eradicated *S. pullorum* from the eastern poultry breeders through a "test and remove" program and insisted the same program should be used for MG and which could not be successful because it was so contagious within a flock. Second was the desire of several University researchers supported by and in competition with poultry vaccine manufacturers to produce a vaccine. MG produced a very poor immune response in chickens and was constantly responsible for re-infection in poultry flocks; and I found it difficult to understand why an industry would be willing to continue to live with the disease even if some help was obtained from vaccine when eradication was so easily available.

With the cooperation of Dr. Moulthrop, the highly respected veterinarian in charge of the Poultry Disease Laboratory in Maryland, I placed a large flock of MG negative Cobb broiler chicks on a farm in that highly concentrated broiler raising area, delayed the bronchitis and Newcastle disease vaccination until the third week (to avoid maternal antibody protection) and the flock was marketed without any evidence of the disease and blood samples tested negative.

When Cobb began offering MG negative breeder stock to the industry, competing breeder organizations used varying methods to eradicate the disease such as heating the hatching eggs, soaking them in antibiotic solutions, and other methods. I suspect that

many of those same eggs if selected from older breeder flocks may have been MG free (by “accident”) had they not been treated.

I believe that Mr. Cobb had been convinced from the beginning that eradication was the desired method of preventing losses due to MG infection, and that other embryo transmitted diseases were a responsibility of the breeder. Certainly the results obtained were responsible for his authorization to build a laboratory in Milton,, N.H. and to staff it with qualified personnel. The original SPF (Specific Pathogen Free) flock used from about 1953 during the MG research was maintained and used in many research projects carried on at the laboratory until after my retirement in 1980 when under ownership of The Upjohn Co. it was closed. I kept two loose leaf 8 x 10 note-books containing graphs of typical flock histories of the occurrence of air sac lesions in unhatched-pipped embryos. I gave one of them to a researcher at the US Poultry Research Center in Georgia and the other to the poultry department at the University of Maine. All of the rest of the research (on Marek’s disease, Salmonella, and bursal virus) were destroyed according to Upjohn policy after I left.

The eradication of MG by a poultry breeder was of far greater significance to the industry than the obvious commercial advantages resulting.

To the best of my knowledge no broiler breeder organization at the time hired a full time veterinarian or poultry pathologist and none maintained an active disease research and autopsy laboratory. As Cobb did when I came with them, all depended upon the State University facilities and on the sales personnel of drug and vaccine manufacturers for assistance in controlling poultry diseases. Breeder emphasis up until this time had been concentrated on and pretty much limited to genetics. Cobb's eradication of MG started a rapid move by both breeder organizations and broiler producing organizations to provide trained personnel, laboratory facilities, and in some cases research activity within the organizations. The University faculties, in the poultry growing areas, became more research oriented, less diagnostic, and industry organizations, from breeders through processors, with trained personnel, took over “health control.”

I consider myself very fortunate to have been involved in poultry disease research for more than 27 years while employed by Mr. Robert C. Cobb and the company he founded.

And while, with the laboratory personnel and facilities, we failed to solve the Marek’s disease problem (though we did maintain the SPF flock free of the disease), we did a great deal of work with Salmonella and maintained company flocks pretty much free of that (those) infection, we also were first to understand and report the immune suppression effects of Bursal Disease (Gumboro).

Again, the discovery of the importance of bursal infection (the age at which infection occurred) came about as the result of observations made in the field at broiler level.

Dr. Dharam Ablashi first headed our laboratory staff and was responsible for equipping it with adequate isolation facilities (individual out door buildings, positive pressure units and isolation pens) and the laboratory equipment for our needs. When he left for a position with the National Institute of Health, Dr. Hernando Botero, a graduate of the University of Georgia, took over as laboratory director. Among his many capabilities, he could and did make antigens for and to and utilize testing procedures for most if not all known poultry diseases. I felt that knowing what diseases were present in our (and customer) flocks was important to know BEFORE a problem occurred and we routinely tested flocks and recorded the data.

A Japanese researcher had recently reported the procedure to produce an antigen for testing for bursal disease and we had added that test to our program. While reviewing some of the testing records on the breeder flock of a "sick" broiler flock owned by Hillcrest Poultry Company in Maine, I noted that the breeder flock had tested negative, the broilers positive, for bursal disease. While it was reported and assumed that all poultry flocks in the concentrated broiler areas (such as Del-Mar-Va) were infected with bursal disease, most Cobb flocks were free of the disease probably due to the isolation practices used to eradicate and prevent MG. And we were also finding that many breeder and broiler farms in the New England area were also free of bursal disease. Having observed the susceptibility of progeny flocks of bursal disease free breeders to acute and chronic respiratory infections following routine vaccinations for bronchitis and Newcastle disease, I researched the literature and learned the importance of the Bursa of Fabricius in the development of immunity. It was then obvious that if chicks from bursal free breeder flocks (with no maternal antibodies for bursal disease) were exposed (from infection in the litter) prior to development of the immune mechanism of the chick (up to two weeks of age) the bursa would be destroyed and the chick unable to develop normal immune response. These chicks, therefore, would be susceptible to all diseases, even to vaccine strains.

The simple method of control was to expose BREEDER FLOCKS just prior to production to the bursal virus when it was not harmful to the development of the immune system and would provide maternal antibody protection for the progeny chicks.

Rather than to use live bursal vaccine, we chose to use a killed vaccine which had to be injected into individual mature birds; but a live vaccine was just as effective and because it could be applied on a flock basis, therefore much more economical, Maternal antibody protection was adequate, regardless of how the breeder flock was exposed.

The details of this research and the method of control were reported in a paper presented at the Poultry Health Conference in Maryland and at the N.H. Health conference in New Hampshire. Researchers at the University of Delaware repeated the research and reported it in Avian Diseases with a foot-note that it was first reported by the Cobb Laboratory.

Again, it was disappointing that efforts were made to produce a vaccine to be used at the broiler chick level (the dollar influence—considerably more vaccine required for broilers than for breeders). The danger of occurrence of mutant and more pathogenic strains from introduction and long use of vaccine always appeared a danger to me; and I would promote eradication of a disease if it were possible. As with MG, field testing proved that bursal disease free breeder flocks occurred by accident even in the congested broiler areas and indicated that the thesis of "if it happened by accident, with knowledge of the disease it could happen by design" might well apply and rid the industry of a very expensive disease.

I have not kept informed of the poultry health conditions since my retirement in 1980; but observing emphasis of research reports at the various poultry meetings, it is obvious that the same diseases (including bursal disease and mutant strains) are still prevalent problems of the industry.

Biographical materials provided by Walter's family

Walter Sylvester Staples, 90, of Tamworth, NH passed away at his home on Turkey St. on August 14, 2004. He was born in Eliot, Maine September, 13, 1913, the son of the late Victor R. and Gladys (Langley) Staples.

The oldest of three, he grew up on a small farm in Eliot where he acquired the love of farming and the outdoors. Like many other young men during the Depression, in 1933 he joined the Civilian Conservation Corps (CCC). He was assigned to a camp in Frenchmen's Bay in Bar Harbor, Maine where he proudly served for "a year, a month, a week, and a day".

While a student at the University of Maine he joined the 1937 Arctic expedition of Admiral Donald B. MacMillan as a student crew member on the Gertrude L Thebaud. The expedition sailed to Baffin Land and the Arctic Circle charting new coastlines, performing medical studies on the native Inuit, collecting wildlife specimens and running aground, nearly losing the ship, in Griffin Bay. Fifty years later he organized a reunion at Bowdoin College in Brunswick, Maine, bringing together all surviving crew and scientists of the 1937 expedition.

After graduating from the University of Maine, Orono in 1938 with a degree in poultry science, he worked for General Mills, eventually managing a feed store in Laconia, NH. Preferring research to retail he left the feed store for a job with Cobb, Inc., one of the world's leading poultry breeders based in Littleton, Massachusetts. While there he created

their disease research department and set the standard for poultry disease research worldwide. After retiring in 1980 he purchased a bulldozer and logged his land in Tamworth, New Hampshire and Wesley, Maine as well as managing 14 acres of Maine wild lowbush blueberries.

Knowing a farm was the best place to raise a family, in 1953 he purchased Pine Top Poultry Farm in Tamworth and moved there with his wife Mildred and their children.

His passion was fly fishing for Atlantic salmon which he indulged with 25 trips to the LaPoile River in Newfoundland and many trips to the Miramichi in New Brunswick. It was while in Newfoundland that he became compelled to write his first book, "The North Bay Narrative", a history of a Newfoundland outport village. He subsequently published several poetry books and "Blueberryland", a book about Maine wild lowbush blueberries. His latest poetry book, "Country Boy", is currently at the printers and his book entitled "Mostly My Maine" is in the final stages of publication.

Authors comments: Walter Staples, trained in poultry science, made two pioneer contributions to the poultry health field. Known as an unusually perceptive and creative thinker, he is generally acknowledged as the first to understand how to control Mycoplasma gallisepticum infection in broilers by hatching only from breeder flocks with no evidence of embryo lesions, thus breaking the cycle of vertical transmission. He is also the first to demonstrate vaccination or exposure of breeders to infectious bursal disease virus prior to egg production so that progeny would have passive immunity and be protected against the immunodepressive effects of field infections. These observations, which are described in his own words (above), rank among the significant accomplishments in poultry medicine but are poorly documented in the scientific literature.

Biography solicited by the Committee on the History of Avian Medicine, American Association of Avian Pathologists.

Additional biographical materials may be available from the AAAP Historical Archives located at Iowa State University. Contact information is as follows:

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