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"ISLE OF WIGHT" OR ACARINE DISEASE: ITS HISTORICAL AND PRACTICAL ASPECTS

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In 1964 we published an article by Dr. L. Bailey on the occurrence of acarine disease and "Isle of Wight" disease during the present century. He concluded that "we have no evidence that any parasite we know today was the cause of the wholesale losses of bees. Having examined the evidence, I suspect that the I.O.W. 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. . . .

We now present an account of the disease by a distinguished beekeeper who himself witnessed the epidemic from 1915 onwards, and who has reared and bred bees for over 50 years. His conclusions are very different from Dr. Bailey's, as the article shows. Ideally, science and practice go hand in hand; where they do not, each side should be given a hearing.

Introduction

In the *British Bee Journal* of 19th July 1906, appeared an Editorial under the heading "The latest bee scare". It referred to a report in the daily press concerning "a new and highly infectious disease" which, according to a statement by the local Secretary of the Hants. and Isle of Wight Beekeepers' Association, had caused the death of a high proportion of the colonies kept on the island. The Editor pooh-poohed the serious aspect of the report, largely because it was thought the disease responsible was bee paralysis—a malady beekeepers had been well acquainted with for more than 20 years. Mr. Cooper, the local Secretary who first drew the attention of the public to the outbreak on the Isle of Wight, is still with us, but the number of beekeepers who had first-hand experience of the I.O.W. epidemic, and the catastrophic loss of bees involved, is dwindling rapidly. As the years pass by after an historical event, it often becomes more and more difficult to get a true assessment of the actual occurrence. It is almost impossible to form an accurate picture of the beekeeping conditions at the time in question merely from hearsay. I witnessed the epidemic from the time it reached Devon, and I have been asked by a number of scientists engaged in research on acarine disease to record my experience and interpretation of the events which culminated in the loss of 90% of the honeybee population of this country in less than 15 years.

The outbreak on the Isle of Wight

In the *B.B.J.* for 8th February 1906, there was a seemingly casual report by H. M. Cooper, of Thorley, Isle of Wight, under the heading "Bee paralysis: is the cause known?" The question-mark was indeed appropriate. Mr. Cooper states: "During a bright, sunny day, recently, the bees on coming out of the hive dropped on to the grass by the dozen, and seemed quite powerless, so far as using their wings". He noted: "The abdomen of the affected bee is not distended in every case, while the wings are often twisted back, having the appearance of

being dislocated. . . . They kept crawling up the blades of grass and on to the alighting board only to tumble off again, and when the sun went down, and the day became colder, the bees collected in little bunches of a dozen or so in each, and soon perished. Another hive since becoming affected with paralysis has developed dysentery, and the bees are rapidly dwindling". In the issue of 19th July 1906, the Editor comments on the tendentious report made to the press by Mr. Cooper, who "estimated that quite half the bees kept in the Isle of Wight are now dead" and that "if it got a footing [on the mainland] it would probably mean the ruin of the bee industry so far as England is concerned". In the *B.B.J.* for 16th August, a further Editorial on "The latest bee scare" is accompanied by some rather disparaging remarks on the activities of Mr. Cooper, who "made himself famous by letters to the Press"; the Editor blandly assumed that the trouble was nothing more than paralysis, and on this basis gives a lengthy excerpt from the *ABC of Bee Culture* on this subject. Meanwhile the disease spread to every part of the Isle of Wight.

In the spring of 1907 Mr. E. H. Taylor of Welwyn became alarmed by the news which reached him from various sources on the Isle of Wight in the course of his business dealings with beekeepers there. At his suggestion Mr. John Silver of Croydon visited the island, in an endeavour to ascertain at first-hand the truth of the matter. Mr. Silver's findings, dated 25th May 1907, and published in the *B.B.J.* for June 6th, contain the first independent confirmation of the extent and gravity of the outbreak. The same issue had an Editorial on the "Bee-epidemic in the Isle of Wight". Roused at last from his apathy, the Editor reprints a long extract from the annual report of the Hants. and Isle of Wight Beekeepers' Association which had been quoted in the *Daily Telegraph* on 21st May. It was entitled: "Bee-epidemic. Isle of Wight scourge", and included the first realization that "a new—or, at all events, not understood—disease among bees has spread east and west, north and south, through the island". It was now fully accepted that paralysis had nothing to do with the outbreak.

Mr. Silver says "I rode down from Croydon on my bicycle", and interviewed over 30 beekeepers on the island, "who three years ago possessed an aggregate of 326 hives of bees"; at the time of his visit "only 29 of their stocks were alive, and 14 of these were not expected to survive long". He writes: "My first impression was that the devastation seen had been caused through carelessness or inexperience, but I learned that many of the sufferers were successful and experienced beekeepers. The Rev. Leslie Morris, Brook, who has lost 28 stocks, has kept bees with success for 30 years. The Rev. John Vicars, Colbourne, who comes of a family of beekeepers, lost all his 16 stocks. The Misses Gibson, Porchfield, . . . lost the whole of their fine apiary of 23 colonies, comprising some of the best strains of bees in the kingdom. . . . Mr. H. M. Cooper, Thorley, . . . has lost 57 stocks, and although he imported three swarms from the mainland, he now possesses but five stocks, two of which are already affected. Mr. Twyman, of Newbridge (a skeppist beekeeper for 40 years), says: 'Soon after the bees swarmed last summer I found them crawling in thousands all over the place, and before I realized what had happened my 14 pots [skeps] of bees were dead!' "

In this same report Mr. Silver states: "It appears that in the summer of 1904 the first symptoms of this malady were noticed in the south of the island at Brook, Brightstone, Wroxall and Shanklin, but it was not until last year [1906] that it spread to the centre and north of the island". As to the origin of the outbreak, some ascribed it to several colonies imported from France or Switzerland about

four years previously. According to Dr. W. Malden the disease was first observed in the autumn of 1904, at a place a little south of Newport. In the following year it made its appearance in a number of villages in the neighbourhood of the original starting place. By 1906 it had spread to nearly every part of the island.

In a lecture given on November 5th 1909, Dr. Malden referred to the outbreak on the Isle of Wight as "the new disease, unknown till five years ago", and stated that there was now not a single stock left of those which had been in the island before that time. When he was there last summer he saw the only two remaining of the original stocks and these had since been destroyed by the disease. He also indicated that the disease had now reached the mainland, having appeared in Hampshire, Sussex, Berkshire, Hertfordshire, and, so he believed, also in parts of Essex. As a matter of fact, on reaching the mainland, it spread like wildfire in every direction and to the remotest parts of the country in the space of a few years.

The cause of the rapid spread

On reaching the mainland, the disease well nigh exterminated the honeybee population within 10 years. The rapid spread was brought about by two factors: the extreme susceptibility of the old British native bee to this disease, and the indiscriminate sale and movement of bees from one end of the country to the other. As a case in point, two lots of driven bees from the south of England were imported to the Outer Hebrides on 28th September 1909. By the end of the following January they showed the first signs of the I.O.W. disease—on the same day, although the colonies were six miles apart. The disease was introduced in a similar way to the Isle of Man, and by midsummer 1912 was found in Ireland. The advertisement columns of the *B.B.J.*, and the numerous authenticated reports of the spread of the disease by the purchase of swarms and driven bees, leave no doubt on this point. How it spread to the Continent—if it did so—we do not know.

The climatic conditions

Climatic conditions have an influence on any disease of bees. A good season tends to retard a disease, and may even help to eliminate it; an unfavourable summer has the opposite effect. But seasonal variations seem to play a minor role with the disease under consideration. The assumption that the outbreak on the Isle of Wight and the subsequent epidemic were brought about by a series of unfavourable seasons is not valid: the seasons from 1900 to 1920 were not more unfavourable than subsequent seasons; on the whole they were rather better.

Mr. H. M. Cooper, the beekeeper who appears to have suffered the severest losses on the Isle of Wight, reports in the *B.B.J.* of 4th May 1905: "My average yield of honey per year for the past four seasons has been rather more than 60 lb per hive, the best return from a single stock was in 1903, when one hive produced 155 lb of surplus". Apparently 1905 was too dry for the best results—a common occurrence before 1936—but yields were reported as averaging 23–67 lb per colony, depending on the district. The next year was a good season, with averages ranging from 60 to 129 lb per colony, and individual yields up to 210 lb. In our own district the maximum was 160 lb from a first-cross Carniolan.

Undoubtedly 1907 was a bad season everywhere; 1908 was good; 1909 was again too dry for white clover, but fruit bloom and lime yielded well, and the year was noted for an abundance of honeydew—our own colonies averaged 66 lb that season. The crop in 1910 was poor, but by no means a failure; 1911 was outstandingly good (probably the best summer experienced by beekeepers this century); 1912 was bad, but it was followed by a series of good seasons.

The rapid progress of the epidemic was virtually unrelated to the incidence of poor seasons. Indeed there are numerous reports in the *B.B.J.* of colonies dying of the disease within a short time of their collecting a good crop of honey. Many accounts lay stress on the fact that often the strongest and most productive colonies were first to succumb to the disease. Practical experience at the time gives no support to the view that, if we had the comparatively regular and abundant nectar flows of North America and Australia, the disease would probably be unknown in this country also.

The beekeeping of the time

Beekeeping practices and colony management undoubtedly have a bearing on disease. The time of the I.O.W. epidemic happened to be the period of transition between primitive and modern beekeeping in this country. Because of this, the view has been put forward that the unusual loss of colonies was in large measure due not to any disease, but to ignorance which led to mismanagement and over-manipulation of bees in modern hives. This assumption would have some justification if the loss of colonies had been confined to modern hives, but bees in skeps were killed by the I.O.W. disease just as readily. Indeed the disease spelled the doom of beekeeping in skeps. Before the disease reached our area, almost every farm possessed its skeps; in the village of Buckfast (which was quite small then), there were a number of cottagers with skeps. However, all native bees in the area died in the winter of 1915–1916, whether they were in skeps or modern hives.

It is also incorrect to assume that the beekeepers of that time lacked experience, skill and knowledge; they were in fact very keen and able, although some of their ideas and methods may seem strange to us.

The use of every conceivable remedy to combat the ravages of the I.O.W. disease may appear to us now to have bordered on hysteria, but these beekeepers were panic-stricken, and in their despair they grasped every glimmer of hope. We can hardly visualize now the ghastly spectacles that faced them. In a colony infected with nosema, dysentery may be present in spring, but with I.O.W. the surroundings of the hives were littered with thousands of dead and dying bees—at any time of the year, and often over long periods. Some of the remedies applied may have done more harm than good, but not obviously so, or they would not have been widely used. The addition of a small quantity of salt added to the syrup—considered beneficial in the time of Huish, a hundred years before—may well be poisonous to bees in the laboratory, but this does not prove that it is so in their normal environment, when they are at liberty to fly. But there is no doubt that, at the time of the epidemic, every unaccountable death of a colony was ascribed to the I.O.W. disease.

From what I can recall of the beekeeping of 50 years ago, I am convinced that the methods and practices in use had no more to do with the wholesale loss of colonies then than at the present time. There can be little doubt that many of

our present-day methods tend to foster nosema disease and impair the well-being of colonies.

In the days of primitive beekeeping, a beekeeper had an exceedingly limited control over the general welfare of his colonies, and occasional heavy losses must have been fairly common. We get summers now and again in Britain when the most hardy, industrious and thrifty bees cannot provide their own subsistence. Losses due to starvation or unsuitable stores must have been very heavy at times, but only in a particular year, or a period of unfavourable seasons. In any event, we have no records of wholesale loss of colonies except during the I.O.W. epidemic. If another such catastrophy had occurred within historical times, surely it would probably have been recorded, since people in years past were more dependent on bees than we are now.

The confusion

The trouble on the Isle of Wight referred to earlier was at first mistaken for paralysis—notwithstanding the fact that the specific signs of this disease were absent; the black, hairless, shiny bees. The disease called May-pest, or *mal de mai,* was next suspected, but this is essentially a malady confined to May and early June. Soiling of the hives, and crawling (resembling that of bees affected by I.O.W. disease) are the main signs of "May-pest". This disease is very uncommon in Britain, although it appears to cause heavy losses now and again on the Continent.

In 1907 Prof. E. Zander discovered the causal agent of the disease we now know as nosema. In the old literature it is more usually referred to as "spring dwindling". The signs of nosema disease and the I.O.W. trouble were to some extent similar, and enquiries were therefore set in train along these lines. By 1911 the team of scientists engaged by the Board of Agriculture came to the conclusion that *Nosema apis* was the cause of the I.O.W. epidemic. There are some similarities between the two diseases, but they cannot really be mistaken one for the other. The losses due to nosema are almost exclusively confined to the spring months; dysentery is often present, but more usually the bees of an affected colony dwindle and disappear, in severe cases until there are none left. There is no mass crawling, and any crawling that occurs is confined to June and early July; moreover it has quite a different character from that in bees affected by the I.O.W. disease. With the latter, the queen usually survived to the last; in nosema-infected colonies she is often one of the first victims, young queens being particularly susceptible and, when infected, dying within a few weeks. Dr. J. Anderson was the first to point out that the two diseases were not identical (1916).

Inevitably, many colonies suffered from nosema and the I.O.W. disease simultaneously—and some from foul brood as well. Both A.F.B. and E.F.B., as well as sacbrood and chalk brood, were far more common diseases at that time than they are today. In addition there were doubtless cases of septicaemia, and of poisoning of one kind or another. Multiple infections were probably the rule rather than the exception. But, judged on the basis of what I observed at first-hand, there is no doubt whatever in my own mind that the trouble we knew as I.O.W. disease was the primary cause of the epidemic, that it did the killing, and well-nigh swept the country clear of bees.

The scientific enquiries

Investigations were instituted as soon as the Board of Agriculture realized that the outbreak on the Isle of Wight was of an unusual nature and magnitude. Dr. A. D. Imms was put in charge of the task in 1907; from him Dr. W. Malden of the Pathological Laboratory, University of Cambridge, took over two years later. Drs. G. Smith, H. B. Fantham and A. Porter, of the same University, shortly after joined the Board of Agriculture investigations. In 1909 Prof. E. Zander published an account of the main facts in the life cycle of the parasite *Nosema apis*. Scientists in England followed up his findings, and by 1913 came to the unanimous conclusion that *Nosema* was the causal agent of the I.O.W. disease. However, some doubts prevailed, and in 1917 Dr. J. Rennie, of the North of Scotland College of Agriculture in Aberdeen, started a series of experiments, the results of which were published in 1919.

As Dr. Rennie's findings have a great bearing on the whole issue, I give the gist of them in his own words: "In a stock infected with *Nosema apis* the behaviour of the bees has in our experience been in striking contrast to that of the members of a colony afflicted with the conditions known as 'Isle of Wight' disease. . . . Loss of flight power has not been found to be a characteristic of *Nosema* infection until the insect is actually dying. In the Isle of Wight disease it is usual for this symptom to appear a considerable time before death, if the bees are prevented from sacrificing themselves by crawling and subsequent death from exposure. We have not observed *Nosema* infected bees to loiter in large numbers about the doorway nor to *gather in clusters on the ground* [my italics], as Isle of Wight crawlers do." It was also pointed out that "the frequency with which loss or death of the queen occurs in *Nosema* disease is in striking contrast to her survival in Isle of Wight disease" (Rennie & Harvey, 1919).

In November 1920, Dr. Rennie and his colleagues were able to give the first details of their discovery of *Tarsonemus woodi* (subsequently renamed *Acarapis woodi*) as the cause of the disease known until then as I.O.W., and subsequently as acarine disease or acariosis. Dr. Rennie wrote (1923): "In the course of now very numerous examinations of diseased bees from all parts of the country, I have found that *at least 99 per cent of the stocks reported to me as failed or failing from what has been popularly termed 'Isle of Wight Disease' harbour the parasite* Tarsonemus woodi."* At the time of the discovery of *Acarapis woodi* only 15 years had passed since the oubreak on the Isle of Wight in 1905. Dr. Rennie was fully conversant with the various aspects of the epidemic, and was well qualified to determine their identity. The main signs of the I.O.W. disease were not different from those of what we now call acarine.

After the discovery of the cause of the great epidemic, Dr. Rennie turned his attention to the finding of an effective cure, but with no great success. Mr. R. W. Frow, of Wickenby, Lincolnshire, was the first to succeed, but Frow's treatment had certain drawbacks. It was not until about 1952 that fully satisfactory acaricides were developed, and tested against *A. woodi*. Folbex seems to be the one now most widely used, but for a complete eradication eight applications are called for, with a week's interval between each fumigation.

In 1922 acarine disease made its appearance in Switzerland. From that time the Bee Department of Liebefeld Institute, Bern, became the main centre of research into the disease, under the direction of Dr. O. Morgenthaler (1932;

* Author's italics.

see also 1960, 1964). A series of valuable discoveries was made: the fact that resistance to the disease is determined by age (*Altersresistenz*), becoming effective when bees are about 5 days old; the discovery of one of the three species of external mites*; the external lodgement, in certain circumstances, of *Acarapis woodi* at the base of the wings (H. Schneider). The application of Folbex was also developed at Liebefeld, by Dr. H. U. Gubler and his staff (1953).

A great deal of research has been carried out in Germany: by Dr. H. Hirschfelder (e.g. 1952, 1957) at Erlangen, Dr. H. G. Sachs at Hohenheim (1952), and Dr. W. Kaeser at Celle (1952); by Dr. J. Svoboda in Prague and by many other scientists. This was partly because acarine continued to be as much of a menace on the Continent as it ever had been. Where adequate steps to combat the disease are not taken, heavy losses often result, as indicated by Dreher (1965). Moreover, remedial measures, however useful and effective, can only be of transient value. The complete extermination of *Acarapis woodi* seems impossible, because susceptible stock will sooner or later become reinfested. So the development of resistant stock appears to be the only long-term solution.

In search of resistance

I must now revert to the time that I.O.W. disease reached our part of Devon, in the spring of 1915. (The first case in Devon had come to light in the latter part of 1912.) No losses had been recorded the previous winter, but crawling was noticed and persisted throughout the summer of 1915. It was a good summer, except for July which was very wet. The weather in May and June was ideal, and enabled us to secure a fair crop from the fruit blossom and white clover. August and September were outstandingly good; it was the most favourable season for the heather I can recall. Nevertheless, the signs of I.O.W. infection persisted, and gave reason for serious alarm by the end of September. The County Bee Expert called early in October and confirmed our worst fears: in his view none of our colonies would be alive by next spring. All the other colonies in the neighbourhood did in fact perish that winter, probably 200 colonies within a few miles of Buckfast, and we were left with 16 out of the 46 we had in the autumn; the colonies that survived were either Italian or of Italian extraction.

The spring of 1916 was fairly propitious, but from mid-May until the end of June the weather could not have been worse. However, the earlier good spell enabled us to build up our colonies to their original number, with the help of queens imported from Italy. From early July the weather became progressively more favourable and we finished the season with an excellent crop of honey and our former number of colonies. These wintered without loss, and in 1917 they were increased to a hundred. We intended to operate these for honey production, but owing to the call for bees from every part of the country we decided in the spring of 1918 to devote our entire resources to meet this need. Many hundred nuclei were sent from our apiary to different parts of the country in 1918 and 1919. The Ministry of Agriculture initiated its own restocking scheme within a few months of the conclusion of the war.

Among the Italian queens raised by us at that time there was one, a first-cross, of outstanding performance, and she became the foundation of our present strain. The performance of this cross was so far ahead of the pure Italian strain that we

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* *Acarapis externus* was discovered by H. Homan, Marburg (1933); *A. dorsalis* by Dr. G. D. Morison, Aberdeen (1931); *A. vagans* by H. Schneider, Liebefeld (1941).

felt justified in running the risk of a recurrence of the disease. We had in fact not long to wait for the tell-tale symptoms, though now manifested in an attenuated form—anyway for the moment. The next season (1920) was not particularly good; 1921 was exceptionally favourable, and the second-generation hybrids surpassed our highest expectations. Two of the best queens, sisters, were selected as breeders for use in 1922.

By now it was widely accepted that the Italian bee displayed a measure of resistance to acarine disease, and indeed the Ministry of Agriculture's restocking scheme was based on this assumption. We were, however, unaware of all the implications of this factor of resistance or of susceptibility. It happened that the offspring of one of our two 1922 breeders proved highly resistant, and the offspring of her sister extremely susceptible. To determine the effect of the drone parentage, half of each batch of queens was mated with pure Italian drones, the other half with drones from second-cross or sister queens. By chance all 36 susceptible queens were introduced (in the early part of summer, after a ten-day period of queenlessness) in an out-apiary of 40 colonies, the 4 remaining colonies being requeened at the same time with queens raised from the resistant breeder. We were completely unaware at the time of the factor of resistance or susceptibility possessed by these two breeders.

By the following spring the majority of the colonies with the susceptible queens were dead, and the others in bad condition. The four colonies with the resistant queens wintered perfectly, without a trace of acarine—as did all our other 120 colonies, dispersed in three further apiaries. The colonies with susceptible queens that were still alive, and gave hope of a possible recovery, were requeened at the end of March with resistant queens, of which we fortunately had a good number in reserve. Three of the colonies with susceptible queens looked as if they would make a full recovery without a change of queen, but did not do so (although 1923 was a good season), and in July they were requeened.

It was apparent that we had by accident made an important discovery—of hereditary susceptibility and resistance to acarine. As the two breeder queens were sisters, the results we had observed were startling, and from the breeding point of view of great consequence. The drones had had no immediate influence on resistance or susceptibility, and this fact was later substantiated many times, by us and by other observers. A single instance of apparent hereditary resistance or susceptibility was of course no proof that it existed; a number of similar cases were needed to put the matter beyond doubt. We had not long to wait for a second and equally interesting example.

In the summer of 1924 I obtained some queens from North America; the bees were of a bright golden colour, and I was assured the strain was outstandingly good. As I was greatly impressed, we raised a fairly large number of queens of this strain, and imported a further supply from the same source in 1925. This was one of the outstanding seasons of the century, and the American strain gave full satisfaction in every way but one: it proved highly susceptible to acarine disease. This susceptibility did not manifest itself fully until July 1926, when it showed up in such an extreme form that we had no choice but to abandon the strain. Again, there was no difference in susceptibility between the offspring of the imported and home-bred queens, nor did the drone parentage affect the issue.

I was always on the lookout for chances to explore the possibilities of cross-breeding, and in 1930 raised a batch of queens from a pure French breeder-queen of proved performance and economic value. These queens we crossed with

drones of our own strain: 83% of the young queens were highly susceptible; the others were partially resistant. The partially resistant first-cross was outstanding on the basis of performance, so we decided to breed from the best of the two first-cross queens. On the maternal side there was a degree of susceptiblity as yet undetermined, and on the paternal side a high resistance. Within about seven years I was able to develop a highly resistant new combination. I was simultaneously working on a subsidiary line of the same cross, which attracted my attention on account of an unusual series of good qualities. The colour was a deep golden I had not seen before; what was far more important, this line was extremely gentle and exceptionally thrifty; it did not swarm, or use propolis; it equalled almost any other race or strain then known to me in honey-getting ability. These qualities (apart from honey production) were all the more remarkable because the French bee is known for its bad temper, and excessive swarming and propolising tendencies; and in place of the black colour we had a new golden hue. Alas! this line proved so susceptible to acarine disease that we abandoned it, notwithstanding its great economic value in other respects.

I was also aware of the good qualities of the native British bee, and its usefulness and potential value in cross-breeding, and I tried to get hold of it again—if it still existed. About 1942 Mr. J. Tinsley, then in charge of the beekeeping section of the West of Scotland Agricultural College, Auchincruive, thought he had discovered a few colonies on an island off the west coast of Scotland, but further tests showed that this was not so. In the summer of 1945 I secured some queens from a secluded place in the extreme west of Ireland, which were claimed to be pure natives. This strain proved to be like the old native British bee in its susceptibility to acarine; we raised a batch of queens but, although they were mated with drones of our own strain, the first-cross as well as the original stock succumbed to acarine.

In our search for material for our cross-breeding experiments, queens of every known race have been introduced into our apiaries since the end of the last war. Needless to say, watch has been kept for any signs of susceptibility to acarine*. Our accumulated evidence points to the fact that all strains (and varieties) of the *intermissa* group, to which the European black bees belong—including our old native British bee—are susceptible. Carniolans are a little less so, manifesting clear signs of susceptibility only in adverse or unusual circumstances. So far we have not come across any Italian strains bred in North America which are not susceptible. A few years ago we imported 30 queens of a strain we had not tested previously. As their performance was not up to expectations, the queens were eliminated, except for the best one of the 30: her colony suddenly succumbed to acarine disease at the end of September 1965, after the queen had been in the hive for two and a half years. Our general experience over the past 45 years is that a great many of the American-bred Italian strains are highly susceptible. A letter received about two years ago from the late M. P. Horguelin in France indicated that he had obtained results apparently similar to ours. Why there should be this lack of resistance we do not know, and all American strains are not equally susceptible.

In 1958 we imported two queens from the U.S.A., of the strain we had tested

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* The findings of Dr. L. Bailey (1961) were based on second- and third-generation hybrids, raised from a Buckfast-Greek cross, not on queens bred from pure Buckfast stock, as he assumed.

between 1924 and 1926, primarily to ascertain whether this strain was still as susceptible to acarine. The queens arrived in July, and were introduced at the home apiary where I could keep them under close supervision. It was soon apparent that this strain still possessed all the good qualities it was noted for, and that the queens were now more prolific. The colonies wintered normally and built up well in spring, but early in July 1959 one of the colonies appeared listless. On the evening of July 22nd (after a heavy honey flow had been in progress for some weeks), I took a stroll around the home apiary, and found the lawn in front of one of the two hives thickly covered in bees to a distance of about 5 feet—a clear case of mass crawling, with all the characteristic signs of acarine disease. I immediately dispatched about 20 bees to the National Agricultural Advisory Service at Rothamsted; the verdict from their examination was: "Every bee heavily infested with acarine; no indication of nosema or amoeba". The mass crawling continued for some days; after about 60% of the bees were gone the colony seemed to pick up again, only to succumb early in winter. The other colony did not show the disease until the following spring, but then collapsed rapidly with all the characteristic manifestations of severe acarine infestation.

At the time of the first mass crawling in July 1959 we had an additional 48 colonies at the home apiary, but none showed any visible indication of acarine infestation. The season in 1959 was moreover exceptionally good, from early May until the conclusion of the flow from the heather, with an average surplus of 172 lb per colony. But these favourable conditions did not prevent severe acarine infestation.

Immunity and resistance

I have cited cases of extreme susceptibility; and of extreme resistance—under identical environment, climate and honey-flow conditions. There can be little doubt that in every case we were dealing with hereditary dispositions. A close study of the honeybee and its way of life reveals an innate power opposing infection, not only acarine, but all others that bees are subject to. However, we should not confuse resistance with immunity to a disease, such as is shown by plants, particularly where self-fertilization takes place. In the honeybee, where the viable unit is composed of a great number of individuals, and where complete genetic purity denotes degeneration and incapacity for survival, immunity is non-existent. But a high resistance can meet the full requirements of the practical beekeeper.

On the Continent an idea is current that a good honey season, causing a rapid change in the population of a colony, is the most effective way of eradicating acarine disease. Another view held is that there is no recovery or spontaneous cure. Either of these views may hold good in certain circumstances, but not in every case. Between the two extremes of susceptibility and resistance I have cited, there may be every possible graduation, even in the same colony. A spontaneous cure would seem to be impossible, even in the most favourable circumstances, where there is extreme susceptibility. But with partial resistance there may be recovery, or complete disappearance of infection, depending on the degree of resistance and whether other circumstances are favourable.

The existence of partial resistance has made the study of the disease very confusing; the problem is made more complex by the fact that we are dealing not only with individual bees, but also with colonies of say 60 000 members, which represent a genetically mixed assembly. A proportion of these bees, taking an

active part in the normal work of a colony, may be infested with *A. woodi*, without any apparent signs of the disease. Infestation will probably shorten the life of the bees affected, but may have little or no bearing on the performance of the colony. This is equally true in nosema disease, yet its pathogenicity is not questioned on that account.

We do not know what factor or factors form the basis of resistance to acarine disease. There is much research in progress in Czechoslovakia, France, Germany and Italy on this problem. Resistance does not appear to be based on structural characteristics of the thoracic spiracles, obstructing in some way the entrance of the mites (though age-resistance is caused by a stiffening of the hairs surrounding the spiracles). Both laboratory tests and practical experience show that, in very adverse environmental conditions, hereditary resistance and age-resistance may break down, as would be expected from general considerations. Up to a certain point we know what causes *susceptibility* in bees less than five days old, but we do not know what can *prevent* infestation before age-resistance comes into operation. It may well be some factor in the behaviour of the bees or their reaction to the mite.

The origin of acarine disease

This question has puzzled scientists and beekeepers alike since the first outbreak on the Isle of Wight in 1905. It is widely assumed that *A. woodi* has been in our midst since the beginning of time. If this were true, we would have to assume that the British native bee, due to some cause or another, suddenly became highly vulnerable to the mite. If it were not true, the native bee would presumably have been exterminated in ages past. We have no records of an epidemic similar to that which originated on the Isle of Wight, nor do we know of one of equal magnitude anywhere else in the world. In the past 40 years there have been sporadic reports of heavy losses in the southern and western sections of the U.S.A., but some of these have been exaggerated, and they are not of the same character as the I.O.W. epidemic.

If *A. woodi* had been common in Europe from early times, it would surely have found its way to North and South America, as well as Australia and New Zealand. Every other known bee disease is found in these countries, presumably imported with some of the original bees brought from Europe. This is also true, at least for North America (Eckert, 1961), of the external mites *A. externus, A. dorsalis* and *A. vagans*, which bear such a close resemblance to *A. woodi* that they could not at first be distinguished from it. It has been argued that perhaps *A. woodi* was imported to these countries, and died out in time owing to some climatic influence. This is a hypothesis to which I might have also subscribed, had I not observed evidence of acarine disease in sub-tropical North Africa and the Iberian Peninsula; this evidence was confirmed by the N.A.A.S. at Rothamsted after examination of samples: "all the bees infested with acarine; no evidence of nosema or amoeba".

Acarapis woodi probably exists today in every apiary of any size in the British Isles. This does not hold good on the Continent; indeed it has not been found in any of the Scandinavian countries, the far north of Germany, or the south-eastern parts of the Balkan Peninsula. It seemingly did not reach the Iberian Peninsula until about 1948, but has since caused heavy losses, so that there has been a decline in beekeeping in southern and eastern Spain. In Central Europe there are extensive areas free of acarine. In Switzerland it is mainly confined to

the western and northern sectors, and there has been no occurrence in most of the south-eastern Cantons. But in the Cantons where the disease has occurred, it has been necessary to treat about 12 500 apiaries with acaricides between 1953 and 1959. In Central Europe, outbreaks in sections previously free from the disease have always been traced to the importation of diseased swarms or colonies. The incidence in the Congo and in India was almost certainly due to importations from Europe. There was a similar occurrence recently on the island of Réunion in the Indian Ocean. An outbreak in South America was clearly caused by the importation of queens from Europe.

Acarapis woodi certainly did not develop spontaneously; it seems exceedingly improbable that it evolved by a mutation, or a series of mutations, from one of the external mites. The most plausible explanation is that it was brought into this country in some way or another, from some place not yet identified. I recently discussed this hypothesis with an acariologist in the U.S.A.; he also favoured this view, and cited a somewhat similar occurrence with a mite affecting reptiles. From the ecological requirements of this mite it was assumed to come from the Malayan Peninsula, with a steamy hot climate, but it was found to come from an apparently unlikely habitat: the Nile valley.

Conclusion

The I.O.W. outbreak was no ordinary epidemic, brought about by a period of unfavourable climatic conditions. Acarine disease causes as severe losses now as it ever did in the past, whenever highly susceptible stock is brought in contact with it. The main signs of the so-called I.O.W. disease were no different from those of acarine disease today.

Not all strains of bees are equally susceptible to *A. woodi*. Bees in use today are endowed with varying degrees of resistance. Nature, by weeding out the more susceptible stock, has to some extent selected and bred for resistance, and bee-breeding programmes towards this end have made their own contribution. Acarine disease is as great a menace as it has ever been, where common European black bees or Carniolans are kept. It would therefore be an inexcusable mistake to drop all precautions and safeguards designed to prevent its spreading. Measures of this kind are not "wasted effort" to anyone in touch with the practical problem. Remedial measures encourage the survival of susceptible stock, but they have their value. High resistance is clearly the long-term solution to the problem of acarine disease.

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