MICROSPORIDIOSIS, A PROTOZOAL DISEASE OF BEES DUE TO *NOSEMA APIS*, AND POPULARLY KNOWN AS ISLE OF WIGHT DISEASE

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I. INTRODUCTION

The year 1904 was rendered memorable to bee-keepers in the South of England by the sudden outbreak of a 'new' and 'mysterious' disease among adult bees. The centre of disease at first was the Isle of Wight, where great precautions were taken to prevent the spread to the mainland. Soon after this, a few cases were reported from Hampshire, but the Island remained the most stricken spot, huge numbers of bees succumbing to the malady.

Early in 1906, through the kindness of friends resident in the Isle of Wight, some dead and some infected, but still living, bees came into the possession of the present writers. A joint investigation was begun, and has continued up to the present time. We found that the dead and sickly bees received from our friends contained the minute Microsporidian parasite since named *Nosema*

apis. Some of the bees contained only young stages of the parasite, while in others, mature, very resistant forms of the parasite, the spores, were present. Experimental work to prove the pathogenicity or otherwise of the Microsporidian was at once commenced. The results will be given later (pp. 154-157), but it will suffice here to say that they fully confirmed the suspicions aroused by finding extensive destruction of the gut-epithelium of the bees due to the action of the parasites.

From the commencement we were hampered by the lack of suitable material, for fear of spreading disease to other districts was rampant among the owners of infected stocks; hence our experiments had to be undertaken on a somewhat small scale. This was not altogether a disadvantage in some respects. More detailed observations of the habits of infected insects can be made with a few insects, where the presence of a large number would cause distraction or distribution of attention, and so tend to confuse the issue. As a rule, spores of the parasite from one set of experiments had to be used at once for further cross-infection experiments.

In 1907, again we found *Nosema apis* in bees from another part of the Isle of Wight, and during 1908-10 found the organism in bees obtained from various districts in Surrey, Hampshire, Middlesex, Hertfordshire, Cambridgeshire, and Devon.

At the commencement of the spring of 1911, there was a great outcry among bee-keepers, who, on opening their hives, found dead bees only. Public comment in the press was a daily occurrence, and plaints of 'no bees, no fruit,' 'a fruitless year,' etc., appeared on all sides. Suggestions had been made by various investigators of the disease, under the Board of Agriculture, that in one case blood-poisoning, and in another a bacillus, was the cause of the trouble. Both cases had broken down. Having revised the accumulation of our experiments, examinations of bees, hives, combs, honey and excrement, we decided to bring forward our results. On April 4, 1911, therefore, microscopic preparations of *Nosema apis*, together with infected bees and combs, were exhibited by us before the Zoological Society of London, where an outline in brief of our results was announced and published. Since that time, owing to becoming members of the Board of Agriculture's Enquiry, we have

examined very many bees, but the result has been merely to confirm all our previous work, without adding any new facts regarding the parasite to our previous knowledge—a disappointing occurrence. On becoming members of the official Enquiry we delayed publishing our results *in extenso*, but we propose in these papers to give an account in full of our pioneer researches, confirmed, as they are, abundantly by subsequent investigations, both of our own and of our colleagues on the Enquiry under the auspices of the Board of Agriculture.

We would take the opportunity here of heartily thanking all those who have kindly helped us in obtaining material and have given us facilities for examining hives, surveying gardens and fields, and conducting general observations in the open country.

We may mention that our researches were begun in the Zoological Research Laboratory of University College, London, and continued in the Quick Laboratory, Cambridge, and in the Liverpool School of Tropical Medicine. We wish to thank the heads of these laboratories for the kindness and courtesy extended to us during our investigations.

We propose now to arrange our results in three papers, each complete in itself, the first dealing with the relation of *Nosema apis* to disease, the second concerned with the morphology and life history of *Nosema apis*, while the third relates to the methods of dissemination of the disease as we have observed them in the open—the only methods of practical value. The present paper, then, is concerned with the relation of *Nosema apis* to disease, together with such general observations on bee-structure and bee-life as are necessary for a ready understanding of the remainder.

II. OBSERVATIONS OF THE HABITS OF BEES INFECTED WITH *NOSEMA APIS*

In order, if possible, to determine definitely the symptoms of disease due to *Nosema apis*—we named the disease Microsporidiosis, as it was due to a Microsporidian parasite—we carefully noted all diseased bees sent to us, and all bees infected artificially, as well as bees belonging to dwindling colonies. In order to avoid errors of interpretation, detailed studies of normal bees were made at the same time.

Naturally, our observations on the subject coincide to some extent with those expressed either separately or in groups by bee-keepers, and can only be summarised here. The question of symptoms is rendered very difficult because the bees vary enormously among themselves, so that there seems, at present, no one great outstanding symptom common to all.

In most cases with which we were personally acquainted, examination of the ground and grass around the hives showed quite a number of bees feebly crawling about, and evidently distressed. If a finger were very gently placed in front of one of these crawling bees, it would usually climb on to the finger and remain there without making any attempt either to fly away or to sting. One of the present writers has collected some sixty such bees in the course of ten minutes without incurring the slightest trouble from stings. However, lack of desire to sting is not universal. Bee-keepers rarely commented on the loss of this power—but one is inclined to question as to how often they had the desire to obtain evidence on this point.

When collecting diseased bees, or handling them, we have noticed that the abdomens of a fair number were distended. Gentle touching of the abdomen with a blade of grass or the finger was sufficient to produce discharge of faeces which bespattered the area round about. Again, this symptom is not constant. When bees are unable to take cleansing flights the abdomen may also be distended with waste pollen, etc. But microscopical examination shows that in the excrement of the *Nosema* victim there may be thousands of tiny, oval, shining spores of the parasite mingled with undigested and indigestible pollen, while in the case of the bee suffering from lack of chance for cleansing flights, pollen alone is present.

Bees normally defaecate when on the wing, but *Nosema* infected bees are incapable of so acting. They defaecate for preference when stationary, and hence the soil, grass, or other low vegetation, the sides of the hive, the alighting board, the very combs and honey itself, all may show splashes of faecal matter, which microscopical examination shows to be highly infective owing to the presence of spores of the parasite.

The communal life has an enormous impress on the life of the

individual bee. Even though heavily infected, a bee will endeavour to perform its work as a forager, and while attempting to start its flight from the alighting board, it is not uncommon for it to fall from the board to the earth, there to crawl laboriously until death overtakes it. Similarly, foraging bees that have left the hive early in the day may have a rapid multiplication of this intestinal parasite occurring within them. They endeavour painfully to fill their pollen baskets, but, weakened both by their load and by the course of the disease, they collapse on reaching the alighting board, fall to the ground, and die there.

On one occasion we observed a number of bees fly very slowly from a hive towards a bed of Arabis. Some hours later none had returned, but in the Arabis bed were a number of fresh bee-corpses, in which various stages, but mainly the young, growing, multiplicative stages, of the parasite were found. In this case, the owner informed us that he had lost nine hives in all, but until then had never seen a dead bee. The dwindling of other colonies without the presence of dead bees being detected may be similarly explained in all probability.

While distended abdomens, dysenteric discharge, falling from alighting boards and crawling are among the most common features observed by us, other features are sometimes present, such as a sort of paralysis and dislocation of the wings.*

Again, bees infected with *Nosema* seem unable to preserve their spotless cleanliness, and become fouled with excrement. Healthy bees attempt to cleanse them, and in so doing ingest the spores of the parasite and themselves become infected.

On some occasions an infected colony seems quite unable to produce normal wax, and the honeycomb may be very rough, mingled with faeces and undigested pollen, or sometimes it rapidly darkens, so that the comb would be considered to be several years old, whereas as a matter of fact it was newly made. We have several such combs in our possession.

Owing to the death of the worker bees, it not infrequently happens that the colony comes to an end, owing to the chilling of the brood. Measures should at once be taken to prevent access of healthy (or infected) bees to such a hive, for there are grave

* Some bee-keepers lay stress on this symptom.

possibilities of the infection of both honey, pollen, and comb, with the result of the disease spreading to the robbers by means of their stolen goods.

III. THE ALIMENTARY CANAL OF THE BEE AND THE MODE OF FORMATION OF THE DIGESTIVE JUICES

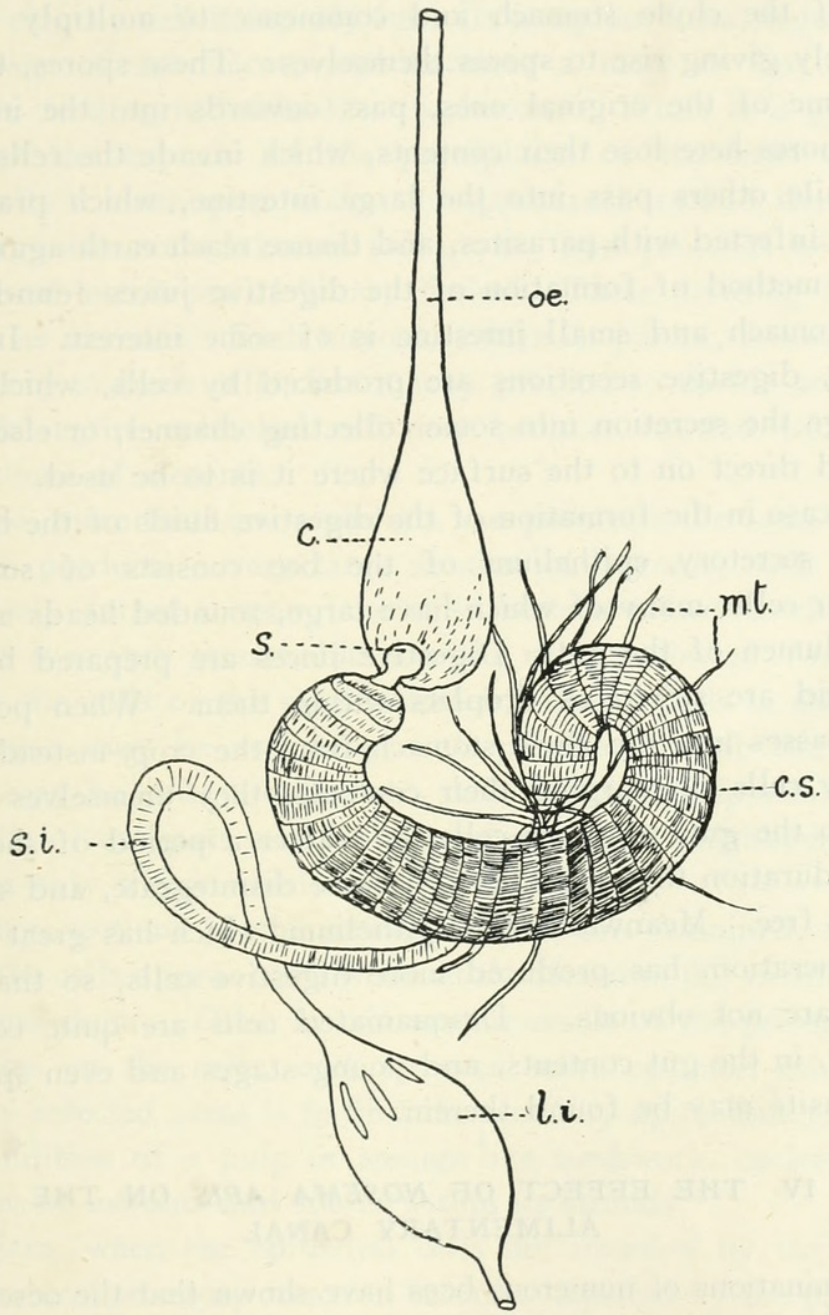
In this section it is not intended to give a detailed account of the well-known alimentary tract of the bee as such, but to indicate its structure and functions only in so far as they are related to disease due to *Nosema apis*.

The main features of the alimentary canal are shown in the accompanying text figure. A narrow oesophagus (*oe.*) passes from the mouth through the thoracic region, expanding as it enters the abdomen into the thin-walled crop (*c.*), or honey-sac as it is termed. This honey-sac serves as a storage place for pollen and nectar. The opening from the storing crop into the digestive region of the food canal is by a small aperture guarded by a complicated structure, the stopper (*s.*). The digestive region is the chyle stomach (*c.s.*), which in a normal bee is reddish in colour and somewhat firm. The inner wall is chitinous, but the chitin ridges allow access of digestive juices to pollen and nectar more readily here than in the crop. At the distal end, the chyle stomach narrows to the small intestine (*s.i.*) which, after a rather short course, widens again to a large intestine (*l.i.*). The dilated portion of the latter serves for storage of faeces, and can become greatly enlarged. It is sometimes called the colon. The actual rectal region is small. At the junction of the chyle stomach and small intestine a large number of very narrow Malpighian tubules (*m.t.*) open into the food tract, the actual perforations being in the small intestine. In the figure a few only of these are shown.

The parts of the food tract infested with *Nosema apis* are indicated in the figure by shading, and an attempt is made to indicate, by the depth of shading, the *average* intensity of infection, though this intensity may vary to a considerable extent.

Nosema apis seems to be a very specialised parasite, and at present almost exclusively an intestinal one, in the broader sense of the word. The method by which *N. apis* gains access to the organs is casual or contaminative, that is, by the mouth. The

spores, when swallowed with food or drink, pass into the oesophagus, where they become mixed with saliva from the bee's salivary glands, and then reach the crop. Here, softening of the



Text-Fig. I. Alimentary Canal of Bee. Infected areas shaded

spores, which have a tough coat or sporocyst around them, occurs. A few of the contained organisms, tiny amoebulae, are able to leave the softened sporocysts, and begin to creep about over the walls of the honey stomach; but this is not very common. After a period of softening in the crop, the majority of the spores pass

into the chyle stomach, where much more powerful digestive juices occur, and under the influence of these the majority of the spores lose their contents and the liberated parasites rapidly invade the walls of the chyle stomach and commence to multiply therein, ultimately giving rise to spores themselves. These spores, together with some of the original ones, pass onwards into the intestine. Some spores here lose their contents, which invade the cells of the gut, while others pass into the large intestine, which practically never is infected with parasites, and thence reach earth again.

The method of formation of the digestive juices found in the chyle stomach and small intestine is of some interest. In many animals, digestive secretions are produced by cells, which either discharge the secretion into some collecting channel, or else empty the fluid direct on to the surface where it is to be used. Such is not the case in the formation of the digestive fluids of the bees.

The secretory epithelium of the bee consists of somewhat columnar cells, many of which have large, rounded heads abutting on the lumen of the gut. Digestive juices are prepared by these cells, and are stored as droplets within them. When pollen or nectar passes into the chyle stomach from the crop, instead of the secretory cells discharging their contents, they themselves are set free into the gut, as entire cells, and after a period of shorter or greater duration they burst, or otherwise disintegrate, and set their contents free. Meanwhile, the epithelium, which has great powers of regeneration, has produced more digestive cells, so that large lesions are not obvious. Desquamated cells are quite common, however, in the gut contents, and young stages and even spores of the parasite may be found therein.

IV. THE EFFECT OF *NOSEMA APIS* ON THE ALIMENTARY CANAL

Examinations of numerous bees have shown that the oesophagus is usually empty, or contains only freshly ingested spores of the parasite. Its tissues remain unaffected. The walls of the crop are, in most cases, in a similar condition, but sometimes we have found the tiny germs or amoebulae, called planonts, that issue from the spore, creeping over the lining of the honey stomach, and penetrating some of its cells. The same occurrence is well seen in

the chyle stomach, where it is far more common than in the honey stomach. The amoebulae at first are very small, and creep over the surface or between the cells, and may begin to penetrate them. If one enters a cell it rapidly loses its irregular form, together with its power of movement, and becomes a more or less rounded, actively feeding individual, a trophozoite. While it is growing it lives at the expense of the protoplasm of the host cell, and gradually a clear space makes its appearance around the parasite. The organism soon becomes full-grown, and proceeds to multiply by several variations of binary fission. These dividing forms are known as meronts. Each meront, unlike its parent, is incapable of migrating to fresh cells, but it may proceed to divide actively, so that the entire epithelial cell may become crowded with several generations of meronts.

Also, more than one amoebula can penetrate the same cell, and as each produces a very large number of meronts, each of which ultimately forms a spore, the cells become crowded with spores.

But it is very remarkable how the distribution of *Nosema apis* among the cells of the chyle stomach varies. Cells in one part of the organ may be swarming with parasites, while the adjacent tissue may be quite uninfected. Also, while multiple infection of the cells of one part may be very common, the neighbouring areas contain only a few widely separated parasites.

When sections of the gut of the bee are examined, in certain areas the cells appear torn, while in the lumen in the vicinity crowds of spores occur. These lesions are the result of the pressure of the parasites on the cells, and an even more common condition in heavily infected areas is to find the secretory epithelium reduced to the condition of a pulp or sponge-like meshwork, enclosing large colonies of meronts and spores within its strands.

Again, when the epithelial cells are invaded by the *Nosema*, their natural secretory function becomes impeded by the presence of the parasite, and ultimately ceases. Consequently, derangements of the digestive processes follow, and the bee is unable to utilise such nourishment as would otherwise be obtainable from pollen or honey ingested by it. The result is that great weakness due to malnutrition sets in, and the exhausted bee falls a victim to the further multiplication of the parasite within.

Externally, when the parasites are able to reach the spore stage, the gut presents certain rather striking differences. It is much more fragile and brittle as a rule than is the gut of the normal bee, and paler in colour. When many spores are present in the chyle stomach, its reddish hue completely disappears, and is replaced by a chalky-white colour, due to the contained spores. The intestine also is much paler, and while the colour of the contents of the large intestine normally are a rather deep yellow, under the influence of the *Nosema* spores the colour changes to a paler tint, and in a few extreme cases it has been found to be a dirty white.

While the abdominal portions of the alimentary canal are the usual seats of the parasites, the diverticula of the canal seem singularly free from them. Up to the present we have found no stages of the parasite in the complicated series of salivary glands that open into the oesophagus, and on one occasion only have the Malpighian tubules been found to be infected.

But the youngest forms of *Nosema apis* are capable of adapting themselves to life in regions other than those of the alimentary tract. They may invade the haemocoelic fluid, and are able both to grow and multiply there to some extent. In some bees the haemocoelic fluid is more heavily parasitised than in others, while in many cases no parasites have been found in it. *Nosema apis* thus affords a study in progressive parasitism with increasing diffuse infiltration.

The effect of *Nosema apis* on the internal organs of the bee is given in greater detail in a subsequent paper (see p. 180).

V THE EXPERIMENTAL IDENTIFICATION OF *NOSEMA APIS* AS THE AGENT OF THE 'ISLE OF WIGHT DISEASE'

From 1906 onwards, when we first found *Nosema apis* in bees, we felt it was of the utmost importance to determine definitely whether the organism were pathogenic or not. Having made a number of observations of the behaviour of both normal and diseased bees, we devised a series of experiments, designed to follow on the lines of natural means of infection as far as possible. Control, healthy bees were kept constantly, and minute examination of all food supplied to the bees was made, for we had found that both infected combs and run honey containing spores of

Nosema apis had been bought by us in the open market. However, sources of infection other than spores supplied by us to the bees were excluded.

The experiments can be arranged in six groups. When we record that bees used by us died from the effects of *Nosema apis* during the infection experiments set forth, we mean in all cases that developing spores, or young stages, or both, of the parasite were found within the epithelium of the bees' digestive tracts—not merely that *Nosema* spores occurred in the lumen, and might only be passing through after being swallowed.

SERIES I. *Healthy bees were fed on honey contaminated with spores of* Nosema *taken from the guts of infected bees.* The bees were allowed to feed as they pleased on the infected food. Usually after two to four days they became quiet, showed a disinclination to move about, and their faeces contained spores of *Nosema apis.*

In June, 1906, twelve bees so infected all died within ten days, while control bees kept under similar conditions remained healthy.

In June, 1907, eight bees were fed on infected honey, and all had died by the end of eleven days. As before, the controls remained healthy.

June, 1910, an experiment using six bees resulted in the death of the bees in a week, the controls remaining alive for more than a month.

SERIES II. *Healthy bees were fed on honey or syrup contaminated with the faeces of bees infected with* Nosema apis. Controls were fed on pure honey or syrup. The results, summarised, were:

June, 1907. Three experiments, using ten bees each time. All died in eight days, ten days, and eleven days, respectively. Ten control bees were used each time, and remained healthy, living in the small experimental cages from twenty to twenty-nine days.

July, 1908. One experiment, using six bees for experimental feeding and six as controls. The experimental food was gross excrement in syrup, the controls being supplied with pure syrup. The infected bees all died between the eighth and tenth day, while the controls remained healthy for four weeks.

SERIES III. *Bees infected with* Nosema apis, *and in a weakened condition, were introduced into cages of healthy bees, and all were supplied with pure honey.*

Result: The healthy bees endeavoured to expel the sickly ones, who thereupon defaecated freely, fouling both their neighbours, the cage, and the food supply. Consequent on this, the hitherto healthy bees ingested *Nosema* spores, became infected, as shown by microscopical examination, and died. Control bees associated with bees dying of overwork lived the normal period.

June, 1908. Six sickly bees ('crawlers') were put with twenty-five healthy bees. Result:

5	bees	found	dead	on	3rd	day.
4	,,	,,	,,	,,	8th	,,
6	,,	,,	,,	,,	10th	,,
3	,,	,,	,,	,,	11th	,,
4	,,	,,	,,	,,	12th	,,
3	,,	,,	,,	,,	13th	,,
2	,,	,,	,,	,,	15th	,,

The remaining bees died within the month. It may be remarked that in order to distinguish the original sickly bees, they were marked with either zinc white or cinnabar powder, and so could be distinguished from their companions. A similar procedure was adopted in each of the preceding experiments. It was also found that sprinkling the bees with flour was a fairly efficacious way of marking them, for the very sickly bees made little attempt to clean themselves, and there was no risk of injury to the bees.

SERIES IV. These experiments were designed to determine whether the use of infected travelling boxes or hives would result in an outbreak among healthy bees placed therein.

Healthy bees were placed in a cage in which infected stock had travelled.

May, 1907. Eight healthy bees, caught on the wing, were placed in a soiled cage, and eight control bees, caught under similar conditions, were housed in a new cage. All the bees in the soiled cage were dead at the end of nine days, and *Nosema* spores were recovered from the corpses. The controls were all healthy for at least a fortnight, when they were killed. No parasites occurred in them. The only source from which *Nosema* spores could be obtained was the excrement in the soiled travelling box.

June, 1908. Twelve healthy bees, caught in the open country, were placed in an infected cage. Six were found dead on the

eighth day, four on the tenth, and one each on the eleventh and thirteenth day of infection. *Nosema* was found in all except the last two. Twelve controls all lived over a fortnight, except one that died on the tenth day, but no *Nosema* was found in any of the controls.

SERIES V. *Some healthy bees had infective excrement smeared or wiped on to their bodies, their controls being similarly treated with normal excrement.* Both sets of bees at once attempted to clean themselves. In so doing a certain amount of faeces was ingested. Experiments were performed in the months of May and June in 1907, 1908, 1909, 1910. The results may be summarised thus: Bees smeared with infected excrement died in from six to twelve days. The bees contaminated with normal excrement were observed from two to four weeks, during which time no deaths occurred. Our experiments confirmed the observation made in the case of sickly bees in a small observation hive. Here the excrement voided by the first set of sickly bees fouled some of their neighbours, and the latter died in a few days.

SERIES VI. *Honey in the comb from a 'dead' hive, of which no bee had survived, was put into a cage containing healthy bees.* Of these bees—twelve was a convenient number for detailed observation—ten were dead at the end of twelve days, and the remaining two had succumbed by the fifteenth day. Control bees, supplied with pure, run honey, remained apparently healthy, and continued so for three weeks. *Nosema* spores were present in the bodies of several of the bees that fed on the infected honey, and some young stages in all of them.

During the course of these experiments—sufficient to demonstrate the pathogenicity of the parasite to any thinking person—we made continuous field observations on the natural methods whereby bees can become infected. These results are set forth in detail elsewhere (pp. 197 et seq.).

Examination of bees obtained from many different localities since—often bees so long dead that they were useless for all except the gross structures, the spores—has shown that the conclusions we formed years ago are accurate. Experiments, using larger numbers of bees, have been made by others, and confirm fully our work. In this connection it may be of some interest to note that

bees suffering from 'Isle of Wight' disease, were sent to Zander* (1911, pp. 23-24), who first found *Nosema apis* spores in Bavarian bees, and that he reported that *Nosema apis* was not present in the English bees. Apparently, Zander diagnosed solely by the presence or absence of spores—the only thing possible in the case of bees long dead. Whether he received living or dead bees we know not, but it is certain that bees may die from the effects of young stages of the parasite, and so do not contain spores.

We may refer readers further interested in the details of the examination of diseased bee stocks to Tables I and II in the recent Report of the Board of Agriculture, wherein are summarised the results obtained from examining over 60 diseased stocks by Dr. Graham-Smith and the present writers in 1911.

VI. OTHER HOSTS OF *NOSEMA APIS*

In order to determine whether *Nosema apis* were a parasite solely of the hive bee, or whether it was present in other Hymenoptera, and whether these latter could act as carriers of the disease, we made as many observations as possible on insects caught at large, and also performed some cross-infection experiments. Owing to some difficulty in obtaining material, our experiments were restricted to cross-infection of mason bees (procured, again, with much difficulty) and wasps.

Mason Bees. These insects are not common in England. Those we used were obtained from a friend who had brought them from abroad. They were placed in a piece of old wall, which was screened for purposes of experiment. Twenty bees returning to the tunnels were captured to act as controls, and others were caught and examined to determine whether there were natural infection with *Nosema apis*. Such proved not to be the case. No Microsporidia were found. Bees dead of *Nosema apis* were then introduced into some of the tunnels in July, 1907. A fortnight after, no mason bees were to be seen. The wall was broken up, and some of the dead bees recovered from among the débris. Post-mortem examination of twenty of them showed that twelve contained spores of *Nosema apis*. Young stages were also found. The controls seemed perfectly healthy, and no form of the parasite was found in their bodies.

*Handbuch der Bienenkunde, Bd. II

Wasps were used in another experiment, as we had received complaints of honey losses due to wasps* robbing the hives.

A small nest of wasps that had been under detailed observation by one of us for other reasons for some time, was used for this experiment. Liberty to the wasps to fly at large was prevented by a muslin screen. After about a fortnight, no wasps left the nest, and when the latter was dug out, both adults and late brood were found to be dead. Examination was made of the wasps, and twenty worker wasps and the queen were found to contain some spores of *Nosema apis* in their alimentary tracts. Observations of young forms were precluded in most cases, as the wasps had been too long dead. When present, they were quite typical.

We have examined both mason bees and wasps from districts where bees have died 'mysteriously,' and have found *Nosema* spores in some cases, but in very few of the insects. Such spores fed to hive bees reproduced the disease. These field observations show that under some circumstances mason bees and wasps can act as carriers of *Nosema* spores. The morphology of *Nosema apis*, whether it be present in hive bees, mason bees, or wasps, seems to undergo no change whatever.

It may be of interest to add that Microsporidia have been found by us in several other British Hymenoptera. For example, we have found humble bees, chiefly *Bombus terrestris*, dying from heavy infection of a Microsporidian in the Malpighian tubules. This parasite resembles *Nosema apis*. Again, recent examination of leaf-cutting bees, belonging to the genus *Megachile*, shows that they are parasitised by another Microsporidian, both the Malpighian tubes and the digestive tract containing the parasite.

VII. PARASITE CARRIERS AND IMMUNITY

During the past six years we have made numerous examinations of hive bees from different parts of England. Many of these consisted of dead bees wherein spores only could be detected, if the parasite were present. In the early stages of our investigations every bee was examined individually, and it has been our experience sometimes to examine as many as 200 bees without finding any spores of *Nosema apis*. Then suddenly, a bee infected with spores was encountered, only to be followed by a long succession of bees without spores.

* Wasps have been observed carrying away bees dead of *Nosema* and then feeding their larvae on the corpses.

Similarly, we have examined large numbers of bees from healthy hives, in which no disease had appeared, and after many bees had been dissected and examined with negative results, one containing either young stages or spores of the parasite would occur. The infected living bees in each of such cases showed no marked difference from the other inmates of the hive, nor was any abdominal distension or weakness noticeable. They, themselves, seemed to have become immune to the disease. But such parasite-carriers may be very dangerous to the community of which they are members, should conditions alter and react unfavourably upon them. This is one of the dangers present in so-called 'recovered' stocks of bees. Some bees have survived attacks of disease, and have become partially immune to the parasite, but when unfavourable conditions set in, the parasite overcomes the resistance of its host, and the latter succumbs.

Judging from the remarks of certain Continental investigators, parasite-carriers seem to be more common abroad than in England, but the direct evidence on this point is lacking.

Regarding the acquisition of partial immunity by bees, there is some hope of an immune race arising, exactly as has happened in the case of silkworms infected with *Nosema bombycis*.

Bolle, investigating pébrine in Japanese silkworms, found that both the silkworm larvae, moths and eggs were heavily infected with *N. bombycis*, and yet the silkworms did not appear to suffer, nor was there much mortality among them. It is thought, therefore, that Japanese silkworms have greater immunity against *N. bombycis* than have European ones. It would be of interest, then, to ascertain what race of bees posseses the greatest immunity to *N. apis*.

Examination of stocks during 1911 by Dr. Graham-Smith and the present writers has shown that partial immunity existed in one special case, and we have information dating from 1907 of one hive that has remained alive and flourishing up to the present, though the hives on either side of it died out. It certainly seems that this hive has acquired some considerable degree of immunity, and it is to be hoped that more cases of the same kind will be reported as time goes on. At present the chief hope of the beekeeper seems to lie in destroying any stock that presents symptoms of disease in any form, and in ensuring complete absence of the parasite from the hive and food and from the soil in the immediate neighbourhood.

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