ROTHAMSTED RESEARCH

Brood Diseases of Bees

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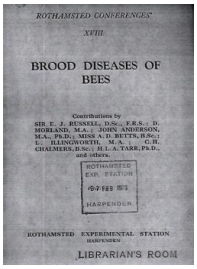

Present Position of the Scientific Investigation of Foul Brood Diseases of Bees

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THE PRESENT POSITION OF THE SCIENTIFIC INVESTIGATION OF FOUL BROOD DISEASES OF BEES

By H. L. A. TARR, Ph.D.

At the present time much confusion exists in the literature dealing with brood diseases of the bee, and although much valuable work has been done there is yet a pressing need for advancement in our scientific knowledge of this subject. I recognize that, as one of at present rather limited experience in this field of investigation, I am as yet not fully qualified to criticise the published papers relating to this subject, and I must therefore ask you to accept what is, on the whole, a non-critical survey of this field.

American Foul Brood

White (1906, 1907, 1920a) first succeeded in isolating the causative organism of this disease, and proved by actual inoculation experiments that pure cultures of this organism actually caused American foul brood. He named the infecting agent *Bacillus larvae*. There is practically no doubt that Maassen's *Bacillus brandenburgiensis* (1906) was identical with *B. larvae*. Since White's discovery Toumanoff in France, Borchert (1930) in Germany, Lochhead (1928a) in Canada and Chalmers and Hamilton (1933) in England, and a good many other investigators have accepted, without much reserve, the findings of the American investigator.

Perhaps the most striking characteristic of *B. larvae* is its inability to grow upon the more common bacteriological culture media, and it was undoubtedly this fact which caused such confusion among earlier workers who attempted to isolate organisms responsible for brood diseases. Such workers as White (1920a), Sturtevant (1924), Lochhead (1928a, 1933) and Toumanoff (1930b) have described media upon which this organism will grow with comparative ease. The spores of *B. larvae* are remarkably resistant to heat, a fact which makes its elimination from the apiary a rather difficult matter.

Recently Sturtevant (1932) has shown that a relatively large initial inoculum of the spores of *B. larvae* is required to initiate a definite infection in a colony of bees: he has estimated that at least 50 million spores fed in one litre of syrup are necessary to infect a colony, and that each larvae required some 10 million spores in 0.01 cc of syrup in order to develop the disease. His results tend to show that commercial honey is probably not a fruitful source of infection in American foul brood.

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It is well known that American foul brood does not usually develop until the larva have been sealed, and Sturtevant (1924) attributed this fact to the inability of *B. larvae* to multiply in the presence of much sugar. He found that concentrations of glucose in the neighbourhood of 5 per cent. completely inhibited multiplication of both spores and vegetative cells of this organism. Lower concentrations of glucose also caused partial inhibition of growth.

The comparatively recent experiments conducted by Toumanoff (1929) are of interest in connection with American foul brood, for he is apparently the only investigator who has questioned the pathogenicity of *B. larvae*. In his experiments he employed aqueous suspensions prepared from young cultures of five different strains of this organism, and fed small amounts to healthy larvae. Of 302 inoculated larvae, 170 were removed by the bees, while the 132 remaining underwent metamorphosis in the normal manner and developed into healthy adult bees. He found that the bees removed some of the larvae when ordinary saline was fed as control in place of the bacterial suspension. He used both vegetative cells and spores. He assumed from his results that it is by no means always easy to infect brood with *B. larvae*, and suggests that his results may be explained by an attenuation in virulence of the organism resulting from cultivation on artificial media. In the light of Sturtevant's work it is possible that Toumanoff's results may be explained by the fact that the number of organisms fed was insufficient to cause disease. There is room for further work along these lines.

European Foul Brood

While American foul brood appears at present to be a relatively well-defined disease, European foul brood is a disease the etiology of which is still in doubt. It is now practically certain that the brood disease attributed to *Bacillus alvei* by Cheshire and Cheyne (1885) is identical with that which was christened European foul brood by Phillips (1906), and which was studied in detail by White (1912, 1920b). White believed that the disease was caused by a lanceolate-shaped, non spore-forming organism, which occurred in large numbers in freshly infected brood, and which would not grow on any of the culture media which he tried. He named the organism *Bacillus pluton*. He assumed that this organism was responsible for producing European foul brood because, when fed in sugar syrup or honey to healthy larvae, typical disease resulted, and because none of the readily-isolable so-called " secondary invaders " (*Streptococcus apis*, *B. alvei*, *B. orpheus* and *Bacterium eurydice*) produced disease when inoculated into experimental colonies. Although many investigators accept White's work, the fact that he was unable to isolate *B. pluton* leaves his conclusion rather open to criticism, and in certain quarters his thesis has not gone unchallenged.

In 1927 Wharton published what appears to have been a premature statement on the etiology of European Foul Brood. He claimed to

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have developed "a medium admirably suitable for the growth of *B. pluton*," and at the same time suggested that this organism is merely a stage in the life cycle of *B. alvei*. Shortly afterward Lochhead (1928b) published a note in which he condemned Wharton for his unauthorized and premature statement. Lochhead himself (1928b) apparently doubted the existence of *Streptococcus apis* as a species distinct from *B. pluton*. He also pointed out (1928c) that it is possible that *B. alvei* dissociates into *B. pluton*, but he has never asserted that this change actually occurs. Thus he stated that, "As yet the identity of the coccoid form of *B. alvei* with the coccoids seen in European foul brood is suggested only on the strength of microscopic comparison." . . . "Our attempts to produce the disease in a colony of black bees through feeding cultures have so far been inconclusive, and consequently no statement can be made at this time regarding the pathogenicity of this form of *B. alvei*."

In connection with the controversy on European foul brood it seems proper to include the recently-discovered disease termed "Para foul brood." Burnside and Foster (Burnside, 1932) (Foster and Burnside, 1933) have recently described this apparently new brood disease, the symptoms and course of which appear to differ from those commonly experienced in American and European foul brood infections. Because of its apparently close relationship to *B. alvei* the authors have chosen the name *Bacillus para-alvei* for the organism which they claim is responsible for the disease. These authors make the mistake of misquoting Lochhead when they say that he actually demonstrated that *B. pluton* is a stage in the life cycle of *B. alvei*. However, if their claim that this has been verified in the Washington laboratory is true, it may be that the problem of what is the infecting agent in European foul brood has been solved. It is to be hoped that a comprehensive scientific report of their work will appear in the near future, and that it will clarify some of the existing confusion in our knowledge of this disease.

Sacbrood

This disease, which is apparently more benign than malignant, was discovered and studied thoroughly by White (1913, 1917). The infected brood presents what appears to be a very characteristic appearance. The most important distinguishing feature from the bacteriological standpoint is the entire, or almost entire, absence of bacterial cells in the infected larvae. This disease, according to White, is due to the activity of a filtrable virus capable of passing through the pores of Berkefeld and Pasteur-Chamberland filters. The porosity of the filters employed in his work is not stated. Apparently no further publication on this disease has appeared since White's original communications, though certain European investigators refer to Sacbrood as a well-defined disease in their publications.

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Rarer Infections of the Brood

In 1921 Sturtevant found that American and European foul brood occasionally occurred simultaneously, but such outbreaks were extremely exceptional. Borchert (1934) claimed that *Bacillus orpheus*, an organism considered by White (1920b) to be a non-pathogenic secondary invader in European foul brood, can infect the brood of bees. The results obtained by Borchert do not appear to be very striking, since the relative amounts of infected brood obtained in his experiments was very small. At present it appears as if this type of infection is more of academic than of practical importance.

Toumanoff (1927) has described a brood disease which appears to differ from any previously described. From combs containing naturally infected larvae he isolated four different organisms: *Colibacillus paradoxus*, *Bacillus agilis larvae*, *Micrococcus luteus liquefaciens* var *larvae* and an unidentified species of *Torula*. He describes the cultural and morphological characteristics of these organisms in some detail, but makes no attempt to explain which of these organisms is the primary infecting agent.

The most recent work dealing with fungus diseases of bee larvae is that of Burnside (1930), though his work is chiefly concerned with diseases of adult bees. He found that the moulds of the *Aspergillus-oryzae* group are largely responsible for fungus diseases of the brood, *A. flavus* being the most common infecting agent. *Pericystis apis* and *P. alvei* have, according to Burnside, never been reported in North America.

The Immune Reactions of Larvae

Borchert (1924, 1930) claims to have demonstrated complement-fixing antibodies in extracts of the larvae and scales from foul brood combs, but he failed to find agglutinins or precipitins for *B. larvae* or *B. alvei* in such extracts. He was able to demonstrate a complete serological difference between these two organisms.

Metalnikoff and Toumanoff (1930) and Toumanoff (1930a), showed that two types of blood cells are present in larvae, namely proleucocytes and leucocytes, the form of which they describe in detail. In normal larval blood 85 per cent. of the cells are proleucocytes and 15 per cent. are leucocytes. In certain experiments they injected number of 3 to 5-day-old larvae with 1/160th of a cc. of a thick suspension of a human strain of *Staphylococcus* The injection was made at the caudal end of the larvae directly into the blood. They observed a considerable decrease in the number of proleucocytes and a simultaneous rise in the number of leucocytes, accompanied by a pronounced phagocytosis by the last-named cells. At the end of twenty-four hours all the inoculated larvae had died of septicaemia, and blood cells were no longer demonstrable. In a subsequent experiment the larvae were immunized with a heat-killed culture of the same strain of *Staphy*

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lococcus twenty-four hours before the injection of virulent cocci. In this experiment after 24 hours ninety-nine per cent. of the total blood cells were leucocytes, and all free bacteria had vanished from the larval blood. The larvae survived two days after this experiment It appears as if phagocytes are important in determining immunity in these simple forms of life.

It is apparent from the foregoing remarks that our scientific knowledge of foul brood and other brood diseases of the bee is by no means in a satisfactory state, and this is especially true of European foul brood. In the case of this disease the controversy as to what is the infecting bacterium must be settled. There is also the question of bacterial diseases of the brood which are of rarer occurrence and of ascertaining whether they are of much practical importance and what the infecting organisms are. In England the distribution of the different types of foul brood must be determined. Again, more effective measures of scientific control of the spread of foul brood infection are badly needed. It is hoped that some of these problems may be solved at this Station.

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Note added August 20th, 1934. Since this paper was read, Burnside (J. Econ. Entomol., *27*, 656, 1934) has published the results of his investigations on European foul brood. In his paper he states that there is, in all probability, no such organism as *Bacillus pluton*, and that it is merely *Streptococcus apis*. He describes experiments in which he succeeded in producing foul brood with both *Streptococcus apis* and *Bacillus alvei*. He also believes that European foul brood is caused by a pleomorphic organism which may assume the form of *S. apis* or *B. alvei*, but the evidence which he presents in support of this hypothesis is rather inadequate.

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