Just look what happens if you don't treat against varroa!

"All beekeepers must manage varroa in their stocks"

Summary

I last treated my local, native and near-native bees against varroa in 2002, since when out of hundreds of colonies, I have lost no more than three or four to the mite. However, since very few mites, alive or dead, were in evidence I was unable to deduce the manner of the bees' apparent resistance. In May 2010 one colony (JB5) led by a dark queen, but containing both dark and ginger-banded workers, developed a heavy infestation, dropping dead mites at a peak rate of 23 per day in late May and affording the opportunity for me to investigate. Just before this peak, the colony swarmed and the swarm was lost. At that time fallen mites in the resident colony included a small percentage with serious injuries typical of those caused by mandibular grooming. As the summer progressed the proportion of damaged mites in the resident colony increased in a complex pattern, but the total mite drop decreased to a negligible level. This account details what I observed and my deductions therefrom. I suggest that the Northern native honey bee, Apis mellifera mellifera, when left to its own devices, may have as many as nine strategies it can utilize to overcome the varroa mite.

Introduction

The most recent comments on the treat/don't treat debate I have seen were in Arnold Desandre's letter in the May 2017 BBKA News entitled "Varroa, to Treat or Not" and he is very concerned. He wrote: "There is great risk in advising to not treat ... beekeepers are not in a position to understand the ramifications of not treating." Clive and Shân Hudson (2016) mention the National Bee Unit's instruction: "All beekeepers must manage varroa in their stocks. If they fail their colonies will collapse and die" (NBU, 2016). However, the NBU support those dire directives with varroa growth curves that do nothing to inspire confidence in the educated beekeeper (Ball et al, 2010; NBU, 2016; APHA, 2017). They are mathematically perfect, smoothly exponential and soaring upwards toward infinity. These are presumably based on the report by Calis et al (1999) that varroa can double their numbers in four weeks. That may be the case, but it does not mean they will keep doing it every four weeks. Biology is rarely mathematically immaculate and Calis et al add that "interaction between the mite population and the colony is evident at high mite populations", which would make a mess of the curves. De-Grande-Hoffman et al (2016), for example, show both theoretical and actual varroa growth curves that look realistic and are very different from those of the NBU (see also Fig. 1). Also, and most significantly, neither Calis nor the NBU report the subspecies of *Apis mellifera* which hosted the mites they studied. That omission conceals a healthy programme of defensive tactics and generally unrecognised aspects of bee behaviour that notably vary between the races (see Pritchard, 2016). If the irritant that elicits it is removed, the bees' defensive programme may well never get started, giving a completely misleading impression of a colony's capacity to look after itself; in my colony the irritant was left in place and a whole alternative scenario of honey bee behaviour was revealed.

I am the one who carried out the statistical analysis of Heaf, Hudson and Hudson's (2015) figures on Welsh winter losses that reported higher survival rates among non-treated than treated colonies, so I have a professional interest in this debate. Referring to their figures I concluded: "taken as they are, they suggest that chemical treatment against varroa is indeed associated with significantly higher winter losses than occur when no treatment is applied." (Pritchard, 2015) Those first four words are important, as there could be circumstantial considerations that might modify the bald statistical conclusion. But read on!

Varroa arrived here in Northumberland in AD 2000 and in 2003 I stopped treating my bees. I did so as I deemed it unnecessary since there were no mites in my hives and I also considered some official advice unacceptably dangerous. Some acaricides become less effective with use, so ever more noxious poisons are needed. As Randy Oliver has commented, "this chemical-dependent treadmill ... places beekeepers in the position of only being the next chemical away from disaster." (Oliver, 2017). Could it be the practice of pouring, squirting, volatilising, vaporising and dusting toxins into beehives to which we can ascribe the present very regrettable situation where, according to COLOSS (see Büchler & Uzunov, 2016) there is scarcely a honey bee colony in Europe capable of surviving indefinitely without veterinary attention? Of the several hundred colonies I have maintained since AD 2000 the loss of only three or four could be ascribed to varroa.

Perhaps you think I'm one of the awkward squad, but my story should help you appreciate my perspective.

My story

I'm a retired lecturer in human and medical genetics and for nearly 40 years I have collected and bred native and near-native colonies of the British Dark Bee, *Apis mellifera mellifera*. When I first took up beekeeping, since nearly everything I knew in relation to my profession had been acquired from books, I decided that with beekeeping, since it is a traditional occupation, I would instead

learn only from people and the bees themselves. Later, when I began teaching beekeeping I looked at a few books and found that nearly all were written by and for people who are trying to maintain Mediterranean bees in the south of England, an uphill struggle at the best of times (Fig. 11). For Northern native beekeepers those books and the popular magazines are misleading in several ways.

Native honey bees are considerably tougher, less prolific and more closely harmonised with their surroundings than the foreign imports. Peak July temperatures are below 15°C north of York, by comparison to those around the Mediterranean at over 20. To reveal their strengths and to select out native type colonies from the foreigners, you need to reimpose the forces of natural selection. So you don't pamper native colonies once they have reached 5 frames of brood. They appreciate being kept cold in winter, without insulation, when they keep themselves warm hanging in a cluster, in an empty shallow below the brood nest ("the best insulation for bees is bees"). We don't sugar feed except in exceptional circumstances and we aim to overwinter strong colonies on nine (instead of eleven) deep frames of heather honey, with a gap at each end for air circulation, and no supers or extra food. In summer we keep them hot, with insulation, to help them secrete and work their wax and raise strong brood. Native bees are very good at looking after themselves when delicate Mediterraneans cannot survive, and we rely on that difference to keep the local breed strong. By following this course, reinstating something like natural selection, before I found natives locally I succeeded in re-creating bees that accorded with all the then recognised characters of native bees, including their mitochondrial DNA.

Since I began writing about bees I have read many original research papers, but have found little relevant British research, although a lot carried out abroad. Much of that knowledge never gets through to British beekeepers unless they are very persistent or have been trained in research techniques. This report deals with aspects of bee behaviour that are well known in Germany and Poland, for example, but almost never spoken of here. The term "emergency bees" is however, my own.

Breeding native bees requires constant vigilance, as here I'm surrounded by honey bee "mongrels", while some of my neighbours actively import exotic or Buckfast queens. This means that every season a proportion of my dark queens produce workers with a ginger band at the fore end of their abdomens (see Figs 9, 10). In the spring of 2010 I found one such colony (JB5) heavily infested with varroa, which were dropping naturally at the rate of 17 a day. This was nearly three times the lethal threshold for May of six; specified by the NBU for treatment (Ball et al, 2010). I almost never see varroa mites in my hives, even at low densities, so I was delighted (nay, thrilled!) at the rare opportunity to investigate if or how my wonderful bees would cope. (So if they could, would it be the bees that are different, or my attitude?)

At a SICAMM conference in Sweden in 1998 I had expressed my eagerness to get to grips with the mite that was so far defeating so many, as I considered breeding varroa-resistant bees a worthy challenge. Long before 2010 I had my varroa-resistant bees. It was not a question of selection for resistance, because the native type bees here are virtually all resistant! However, because there were no mites in their hives to examine I didn't know how they were doing it. When the aberrant JB5 appeared, I considered it might provide the answer. The practical details of what followed and my earlier deductions are in the July issue of the new magazine, *Natural Bee Husbandry* (Pritchard, July 2017), but here is an update.

The observations

In short, when left to their own devices, far from giving up the ghost, within several weeks, colony JB5 learnt to cope with the mites, destroying them almost completely, apparently by biting with their mandibles, as *Apis cerana* do (Ruttner & Hänel, 1992). This behaviour was however not spontaneous, they apparently had to learn what to do, or how to do it and the activity built up over time. The mite drop reached a peak at 22.6 per day, following swarming in late May. This is off the NBU scale for action (APHA, 2017, Fig. 56), but thereafter it dropped to a daily average of around five from the middle to end of June (Table 1; Fig.1). There had been a steep (possibly exponential) increase in mite numbers up until departure of the swarm, but soon after this the bees' fight-back overtook the mites' capacity to increase. There was no other indication that these mites could double their numbers in four weeks and multiply exponentially without check, as the NBU imply.

Dates	9/4	22/4	30/4	9/5	14/5	19/5	21/5	31/5	2/6	11/6
Brood frames	4	7	8-1#	9	9-1#	(9.5)	10*	10-2#	10	(6.5)
Time span										
Data point date					17/5		26/5		6/6	
Days elapsed					5		14		9	
Data point no.					1		2		3	

Dates	11/6	19/6	27/6	8/7	20/7	25/7	1/8	19/8	14/9
Brood frames	(6.5)	(3.8)	3	(5.8)	(8)	9	9	6	(4.5)
Time span									
Data point date		15/6	30/6	14/7		26/7	10/8		1/9
Days elapsed		8	19	12		12	18		26
Data point no.		4	5	6		7	8		9

*: Swarm departed
#: frames removed
numbers in brackets were estimated from Fig. 1.

Table I. Major events in hive JB5 and establishment of data points. Numbers in brackets were estimated from Fig.1 in Pritchard, 2017.

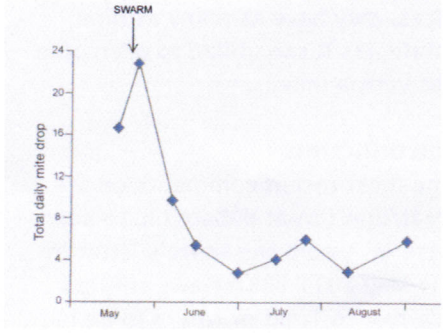

Figure 1. Total natural mite drop over the summer of 2010
The colony swarmed in late May and the new queen commenced laying in late June.
Total mite drop fell from 22.6 per day just post-swarming, to average around 5.2 per day in June - September.

What was really impressive though was the proportion of fallen mites that showed severe physical damage. Initially 13% were damaged, but this peaked at 70% in mid-June, fell to a trough at the end of June, then in-

creased and remained high to the end of the season (Fig. 2).

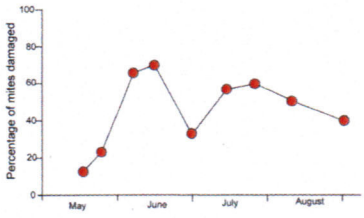

Figure 2. Percentage of naturally fallen mites that were severely damaged.
The percentage of damaged mites rose from 13% in mid-May to peak at around 60-70% in mid-June, fell to 33% at the end of June, then rose again to ~60% in late July, before falling again to ~40%.

The damage to the mites was not just dents in their carapaces (or "idiosomas"), it was severed legs and major gashes that would definitely kill them by loss of body fluid (see Fig. 3); minor dents were not recorded. This was similar to that reported by Ruttner and Hänel (1992) as being caused by the mandibles of *A. cerana cerana* and *A. m. carnica*, being used like shears. Wallner (1990) reported that if 60% of mites falling from his *Apis cerana* colonies should show severe damage, those mites are doomed, as indeed were mine, as I think no mites in this hive survived the winter. (Nor, incidentally did those in two nukes I set up from four brood frames removed from the brood nest at around the time of swarming).

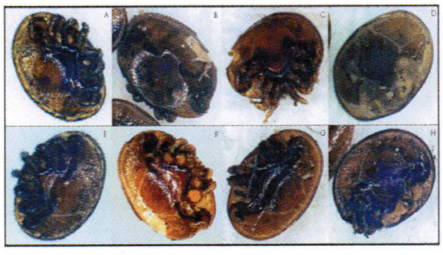

Figure 3. Photographic evidence of damage to mites.
A, E: Ventral aspect of mites with no obvious major damage.
B, F: Dorsal and ventral aspects of the same severely damaged mite, showing a large gash in the idiosma and loss of the third left leg.
C, G: Mites showing loss of legs and a gash in the idiosoma edge of C.
D, H: Ventral aspect of mites that have lost all or several limbs.

Details of the practical aspects of this study are given in Pritchard (2017). Table II shows a summary of data collected, which included estimation of the percentages of mites showing serious damage at each data point, and from this the calculated total numbers of damaged mites.

Data points	1	2	3	4	5	6	7	8	9	
No. frames of brood	9.5	10	6.5	5	4	8.5	9	7	4	See Table I
No. fallen mites	87	317	87	44	52	47	71	49	~150	~900 total
No. fallen mites per day	17.4	22.6	9.7	5.5	2.7	3.9	5.9	2.7	~5.8	~7.0 See Fig. 1
% mites damaged	13	24	66	70	33	57	60	51	40	38.4 overall, see Fig. 2
No. mites damaged	11	76	57	31	17	27	43	25	60	347, values estimated
No. mites damaged per day	2.2	5.4	6.3	3.9	0.9	2.3	3.6	1.4	2.3	
% mites damaged per brood frame	1.4	2.4	10.2	14.0	8.3	6.7	6.7	7.3	10.0	See Fig. 7

Table II. Development of the brood nest of colony JB5, empirical counts and derived values.

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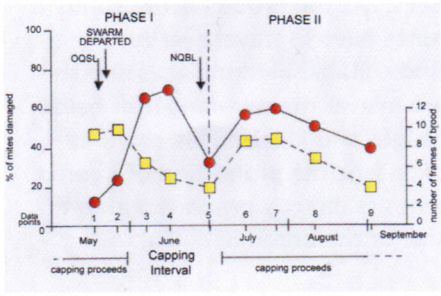

Figure 4. Development of the broodnest and percentage of fallen mites showing severe injury.
Yellow squares indicate numbers of frames of brood and/or eggs, red disks show the percentage of injured mites. Data points are indicated (1-9). OQSL: old queen stopped laying. NQBL: new queen began laying. Phases I and II are indicated.

Fig. 4 shows comparative plots of the percentages of mites damaged, against time, and the number of frames of brood and/or eggs at the same data points. The dates of departure of the swarm, when the old queen stopped laying (OQSL) and when the new queen began laying (NQBL) are also shown. From these, the interval in brood capping was calculated and is shown below the graph.

Distinctive features of the data are revealed by this representation, which allows us to divide events into two phases. In Phase I (Data Points 1-5) no obvious relationship can be discerned between the number of frames of brood and the extent of mite damage, but there is a very obvious coincidence between the peak in mite damage and the interval in capping of the brood. By contrast, in Phase II, after the new queen had begun laying (DPs 5-9), there is a striking correspondence between the degree of mite damage and the number of frames of brood and/or eggs.

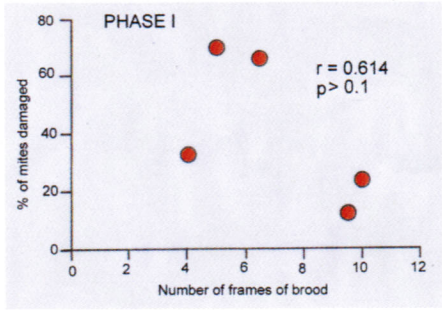

Figure 5. Relationship between percentage of damaged mites and size of the broodnest during Phase I.
The percentage of damaged mites shows a random relationship with broodnest size. DPs are indicated.

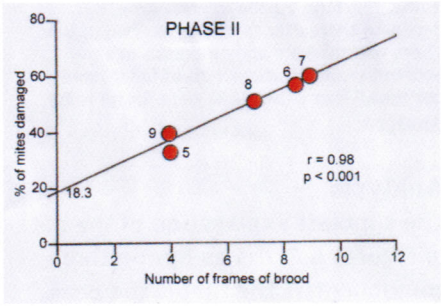

Figure 6. Relationship between percentage of damaged mites and size of the broodnest during Phase II. Data Point 5 is shown in both Figs. 5 and 6. This straight-line relationship fits the equation: y = 18.3 + 4.6(x).

Figure 6 shows the data in Phase II as a plot of percentage of damaged mites against the number of frames of brood. This is a straight-line graph, which fits the equation:

% of mites damaged = 18.3 + 4.6(number of frames).

The two sets of figures show almost perfect correlation (Pearson coefficient of correlation, r = 0.98) which is very highly significant ($p < 0.001$; 4 df), showing that in Phase II the proportion of damaged mites is strictly proportional to the size of the brood nest. Note that the critical level for mite damage of 60% at Data Point 7 occurs when the brood nest occupies nines frames (Wallner, 1990). By contrast, as shown in Fig. 5, in Phase I there is no such correlation, where r=0.614 and p>0.1(4 df).

When normalised for brood nest size (i.e. per frame of brood) over the whole of the summer, a plot of the percentage of mites damaged against time reveals a striking pattern, with a massive peak in mid-June, coinciding precisely with the capping interval, followed by a trough in mid-July, then a steady rise into September (Fig. 7).

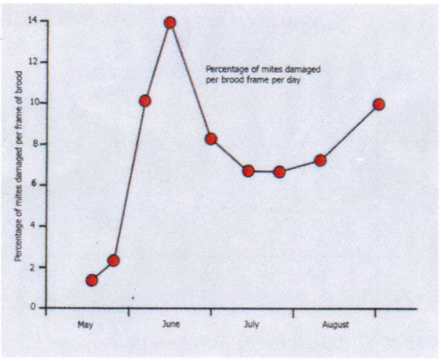

Figure 7. Percentage of damaged mites normalised for brood nest size, against time. This plot shows a simple, but striking pattern in the percentage of damaged mites, per brood frame, reaching a peak in early June, but declining thereafter till the beginning of July. There was then a steady increase to early September, when about 10% of fallen mites per brood frame (i.e. 40%) were found to be damaged.

Analysis

The simplest explanation of the data in Figures 6 & 7 is as having three components. The first is the peak, corresponding precisely in time to "the capping interval", i.e. the period when no brood was being capped (see Fig. 8). At this time all the newly emerged mites would have been in the phoretic state, and therefore at serious risk of injury. There is also a gradual background increase over time in the proportion of damaged mites, extending throughout the entire period May - September, and accounting for a total increase of 8% per brood frame. An explanation of the latter could be that the bees were improving their anti-varroa grooming efficiency, or perhaps recruiting progressively more workers to the grooming force.

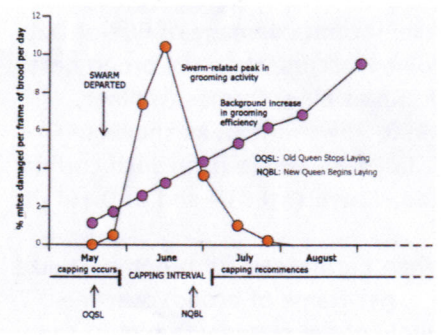

Figure 8. Analysis of pattern of percentage of damaged mites per brood frame.
The data in Fig.7 is interpreted as having two components: a massive peak in the percentage of mites damaged soon after swarming (orange spots) and a gradual increase, accounting for a rise of ~8% over a period of 4 months (purple spots). The position of the peak relates to "the capping interval".

The third component relates to the almost complete correspondence between mite damage and brood nest size during Phase II (see Fig. 6). This is puzzling, but there is an explanation in similar terms. Emerged mites are always at great risk when in the phoretic state, and in a small brood nest newly emerged mites do not have to look far for the new safe haven offered by a brood cell about to be capped. In a small brood nest they are therefore at minimal danger of being caught and killed by house bees. As the brood nest expands, mites have to travel ever further to find suitable hiding places and so are in ever greater danger of being caught and killed. This could explain why the proportion of injured mites is directly proportional to the size of the brood nest (Fig. 6).

Recap

This analysis so far therefore suggests three components to the bees' anti-varroa grooming behaviour: the post-swarming peak, when the majority of the mites left behind are in, or soon progress into, the phoretic state and highly vulnerable to competent broodnest defenders; an increase in the effectiveness of grooming, presumably either due to improving efficiency by individual house bees, or to an increase in the number of bees capable of destroying mites by allogrooming (see below); and thirdly, the colony expansion phenomenon, when mites have progressively increasing difficulty in finding their next vacant brood cell as the broodnest enlarges.

Entombment

A curious feature I did not notice at the time is that this broodnest always contained at least three frames of brood, when none would be expected at ~4 weeks after swarming (the reported value of four frames at DP 5 is the mean of three readings; see Table 1). My only explanation at present is that three frames probably contained brood that never emerged, possibly because it was dead due to infestation by mites. A corollary of this deduction is that the bees in colony JB5 probably did not impose varroa sensitive hygienic (VSH) behaviour (see below).

VSH is one of the most potent means of defence shown by some colonies, when house bees uncap parasitized pupae and eject them, together with the mites in the same cell. But if JB5 does not perform VSH, this suggests another means by which they, albeit inadvertently, combat varroa. Absence of VSH behaviour, combined perhaps with a greater susceptibility of the brood to parasitism, could mean that some mites do not emerge or re-emerge from brood cells and remain entombed in the brood nest. Absence of VSH behaviour could therefore constitute a fourth means by which these bees escaped further harm by the mites.

Brood breaks

When the queen goes off lay (e.g. in the context of swarming) this presents two advantages to the colony with regard to their battle with the mites. About ten days after she ceases to lay there is a dramatic absence of cells containing larvae about to be capped, in which female mites would normally hide until the young adult bee emerges. Cessation of laying by the queen therefore induces a brood break for both the bees and the mites. In addition, because their normal hiding places are denied them, the mites remain in the phoretic state, feeding on the haemolymph of the house bees, but in a grooming colony, openly exposed to attack by those house bees. So, in colonies with defensive grooming, a bee brood break creates not one, but two obstacles to mite survival. The post-swarming mite brood break constitutes a fifth means of defence.

Northumberland bees typically also have an autumn-winter brood break of two months or more and often also (but not in all colonies) a break of three weeks or so in July, corresponding to the June forage gap in the south of the country. Both of these present similar barriers to the progress of the mites, so that typical colonies would have one or two

brood breaks in addition to that associated with swarming. This raises the total number of circumstances when mites are or could be controlled by two or four, i.e. in total from five, to seven or 9nine!

Swarming

There seems to be a curious lack of curiosity within the bee research community as to the relationship between varroa infestation and swarming. You would think someone would ask: "does varroa infestation affect when or how swarming occurs?" Or, "does swarming have any effect on infestation by varroa?" My observations suggest both. Colony JB5 swarmed before raising queen cells, which is highly unusual; so were those swarm cells, or emergency cells? I am inclined to consider them the latter. Those queen cells were raised because the colony recognised the absence of a queen, not the need to swarm.

Figure 9. British native dark bees, Apis mellifera mellifera L. from the author's apiary. The bees are in a cast and in "testudo formation" following spraying with water. Their native (or near-native) status is deduced from body colouration, tomentum width and colouration, abdominal hair length, wing morphometry, brood nest pattern and behaviour, confirmed in some cases by mitochondrial DNA analysis.

As to the second issue, in the case of JB5, swarming certainly affected the varroa population and indeed dealt it a death blow in the parent colony. I have no knowledge of what happened within the swarm. Fries and Camazine (2001) wrote that colonies which had swarmed shed fewer mites later in the season than those which had not swarmed, a finding echoed by Seeley and Smith (2015), but in neither case was an explanation offered. Were those mites that fell damaged? If they were, that suggests the reduced numbers of fallen mites were due to their reduction by grooming by the bees; if they were not damaged, that would perhaps suggest reduced reproduction of the mites.

We go to great trouble to prevent our bees swarming, but perhaps if we tried less diligently, they would be generally more effective at mite control.

Emergency defenders

There is documented evidence of specialist allogroomers (i.e. house bees who groom other bees) in some hives, one ("Red 93") being recorded as spending 84% of her life just grooming her nest mates while others of her age cohort averaged only 15% (Moore et al, 1995; Kolmes, 1989). She began this activity at seven days after emergence. There are also reports of "hit-squads" of such specialist groomers that rove the brood box on missions of extermination (see Pritchard, 2016). They would be the most potent agents of mite demise.

During their development alongside pupating bees, varroa mites incorporate bee proteins and other materials into their external integument. This creates an odour camouflage that renders them difficult to identify. Allogroomers however can detect them in two ways. Their attention is first attracted by a nest mate struggling to scrape a mite off its own body with its legs, this action being interpreted as a "grooming invitation dance" to which groomers respond. Dedicated allogroomers also seek out mites by palpating the abdomens of their nestmates. They then pull them off using their own legs and mandibles and dispatch them with the latter. It is only by use of the mandibles that serious harm can be inflicted and their assault is concentrated on the forelegs of the mites, to a lesser extent on the other legs and the idiosoma (see Pritchard, 2016). Major loss of haemolymph ensures their death.

Arnold Desandre (2017) objects that evolution proceeds too slowly for honey bees to develop mite resistant behaviour in just a few seasons. In this he's probably wrong, but the bees' solution is quite different from what that statement implies. It's not a question of evolving a new capacity, but of a colony under stress transiently creating these specialist "emergency bees" that deal specifically with the problem causing their stress.

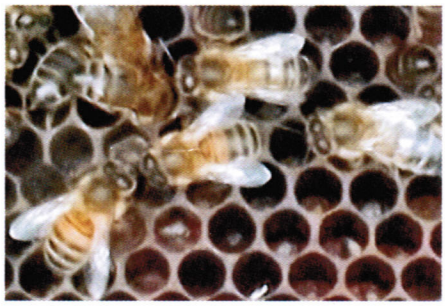

Figure 10. The most common obviously hybrid bees seen in the author's apiary. This shows ginger banded workers, as described in Colony JB5.

Bak and Wilde (2015) carried out a comparison of autogrooming behaviour in *A. m. caucasica*; *A. m. mellifera* of the Polish Augustowska line and an *A. m. capensis* - *A. m. carnica* hybrid. The *A. m. mellifera* bees showed by far the strongest autogrooming tendency, 98% of its workers making serious attempts to dislodge mites placed on their own thoraces. Frustrated autogrooming behaviour is similar to what has been considered a "grooming invitation dance" (see Pritchard, 2016). In responsive nestmates this triggers mandibular allogrooming, which can result in lethal injury to the mites they attack. Responsive nestmates may not be present in significant numbers when the colony is unstressed, but are generated from normal young bees as a consequence of the unusual stress.

The subspecies and ecotypes of *Apis mellifera* have evolved a variety of emergency behaviours towards which young bees can be directed to cope with stressful situations that arise from time to time such as shortage of water, the need for undertakers and invasion by parasites and competitors (see Kolmes, 1989; Moore et al, 1995). Such defensive behaviours develop as a response to

those stresses, in the physiological context of changing titres of juvenile hormone, creating transient sub-populations of specialist "emergency bees" that work singly, or in collaboration, to remove the agent that is causing the stress. I have seen films of mites emerging from under pupal caps as the young adult bee cuts her way out, and they move fast! It takes a speedy bee to catch them, but they floor them in a flying tackle, bite them and rapidly drop them. This is another form of defence by bees against mites, but not necessarily involving grooming.

Conservation of defence related genes

As a result of human management designed to override life threatening emergencies, many of the responsive genes may well have been lost from the genetic repertoire, especially in commercially managed stocks. It is our duty to future generations of beekeepers to ensure that these ancient defensive behaviours are retained and not sacrificed to well-meaning, but misguided "care" by their owners. I suggest that their retention with regard to varroa resistance requires us to discontinue management practices that artificially reduce the numbers of mites in our hives.

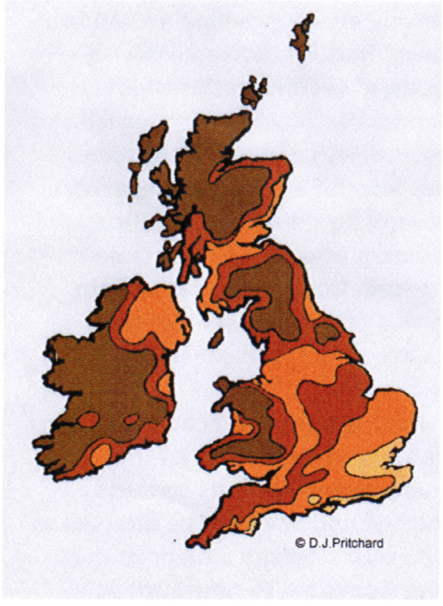

Figure 11. The distribution of 'nativeness' in British bees.
This map shows the frequency distribution of native wing morphometric characters in Britain in the 1960s (redrawn from Cooper, 1986, with permission.)

The ten "classic" mite-resistant populations (see Locke, 2012) illustrate that a capacity to develop resistance is present in honey bee populations throughout the world and these often utilise a combination of inherited behaviours. Where such assemblies exist, however, performance of the resistant behaviour may still require the appropriate pressures for it to be brought into operation. This probably involves actual varroa infestation and this at a level that threatens their extinction. If that is the case, then artificial depletion of mites with acaricidal chemicals may mean that neither selection of resistant alleles, nor their triggering into expression will occur, and the colony will be dependent on acaricides for its continued survival. It would seem that allowing the mite population to build up to massive levels is what is needed to bring emergency bees into existence. This then allows the colony itself to take control and exterminate the mites by natural biological means.

I suggest that in those geographical areas of Britain where native *A. m. mellifera* characters are well represented we might perhaps expect to find strongholds of varroa resistance, partly because these bees are well adapted to their home environment, but also because they have probably been "managed" to a lesser degree than imported strains. Mite resistant grooming and other emergency behaviours might be expected to be less common in the lighter coloured regions of the map (Fig. 11), where the native component is poorly represented and imported commercial strains may have lost their defensive instincts. However, the foreign literature abounds with accounts of subspecies other than *A. m. mellifera* resisting varroa in a variety of ways and to varying degree (Locke, 2016; Pritchard, 2016), so there may be greater scope than I am suggesting for foreign bees now found in Britain to cope with varroa.

The COLOSS survey

I have just read a recent perspective on the COLOSS survey of honey bee survival in non-medicated colonies (Büchler & Uzunov, 2016). The striking overall conclusion was that in apiaries tested all across Europe, in the face of varroa, when medication was withdrawn entirely the local bees survived best, on average 83 days longer than foreign introductions. With my mind-set, something like this is just what would be expected, but what struck me much more forcibly was the pathetically tiny proportion of colonies that managed to survive at all to the end of the experiment at 30 months. Among exotic stocks it was an abysmal 7%, among natives it was more than twice as high, but still only 19%! We therefore have to recognise that without medication, fewer than 10% of foreign and 20% of native European colonies are capable of survival for even as little as two and a half years! My locally adapted native and near-native British *A. m. mellifera* have been going for 16 years now with varroa all around them, but without medication — and they are very healthy!

Gareth John (2016/17) withdrew treatment from his collection of mixed-race feral colonies, but did nothing more. He wrote: "thus, without interference from myself, over the course of the last five years, it seems the bees in my apiary have increased their varroa resistance and have also increasingly expressed the characteristics of native British bees. That which I thought might not be possible turned out to be entirely possible and has been achieved not by me actively breeding but by the bees breeding themselves ... all I had to do was to have the courage to trust them."

Conclusions

The British beekeeping literature overwhelmingly asserts that honey bees are virtually powerless to resist varroa mites, but, on the contrary, my observations over 17 years indicate that locally adapted native and near-native British honey bees can easily overcome mite infestations and may use as many as nine or ten "strategies" to do it (and that is ex-

cluding VSH). This interpretation has very important implications for the health and survival of honey bees in general. Moreover, I believe it is more a matter of intelligent management than genetic selection.

A healthy native honey bee colony should be a vigorous, dynamic and essentially self-sustaining community. Maintaining it in good fettle is less like running an old folks' care home (as most advisers persuade us!), more like training a rugby team! You have to charge your team with the responsibility to look after themselves — then trust them to get on with it! If some colonies don't make it, those are not the ones you wanted anyway. But in my experience, although varroa presents honey bees with a challenge, it does not kill them if they are left to work out their own solutions. And their strength in that endeavour gets reinforced by success.

My approach of aiming to reinstate something like natural selection may sound tough, but it works. If you eliminate chemicals and restore natural selection, perhaps you could enjoy bees like mine and beekeeping would again be just like it was in the old days.

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Addendum:

The Probable Sequence of Events

Low grade mite infestation.
↓
Death of "dark" brood and their mites, but survival of infested hybrid brood.
↓
Occupation of all brood space in brood nest.
Absconding of old queen and many bees taking phoretic mites with them.
↓
Emergency queen cells form during 9-day extended brood break.
Mites remain in the phoretic state, unable to reproduce.
↓
Attacks by phoretic mites on young bees induce hormonal changes in them, triggering auto- and allo-grooming behaviour.
Bees attack phoretic mites, killing them in massive numbers.
↓
Re-commencement of egg laying.
8-day brood provides hiding places for mites.
Many potential allogroomers are created from new brood.
As brood nest expands, new havens become ever more distant, allowing increased number of allogroomers to catch and kill progressively more mites.
↓
Mite reproduction reduces and eventually fails altogether.
Colony returns to normal and the number of allogroomers is reduced.

It is suggested that in normal native "dark" *A. m. mellifera* colonies of the north of England, pupal stages are hypersensitive to infestation. Low grade mite invasions are therefore arrested by the early death of infested capped brood, providing a high basic level of resistance. Phoretic mites are also destroyed by aggressive grooming by house bees, so that these colonies are virtually completely resistant to *Varroa destructor* and the viruses it carries.

Note
An additional explanation for the close correlation between percentage of mites damaged and size of the brood nest in Phase II is that the number of newborn bees, including potential allogroomers, would increase and decrease strictly in proportion to brood nest size (Fig. 6).

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