YOUR NEWSLETTER

FEBRUARY 1984

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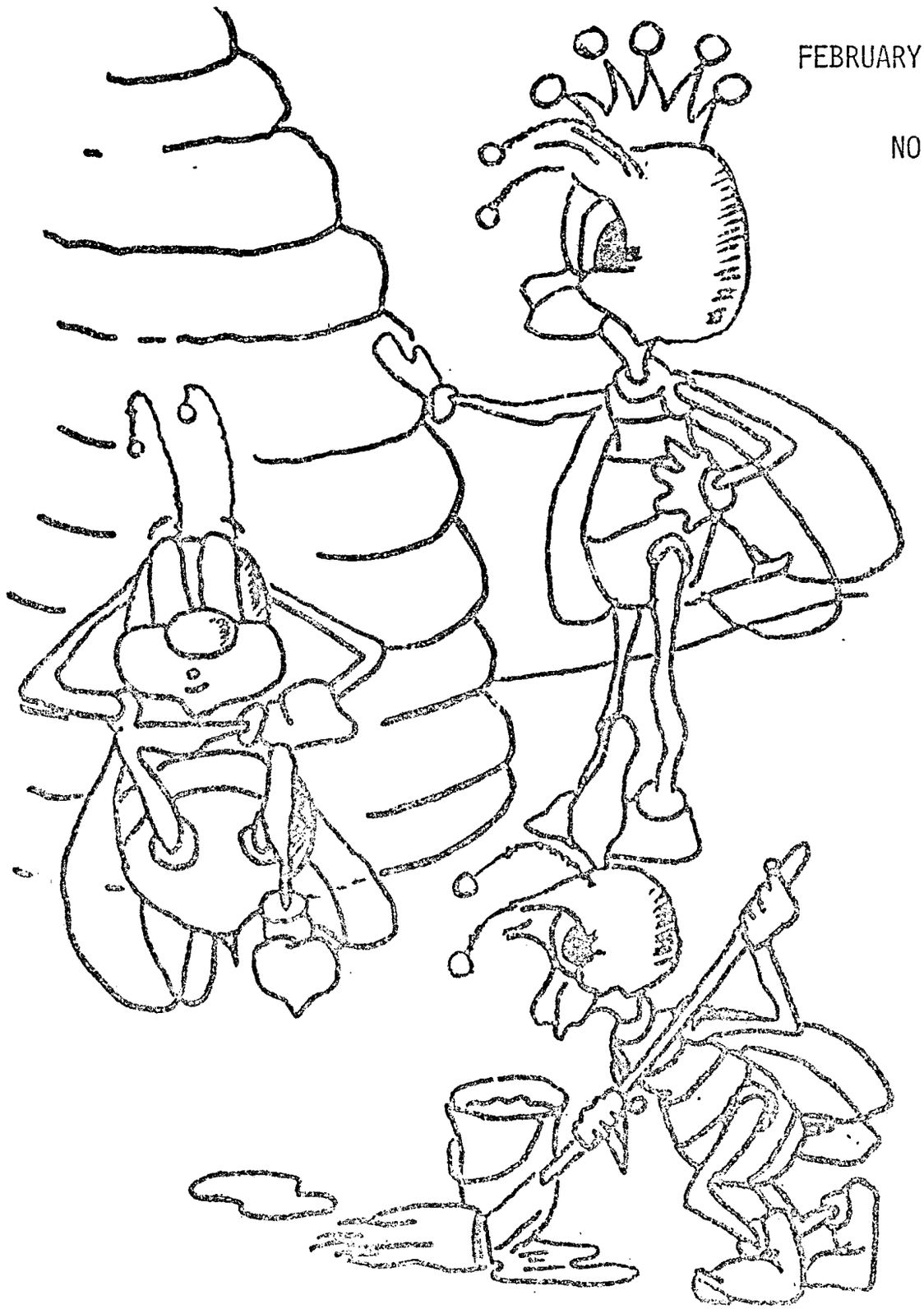
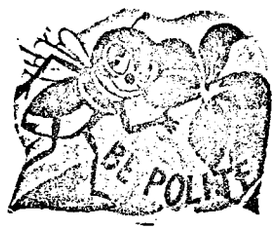


Ministry of Agriculture & Fisheries
Private Bag
Tauranga

T G Bryant
Apicultural Advisory Officer

YOUR NEWSLETTER

This issue is printed earlier than usual, firstly so that you, the beekeeper, may be better informed about the Chalk Brood disease outbreak in Northland; secondly as a direct result of difficulties experienced by many beekeepers this spring to get you thinking about the very serious questions of wintering bees; and thirdly, if you raise your own queens or buy queens all beekeepers need to give much more thought about their breeding stock and in particular, resistance in their bees.



The Chalk Brood (CB) outbreak in Northland raises many important issues nationally and internationally for the industry. It is fortunate that CB is not considered a serious disease of bees, the industry could certainly have done without it as it will make the job that little more difficult. But it will not have the impact of say European Brood Disease nor should it affect the ability of the beekeepers to service those crops requiring pollination.

Factors highlighted by the exercise in Northland:

(i) The need for all beekeepers to notify MAF promptly should anything untoward be observed in the hive; ie get a diagnosis.

(ii) Early detection and diagnosis is essential if a potentially dangerous disease is to be eradicated.

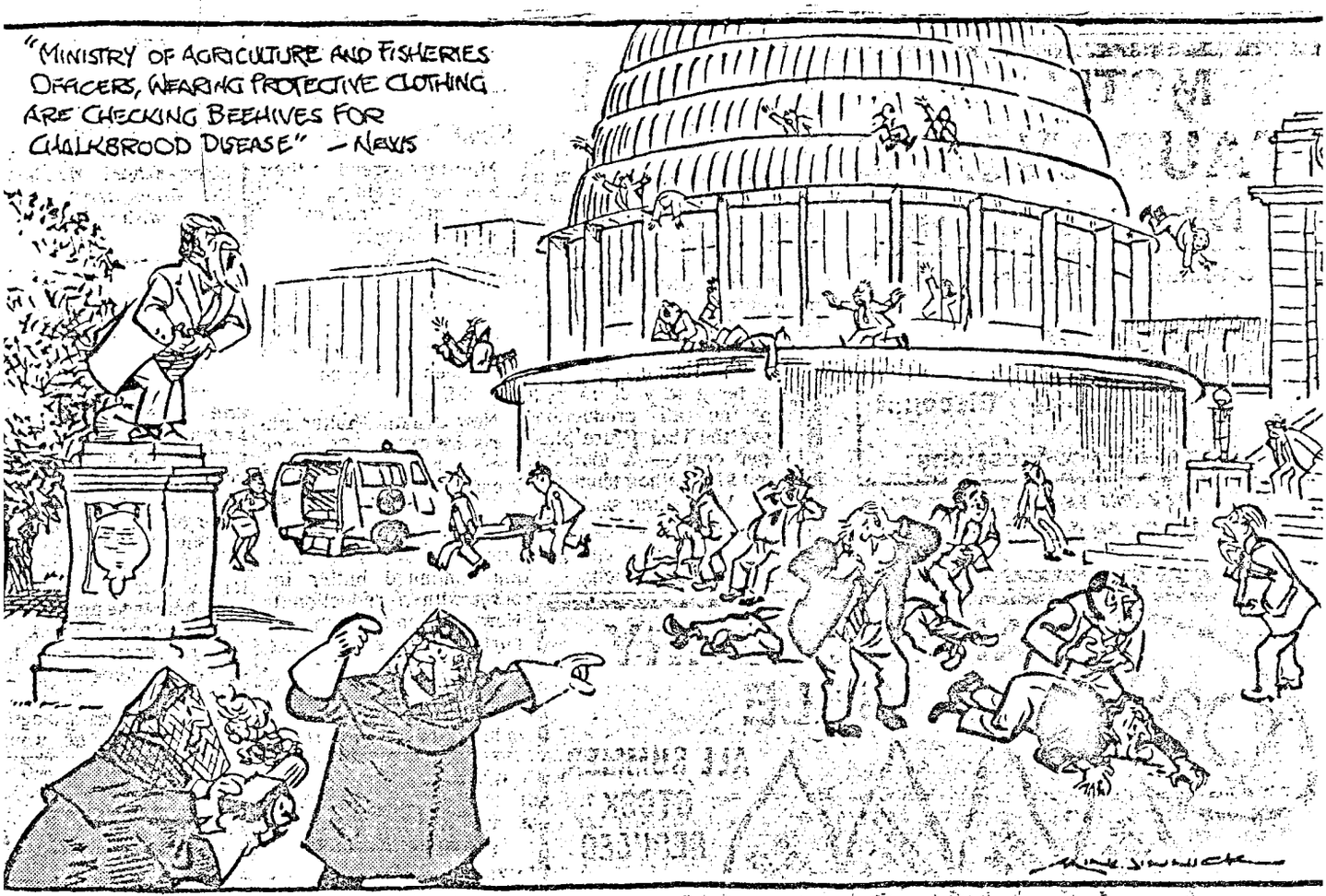
(iii) All apiaries must be registered and clearly identified with a registration number if a similar exercise is ever carried out.

(iv) Media hysteria must be avoided; ie know the whole story and its real potential impact to avoid panic or exaggeration of the facts.

(v) There is a need for a honey bee pathologist and diagnostic clinic in New Zealand.

(vi) An operation to combat an exotic disease outbreak can be carried out quickly and efficiently; co-operation between MAF and the industry is excellent but unless an early diagnosis is made it is all for nought.

I could go on at great length but you will now have to accept the fact that CB is here to stay; know what it is, how it works and how it can be kept at a level which should not affect the productivity of your colonies. Keep in mind that American Brood Disease is still the most serious bee disease in New Zealand, that wasps cause greater financial loss than AFB and that good beekeeping management and husbandry can minimise the effect of all these problems.



"Bring up the bee smoker, Harry, and pray Heaven we are not too late!"

THE DISEASE SITUATION - AFB

MAF inspectors inspected 275 apiaries and 4385 colonies this spring, detecting 110 colonies with AFB. Beekeepers reported 74 apiaries with 173 colonies affected. District totals - 110 apiaries, 283 colonies; District percentage 0.76%, an increase of 0.39% over 1983 figures.

Sac Brood and Paralysis were observed throughout the region, wasps caused problems in the autumn and winter but the difficult spring conditions have made a marked impact on populations and build-up reducing numbers significantly.

Chalk Brood has not been found yet in the region.

Pesticide losses have been minimal.

CHALK BROOD DISEASE

I have reproduced in full an excellent review from Bee World, 63 (3) 1982; 119-130, deleting references and a list of possible prophylactic methods of disease control attempted. If any beekeeper should like a copy of the complete article then please do not hesitate to contact me.

DEVELOPMENT OF CHALK BROOD IN A HONEYBEE COLONY: A REVIEW

by L. A. F. Heath
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Introduction

Chalk brood in honeybees (*Apis mellifera*) may be easily recognized in cells uncapped by the workers. At first, dead larvae are covered by a fluffy white mould and swollen to the hexagonal shape of the cell. Later they dry and shrink into 'mummies', and may become grey/black if spore cysts form. The only condition with which this might be confused is mouldy pollen, but the latter mostly occurs early in the spring, and the mouldy mass breaks up easily when probed, whereas a mummified larva infected with chalk brood retains its identity when similarly treated[4]. The dead brood consists mostly of stretched larvae, the head projecting from the fungal mantle[44]. In severe infections, many cells containing mummies may remain capped. Mummies in course of being ejected may be found on the floor, or on the alighting board, of the hive[16]. Heavily infected hives emit a yeasty odour, although attempts to use this for diagnosis do not appear promising[30].

Early accounts spoke of the drone larvae being preferentially attacked[7, 10, 40], but more recent reports make it clear that this may have more to do with their position on the comb than with any intrinsic difference in resistance[5, 16, 35], or with the fact that colonies under pressure neglect cleaning out drone cells[83]. Worker larvae certainly can be attacked early and extensively in the development of the disease[3, 19, 77]. Betts[7] commented that she had not seen queen larvae with chalk brood, but Cury[12] and De Jong[16] refer to infected queen larvae, and 7 out of 300 questionnaires returned by British beekeepers reported having seen queen larvae with the disease[12]. However, Woyke and Bobrzecki[85] report that 20% of queen larvae in 'long queen cell disease' appear to have chalk brood symptoms, and this may perhaps introduce an element of confusion.

Time of occurrence

Diseased larvae may be found by the beekeeper from April to October in the northern hemisphere with a peak in May-June[4]. Maurizio[44] and Morgenthaler[55] reported a June peak for Switzerland; Toumanoff[80] in France and De Jong[16] in New York State, USA, indicated that the disease reached a maximum in July. A survey of apiary inspectors in the USA found the majority reporting most symptoms in April, May and June[50]. It seems clear that chalk brood is a disease which manifests itself most noticeably as the colony expands in early summer.

Effects

The mummified larvae seen by beekeepers are only a small proportion of those infected, as workers clean out infected larvae—with a degree of efficiency which seems to vary with the strain of bee and is probably inherited[16, 69]. De Jong[15] demonstrated that in colonies with less than 12% infection, mummified larvae are not seen during normal inspections, and that chalk brood in these colonies is only detectable by fitting a dead-bee trap to the hive.

Chalk brood rarely kills a colony, although cases have been reported[5] of every worker larva being killed by the fungus, and Roussy[70] counted 113 mummies per dm^2. More commonly, the loss of larvae leads to a weakening of the subsequent foraging force, and hence to a fall in the honey crop[6, 17, 47, 70]. Estimates of the extent of this loss vary from 1-5% reduction in the honey crop[57] to 23% lower strength of colonies and 49% reduction in foraging capacity[67]. It has also been suggested that poor winter survival may result from infection, due to the reduction in the proportion of young worker bees going into winter[48].

Etiology of chalk brood

Cause of the disease

The fungi *Ascosphaera apis* (Maassen ex Claussen) Olive and Spiltoir, *Ascosphaera major* (Prökschl and Zøbl) Skou, and *Arrhenosphaera cranei* Stejskal, have been isolated from mummified honeybee larvae[44, 45, 66, 74]. *A. apis* has been fed to larvae which have subsequently developed chalk brood symptoms[15, 24, 54]. As far as the author is aware, no similar indication of pathogenicity to the honeybee has been obtained for the other two fungi.

However, larvae may be fed *A. apis* and not develop the disease, even when development of the fungus on the faeces shows that it has passed through the larval gut[5]. Furthermore, larvae from apparently uninfected colonies can develop the disease when incubated in the laboratory[44]. The fungus is clearly widespread, and its presence in the larvae does not necessarily cause the disease to appear. It seems that one or more predisposing conditions must occur at the same time if the disease is to develop.

Predisposing conditions

Betts[7] commented that hot weather appeared to encourage the disease, but Roussy[70] that hot weather controlled it! Lunder[41] thought that the disease was most prevalent in damp regions and where the water is acid. Dreher[18], Roussy[70] and Dallman[13] also blamed damp weather, but Tabarly and Monteira[75] associated the disease with apiaries in which the water content of honey exceeded 19%, or excessive syrup feeding had taken place. Albisetti and Brizard[1] attribute a June peak in the quantity of dead larvae to the dampness in the hives resulting from the evaporation of water from nectar. Wille[83] is sceptical about the effect of high humidity, and Moeller and Williams[53] point out that inside an active colony the humidity is relatively constant, regardless of external conditions.

Seal[72] thought that darker honeybee races were more susceptible to chalk brood, possibly due to their 'excessive swarming', which tended to leave too large a brood nest for the remaining bees to care for. Lunder[41] considered Carniolan and Italian bees to be more resistant than Nordic, and Nelson[60] demonstrated that in Alberta, New Zealand × Californian crosses were more resistant than local stock. Moeller and Williams[53] stated that inbred lines were excellent targets for chalk brood. Murdoch[59] reports mother-to-daughter transmission of susceptibility. There are grounds for believing that bees vary in their resistance to the disease, but whether this character is linked to pigmentation remains uncertain.

De Jong[15] reported that colonies with inadequate pollen supplies were more susceptible; and Herbert[34] showed that feeding with pollen two years old enhanced the development of symptoms. Mehr[47] also used pollen two years old in the induction of chalk brood disease. Haydak[31] showed that, after two years, pollen had lost as much as 76% of its biological effectiveness in promoting hypopharyngeal gland and body development, so bees in a colony fed with this might be inadequate secretors of brood food and hence induce protein or amino acid deficiencies in the larvae.

Deans[14] suggested that, in severe cases of chalk brood, some other circumstance such as acarine disease or paralysis had already weakened the colony. Borchert[9] also commented on the frequency with which chalk brood occurred in combination with conditions such as EFB, AFB, laying workers, drone-laying queens, or chilled brood, which weakened the colony. Mehr[47] noted that nearly half of the test colonies contracted sac brood when chalk brood infection was being induced by feeding pollen two years old containing *A. apis*, and Moeller and Williams[53] and Mehr[46] suggested that larvae weakened by sac brood may be more susceptible to chalk brood. Woyke and Bobrzecki[85] reported that 20% of queen larvae with long queen cell disease exhibited chalk brood symptoms, and it may be that this other disease permits the development of *A. apis* spores already present. Wille[83] regarded the fungus as a secondary invader, infecting larvae having other infections, or otherwise enfeebled. Gilliam[22] has made it clear that *A. apis* is normally a pathogen and not merely a secondary invader of larvae killed by other events. Lunder[41] suggested that weak colonies are more easily infected because they cannot maintain the optimal brood temperature. Bailey[5] linked development of chalk brood infection with chilling of the brood at about the time it is sealed, and this link could account for many of the above observations, together with the fact that chalk brood is common in observation hives[3] and mating nuclei[23]. Cooper[11] has suggested that in some types of bees the brood-nest temperature fluctuates between 35·5°C and 18°C, making them more likely to show chalk brood symptoms through chilling.

On the other hand, records of chalk brood infections exist where it is difficult to understand how chilling played a major part. Thus Gilliam[22] recorded heavy infections at a time when the average monthly temperature was 29°C, and Mehr[47] also reported infections throughout the brood nest during hot dry weather. This has led to the suggestion that the North American strain of the fungus is a more virulent mutant than elsewhere[53], or that genes enhancing susceptibility to chalk brood have been introduced into the gene pool of honeybees in North America[58, 84]. It has also been suggested that the widespread use of oxytetracycline in North America, to control AFB, might be contributing to the increase in chalk brood[20, 71]. However, it has been shown[51, 52] that oxytetracycline neither aggravates nor controls chalk brood symptoms.

Epidemiology of chalk brood

Infection

It seems to be generally assumed that infection of the larva is initiated by ascospores, although Bailey[4] suggested the possibility of infection by mycelium: Borchert[9] and Claussen[10] reported mycelium spreading over the comb from uncapped cells. Betts[7] indicates that the fungus cannot penetrate wax cappings. In the light of Maurizio's[44]

finding that 10 of 38 combs she examined bore infections of only one sexual strain of the fungus, perhaps the possibility of infection by hyphal fragments should be further investigated.

In the following account, the word 'spores' is only used where the investigator's recorded method eliminated the possibility of infection by hyphae. In other cases, more general terminology is employed. Gilliam[24] demonstrated that infection can occur either from ingested material or from surface inoculation of the larval cuticle. In the light of Thomas and Luce's[77] demonstration that spores will not germinate aerobically, it seems likely that the Gilliam's cuticular infections arose from hyphae in her inoculum, and not directly from spores on combs as Matus and Sarbak[43] had suggested. Roussy[70], however, said that he observed spores germinating on the larval cuticle. There is therefore a direct conflict of evidence on this point. Maurizio[44] rejected the possibility of surface infections because when she removed apparently healthy larvae from the combs and kept them on agar, many developed the infection. These, she believed, must have been infected internally. De Jong[16] discounted the idea of infection through the body surface, because Huber[38] could not demonstrate the production of a chitinase by *Ascosphaera apis*. Although Huber's experiments were open to objection because of the constitution of the media he used, Gochnauer and Margetts[27] have recently also failed to demonstrate chitinase production by *A. apis*. It does seem that further elucidation is needed of the mechanism of penetration of the larval cuticle in the absence of chitinase enzymes, and apparently in the presence of oxygen.

Bailey[5] demonstrated that spores could germinate at brood temperature and in the anaerobic conditions to be expected in the larval gut, and that small mycelia could be seen there. He reported that the youngest larvae are not very susceptible to infection, perhaps because the mycelium does not easily survive prolonged anaerobiosis until the larvae become sealed. Larvae 4 to 5 days old appeared to be resistant because there was not time for the spore to germinate and grow before it was voided when the midgut-hindgut junction opened prior to pupation. In normal circumstances all germinated spores are also voided at this time, because the mycelium cannot grow anaerobically. When larvae are chilled to 22°C immediately after sealing, oxygen penetrates the gut, and the fungus is reactivated and grows out of the gut and through the tissues.

At the stage at which penetration of the gut wall usually takes place, the larval cells are under autolytic attack from lysosomal phosphatase, as a normal part of metamorphosis. The basal region of the cell is attacked, and decomposed cell components are released into the intestinal lumen. The monolayer propupal midgut is then formed from small regenerative cells[65]. It is probable that, at this stage, that the larvae are very susceptible to infection from the gut, and that any major physiological disturbance may interfere with regeneration and provide the fungus with its opportunity.

Claussen[10], however, said that the fungus can infect bees in all stages from egg to pupa inclusive. Betts[7] also thought that the fungus could attack eggs and, rarely, pupae. Roussy[70] reported pupal infection in 1962. However, Nelson & Gochnauer[61] failed to isolate *A. apis* from the gut of pupae, and Gilliam[22, 24] demonstrated that eggs and pupae are not susceptible to laboratory infection. She also showed that larvae 3-4 and 4·5-5·5 days old were equally susceptible to infection in petri dishes held at 25°C. The temperatures employed probably hold the clue to the apparent discrepancy between

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these and Bailey's results.

Claussen[10] said that if eggs or small larvae are infected, the cells normally become filled with hyphae only up to about half their depth. If the larvae are large, then the hyphae fill the cell. Maurizio[44] complained that, in her experience, this description is inaccurate but that it had been included in all textbooks except that of Angelioz-Nicoud[2] who, like Maurizio, describes mummification as limited to the stretched larva stage. Hitchcock and Christensen[35], however, reported mummification of coiled larvae, and Wille[83] was convinced that larvae may be seen to be infected at a very early stage. These early infections, according to him, are detected by the nurse bees which then eject the larvae. Consequently, sparse brood may be the only symptom visible to the beekeeper. One aspect of this is supported by Gilliam[24] who showed that larvae with the early stages of infection are removed from their cells within 16 hours.

Disease development

Maurizio[44] carried out histological studies on the development of the disease in a larva, and showed that the fungus is initially restricted to the lumen of the hind end of the midgut. It then grows through the gut wall to the fat-body, and finally ramifies through the posterior two-thirds of the larva. Cuticle, tracheae and oenocytes are the last to be attacked. The fungus does not penetrate the anterior portion of the body, and the larval head is left projecting from the fungal mantle after mummification. Gochnauer and Margetts[28] showed that *A. apis* lacks many of the lytic enzymes common in other insect pathogens, and considered it 'a relatively non-invasive parasite that kills the host by competition for primary nutrients', so the mechanism of its spread through the larval body remains to be elucidated. The mummies contain about 10% of the glycogen and less than 20% of the glucose found in a healthy larva. Maurizio also demonstrated that the fungus had no effect on the length of life of adult bees when it was fed to them in syrup.

Transmission within the colony

The fungus can survive the winter in honey: this has been demonstrated by its isolation from honey after two years' storage at both 20°C and 30°C[44]. It has also been isolated from imported honey, where it appears to have survived processing[81, 86], and found in bee-stored pollen[61]. Borchert[9] and Barthel[6] say that it may survive the winter in the adult bee midgut, but the author can find no record of experimental proof of this. Nelson and Gochnauer[61] isolated *A. apis* from the midgut of adult bees, but not from the honey sac or rectum. De Jong and Morse[17] and De Jong[16], however, demonstrated that summer bees from colonies with 2-100% infection all carried the fungus in the honey sac and transmitted it to uninfected workers through 3-mm wire mesh. The continuous food sharing in a colony provided a mechanism by which infective units can rapidly spread among adult bees, including those feeding brood. Maurizio[44] demonstrated that the honeybee intestine contained the fungus until well after obvious symptoms of the disease had disappeared from the colony. Vandenberg[82] recently demonstrated by electron microscopy that *Ascosphaera* spores are carried on the surface of the leaf-cutting bee *Megachile rotundata*. It seems probable that similar transport of the viscous spores of *A. apis* could occur in *Apis mellifera*, as suggested by Moeller and Williams[53].

Transmission between colonies

Thorstensen[78] considers that spores may be distributed by wind, rain, bees, birds or beekeepers. Beekeepers certainly appear to be agents of transmission, as Herbert[34] demonstrated that transference of queens, workers, or sealed or unsealed brood from an infected to an uninfected colony could transmit the disease. Moeller and Williams[53] and De Jong[16] have also demonstrated that requeening with an infected queen may transmit the disease.

A mummified larva may produce 10^8-10^9 ascospores[5, 61, 78] and mummies bearing these spores are removed from the hive by workers. Following removal, the cells are efficiently cleared by the bees, and Nelson and Gochnauer[61] failed to isolate *A. apis* from swabs of brood cells. Thorstensen[78] and Moeller and Williams[53] suggested that ascospores from mummies may be subsequently wind dispersed: this seems unlikely, as the dead larvae are likely to fall among ground vegetation where air movements are slight, and the viscous surface of the spore[73] is not suited for wind dispersal.

Moeller and Williams[53] also suggested that spores could be picked up by foraging bees at sources of nectar, pollen or water, and infected pollen can apparently cause the disease[47]. Nelson and Gochnauer[61] failed to isolate *A. apis* from fresh nectar in the hive, but did isolate it from pollen collected at the hive entrance; probably the pollen was infected when the bee combed it from her body hairs into her corbiculae. This would fit well with Vandenberg's observations[52] for other bees and other species of *Ascosphaera*, and suggests that pollen foragers could transmit spores via flowers. In addition, the demonstration of the presence of the fungus within the gut of adult bees from infected colonies[44, 61], and of transmission between bees by food-sharing behaviour[16], make it clear that the disease can be transmitted by drifting bees—as suggested by Borchert[9] and Barthel[6]—whether surface transport is confirmed or not. Drifting is particularly common in young bees[8] and could be the main path of spread within an apiary. Robbing and drifting of drones provide similar routes. The spread of spores appears to be well catered for, and it seems probable that most hives in infected areas will contain the spores of *A. apis*. Betts'[7] observation that the disease does not spread easily in a district, or even in the same apiary, is probably a reflection of the need for the predisposing conditions to be present in any hive before infection becomes obvious to the beekeeper.

Control of chalk brood

Natural control

De Jong[16] demonstrated that larvae differ in their resistance to infection, and that strains of bees vary in the efficiency with which they remove larval mummies. Rothenbuhler[69] has investigated the inheritance of this latter characteristic with regard to AFB. The uncapping of the cells and the removal of the larvae appear to depend on separate genes. There seems no reason to suppose that the behavioural inheritance affecting the honeybee dealing with dead AFB larvae is in any way different from that which controls it when dealing with chalk brood mummies.

Artificial control

A number of recommended methods of control are based on the assumption that hive

dampness is a major predisposing condition. For example, Borchert[9] recommends the avoidance of damp sites, and Seal[72] treatment by hive ventilation. Apparently in response to Bailey's 'chilling' hypothesis, Pedersen[62] showed that warmth provided under the hive in spring reduced infection. As the fungus clearly exists within colonies which appear to be healthy[24, 44, 49], destruction of infected combs alone is not likely to lead to successful control. Indeed, Anderson[3] reported on extensive chalk brood infection in a comb so new that it was not fully built. Bailey[5] showed that spores can pass through the gut of healthy larvae and be voided with the faeces immediately before pupation. These faeces thus constitute a potential source of later infection. They are sealed from immediate contact with larvae or bees by the laying down of the cocoon, but if the comb is broken later, the infected faeces become exposed. Nelson and Gochnauer[61] note that infection is greater in old than in new comb, and that propionate-sorbate treatment is more effective on old comb. Combs may be successfully disinfected with 40% formalin fumes for three weeks[40], or with ethylene oxide for 15 hours at 22°C[21, 77] or with pure ethylene oxide for 30 min at 35°C[29]. It is not clear whether these treatments kill any *A. apis* sealed between the cocoon and the cell wall. However, treatments aimed at the elimination of the fungus from combs seem doomed to fail in field conditions, because of the widespread occurrence of the pathogen and the ease with which it is transmitted.

De Jong[16] showed that manipulations which decreased the brood-to-adult ratio in the colony decreased the severity of the disease. Consequently, strengthening a colony should offer a degree of amelioration. Requeening, a treatment recommended by Roussy[70] and Mraz[58], may work by introducing resistant genes into the stock, or—given an active young queen—by increasing the proportion of young house-cleaning bees in the hive.

Table 1 lists chemical treatments that have been attempted; as Wille[83] points out, positive results have not been repeatable under different conditions. Various chemicals, such as thymol, when sprayed onto the combs, will enhance cell cleaning, but this can be achieved equally well by spraying sugar syrup[6].

There are also a number of contrary reports about the efficacy of these chemical treatments. Thus Nelson and Gochnauer[61] found benomyl and mycostatin ineffective; Menapace and Hale[49a] reported that citral and a combination of sodium propionate and potassium sorbate did not control chalk brood. Heath and Heath[33] failed to inhibit the growth of *A. apis* on 2% malt agar with griseofulvin, sorbic acid and sodium propionate separately, each at 0·05%; of the 10 agents tested, they obtained inhibition of the fungus only with 0·0025% cycloheximide. Cycloheximide inhibits protein synthesis in eukaryotic ribosomes, and hence may be expected to inhibit honeybee larval growth almost as effectively as fungal growth.

A compound for the control of chalk brood must be both convenient to use and not more expensive than the natural loss due to the disease[49a]. This would appear to require a cheap effective antifungal agent which is nontoxic to bees, and which can be steadily released in the hive throughout the season. In addition, it should not stimulate the production of resistant strains of fungus. Given these facts, the present author is not optimistic about the development of chemotherapy for the control of chalk brood.

Conclusion

Experimentation with this disease is dogged by the difficulty that control colonies frequently show high levels of chalk brood infection[49a]. Unless very large numbers of colonies are used, it is difficult to detect whether any manipulation or chemical treatment is having a significant effect. Many published results are suspect for this reason. In the author's experience in south-west England, it has been possible to detect *A. apis* in any colony of bees so far investigated if the search is diligent enough. It appears, therefore, that we may well be dealing with a disease where exposure to the pathogen is the usual situation for a larva, and the development of infection is mainly dependent on the physiological and environmental conditions of the larva at the time. *A. apis* is therefore best regarded as an opportunistic pathogen which is efficiently dispersed and very widespread. At our present state of knowledge it seems that the selection of resistant strains of bees is most likely to advance control of the disease.

DETERMINING THE RESISTANCE OF HONEY BEES TO BROOD DISEASES

Disease resistant (DR) strains of bees can be determined without the need to have any disease present in your colonies. DR is determined by the hygienic behaviour of the brood nest; ie the ability of worker bees to remove sealed dead brood quickly.

To test for DR you require:

1. Colonies with brood in all stages of development.

2. A minimum of 10 colonies at a time.

3. Sealed brood killed by freezing; be sure all brood is killed at same time, stored in same place and is free of honey or pollen residue.

4. Insert small samples of 40 mm^2 dead brood into centre of the brood nest rather than use whole comb; ie cut out square from brood comb and insert sample.

5. Be sure each test is carried out in exactly the same manner and all dead brood has been treated exactly the same.

6. Be sure the bees you are testing are from the queen resident in the hive, wait say two months after introducing the queen or selecting the potential breeder, mark the queen to be certain.

7. Examine test colonies in 24 hours and again in 48 hours (you may want a further examination again 24 hours later.)

8. Expect to find about 1 in 10 (20) colonies that will show the clean up characteristics of uncapping and removal.

9. Expect differences in the rate of uncapping and removal due to conditions and colony placement (site); ie spring build-up, honey flow.

10. Colonies exhibiting DR should uncap and remove most of the dead brood within 24 hours and all in 48 hours.

11. Be sure to select for other characteristics at the same time. You don't want DR bees at the expense of honey production for example.

12. Do not use samples of actual diseased brood to test for DR. Use only healthy brood you have killed.

REFERENCE:

Taber S, 1982. Bee Behaviour, Determining Resistance to Brood Diseases. Am Bee Jrnl. 122 (6); 422-425.

NB: The first rule in rearing queens for increased production is to improve the environment in which you raise that queen; ie raise queens in the best possible way. Nutrition is paramount - carbohydrate (sugar syrup or honey) and pollen. Pollen must be as close to cells as possible as bees rarely move it about the hive. Such things as selection, age of larvae, mating are of no value if the queen is raised on a poor diet.

WINTERING

The over-wintering of bee colonies is a very neglected aspect of beekeeping in this part of the world and many of the problems beekeepers have to overcome in the spring are directly attributable to lack of thought the previous autumn.

If your winter losses are consistently higher than 5% then brush up on your wintering, take losses on the nose in April. Eliminate queenless hives, failing queens, drone layers and weak colonies - they are going to die so don't waste their stores which can be more profitably used on colonies that will survive.

Don't leave winter preparations until the onset of winter, you should have taken steps to ensure your hives had plenty of bees which are going to over-winter back in March.

Hives should be weighed to ensure there are plenty of stores. Tare weight of the standard two brood box hive, lid, floor and bees is approx 32 kg. If the total weight of the hive is 50 kg, there is approx 18 kg of honey available for winter feed. Tare for three-quarter depth equipment is 29 kg.

Ensure there is plenty of pollen in the hive and that in March the queen has room to lay - often after the honey flow brood nests can be choked out with honey and pollen, leaving little room for the queen to lay out. You want winter bees, not summer bees which will die in the winter.

Site preparation is important - anything you do to keep the bees from being stressed helps bees overcome the adult bee disease Nosema, dysentery, stops Chalk Brood getting out of control and results in a healthy, populous colony in the spring which are then easily managed.

If you have to feed bees sugar syrup, don't wait until stores are depleted, feeding should supplement stores in the hive; when all are gone you are not helping the colony although you may keep it alive.

REMEMBER - bees don't freeze to death, they starve.

BITS & PIECES

* *You need not fear Chalk Brood, with good beekeeping practices it is no worse than Sac Brood.*

* *Avoid stressing your bees, requeen in the autumn, make any increase in the late summer (February).*

* *Pollination - Gisborne: 2020 hives went into orchards;*
  BOP: 19777 hives into orchards.

* *A Budgeting & Financial Management Workshop is planned for June/July. If interested contact me or watch for details.*

* *District honey crop is estimated at 682 tonnes - 18 kg surplus for all registered hives in the region; ie some will do better than others.*

* *You have seen something in your hives you are not sure about!! Report it.*

* *Get those registered numbers out on all your apiaries.*

* *NBA branch meetings and AGMs are fast approaching. Have you thought about office bearers/remits etc, etc? Now is the time to get those thinking caps on.*

* *It is not the MAF's responsibility to collect hive levies. If you own 50 plus hives you must pay the levy. For details write to NBA Secretary, PO Box 4048, Wellington. It's up to you, not us in MAF.*

* *Any information in your annual Statement of Inspection re apiary sites, hive numbers etc is confidential between MAF and you. Nor is any information or details regarding pollination contracts available to anyone other than MAF. This information is however essential and is required by law; ie anywhere where bees are kept must be registered as an apiary with MAF.*

Keeping in mind what happened in Northland it is imperative that you as beekeepers keep us informed. Despite letters, many beekeepers failed to keep us informed of their pollination activities until long after the event. There are one or two still outstanding but I have been able to get details such as total hives used and orchards pollinated so the total quoted is fairly accurate, but such a situation should not have occurred.

If you were a carrier of any disease or were in contact (flight range) with diseased hives how are we supposed to help if we don't know where you are. Be a responsible beekeeper, think of your industry and others, not just yourself.

The book 'Honey Bee Brood Diseases' by H Hansen has excellent colour plates of all brood diseases including CB and is available from most suppliers of beekeeping wares. A colour photo insert will be included with the next issue NZ Beekeeper. An AgLink updating brood diseases is being printed and will be available shortly.

TREVOR.

T G Bryant
Apicultural Advisory Officer