The "Isle of Wight Disease": The Origin and Significance of the Myth

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THE "ISLE OF WIGHT DISEASE": THE ORIGIN AND SIGNIFICANCE OF THE MYTH

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by Dr. L. BAILEY

THE REASON I have chosen the old and celebrated subject of the 'Isle of Wight disease' (IOW disease) for this lecture is because I believe it has established, more than anything else, a common but false attitude of bee-keepers and research workers towards diseases of bees. I have called the IOW disease a myth—not derisively, but according to an Oxford English Dictionary definition, meaning 'a primitive explanation of a natural phenomenon'. Like all myths, however, it has a simple appeal, which can easily suppress doubts raised by awkward facts. I propose to examine these facts in the light of present knowledge and I hope that my conclusions will help future bee-keepers to avoid the wasted efforts that belief in the IOW disease engendered and which still continue.

The IOW disease was alleged to affect adult bees and was said to have reached epidemic proportions in the British Isles on at least three occasions between 1905 and 1919. The main symptom usually given was very many bees crawling and dying on the ground outside their hives. The first major outbreak was said to have been in the Isle of Wight in 1906. The disease was then believed to have spread to the mainland in the south of England in 1909 and, according to Herrod-Hempsall (1937), by 1918 'not a bee-keeping district in Great Britain was free from scourge [and] . . . eventually the parasite [*Acarapis woodi*] invaded Ireland as well as European countries'. This is the common belief, and it is typified by a statement about *Acarapis woodi* issued within the last four years by the American Beekeeping Federation which says 'This [IOW] disease is considered by apiculturists in the countries where it does exist to be far more serious than American foulbrood'.

There is no doubt that some bee-keepers lost most of their bees in the Isle of Wight in 1906, which, apparently, was the worst of two or three consecutive bad years. It was then assumed, however, without any evidence, that the cause of the losses was an infectious disease. This

idea was then promulgated by sensational but uninformative articles, which I have read, in the *Standard,* a now defunct London morning paper, and in several provincial newspapers. This publicity, as usual, helped to fix the belief firmly in the public mind.

The first professional investigation was made by Imms in 1907. He examined bees in the Isle of Wight which were said to have the IOW disease and found they had 'enlargement of the hind intestine', which Imms, who at the time seemed unfamiliar with bees, thought abnormal. His diagram, however, represents very clearly the intestine of any normal bee that has been long confined to the hive. Malden (1909), the next professional investigator to visit the Isle of Wight, pointed out that the intestine of healthy bees confined to hives for a few days very closely resembled those of diseased bees. He had accepted the idea that there was an infectious disease, however, and he obtained a colony, said to have the IOW disease, and confined them in a 'warm room' in a muslin cage on 27 June 1908. By 10 August, he said, they had ceased to fly; and the colony was dead by 26 October. To keep them for so long under such conditions, however, would have been difficult had the colony started in the best of health. Malden examined minutely the anatomy of bees said to have the IOW disease, including their tracheae and air sacs, but all he found were more bacteria in the gut of diseased bees than in healthy ones: he failed to show that these micro-organisms were pathogenic. Bullamore (1922) also pointed out that bees prevented from flying sometimes develop symptoms, described as crawling with bowel distension, which are indistinguishable from the IOW disease. Now in 1906, according to newspaper accounts, there was a disastrous April for agriculture with frost (-5° C in London on 2 May) and snow *after* a very early spring which had been hot enough to draw crowds to the seaside resorts. This very unusual weather might have accounted for trouble with bees, which, being suddenly confined to their hives, possibly with freshly gathered nectar, may well have become very dysenteric. The only photograph of bees suffering from IOW disease I have been able to find was taken in 1911 by G. W. Judge. A print is in the Bee Research Association library, and it shows what appears to be a colony with severe dysentery—not a very unusual event after winter even today.

As far as I know, there is no more recorded evidence about the

disease in those early days in Britain. There are, however, descriptions of the death of numerous colonies between 1901 and 1905 in several other countries, including Italy, Brazil, Canada, and the United States, and all the bees had symptoms exactly like those described for the IOW disease in Britain (see Bullamore, 1922). One incident was the loss of 20,000 colonies in Utah, with the bees dropping to the ground, mounting blades of grass and twigs with great difficulty and then dying. Had this disaster occurred in Britain at the same time it would have been attributed unreservedly to the IOW disease. In recent years there have been further reports of large scale losses of bees in many parts of the world, particularly in Australia and South America, with bees crawling and dying in front of their hives and with no known parasite present in sufficient numbers to be the cause. Poisonous nectar or pollen is suspected, but this trouble would certainly have been classified as the IOW disease by bee-keepers in Britain fifty years ago.

There are all kinds of possible reasons for the death of bees, apart from infections, and there is little doubt that bees dying of non-infectious diseases were often included in the IOW disease casualties. Imms (1907) found the most successful remedy was 'feeding cane sugar' and in Cumberland, where IOW disease was said to be serious in 1915 and 1916 with between 5 and 20% of colonies 'affected' according to a report of their bee-keepers' association at the time, it was said that '1916 was a poor season, many colonies were insufficiently provided for winter, and sugar was practically unobtainable'. I rather think, therefore, that starvation was often to blame for some losses included in IOW disease casualties.

So-called treatments for the disease must have killed numerous colonies. One official report said diseased bees were short of nitrogen, because their distended rectums contained much pollen. This followed the mistaken belief that adult bees usually did not need protein food and, when they did, then pollen was unsuitable; so it was recommended that all pollen combs should be removed in autumn and the colonies fed beef extract to make good their supposed nitrogen deficiency. This would certainly kill or seriously cripple any colony because pollen is essential for adult bees and beef extract is poisonous for them, probably because of its salt content. The ruinous idea of removing so-called 'pollen-clogged' combs persisted, however, and was widely

practised for many years. Other remedies that were recommended were phenol, formalin, 'Izal', sour milk, salt and other lethal chemicals—all to be fed in syrup to ailing colonies and as preventives to healthy ones. Other reports describe colonies which clearly were crippled with foul-brood; and poison sprays were certainly used, probably with less consideration for bees than they are today. After perusing all the British bee journals from their beginnings until about the 1920s I find it requires very little imagination to see that many bee-keepers eventually attributed all colony deaths, which had no other obvious cause, to the IOW disease. Some bee-keepers were sceptical; they pointed out that the symptoms were not specific: for example, they resembled those of the fairly well-known disease called paralysis, for which there was no known cause, but which had been described from time to time at least half a century before the IOW disease. The final opinion of Rennie (1923), a co-discoverer of *Acarapis woodi* who had much experience with bees said to have the IOW disease, was that 'under the original and now quite properly discarded designation "Isle of Wight disease" were included several maladies having analagous superficial symptoms'.

The publicity had won long ago, however: by about 1912 almost everyone had accepted the idea that the IOW disease was infectious and thought that only the identity of the one supposed infectious agent was needed. This encouraged a burst of activity which culminated in the discovery of *Acarapis woodi* in December 1919 by Rennie *et al.* (1921) who at first considered this mite to be the cause of the disease. Their own results did not support this, however; on the contrary they made it clear that *A. woodi* was widespread, occurring in many normal colonies. They found it occurred in all colonies they believed to have the IOW disease; but I shall return to this point later. The significance of their report was that it showed many bees from both diseased and healthy stocks behaved and flew normally, even though they were infested with mites and some of them had pronounced blackening and hardening of their infected tracheae. Normal nectar- and pollen-gathering bees from stocks in which 'crawling and other symptoms were well established' were found heavily infested, 'quite as badly as anything ... observed in crawling bees'. In fact 'flying workers were frequently more heavily parasitized than were bees of the same stock

which were unable to fly'. This evidence shows that *A. woodi* was not obviously pathogenic and certainly could not have been causing the observed sickness which was considered to be the IOW disease. It seems the mite was then much as it is today—its only significant pathological effect being to shorten very slightly the life of bees, but usually causing no obvious sickness in spite of the abnormal appearance of infested tracheae (Bailey & Lee 1959). Why *A. woodi* became so firmly established as the cause of the IOW disease in the face of this evidence is hard to understand. It may have been partly because the size of the parasite, its incidence, and the appearance of infested tracheae were startling; but its restricted habitat in the thoracic tracheae, which are neither easy to see without a special dissection technique, nor of obvious interest, make its late discovery understandable. Perhaps some thought it was the last adult bee parasite that would be found, and as the other parasites known at the time—*Nosema apis* and *Malpighamoeba mellificae*—did not seem particularly dangerons, then *A. woodi* must be the cause of the IOW disease. This ignores the possibilities of other pathogens, especially of bacteria and viruses, which we now know to exist and cause diseases with symptoms resembling those reported to be of the IOW disease.

The confusion of thought about *A. woodi* is illustrated by the account in the book by Herrod-Hempsall (1937), which perhaps best reflects the popular beliefs of those days. He stated that *A. woodi* spread from the Isle of Wight to European countries after 1918. Yet in the same account he wrote 'there is little doubt that [*A. woodi*] has infested the honey bee in a number of eountries for several centuries'. I suggest his second statement is nearing the truth but I should guess the mite has infested honey bees for several thousand millenia: it almost certainly has no other host and it is extremely closely adapted to lead a complicated life with honey bees. It is widespread and has been found in India and Africa as well as Europe, Russia and S. America. It was found in France and Switzerland in the winter of 1921–2 and even in Tula, south of Moscow in 1922 (Perepelova, 1927), which is most remarkable progress if it started from England, especially considering the difficulties there must have been in transporting bees during World War I and the Russian Revolution. *A. woodi* is unknown in North America and Australia, but so it probably would be in Britain if we

enjoyed their comparatively regular and abundant nectar-flows. For it is in these conditions that mites decrease in number, quite possibly to become extinct (Bailey, 1961). And it is in the opposite circumstances—in poor seasons when colonies are having a lean time—that *A. woodi* multiplies and spreads: these were the seasonal conditions in which Rennie and his colleagues discovered the mite, and his colonies that had suffered the worst conditions developed the largest mite infestations. Mite infestation increases in colonies as a *result* of their poor circumstances: it then merely adds to these, occasionally being the last straw, but usually it dwindles dramatically when the environmental conditions for bees improve enough to make them forage actively (Bailey, 1958).

The striking feature of bees is the resistance they have towards the multiplication and spread of all their common parasites. Bees and parasites probably evolved to their present state long ago and they now survive in spite of each other. The parasites are by no means harmless, however; bees are better off without even the least harmful ones and when the mechanisms that suppress their spread in normal colonies break down then the parasites can overwhelm and destroy the colony. But they are usually effectively checked. The result is that infections, particularly of adult bees, are common and yet obvious sickness caused by them is rare. If this is not appreciated, however, as in the days of the IOW disease and all too often today, then bees not obviously sick are thought to be free of parasites; conversely when a visibly diseased colony is found with a recognizable parasite, the disease is attributed to that parasite. It is further assumed that should a normal colony become infected with the parasite a serious disease will be caused; also that when a colony dies without obvious physical cause it must have been killed by a parasite. Even von Frisch in one of his popular books (von Frisch, 1954) remarks that honey bee parasites are 'those horrid creatures that spell disease and doom to the bees'. This is sensational and has considerable impact on bee-keepers, but it is very misleading. Not only is obvious sickness caused by infections in adult bees rare but it is very difficult to cause by deliberately infecting colonies. It has been achieved only with *Nosema apis* and even with this happens only when colonies are heavily infected in autumn when bees are least able to clear it up (Bailey, 1955). The mechanisms by which bees resist

infection differ for each type of parasite, but for adult infections it is generally a dynamic process depending on the normal actions of colony life, with infections being flushed away by the normal short life and expendability of individual bees. Brood infections, which cause diseases that are often easy to see, unlike diseases of adults, are also often naturally suppressed. Even the spread of *Bacillus larvae,* potentially the most destructive of all bee parasites, is opposed in many ways. Most larvae are innately immune to infection; only those less than about a day old are easily infected and adult bees detect and eject most of these before infective spores of the bacillus have formed. Contrary to common belief, it is difficult to cause American foulbrood unless the remains of more than about 100 diseased larvae are placed in the broodnest of a colony (Woodrow and States, 1943) or a colony is fed 500 ml. of syrup or honey containing more than about 10^{11} spores—about the number in the remains of 100 diseased larvae (L'Arrivée, 1958). The factors causing resistance to American foulbrood—the innate immunity of most larvae, the ability of bees to detect infected larvae and the efficiency of bees to clear away dead larvae without spreading their spores—all differ somewhat between different strains of bee. Rothenbuhler (1958) showed that resistance is genetically controlled, but no one factor causes a very striking increase of resistance in strains of bees that have been selected for resistance, when these are compared with ordinary bees or even with strains selected for susceptibility. In other words, all bees are almost as resistant as those that have been specially selected for resistance. No scientific evidence is available about differences in resistance between bee strains to other parasites, but I expect they have the same kind of slight variability in their generally high resistance towards them as they have towards *B. larvae.* From the practical point of view, even if all the resistant factors were collected into one strain of bee, and all the accompanying genetical faults could be weeded out, the bees would probably still only be resistant, not immune. It would probably be only a matter of time before more virulent parasites evolved and re-established the original balance.

Although it is clear that bees resist their common infections when they are in normal circumstances they are only in these when they are in the wild, surviving by the food they collect. As kept by bee-keepers, bees are not in normal circumstances, but they are probably not far

removed from them; certainly not so far removed as cultivated plants and domesticated animals are from their wild origins. We do little more than try to find them the environment in which they survive best, and then exploit them: improvements in bee-keeping are almost all for our convenience. So the natural mechanisms that oppose infections probably operate in our colonies very effectively, provided we do nothing to oppose them. But we often do oppose them by some of our bee-keeping activities. Many things we do that hinder normal activities of bees can be expected to give parasites more opportunity than usual to spread. Some bee-keeping methods directly transmit or preserve parasites. The most obvious one is to use and store movable combs. This transmits and preserves parasites that have stages in their life histories able to survive on comb—*Bacillus larvae* and *Streptococcus pluton,* which cause American and European foulbrood respectively; and *Nosema apis* and *Malpighamoeba mellificae,* parasites of adult bees —these all depend on having resting stages on combs for their survival from year to year. Bee-keepers often take such combs away from colonies before the times in the year when the colonies would normally have cleared away most of their infections, and give them back uncleaned. The abundant introduced pathogens then cannot be stopped from multiplying because that seasonal phase of colony development which normally clears most of them away has passed.

To sum up, we have no evidence that any parasite we know today has been the cause of wholesale losses of bees. Having examined the evidence, I suspect that the IOW disease was assumed to be the cause of all the losses for which there was no obvious explanation at the time. In this sense it was truly a myth. Bee-keepers saw their bees die; they knew that infections caused sickness and death in other animals and, lacking knowledge, yet feeling the need for an explanation, they assumed an infectious disease was killing their bees. They did not know of the many different parasites bees have, or of the many possible causes of non-infectious disease, or that sick adult bees all look and behave much the same to the inexpert eye whatever the cause of their disease. They were not aware of how disastrous their own bee-keeping activities could be; and these may have been excessive because it was still a time of great change for many bee-keepers from skeps to hives with movable frames. Over-enthusiastic bee-keepers were, and still are, a major hazard for

bees. Whatever the causes of their bees' misfortunes, however, bee-keepers used *Acarapis woodi* after it was discovered as the scapegoat and so it inherited the aura of the myth of the IOW disease. The significance of the myth is that lack of sufficient knowledge allowed it to develop and dominate thought and so cause much unnecessary apprehension and wasted effort. The moral, I trust, is obvious.

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