THE AFRICANIZED BEES OF BRAZIL HAVE BECOME TOLERANT TO VARROA

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Brazilian apiculture grew slowly since the beginning of this century. Bees of European races were kept, and these often suffered from the effects of bee diseases. During the 40s, before African bees were introduced, Brazil was not an important honey producer. The total annual yields were quite low (around 5,000 tons of honey per year) and Apiculture in the country could be described as a kind of sideline activity, with apiaries that were not moved to take advantage of seasonal flowering. In general, beekeeping was not an economically important activity, despite the strong European influence, especially in the southern states. During this period the first problems with diseases were reported and characterized. The Nosema disease (*Nosema apis* Zander) was found in the bees. A few years later, around 1950, another problem, the Acarine disease (*Acarapis woodi*) was also found to debilitate the bees. Soon afterwards, the first cases of European Foulbrood (*Melissococcus pluton*) were recognized. Among these disease problems, only the latter is still reported, but it is not considered important.

The finding of the parasitic bee mite *Varroa jacobsoni* in Brazil in 1978 (MORSE and GONÇALVES 1979) was a great shock for apiculture in our country. It was apparently introduced into Paraguay, from Japan, about 1969, and from Paraguay into Brasil (by a beekeeper) in 1972 (DE JONG and GONÇALVES 1981). Soon after, in 1979 and 1980, researchers from the Genetics Department of the Faculty of Medicine of the University of São Paulo in Ribeirão Preto, sampled colonies in apiaries throughout São Paulo state, and found infestations that ranged as high as 50 mites per 100 adult bees.

This mite was greatly feared throughout the world, as it had already killed hundreds of thousands of colonies in Eastern and Western Europe. There was an immediate concern that there would be serious damage to beekeeping in Brazil. In 1979 Brazilian technicians and scientists attended the APIMONDIA meeting in Athens, Greece, hoping to find out what steps should be taken to control this new mite pest. Several chemical controls were already available, and companies saw an opportunity for a huge new market in Brazil. However, before allowing the importation of tons of chemicals to treat the more than two million colonies in Bra-

zil, Brazilian scientists determined that it would be best to test samples of the chemicals offered to see whether they would work under Brazilian conditions.

It was decided to make the tests in one of the universities (UNESP in Rio Claro), while another institution (USP in Ribeirão Preto) would concentrate on biological research. An attempt was made to determine the damage that the Varroa mite caused to the colonies, so that a proper pest management strategy could be generated. Colonies with and without Varroa were monitored for growth parameters and honey production to determine the economic injury occasioned by the mite infestations.

The first chemical control tested in Brazil, SINEACAR, which came from Romania, was found completely ineffective (STORT et al., 1981). Jokes were made that the mites were eating this powdered medicine. Other of the early chemical controls tested included tobacco and formic acid. These had varying degrees of effectiveness, but they had to be adapted to the hot, often humid conditions found in Brazil. Tobacco was time consuming and difficult to apply, since effective doses also knocked down many bees in the hive, and formic acid was both dangerous for the beekeeper handling this very reactive acid, and for the colonies, since a turned over flask within the hive could result in death of he colony.

The big surprise came when we tried to determine the rate of infestation which would cause colony damage and death. In fact we did not find that any colonies died, nor was it clear that there was damage detectable at the colony level, in terms or reduced bee populations or honey production. Reports from Europe indicated that the infestation levels in the colonies increased gradually during two to four years, finally resulting in massive dieing off of colonies. We found some high infestation levels in the first years, with as many as 50 mites per 100 adult bees in some colonies (DE JONG et al., 1982), however the rate of infestation fluctuated during the year, and instead of gradually increasing to an insupportable level, was actually decreasing, so that by 1983, the mean infestation rates were around five mites per 100 bees. In that year, five years after the discovery of the mite in Brazil, and about 12 years after the mites were apparently introduced, we still had no confirmed death of a colony due to Varroa infestations (DE JONG et al., 1984).

In the meantime the beekeepers were anxiously awaiting a solution. We had decided that we would wait until we had confirmation of deaths of colonies, or at least damage levels that would justify the expense of treatment. When we had such a situation, that would justify treatment, then we would advise the beekeepers on the best of the control solutions, among those we had tested in Brazil. In fact, as we saw that the infestation levels were actually going down, in-

stead of up, we began to suspect that the long awaited day for the beginning of a national control effort against Varroa would never come.

Though we could see no colony-level damage, virtually all colonies in infested regions were in fact infested. So we began to look for other ways of determining the economic impact of Varroa on the bees. Individual bees which were infested during their development period, had a reduced weight at emergence, weighing on average 6% less when infested by a single female mite, and about 10% less with two female mites (DE JONG et al, 1982). Yet few of these bees had the damaged wings and crippled legs that were widely reported from damaged colonies in Europe (RITTER, 1981). A further investigation of bees infested during development revealed that such bees had a shorter life span. Bees infested with a single mite lived a mean of only 19 days, compared with a normal life span of 30 days. Those which hatched from brood cells, together with two or more mites, lived a mean of only about five days (DE JONG and DE JONG, 1983).

Since we had no way of determining economic injury levels according to infestation levels, we made an estimate based on the shortening of the life span of the bees. On average, the bees which had been infested while in the brood stage lived about half as long as normal bees. So we estimated that a given percentage of infestation, would effectively reduce the colony population by half of that percent. So, with an average infestation rate of 4% around 1983/84, we estimated a population loss of about 2%. Assuming a linear effect on honey production of this population loss, and with an average production of 30 kg of honey, we could anticipate a loss of about 600 g of honey per colony. At an average beekeeper price of US$2 per kg of honey, that would result in a loss of $1.20 in income per colony. Since that value was less than the cost of the controls available, without even accounting for labor and transportation costs, as well as deleterious side effects on the bees, and contamination of the colony products and of the beekeeper himself. Additionally there was evidence that the mite populations were gradually declining, and might decline even more. Treatments could have interfered with this tendency towards adaptation of the host-parasite relationship. MORETTO et al. (1993), working with Africanized and European bees in Brazil, found that the Africanized bees were seven times as efficient as the Europeans at ridding themselves of the mite *Varroa jacobsoni*, when they were artificially infested. So we could conclude that the treatment alternatives available were not suitable for Brazilian conditions, and could not be justified on an economic basis (DE JONG et al., 1984).

Though the damage level on a colony basis was low, it added up to a considerable sum when we consider that there were over two million colo-

nies in the country. Two million times US$1.20, comes to US$2,400,000 damage per year. This level of damage justified a continued research program concerning *Varroa jacobsoni*, and the mechanisms of natural control in the Africanized bee colonies.

Currently, in 1997, we find that mean infestation levels are less than two mites per 100 adult bees. It is extremely rare to find colonies with as high as 10% of the brood infested. Often we need to open hundreds of worker brood cells to find a single mite. Initially we were concerned about the cooler regions of Brazil. As it was found that both bee race and climate has an effect on the infestation rates (MORETTO et al., 1991). ROCHA & LARA (1994) found that the infestation rates were highest during the coldest months in Brazil, and lowest during the hottest months, with a negative correlation ($r = 0.6474$, $p < 0.01$) between temperature and rate of infestation by Varroa. Some beekeepers in the southernmost states of Santa Catarina and Rio Grande do Sul indicated that they had very high infestation levels. In fact we could see that infestation levels increased as we traveled south to southern Brazil, reaching critical levels in the Pampas region of Argentina (DE JONG et al., 1984). As we wondered how the degree of Africanization of the bees in these different regions might affect the Varroa infestation rates, we decided to test Africanized and first generation European / Africanized crosses in a warm region of Brazil (Ribeirão Preto, São Paulo State) and in one of the coolest regions of the country (São Joaquim, Santa Catarina State, 1600 m altitude), where it sometimes snows. We found that the infestation levels were much higher in the cool region, even though we had reared and mated all the queens in the same apiary. The infestation rates in São Joaquim reached 25 mites per 100 bees, while in Ribeirão Preto, they stayed around 5 mites per 100 bees. This led us to fear for some of the cooler parts of the country (MORETTO et al., 1991). However when we examined these and other regions in the south in subsequent years, we found that the mean infestation levels were going down (MORETTO et al., 1995). We had the impression that the relative tolerance of the bees appeared first in the warmer regions, and later reached the cooler areas. As the Africanized bees have a shorter brood development time (about 24 hours shorter), this probably contributes to the relative resistance to or tolerance of *Varroa jacobsoni*, since many of the female offspring do not have time to reach the adult stage and figure as lost reproduction. About 50% of the female mites that invade worker brood cells in Africanized bees in Brazil do not reproduce, as opposed to 10 - 20% in European bees (RITTER and DE JONG 1984; ROSENKRANZ and ENGELS 1994).

Varroa jacobsoni was discovered in Brazil nearly 20 years ago, and apparently has been in the country for 25 years. No one has treated colonies

except on a very limited experimental level, and perhaps a few small scale beekeepers in the south who treated in the early 80s, influenced by their cultural ties with Europe. Nevertheless, even they have ceased such efforts, as they realized it was not necessary.

We can still find Varroa in all, or nearly all honey bee colonies in Brazil, but at very low infestation levels, that do not cause detectable colony-level damage. Although infestation levels initially increased to high levels, they fell to below 5% of bees infested within 10 years after introduction, and just three years after were found in our university apiaries in Ribeirão Preto. Happily, Varroa did not cause economic damage to beekeeping in Brazil. The present annual production is about 40,000 tons of honey. Beekeeping continues to expand into the central, northern and northeastern regions of the country. We can therefore conclude that the Africanized bees in Brazil have become tolerant of Varroa, and that this 'evolution' towards tolerance occurred in a short period of time. Furthermore, we can conclude that honeybee colonies can become tolerant to Varroa with little or no differential colony mortality, implying therefore that selection for tolerance was driven essentially by differential reproduction.

Acknowledgements

Much of the work done in Brazil, cited in this paper, was funded by NSF from the USP, CNPq, FINEP, FAPESP from Brazil, and Probral Project of CAPES-MEC (Brazil) and DAAD from Germany.

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