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History of Biological Control of Poultry Diseases in the U.S.A.

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Reflection on the control of poultry diseases in the United States brings to mind the extensive cooperative forces that were brought to bear to promote the growth of the poultry industry, which provides our nation with an inexpensive source of protein and aids in feeding people around the world. That cooperative spirit radiated from the Agricultural Experiment Stations at the land grant colleges, worked in close association with the U.S. Department of Agriculture, and it did not exclude the help and expertise of the commercial vaccine–producing laboratories.

Poultry rearing became widespread in the United States in the latter part of the 19th century. Increased demand for chicks resulted in the development of artificial incubation of eggs and along with it the dissemination of a disease referred to as bacillary white diarrhea (BWD). The cause of the disease was discovered by Rettger (17) in 1899 and became classified as *Salmonella pullorum*, hence the name pullorum disease. The poultry men looked to the scientific personnel of the experiment stations to resolve the problem of the high chick mortality. It was discovered that this was an egg-transmitted infection, and the secret of its control was identification of the infected layers and elimination of such layers from the flock. The search for a biological test to identify the carrier birds in a flock was initiated at several experiment stations. The first major biological control for poultry diseases took the form of a tube agglutination test, developed by Jones (14) in 1913 for the detection of carriers of *S. pullorum*. This test set the stage for future developments.

The first concerted effort to organize a routine testing procedure to eliminate the disease was initiated by Drs. W. R. Hinshaw and J. B. Lentz of the University of Massachusetts, who called a meeting of the research investigators of the six New England states in 1928. The conference was called the Northeastern Conference of Laboratory Workers in Bacillary Diarrhea. These conferences were held annually to present information on preparation of test antigens, to compare test results, and to establish test procedures. Advances were made in antigens, from that used in the macroscopic tube agglutination test to a rapid serum plate test and eventually to a stained antigen whole blood test (18).

Since *S. pullorum* and *Salmonella gallinarum*, the causitive agent of fowl typhoid, possessed some common antigens, the test became known as the pullorum/typhoid test. An antigenic variation in *S. pullorum* was recognized by Younie in 1941 (23), and because approximately one third of the isolates in the United States were of the variant type, this strain was included in preparing a polyvalent antigen.

Flock testing methods were also greatly improved over the initial procedure that involved catching the birds, banding each with a number, and then taking a blood sample (which is placed in a numbered tube and sent to the laboratory for the serum test). If positive birds were detected, the flock had to be revisited and birds again caught in order to identify the numbered reactor and to remove it from the flock. The stained antigen whole blood test greatly improved the efficiency of the test procedure in chickens because it allowed one to read the results within minutes and remove the reactor immediately. However, the stained antigen whole blood test was not effective in detecting infected carriers in turkeys.

It is interesting to note the events that subsequently occurred as a result of calling a meeting to discuss the means of controlling pullorum disease. Others, outside of the poultry men in the six New England states, were having problems with pullorum, and the word of the first meeting spread, as a result of which 12 laboratories from the Eastern states plus Canada sent representatives to the second meeting. It wasn't long before representatives from various regions of the United States began attending these annual meetings. It soon became clear that

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much was to be gained from these group discussions, which resulted in regional conferences held annually in the southeastern, north-central, and western regions of the country.

In 1957 the northeastern group voted to change their name to Northeastern Conference on Avian Diseases (NECAD). As would be expected, when a group got together to discuss a specific disease, other disease problems would come up for discussion. At the third annual meeting a policy was adopted to include additional diseases in the discussions but with the proviso that the agenda should be limited to poultry.

At this point I would like to interject a departure from our topic of discussion. In reviewing Dr. Henry Van Roekel's report on the history of NECAD, I was amused by a statement in the report of the 34th meeting (1962), which reminded me of how slowly the wheels of progress do turn. The statement read: "The question arose in regard to expanding membership to persons engaged in poultry disease work but are not associated with the state and federal government or private educational institutions. A committee was appointed to review the situation and to submit a report at the next annual meeting." Prior to 1953 I attended a number of NECAD meetings, but after I became employed by a commercial laboratory, I was excluded from those meetings. Dr. Glenn Snoeyenbos succeeded Van Roekel as the reporter of the NECAD meetings, and in his 50th anniversary report in 1963, he made no mention of policy changes in membership. The next time membership was mentioned was at the 40th meeting in 1968, 5 yr later. It read: "A major policy change to open future meetings to all AAAP members residing in the region was voted with less than unanimity. It was a type of modification of membership requirements which had been considered at various times in the previous decade." Nine years later, in 1977, Dr. Snoeyenbos wrote: "The membership voted to amend prior policy and to welcome all AAAP members to future meetings." It was good to learn that the policymakers of the conference finally realized that more was to be gained by cooperation than by exclusion and that the commercial laboratories had more to offer than financial support for meetings.

Now to return to our topic, pullorum disease not only caused a high rate of mortality in chickens but likewise became a significant cause of mortality in turkeys. Once the cause and method of control was determined, many states, in coordination with the Federal government, developed testing programs. The poultry breeding and hatching industries, realizing the importance of pullorum/typhoid control programs, pushed for a nationwide poultry improvement program. In 1934, by an Act of Congress, the National Poultry Improvement Plan (NPIP) was created. The plan was developed to apply new technology for the benefit of the poultry industry. Controlling disease was a significant part of the program, and the testing procedures for eradicating pullorum/typhoid in chickens and turkeys were established. This program represented a cooperative effort, with the U.S. Department of Agriculture (USDA) providing program coordination and certain services. Most states have subscribed to the NPIP program, and as a result of this concerted effort to control pullorum/typhoid, the incidence of these diseases has been markedly reduced to the point that these infections are seldom seen.

In the 1940s, infection with *Mycoplasma gallisepticum* became recognized as having a significant role in the condition referred to as chronic respiratory disease. This disease, like pullorum, was an egg-transmitted disease, so when it was determined that it could be eliminated by use of an agglutination blood test, a testing program was established under the umbrella of the NPIP in 1966. When *Mycoplasma synoviae* was also found to be an egg-transmitted infection, it was also added to the NPIP in 1974. Since then, NPIP has included oversight of programs on *Salmonella enteritidis* and *Mycoplasma meleagridis*. NPIP is a very active organization, with members representing various aspects of the poultry industry, and it has been a tremendous aid in maintaining the health of the poultry industry.

Before delving into the virus diseases I would like to make some general observations and statements that may not be substantiated as scientific fact but that I think are worthy of consideration. Viruses, like all living things, are continuously multiplying, and like a moving stream, they are not constant and do change direction from time to time. Some virus populations are like placid pools and don't change much. Other populations are restless and keep changing from time to time. Now that we are more knowledgeable about the makeup of living entities, we cannot be so smug about their constancy. Anyone who has worked with the evolving nature of viruses such as infectious bronchitis or influenza has little difficulty in accepting the theory of evolution. Researchers, whether they labor in academic or commercial laboratories, need to be

constantly on the lookout for these shifts in virus populations. Producers of vaccines for the poultry industry are aware of the importance of selecting seed viruses and maintaining them to prevent changes in their qualities as a vaccine. We also need to realize that the bird population is a living entity and varies in identity. Although 100% protection from use of a vaccine is the goal, 90% to 95% protection is quite acceptable when all the variables are taken into account. When shifts in virus populations do occur, these have to be met by adjusting the composition of the vaccine strains. Now let us take a look at some of these factors as they apply to specific poultry diseases.

FOWL POX

During the period that pullorum/typhoid eradication was getting much attention, control of certain virus diseases became a part of flock management. One of the first to be contended with by vaccination was fowl pox, frequently referred to as the roup. Prior to 1931, when Woodruff and Goodpasture (22) introduced the propagation of poxvirus in embryonating eggs, vaccines were crude preparations comprising ground-up skin lesions. One of the pioneers in attempting control of pox by use of crude preparations was Dr. Arthur D. Goldhaft (10), the founder of Vineland Poultry Laboratories. Results were variable and unpredictable, as might be expected with a lack of standardization. Vaccines were greatly improved when pure cultures of virus were propagated in embryonating eggs, and lyophilization of the vaccine prolonged its viability over longer periods of time.

For the chicken industry, two types of poxvirus were used, that obtained from fowl and that obtained from pigeons. Various methods of application were tried: injections were given subcutaneously, intramuscularly, and intravenously, but the most effective application was found to be controlled application directly to the skin of the bird. For the pigeon poxvirus, this process was accomplished by plucking a few feathers from the thigh of the bird and brushing the vaccine into the feather follicles. The preferred application for the fowl pox vaccine was the wing web stab method. The applicator contained two grooved needles (sewing machine needles were used initially), which, when dipped into the liquid vaccine, retained sufficient fluid to deposit on the skin when the needle was pierced through the web of the wing. The procedure was quick and effective.

Field strains of virus that were used early in the production of vaccines would frequently produce a systemic reaction in the birds, resulting in death. I can recall many occasions, while working in Dr. Beaudette's laboratory, when poultry men would come in with a sack of dead birds. Upon necropsy one would routinely find liver lesions of blackhead (histomoniasis), and when the owner was questioned as to whether the flock had recently been vaccinated for pox, the answer was "yes." The recommendation in these cases was to use pigeon pox vaccine applied by the follicle method, which produced little systemic reaction. In later years, fowl pox strains were identified that were less pathogenic, and these were adopted by the vaccine manufacturers.

For more details on the development of pox vaccines, I would recommend the article by Dr. F. R. Beaudette, "Twenty Years of Progress in Immunization Against Virus Diseases of Birds" (2).

LARYNGOTRACHEITIS

In the early stages of the appearance of a new disease, the naming frequently became confused. Such was the case in the late 1920s and early 1930s, when laryngotracheitis and infectious bronchitis, both being respiratory infections, were making apperances in poultry flocks. Isolation and cultivation of each of these viruses in pure culture in embryonating eggs became possible. As evidence of how confused the naming of laryngotracheitis and infectious bronchitis had become, the first vaccine license awarded for laryngotracheitis was under the name of infectious bronchitis. In 1931 a special committee of the American Veterinary Medical Association adopted the name infectious laryngotracheitis for the clinical disease, the signs of which are most readily recognized.

The early investigational work on controlling laryngotracheitis followed a similar pattern to that of fowl pox. Crude preparations were made by harvesting tracheal scrapings from infected birds; these scrapings were used in suspensions to try various methods of inoculation to immunize birds. Attempts failed when the virus material was applied cutaneously (to feather follicles), subcutaneously, intramuscularly, orally, or intravenously. Eventually, Hudson and Beaudette (12) hit upon a method of applying the suspension to the mucous membrane of the vent. This tissue supported the growth of the virus as well as the epithelial tissue of the trachea, stimulating early immunity without impairing the

health of the bird. At first some scarification of the mucous membrane was thought necessary for a good vaccination take, but it was later shown that a drop on the vent mucosa was just as effective and provided for a much cleaner procedure.

Dr. C. S. Gibbs (9) did considerable pioneering work demonstrating the practical application of using crude tracheal suspensions applied to the vent mucosa as a means of flock vaccination for laryngotracheitis. When embryo propagation of the virus was developed, field isolates of the virus were used first, but later, attenuated strains were identified as being preferable for vaccine use. There was no good evidence that virulence of a strain was a necessary ingredient to produce immunity. More important was the virus titer of the vaccine and the method of application.

As time and use of vaccines progressed, attenuated strains of laryngotracheitis were identified and other methods of application were adopted. Labor-saving methods of application received the most attention. Since drinking water administration was used for other diseases, this known method was used by many poultry men and was quite effective if handled carefully using a high-titered vaccine. Another labor-saving application was tried, one that involved spraying the virus and relying on contact with eyes and respiratory mucous membranes. This method was risky as an initial vaccination, sometimes producing excessive respiratory reaction. The method was more useful as a revaccination procedure.

Laryngotracheitis, a respiratory infection readily spread by aerosol, is not always preventable by biosecurity measures practiced in the industry, which makes it necessary to resort to vaccination as a means of control.

INFECTIOUS BRONCHITIS

That respiratory infection of chickens that was once confused with laryngotracheitis received its own identity in the 1930s. First identified by Schalk and Hawn (19) in 1931 as a chick disease capable of causing some mortality, it was soon discovered that its greatest potential damage was infection in a laying flock, which in turn causes a drop in egg production and results in poor-quality eggs. The remedy to prevent these losses was to immunize the flock against the disease before the birds reached sexual maturity.

Like the previously mentioned virus infections, bronchitis was also adapted to embryo propagation. One of the first control programs was initiated in the state of Massachusetts by Dr. Henry Van Roekel. The procedure practiced was to expose birds to the embryo-propagated virus during the middle of the growing period of the flock. With a cotton swab dipped in the virus suspension, or a squirt of virus suspension from a syringe, approximately 5% of the flock were exposed to virus in the tracheal area and were then released into the flock to infect the remaining flockmates. This worked well as a means of immunizing the flock, but if concomitant infections such as mycoplasmosis were present at the time of exposure, the flock might end up with what was then known as chronic respiratory disease. Other nearby states adopted the practice, but it soon became apparent that there needed to be a search for attenuated virus strains that produced a milder respiratory reaction. Such screening procedures did identify strains more suitable for vaccination, and these became available from vaccine laboratories in the early 1950s.

Prior to that time, it was assumed that all infectious bronchitis strains were similar antigenically. That bubble was burst in 1956 (15) when researchers at the Connecticut Experiment Station published results to show that distinct antigenic differences did exist. That first identified variant strain was known as the Connecticut strain, and since then a number of variant strains have been identified from various parts of this country as well as in other countries. Where these strains have caused significant respiratory problems, the vaccine laboratories have responded to the problem by providing specific vaccines or combining strains that provide broader cross protection.

To return to my analogy that virus populations change like a moving stream, infectious bronchitis has taken its place in the swifter currents, and researchers and vaccine producers have their work cut out for them to keep pace with these changes.

NEWCASTLE DISEASE

Even before Newcastle disease was identified in the United States, its potential threat to the poultry industry was acknowledged by the War Department Commission during World War II. To be prepared for its possible use in biological warfare, a war research project was initiated to explore methods of protecting poultry by use of inactivated virus vaccines or by enhancing the immunity with a modified live virus vaccine (4). Unbeknownst to

the warfare planners, old Mother Nature had her own scenario for implementing biological warfare.

Newcastle disease initially slipped undetected into the United States and was first reported on the west coast in the early 1940s by J. R. Beach (1), who described the disease as pneumoencephalitis. Shortly thereafter, in 1945, F. R. Beaudette and J. J. Black (3) on the east coast identified the same disease as Newcastle, which matched the description recorded in the literature. The race was on to develop a means of identification and immunization for a respiratory infection that was rapidly spreading in poultry flocks throughout the United States. Early on there were some attempts to immunize against the infection with inactivated virus vaccines, but these efforts were soon superceded by the search for live virus vaccine strains, which at that time were believed to offer lifelong immunity. Dr. Beaudette, a strong proponent of live virus vaccines, made numerous isolations of viruses in embryonating eggs and proceeded to search for a strain suitable for use as a vaccine. Being a zealous student of the poultry disease literature, he had determined that the way to control the disease was to select a strain of virus that could be safely administered to birds around 4 wk of age. The reasoning behind this was that baby chicks would acquire parental antibody through the yolk to provide protection until 4 wk of age. With this in mind, Beaudette proceeded to screen 105 virus isolates in chickens of approximately that age. As a result of this effort, he selected a strain known as Roakin, which was considered suitable for a vaccine to be administered by the wing web stick method. Vineland Laboratories prepared a vaccine from this strain, and in 1948 it was approved for marketing.

Simultaneously, Lederle Laboratories was given approval for a vaccine prepared from a strain, MK107, that Dr. Van Roekel had selected as suitable. These vaccines performed much as was expected in chickens 4 wk of age or older, but the vaccine left the younger chicks subject to high mortality if infected. This gap in protection was remedied with the discovery in 1947 of the B-1 strain of Newcastle virus (11), which was so apathogenic that it could be administered safely to day-old chicks and provide a high degree of protection. Competing laboratories soon acquired the strain, and by 1950, Salsbury's Laboratories and Lederle Laboratories were given approval to market the B-1 strain.

Administration of the B-1 vaccine was done initially by a drop to the nostril, but it was soon found that a drop on the eye was easier and just as effective. Labor-saving methods of application were sought and developed, resulting in water administration and spraying of the vaccine. Lederle Laboratories developed a method of applying the virus as a dust, but this method was never widely accepted. In 1952 Vineland Laboratories introduced a mild strain of virus for intramuscular injection, but it was soon found it could be administered by the same routes as the B-1 virus. This strain, known as the La Sota strain, was made available by a number of laboratories and found its place in many vaccination programs, especially for revaccination, as have some other lentogenic strains.

Overall, Newcastle vaccines have kept the disease fairly well under control. However, one should realize that we are dealing with a sleeping giant. Newcastle disease slipped into this country under the disguise of infectious bronchitis in the 1930s. It has appeared in many levels of pathogenicity, as has been exemplified by the classification of virus strains as lentogenic, mesogenic, and velogenic. Every once in a while it rears its ugly head, as it did in the 1972–73 California outbreak. Fortunately it does not seem to be as prone to changes in antigenicity as infectious bronchitis. Nevertheless, it is a disease that requires constant vigilance to keep it under control.

AVIAN ENCEPHALOMYELITIS

Avian encephalomyelitis (AE) was first reported by Jones (13) in 1932 as a chick disease causing ataxia and tremors of the head and neck. The disease has great significance for poultry breeders, because if susceptible laying flocks become infected, not only does reduced egg production result, but there is also transmission of the virus through the egg, resulting in infection in the hatched chicks.

It was Dr. Kermit Schaaf of Kimber Farms who devised a practical control program for AE by making sure that breeding flocks were exposed to the virus during the growing period. This practice, immunized the flock before laying started, thus avoiding egg transmission. To make the procedure universally available for all breeders, a marketable vaccine was needed. Virus-inactivated vaccine was shown to be possible, but because administration required catching and injecting individual birds, this method of immunization did not become popular. After a long ordeal involving the efforts of several individuals, a live virus vaccine became available that could be administered orally.

The vaccine of choice was that developed by Dr. B. W. Calnek (6) using an embryo-propagated strain

designated 1143, a strain that was capable of immunizing birds when administered by the oral route in the drinking water. Vaccination should take place after the birds are 8 wk of age and 4 wk before the start of egg production. Virus propagation for vaccine production is done in eggs from AE-susceptible flocks, and it is important that the seed virus not become so overly adapted to embryos that it loses its ability to offer immunization via the oral route.

INFECTIOUS BURSAL DISEASE

The identification of a new disease in poultry by Dr. A. S. Cosgrove (8) in 1957 resulted in confusion in naming the disease and determining the exact etiology. The unraveling of the early history of the disease has been well presented by Lasher and Davis (16). The knowledge that the early infection of susceptible baby chicks had a predilection for the bursa, resulting in severe immunosuppression, pointed to the question of how the disease should be controlled. The answer was the immunization of the parent flock, permitting the transfer of maternal antibodies through the yolk to the chick, thereby resulting in protection during the critical first 3 wk of the chick's life.

Early attempts to immunize flocks were accomplished by spreading infected litter in a house and letting nature take its course or by exposing birds to a suspension of infected bursal tissue. The first licensed vaccine, Bursa Vac, was prepared from a mild strain adapted to embryo propagation by Snedeker, Wills, and Moulthrop (20). Other embryo-adapted strains have since been introduced for vaccine production, as have tissue culture–adapted strains. The greatest passive antibody protection in chicks has been found to occur by live virus exposure at 3 to 5 wk of age in chicks destined to become breeders; this exposure is followed, at 16 to 18 wk of age, with administration of an oil adjuvant killed virus vaccine.

MAREK'S DISEASE

My first encounter with this disease was as a young boy on the farm when our range-reared flock of chickens would develop lameness and eventually complete paralysis of the legs. At the first indication, treatment at the time was to catch the bird, escort it to the woodpile, and with the aid of an ax, relieve it of its suffering. Many such birds found their way to the family dinner table. To the best of our knowledge at the time, the birds were affected with the avian leukosis complex. It took thirty more years before I learned that fowl paralysis was a single disease entity caused by a herpesvirus. The story of the unravelling of the cause of Marek's from the avian leukosis complex is too important for me to try and cover in the time allotted, so for a complete coverage of the story, I would refer you to the excellent chapter on Marek's disease by Drs. Calnek and Witter (7) in the ninth edition of *Diseases of Poultry.*

To briefly mention some of the highlights as they pertain to control, the cause of the disease was identified in the mid-1960s as a herpesvirus. One of the early vaccines found to aid in the control was introduced by a team at the U.S. Laboratory in East Lansing, Michigan. This laboratory was established in 1937 for the express purpose of unravelling the avian leukosis complex. In my estimation, the discovery of a turkey herpesvirus that could be used as a vaccine to help control Marek's disease was a crowning achievement in the history of the laboratory. Of course, as was learned with other viruses, Marek's virus wasn't a single antigenic entity but rather had relatives, so that we soon heard about serotypes 1, 2, and 3. Also, other virus strains suitable for vaccine use appeared on the scene. Knowing that field strains varied, mixing of serotypes in the vaccines became a practice to give greater protection in the field.

Administration of the vaccine underwent several changes over time. Because the virus is a cell-associated virus, the first vaccines were frozen and shipped in the frozen state and then thawed for use. Administration was first applied by subcutaneous injection with syringe and needle. A labor-saving innovation was the development of an automatic injection machine. Further innovation was accomplished by embryo inoculation at 18 days of age. Another innovation was made (5) in finding that cell-free virus could be extracted from cells and lyophilized, giving added advantage in the storage and transportation of the vaccine.

Vaccines have not eliminated the Marek's disease problem, but their use has greatly reduced the losses from death and condemnations at the dressing plant. Much progress has been made in the 35 yr of dealing with Marek's disease, and we are now in a much better position for handling problems that may arise in the future.

DUCK BIOLOGICS

A segment of the poultry industry that should not be overlooked is the duck industry. For years this

was centered on Long Island, and as a consequence of its location in New York State, when disease problems surfaced, the growers turned to the Veterinary College at Cornell for help. As a result, the Long Island Duck Research Cooperative, Inc., constructed a laboratory for research on diseases, nutrition, and management of ducks. The research on diseases was under the direction of the Department of Avian Diseases at the Veterinary College and has been managed on Long Island by Dr. Tirath Sandu.

As a result of the research activities in connection with that laboratory, the USDA Center for Biologics approved an Establishment License in 1977 for the production of biologics. They are now approved for production of Duck Virus Hepatitis and Duck Virus Enteritis vaccines, a Duck Virus Hepatitis yolk antibody preparation, *Pasteurella anatipestifer* bacterin, a combination *Escherichia coli–Pasteurella anatipestifer* bacterin, a *Pasteurella anatipestifer* live vaccine, and an autogenous vaccine. These products are not only available to the duck producers on Long Island but also serve growers in other states and in Canada.

TENOSYNOVITIS

This disease is caused by a reovirus that may be transmitted from breeders through the egg to the chick or horizontally within the flock. A live virus vaccine prepared with an attenuated strain of reovirus designated as strain 1133 was developed at the University of Connecticut (21) and is now available for use by drinking water administration. The product is recommended for use in breeder replacement flocks that are 10 to 17 wk of age. By immunizing the breeder flock, the maternal antibodies are transmitted to the baby chicks, thereby protecting them against infection at the most critical age. By further attenuation of the 1133 virus, an injectable reovirus vaccine was developed for young chicks. This is just another example of what can be accomplished through the cooperative effort of a research institution, the production laboratory, and the USDA Center for Veterinary Biologics.

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