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The Early History of Infectious Bronchitis

Julius Fabricant

Unit of Avian Medicine, Department of Microbiology and Immunology, Cornell University, Ithaca, NY 14853

Abbreviations: IB = infectious bronchitis; LT = laryngotracheitis

In 1931, Schalk and Hawn (22) described "an apparently new respiratory disease of chicks" in North Dakota. The disease occurred in chicks from 2 days to 3 wk of age and was characterized by gasping and listlessness. Mortality rates as high as 40%–90% were observed. Postmortem findings included congestion and mucous exudate in bronchi and trachea and occasionally in the nasal passages. The disease was shown to be readily transmissible by contact exposure or by transfer of bronchial exudate to healthy chicks. The nature of the infectious agent was not determined at that time. A similar clinical entity soon became widely distributed in the central United States.

Two years later, Bushnell and Brandly (3) reported on an essentially identical disease. They established that the causative agent was a filterable virus because the disease could be transmitted by Berkefeld-filtered material. Because the symptoms of respiratory distress were caused by a filterable virus, Bushnell and Brandly regarded the disease as a form of infectious laryngotracheitis (LT). Further critical tests were not made to confirm this identity.

Bushnell and Brandly's report (3) was considered by most later research workers in avian respiratory diseases to actually describe further outbreaks of infectious bronchitis (IB). It was not until much later that the possibility could be considered that some or all of their cases could really have been cases of the mild or low-virulence type of LT described by Cover and Benton (4). A further source of confusion was that, in the 1930s, the name infectious bronchitis was used in some papers actually describing LT.

This confusion was cleared up a few years later by Beach and Schalm (1), who proved by cross-immunity studies in chickens that IB virus was distinct from infectious LT virus and also from coryza due to *Hemophilus gallinarum*. Such infection and immunity studies in susceptible hosts were the only tool for studying viruses at that time once the determination had been made that the causative agent was filterable. Beach and Schalm (1) also reconfirmed the filterability of the bronchitis virus and demonstrated that serum from IB-recovered birds was capable of neutralizing the infectivity of the virus in tracheal exudate suspensions.

Beaudette and Hudson (2) first cultivated bronchitis virus in chick embryos inoculated by the chorioallantoic route. They reported that bronchitis virus could cause embryo death but did not cause the distinctive lesions on the chorioallantoic membrane associated with either fowl pox or infectious LT virus. Beaudette and Hudson (2) also observed that, with continued passage in chick embryos, IB virus became increasingly and more consistently lethal for embryos. Delaplane and Stuart (10) confirmed the fact that bronchitis virus increased in embryo pathogenicity after prolonged embryo passage. They also noted that embryo passage modified the virulence of bronchitis virus for chickens, and eventually the virus that had become highly lethal for embryos became noninfectious and nonantigenic for chickens.

Van Roeckel *et al.* (24) made the serum neutralization test for bronchitis a practical reality by applying the procedure in embryonating eggs rather than chickens. The procedure was carried out with the highly embryo lethal strains previously described by Beaudette and Delaplane.

A new appreciation of the economic importance of IB virus resulted from the observations of Delaplane and Stuart in 1939 (9) that IB was a common respiratory disease in semimature and older chickens in Rhode Island and could result in significant losses in egg production. Similar observations were made through-

out the northeastern United States in the next few years.

Meanwhile, other characteristics of IB virus and the disease it produced were studied. Hofstad (14,15,16) described the pathology of chickens infected with IB and some aspects of IB epidemiology. Cunningham and Stuart (6) examined the effects of certain chemical agents on IB virus and also the pH stability of the virus (7).

Jungherr and Terrells (19) found serum-neutralizing antibodies to IB virus in the yolk of chick embryos and in day-old chicks laid by hens that had been exposed to IB. They assumed that this antibody would protect these chicks. Hofstad and Kenzy (17) reexamined the role of maternal antibody and found that chicks hatched from recovered hens could still be susceptible to IB.

A strong impetus to research on IB resulted from two factors. First, the widespread appearance of outbreaks of the respiratory form of Newcastle disease in the mid-1940s made differential diagnosis of Newcastle disease and IB an important field problem. Second, the increased practice of providing separate housing for growing pullets and laying hens (all-in, all-out management) resulted in a marked increase in the population of IB-susceptible laying hens and thus a marked increase in egg production losses associated with IB.

These stimuli soon produced a response in the form of better diagnostic methods for IB. The isolation of IB virus in embryonating chicken eggs and the effects of IB virus on the chick embryo were studied by Cunningham and Stuart (6,7), Fabricant (11), and Loomis *et al.* (20). The serologic diagnosis of IB by the serum neutralization test was defined and refined by Cunningham (5), Fabricant (13), and Luginbuhl (21). The essential purpose of Fabricant's work (11,12,13) was to standardize these diagnostic procedures and to define their use and diagnostic and epidemiologic interpretation.

Fabricant (11) suggested that the "stunting and curling" reaction in chick embryos could be considered a "pathognomic" lesion. He was cruelly disillusioned of this concept by the observation of S. B. Hitchner (pers. comm., 1951) that the B1 strain of Newcastle disease virus could also produce this lesion.

Careful studies with these techniques confirmed previous clinical observations and gave us a clearer picture of the epidemiology and disease patterns of IB and pointed out the need for effective control programs.

Field observations indicated that chickens infected with IB between the ages of 8 and 16 wk showed only mild respiratory symptoms and no adverse effects. Such chickens, when they reached laying age, were immune to infection and its consequent losses in egg production. Laboratory studies by Van Roeckel *et al.* (24) confirmed these observations and led to a program to immunize the growing stock so the flock would be protected against the disease during the laying period. This protection was accomplished by inoculating a few birds with egg-propagated virus. These birds were released in the flock to naturally expose the remainder of the flock. Similar "planned exposure" programs for the control of IB in layers were used in the northeastern United States with virus supplied by the various state agricultural experiment stations until federally approved commercial IB vaccines became available.

Although this history ends at the point where commercial IB vaccines became available, a short footnote is in order. The advent of practical methods of vaccination with commercially produced vaccines was generally expected to prove an effective prophylactic method for IB control. However, within a few years, a series of entirely new factors led the IB problem into new areas that still persist. The rapid growth of the broiler industry and the role of IB virus in chronic respiratory disease entirely changed the requirements for a successful disease control program. Just as important was the recognition by Jungherr *et al.* (18) that there were immunologic differences in IB strains sufficient to prevent cross-protection.

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