Know thy enemy and know thyself, find naught in fear for 100 battles. Know thyself but not thy enemy, find level of loss and victory. Know thy enemy but not thyself, wallow in defeat every time.

-- Sun Tzu, The Art of War

Last month we talked about a threat to honey bees that, in my opinion, has been blown way out of proportion (Love a Beetle?). Small hive beetles are easy to see and fun to squash but are easily kept in check and do not cause the demise of strong colonies. This month we'll discuss an underappreciated threat, one that every competent apicultural authority will agree is the single most important cause of unnatural death to modern honey bee colonies. But the culprit is not easy to see, both because it is small and it is also elusive. This fact, I believe, naturally leads to apathy and neglect.

Just as it took many years of community outreach to convince The Public that unseen germs cause disease, misery and death, it also takes an education campaign to inform beekeepers about their greatest foe, the varroa mite. Realization of what it takes to control this enemy can lead to a better understanding of who you are as a beekeeper (e.g., bee-*keeper* versus bee-*haver* versus bee-*killer*?). Put the two understandings together and Sun Tzu says we'll be unstoppable.

Where did they come from?

It is important to understand the origin of the varroa mite (varroa destructor) in order to appreciate its threat to European honey bees (Apis mellifera). This mite is a natural parasite of the Asian honey bee, Apis cerana. In its native habitat, Apis cerana is the obligatory host of varroa destructor; the mite cannot survive without the bee. Over eons, the mite has evolved such that its reproductive physiology is precisely tailored to fit within that bee's life cycle. It is a successful parasite, in that it co-exists with Apis cerana without killing the host. It only parasitizes Apis cerana drone larvae and there are never so many mites that they cause any problems for the colony.

The mite was first identified on Apis cerana in 1904. Nobody cared about it because it was only a harmless parasite on its host. However as the movement of bee colonies greatly increased in the latter half of the 19th century and throughout the 20th century, the range occupied by European honey bees (Apis mellifera) and Asian honey bees (Apis cerana) began to overlap along the China-Russia border. At some point the mite was able to jump hosts and successfully survive on Apis mellifera. By the mid 1950s, massive bee losses were being witnessed in that region. No one could explain them until 1965, when a Chinese researcher, Jan Tsinhe, discovered the connection between the mite and colony deaths.

By this time the mite was racing through Europe as fast as commercial beekeepers could carry them. Czechoslovakia and Romania responded by strategically destroying 30,000 hives between 1980 and 1983, but that had no impact on the mites' spread. The pest quickly moved into Macedonia, Greece, Yugoslavia and Lower Austria, into Central Europe and Italy. It was in Germany even earlier; it was found in hives there in 1975. Now it is found everywhere in the world with the notable exception of Australia.

The mite was first discovered in the US in September, 1987 in hives transported from Florida to Wisconsin. They had most likely hitchhiked on bees illegally smuggled here in direct violation of US law. The US has been closed to the importation of honey bees from most countries since 1922, expressly to prevent this type of crisis. The overall ban has worked for many years, but the varroa mite issue illustrates that a tiny leak through callous disregard for both the law and common sense can cause catastrophe.

By spring of 1988, the mite had been found in 12 states. It was detected in North Carolina in 1990. All attempts at eradication or quarantining the pest failed.

Perhaps the most significant impact of the varroa mite has been the elimination of feral (non-managed) honey bees in the United States. Without treatment, colonies do not usually survive more than a year or two. The feral bees we see these days are recent escapes from managed hives, not bees that are part of a self-sustaining "wild" population. This is why backyard gardeners can no longer depend on free and abundant honey bee pollination like they used to be able to thirty years ago.

Life cycle

The chart on the last page illustrates the life cycle of varroa mites. The females travel around on honey bees, frequently moving from one to another whenever they get a chance, for example when two bees rub against each other while foraging on the same flower. They'll do this for about a week between baby-raising sessions. This behavior, coupled with drifting and robbing by bees, ensures that the mites spread far and wide. The mites feed on the host bee's hemolymph (bee blood) while they are riding it.

When the time is right, a female mite drops off a host nurse bee into the cell of a bee larva that is almost ready to be capped. To hide, she'll bury herself beneath the brood food in the bottom of the cell, breathing through a snorkel-like appendage called a peretrime. Once the cell is capped and the prepupa bee spins a cocoon, the mite cuts an oozing gash in the young bee's integument. This will feed her and her offspring during their stay in the cell.

The female first lays an unfertilized egg which develops into a male. Then at 30 hour intervals, she lays fertilized female eggs which take six days to mature. The mature male offspring mates with his mature sisters. When the host bee fully matures and emerges, the original female and all of the female offspring that have had time to mature emerge also. The male and all immature females remain and die in the cell. The original and new females ride around on bees until starting the process again. A female mite can do this over and over for six months or more.

Figure 1 illustrates the time constraints for this process in both Apis mellifera (A.M.) and Apis cerana (A.C.). Note that on Apis cerana worker brood, mites do not have enough time to create mature offspring. Therefore the mites do not infest Apis cerana worker brood, only drone brood. However with Apis mellifera, workers pupate a day longer, giving mites a chance to successfully reproduce. As with Apis cerana, Apis mellifera drone pupae are still best and are preferred by mites, but worker pupae serve the purpose quite well too.

Figure 1. Mite and bee development timeline

Hour	Bee	Mite
1	Spins cocoon	
5		Foundress female (FF) begins feeding
70		FF lays **male** egg
100		FF lays **female** egg
130		FF lays **female** egg
160		FF lays **female** egg
190		FF lays **female** egg
210		FF lays **female** egg
240		FF lays **female** egg
264	A.C. worker emerges	
274		First female matures
288	A.M. worker emerges	FF and 1 mature daughter exit
304		Second female matures
334		Third female matures
336	A.M., A.C. drone emerges	FF and **3 mature daughters** exit

(Data from Z. Huang, Varroa Mite Reproductive Biology.)

Geometric versus arithmetic growth

Figure 2 shows a simplistic comparison of the relative growth in bee population versus varroa population over five bee brood cycles (15 weeks for workers). Note that honey bees do not live 15 weeks but varroa mites can live much longer, so the actual bee population is overstated in the graph. The figures are based on varroa reproduction on worker bee brood; reproduction on drone brood is significantly greater.

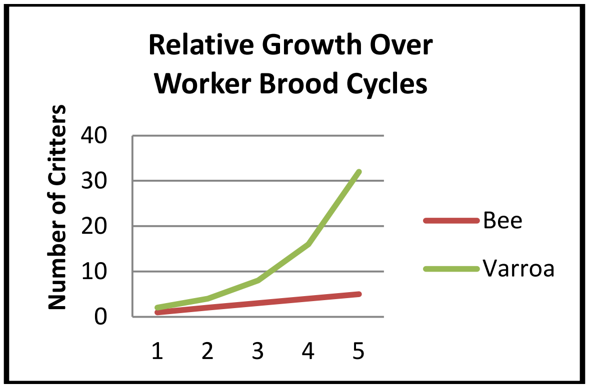

Figure 2. Relative growth in bee versus varroa populations

The conclusions are clear regardless of the nuances of the graph's assumptions: left unchecked, varroa population growth must overwhelm the honey bees over a fairly short time.

Why do they harm our bees?

If vampire bats were to bite a slash in your baby and drink its blood throughout its crib stage, that wouldn't be a good thing. The physical trauma and loss of essential body fluids would certainly reduce body weight and vigor.

This happens to our bees when infested by varroa, however it isn't the main source of harm.

The real devastation comes from the viruses that mites transmit and activate. The mite acts like a tiny hypodermic needle that travels from victim to victim without being sterilized in between. Remember how the AIDS virus swept through the drug-using community in exactly the same manner? This is what happens in our colonies. Just a few of the deadly viruses that mites vector include Deformed Wing Virus, Chronic Bee Paralysis Virus (responsible for Hairless Black Syndrome), Israeli Acute Paralysis Virus, Kashmir Bee Virus and Black Queen Cell Virus. As more and more virus-ridden mites attack more and more bee pupa, the viral loads increase higher and higher within the mites and the bees. If the viral loads in your colonies are high enough for you to see the symptoms of any of these viruses, go ahead and order replacement packages for next spring. At that stage, getting rid of the mites is like closing the barn door after the cows have already devoured your flower patch.

Treatment philosophy

The philosophy behind varroa treatment has evolved over the past 25 years. When varroa first hit the US hard in the 1990s, beekeepers aggressively treated their bees with the only things that were available: fluvalinate (Apistan) and coumaphos (Check-Mite). This action saved the American beekeeping industry.

Unfortunately these remedies weren't perfect. The mites developed resistance to them and the chemicals lingered in the wax. More recently, treatments with more appealing profiles were developed. The most notable are the two thymol-based products, ApiLife Var and ApiGuard, and a formic acid treatment, MiteAway Quick Strips. These are highly effective, reasonably easy to use and are minimally disruptive to the hive when the directions are carefully followed. They are also very affordable for hobbyists.

With better treatments available and fearing that resistance would develop as it did with the earlier treatments, beekeepers were advised to only treat their hives when mite levels became very high or symptoms developed from varroa-borne viruses. As we have gained even more practical experience and knowledge concerning varroa and associated diseases, wise beekeepers are once again treating their hives in a more proactive fashion before varroa infestations get out of hand in the first place. This isn't a complete return to the original treatment philosophy of hit 'em hard and hit 'em often, but instead is an adjustment to the counter-philosophy ("only treat when losses are eminent") that proved ill-advised. Beekeepers should still assess the level of infestation before applying treatments, but a level that was considered acceptable several years ago is now once again viewed as a threat.

The bottom line

Left unaided, how can our bees win against mites? I hope that it is obvious that they cannot. Those who think we should let nature run its course and the result will be a mite-tolerant bee are starting from a false premise, namely that the honey bee must somehow survive. That didn't prove true for the passenger pigeon, ivory-billed woodpecker, Carolina parakeet or the dodo bird; they totally succumbed when an exotic predator came on the scene. Sheep have been prey to wolves for thousands of years and haven't yet developed tolerance to being eaten. Why would we think that honey bees should be any different?

Now that we understand the issues, what do we do about it? Specific treatment options have been discussed previously (see How Might We Smite Mites?). All that is left is for each of us to understand what kind of beekeeper we want to be: one who wants to keep varroa mites and feed them bees to ensure that they thrive, or one who wants to keep healthy and productive bee colonies and realizes varroa mites cannot be part of that equation. Once we know the enemy and know ourselves, we will "find naught in fear for 100 battles."!

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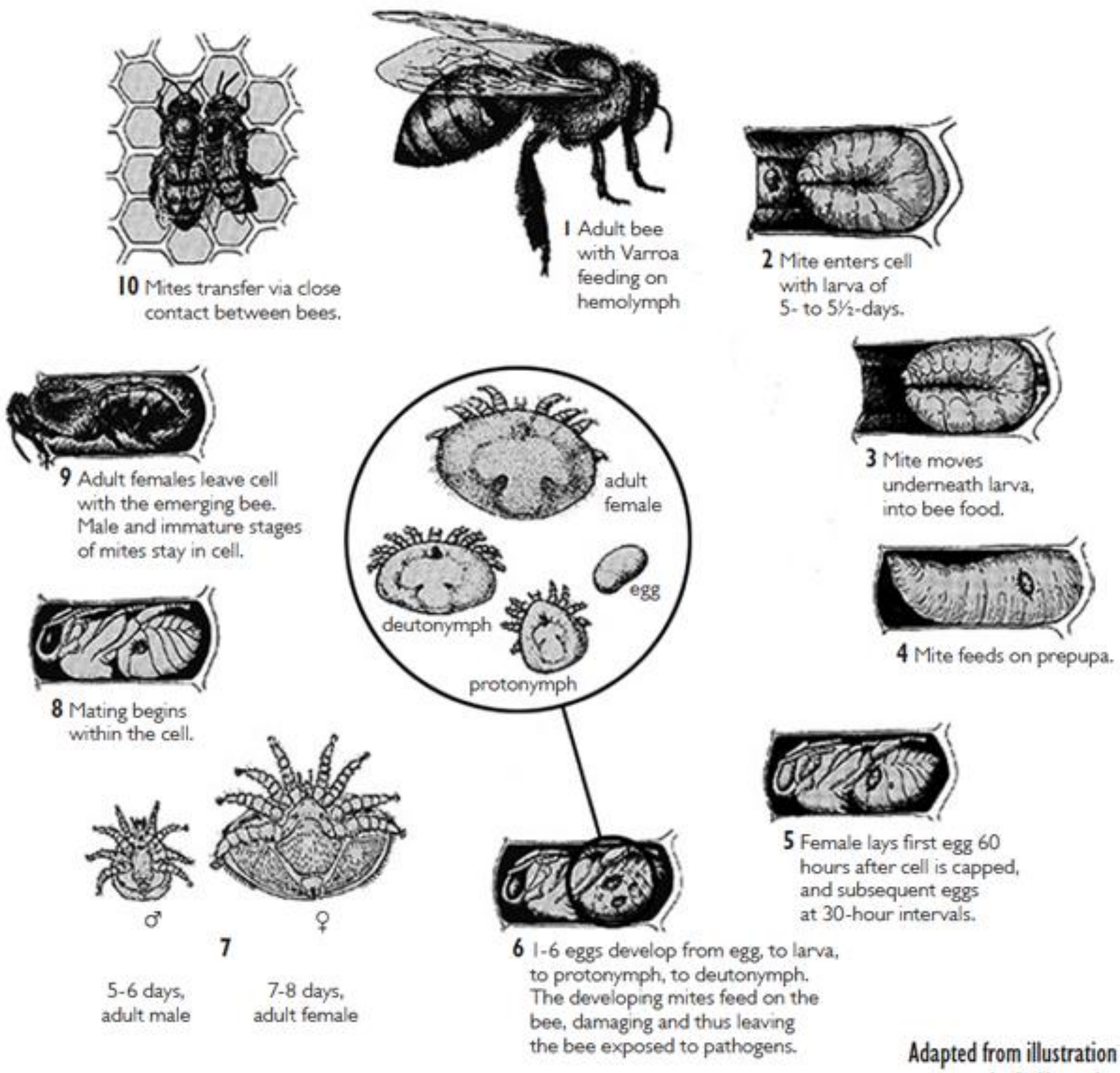

Adapted from illustration by B. Alexander