Apidologie 37 (2006) 67–74
© INRA/DIB-AGIB/ EDP Sciences, 2006
DOI: 10.1051/apido:2005053

Original article

Mortality of mite offspring: a major component of *Varroa destructor* resistance in a population of Africanized bees[1]

Luis MONDRAGÓN[a], Stephen MARTIN[b], Rémy VANDAME[a]*

[a] El Colegio de la Frontera Sur, Línea de Investigación "Abejas de Chiapas", Km 2.5 carretera Antiguo Aeropuerto, 30700 Tapachula, Chiapas, Mexico
[b] University of Sheffield, Department of Animal and Plant Sciences, Western Bank, Sheffield, UK

Received 22 November 2004 – revised 30 May 2005 – accepted 24 June 2005

Abstract – The role of *Varroa destructor* reproduction in the overall resistance to these mites by a population of honeybees in Mexico was studied. Since previous work showed the number of mated female offspring produced per foundress mites on worker brood (*Wr*) to be a main factor of resistance, we evaluated the changes in mortality of mite offspring in worker and drone brood throughout a year in groups of Africanized and Africanized × European honeybee colonies, which have a similar level of mite resistance. The variation in the *Wr* was highly correlated with the mortality of the first (male) and the third (female) offspring ($r^2 = 0.67$ and $r^2 = 0.53$ respectively). Moreover, the fecundity of mites in single infested worker and drone cells was greater than in multiply infested cells. The *Wr* and the *Dr* decreased between cells infested by 1 and 3 foundresses (from 0.9 to 0.4 and from 1.8 to 0.9 respectively). These data show that in the honeybee population studied, mite reproduction is strongly affected by offspring mortality during certain periods of the year, and that it is probably dependent on the foundress density.

***Varroa destructor* / *Apis mellifera* / mite reproduction / resistance**

1. INTRODUCTION

The ectoparasitic mite *Varroa destructor* Anderson and Trueman is currently the most important pest of the honeybee *Apis mellifera* L., causing the death of millions of colonies during the past 50 years. The only genetic line of *A. mellifera* which has consistently been found to be resistant to *V. destructor* is the Africanized honeybee (AHB) (Ritter and de Jong, 1984; Guzmán-Novoa et al., 1999; Vandame et al., 2000; Martin and Medina, 2004). Mite resistance was first observed in Brazil and the low level of mite fertility was considered to be the reason (Ritter and de Jong, 1984; Rosenkranz and Engels, 1994). More recently it was suggested that the less virulent Japanese mite genotype found in Brazil was the main reason for mite resistance of AHB (Anderson and Trueman, 2000). Three independent studies carried out in different tropical regions of Mexico (Veracruz, Vandame et al., 2000; Yucatán, Medina and Martin, 1999; Chiapas, Mondragón et al., 2005) showed that AHB colonies were able to survive without the application of any mite control measures, while the mite population cycled between 1000–8000 mites. In sharp contrast, European honeybee (EHB) colonies kept in the same region, died within a year because of the rapidly increasing mite population (Vandame et al., 2000). Because the mite genotype (Korean) and mite fertility levels were similar between EHB colonies in the UK and AHB colonies in Mexico, this suggests that other factors,

---

* Corresponding author: rvandame@tap-ecosur.edu.mx
[1] Manuscript editor: Stefan Fuchs

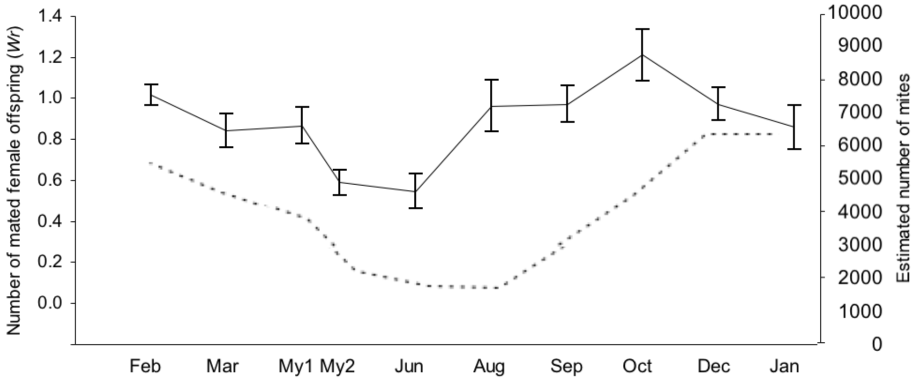

Figure 1. Number of mated female offspring per mother mite (*Wr*) and estimated number of mites per colony, in 18 honey bees colonies, from February 2003 to January 2004 (from Mondragón et al., 2005).

different from those found in Brazil, underlie the resistance of AHB to *V. destructor* (Guzmán-Novoa et al., 1999; Corrêa-Marques et al., 2003; Vandame et al., 2002).

Medina and Martin (1999) found that the number of mated mite offspring produced in worker brood cells per foundress during each reproductive cycle (*Wr*) in AHB from Mexico (0.73) was lower than in EHB from the UK (1.01). This difference was theoretically still insufficient to explain the seasonal cycling of mite populations around a constant average level in AHB, unless combining *Wr* with the density dependent mortality of mites in the drone brood (Martin and Medina, 2004). However, this hypothesis has not been tested yet. Mondragón et al. (2005) conducted a multifactorial study of the resistance mechanisms of AHB and mixed EHB × AHB colonies, showing that 73% of the yearly variation in the mite population could be explained by the variation in the *Wr* (Fig. 1). This was the first study to show the *Wr* cycling as a possible central resistance factor.

The aim of the present study was to unravel which aspects of mite reproduction may explain the yearly variation in *Wr*. This was achieved by a detailed analysis of mite reproduction and offspring mortality in worker and drone brood cells from the colonies previously studied.

2. MATERIALS AND METHODS

The study was conducted from February 2003 to January 2004 near Tapachula, state of Chiapas, Mexico (14°50'N, 92°16'W), a region with a subhumid tropical climate, mean annual temperature of 26 °C, and average yearly rainfall of 1346 mm. The rainy season runs from May to November. The nectar flow runs from December to April. The bees may collect pollen during the rainy season, particularly in August and September.

2.1. Honeybee colonies and *V. destructor* mites

In 1999, 30 feral colonies were sampled in the study region and were analyzed morphometrically using the USDA-ID 2.0 program (Rinderer et al., 1993). According to this multivariate analysis, the projection on the first discriminant axis had a mean factor of –2.12 ± 1.13, showing that the population was Africanized. Since no major introduction of bees in the region has occurred since then, the honeybee colonies used in this study most likely had the same level of Africanization as they did in 1999. Moreover, the colonies have never been treated to control *V. destructor* since the arrival of the mite in the region in 1994.

During 2003, the mite offspring mortality in 18 colonies (6 Africanized and 12 mixed Africanized-European colonies) was measured. The data from these 18 colonies were pooled for analyses because no significant differences were found in the reproductive variables between them (Mondragón et al., 2005).

2.2. Mite reproduction and offspring mortality

Every five weeks (except in May when two samples were taken), a sample (5 × 7 cm section approximately) of worker and drone sealed brood, close to emergence was collected from each colony. The

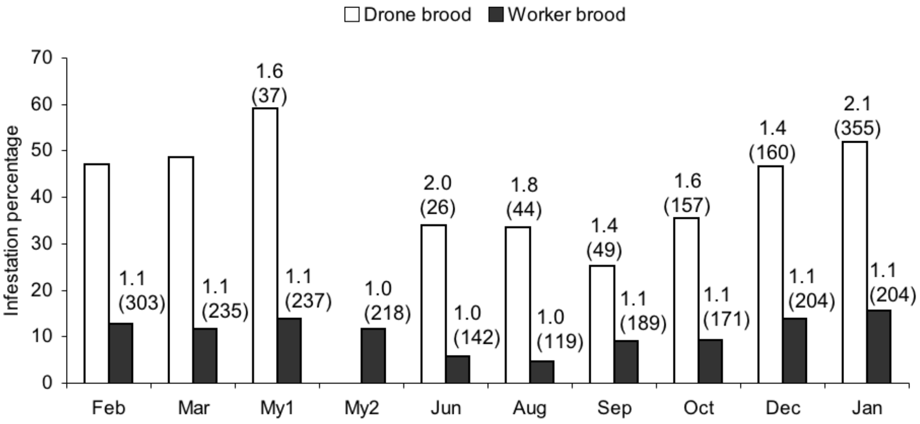

Figure 2. Mean infestation level in worker and drone brood during 2003. The numbers indicate the average number of foundresses present per infested cell; the sample size of cells analyzed is given in parentheses. These data were not collected for drone brood during the first two months.

comb sections were immediately stored at –5 °C to kill and preserve the pupae and mites. In the laboratory, 100 to 300 worker cells, and all drone brood cells were carefully opened and infestation level determined. The pupae and the complete mite family were removed from infested cells, and examined through a microscope. The age of the pupae were determined, and members of the mite family in each cell were arranged in birth order using the ontogenic developmental charts in Martin (1994, 1995a) and pictures in Ifantidis (1983). Mite offspring mortality was determined only in cells invaded by a single mother mite and in the mite families who followed a normal course of reproduction (Martin, 1994) and, to increase the accuracy of the data (see Medina and Martin, 1999), we used brood older than 270 h from cell capping in worker cells and 340 h for drone cells (molting stage). The offspring was considered dead if it was crumpled or shrunk. Foundress mortality was determined from all capped cells irrespective of time since capping.

The number of mated female offspring produced per foundress mite on worker brood (*Wr*) is the crucial value since it represents the average reproductive ability of the population of mites being studied. The *Wr* and the fecundity (average number of eggs produced per foundress mite) in single infested worker brood cells have been previously determined in the study colonies (Mondragón et al., 2005) and are shown again here (Fig. 1). In this study, *Wr* and fecundity were additionally calculated for multi-infested worker brood cells capped for at least 270 h and in single and multi-infested drone brood cells capped for at least 300 h (gray wing pads stage; Martin, 1994). The presence of the exuviae was used to confirm the adult stage of a male or female mite offspring. As mating takes place just after its maturation (Donzé et al., 1996), we assumed that mating had taken place, although this may overestimate the true number of fertile females as all males may not be fertile or mate successfully (Harris and Harbo, 1999).

3. RESULTS

3.1. Brood infestation

The infestation level of the sealed worker brood tracked the development of the mite population (Fig. 2), while the infestation level of the drone brood increased during the period when the mite population was declining, dropped sharply due to the lack of drone brood and rose steadily as the mite population increased again (Fig. 2).

3.2. Mite mortality

The mean proportion of dead foundress mites in worker cells was 3.5% ± 2.08% with 64% of them found trapped in the bottom of the cell between the cell wall and the cocoon. There was insufficient data to reveal any yearly pattern.

Mite offspring mortality was determined in 601 worker and 142 drone, singly infested, brood cells. The pattern of mite offspring mortality in both worker and drone cells was similar (Fig. 3). The mortality throughout the year in worker brood appeared to be variable, but with a tendency to increase from March to June

Figure 3. Annual mean mortality of *V. destructor* offspring in 601 worker (clear bars) and 142 drone (grey bars) brood cells of the 18 study colonies.

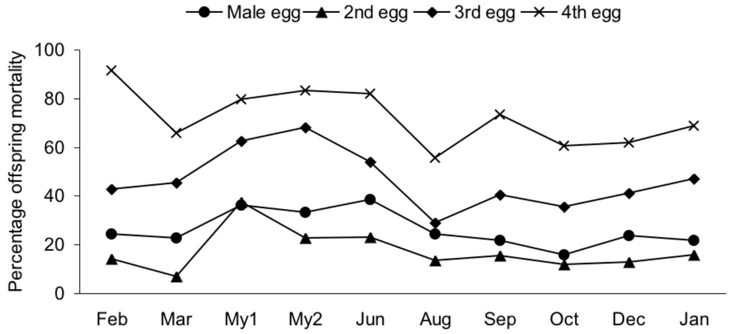

Figure 4. Mean offspring mortality throughout the year in 601 worker brood cells of the 18 study colonies.

(Fig. 4). A simple regression analysis made for each offspring (Fig. 5) showed that the correlation was significant between the first (male) and third offspring (second daughter) mortality and *Wr* (r^2 = 0.67; P = 0.003; n = 10 for the male; r^2 = 0.53; P = 0.02; n = 10 for the third offspring). This correlation was not significant for the second offspring (r^2 = 0.18; P = 0.2; n = 10), neither for the fourth offspring (r^2 = 0.22; P = 0.16; n = 10).

3.3. Effect of multiply infested cells on mite reproductive success

Using all stages of sealed brood, we calculated the number and reproductive success of foundress mites in 2131 infested worker cells and 336 infested drone cells. The average number of foundresses per brood cell throughout the year is shown in Figure 2. We observed a significant decrease in the fecundity, *Wr* and *Dr* when the number of invading foundresses increased (Tab. I). We are confident that there was no effect of the different bee groups (AHB and AHB × EHB) since there was no significant difference among them in the *Wr* (F = 0.23; df = 2, 2020; P = 0.8) or in fecundity (F = 0.32; df = 2, 2022, P = 0.7).

4. DISCUSSION

The central question of this work was why in the AHB and AHB × EHB colonies studied, the number of mated offspring in worker and drone brood (*Wr* and *Dr* respectively) was low and, in the case of *Wr*, cyclic. Solving this problem is fundamental as it may unravel a key aspect of the resistance of Africanized honey bees to *V. destructor*. We found two mechanisms that may explain these findings.

The first mechanism may be due to the increase in offspring mortality which led to a reduction in *Wr*. Figure 5 shows that the correlation found in this study between male mortality and *Wr* fits well with the data found in

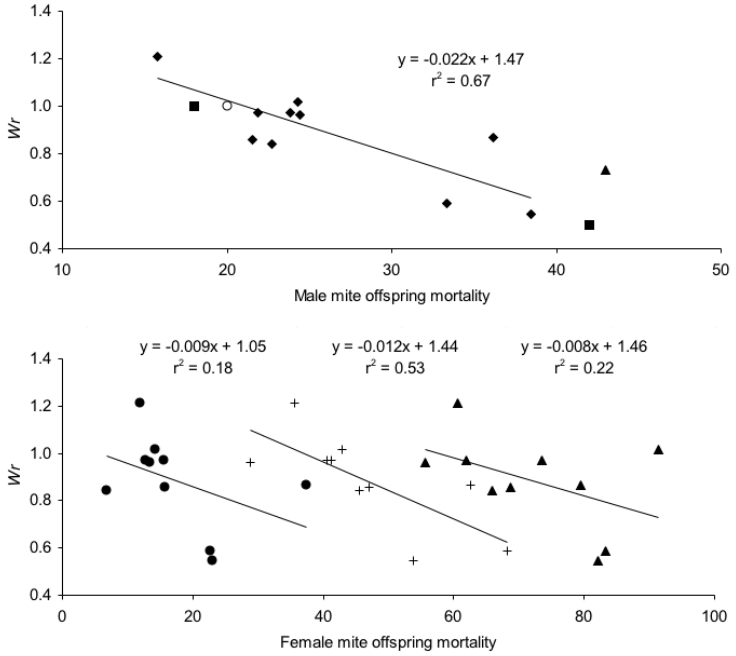

Figure 5. **(a)** Relationship between the mortality of the male mite offspring and the number of mated females produced per foundress mite in worker brood (diamonds), compared with data points from Medina et al. (2002) (squares), Medina and Martin 1999 (triangle) and Martin 1994 (empty circle). The regression line is fit only to the data from this study. **(b)** Relationship between the mortality of female mite offspring (2nd egg, circles; 3rd egg, crosses; 4th egg, triangles) and the number of mated females produced per foundress mite.

previous works (Martin, 1994; Medina and Martin, 1999). For example, the variation in the *Wr* found in the UK, from 0.5 in winter to 1.0 in summer, had a similar negative correspondence with male mortality (42% in winter, 18% in summer) (see Fig. 5). In both cases, the level of male mortality played a central role in explaining a drop in mite reproduction. Moreover, in this study, we found the mortality of the second female offspring (third offspring) explained the variability in *Wr*, such that the variability in *Wr* seems to be determined by the mortality of offspring as a whole.

Though we are currently unable to explain the link between any factor and offspring mortality, there are three possibilities. (1) Offspring mortality is linked to honeybee genetics; but as this factor is similar in all the colonies studied, it would then be due to a dominant trait of AHB. In previous works on EHB in tropical climate (Vandame et al., 2000; Vandame et al., 2002), there were strong differences between AHB and EHB colonies, which strengthens the hypothesis of a dominant trait of AHB. (2) Offspring mortality is linked to mite genetics; this is unlikely as mortality rates change in the various studies of the same mite genotype (Korean). (3) Offspring mortality is linked to environmental factors, e.g. the combined effect of high temperature and low humidity. This hypothesis is the most likely, since the offspring mortality cycles mirror changes in the external environmental conditions.

For the observed variation in the *Wr* to explain the cycling of the mite population, the average number of mite reproductive cycles (the average number of times a mite invades a cell to reproduce) must be between 1.7 and 0.8, because values outside this range would cause the mite population to either continually increase (1.7 × 0.6 (Min. *Wr*) > 1) or decrease (0.8 × 1.2 (Max. *Wr*) < 1). The average number of reproductive cycles in AHB is unknown and difficult to determine, but in EHB it is estimated to be between 2–3 (Martin, 1994). Because the proportion of phoretic mites was

Table I. Comparison between the number of mated female offspring produced per foundress mite in worker (*Wr*) and drone brood (*Dr*), and average number of eggs laid (fecundity) per invading foundress in cells containing increasing numbers of foundresses.

Brood	Source	Item	Number of foundresses invading a cell				
			1	2	3	4	>4
Drone	AHB and EHB × AHB mixed colonies (This study)	*Sample size*	*173*	*94*	*31*	*21*	*17*
		Fecundity	4.2	3.5	2.3	2.05	1.5
		Dr	1.83	1.64	0.94	0.79	0.61
	EHB (Martin, 1995b)	Fecundity	5.5	5.3	5.1	4.8	
		Dr	1.9	1.9	1.6	1.4	
Worker	AHB and EHB × AHB mixed colonies (This study)	*Sample size*	*2022*	*86*	*18*		
		Fecundity	4.1	2.9	2.8		
		Wr	0.88	0.8	0.43		
	EHB (Martin, 1995b)	Fecundity	4.7	4.3	4.1	3.5	3.6
		Wr	1.1	1.1	1.1	0.7	

greater in our study, it is possible that this could be associated with fewer reproductive cycles.

The second mechanism that can help to explain the low *Wr* is the density dependent reproduction in drone brood (Martin and Medina, 2004). The present study is the first to measure the effect of multiple infestation on the number of mated offspring produced per foundress mite in drone (*Dr*) and worker brood (*Wr)*, and on the number of eggs produced in AHB or AHB × EHB colonies. When fecundity, *Dr* and *Wr* were compared with data from EHB (Tab. I), it became clear that there was a large difference between AHB and EHB colonies, because the mites invading AHB (or EHB × AHB) drone cells became unproductive (i.e. $Dr < 1$) when more than two foundresses invaded the same cell. A 32% drop in the fecundity and 45% drop in the number of *Wr* offspring produced in worker cells invaded by three mites was also observed, but this had little biological relevance since multiply invaded worker cells rarely occur in AHB colonies (the maximum average number of foundress mite per worker infested cell was 1.1). However, in drone cells a very high infestation level was observed throughout the year (Fig. 2). This led to numerous cells being invaded by many foundresses, especially when the mite populations were at their highest levels (see Fig. 2). For example, in one colony in January, 74% of the mites in the drone brood were found in cells containing three or more foundresses. This resulted in a decline in the number of eggs produced per foundress mite and, consequently, a decrease in the mite population reproducing in drone brood during this time. This finding is an indication that density dependent effects leading to low reproductive capability of mites can play a role in drone brood at high mite populations. However, more complete field tests are needed before the role of density dependent effects in controlling the mite population in AHB are fully understood.

It is clear that the cumulative effect of many behaviors, such as damaged mites through hygienic and grooming behavior, as was found in Mexico by Vandame et al. (1999) and Arechavaleta et al. (2001) respectively, work together to make AHB resistant to mites, or can be used to select EHB queens resistant to *V. destructor* (Spivak and Reuter, 1998). Environmental factors may also impact the mite reproduction or population dynamics (Harris et al., 2003). In this work, the intent to explain the low and cyclic *Wr* reported as a main resistance factor in a previous work (Mondragón et al., 2005) led us to show the importance of two more features in the resistance of AHB to the mites. These factors are the death of the male and female offspring, and the reduction in the reproductive capability in drone cells due to increasing density of foundresses. The genetic or more probably environmental origin of these new factors still has to be elucidated.

ACKNOWLDEGMENTS

The authors acknowledge Agustín Méndez and Miguel Cigarroa for their great help in the field work. L. M. was granted by CONACYT during his Ph.D. studies, and the work was supported by a grant from Fondo Mixto CONACYT / Gobierno del Estado de Chiapas.

Résumé – Mortalité de la descendance des acariens : un élément majeur de la résistance à *Varroa destructor* dans une population d'abeilles africanisées. Une étude sur un an a été réalisée en 2003 sur des colonies d'abeilles africanisées (*Apis mellifera*) et des colonies hybrides africanisées × européennes. Le but était d'élucider les facteurs de la reproduction de l'acarien *Varroa destructor* liés à la variation du nombre de descendantes fécondées produites par fondatrice sur couvain d'ouvrières (*Wr*) et le rôle joué par la densité des acariens sur leur reproduction. Les 18 colonies utilisées ont été infestées naturellement. Des échantillons de couvain d'ouvrières et de mâles ont été prélevés dans chaque colonie toutes les cinq semaines. Les acariens ont été éliminés de chaque cellule infestée et l'âge des nymphes a été déterminé. Le taux de mortalité des descendants a été déterminé dans 601 cellules de couvain d'ouvrières et 142 cellules de couvain de mâles. Dans les deux types de cellules il y a eu plus de morts dans les descendants du troisième œuf produit que dans ceux du premier (Fig. 3). Pour le couvain d'ouvrières, la mortalité des acariens au cours de l'année a semblé augmenter de mars à juin (Fig. 4). Une analyse de régression simple a montré que la mortalité du premier descendant (mâle) et du troisième (femelle) rendait compte respectivement de 67 % et 53 % de la variation de *Wr*. (Fig. 5). On a observé une forte réduction du nombre moyen d'œufs produits et des facteurs *Wr* et *Dr* dans les cellules mono-infestées par rapport aux cellules pluri-infestées (Tab. I). Les résultats obtenus montrent l'existence de deux mécanismes qui peuvent expliquer les fluctuations cycliques de *Wr* et donc la résistance des abeilles africanisées à *V. destructor* : la mort du premier et du troisième descendant ainsi que la réduction des capacités reproductives due à l'accroissement de la densité des fondatrices.

***Varroa destructor* / *Apis mellifera* / abeille africanisée / résistance / reproduction des acariens**

Zusammenfassung – Sterblichkeit des Milbennachwuchses: Eine wichtige Resistenzkomponente gegen *Varroa destructor* in einer Population afrikanisierter Bienen. In einer 2003 durchgeführten einjährigen Studie an afrikanisierten und afrikanisierten × europäischen Bienen waren die mit der Anzahl begatteter weiblicher Nachkommen pro Gründerweibchen (*Wr*) in Verbindung stehenden reproduktiven Faktoren und die Rolle der dichteabhängigen Milbenreproduktion untersucht worden. Die 18 in der Studie verwendeten Völker waren natürlich infiziert. Aus jedem Volk wurden alle 5 Wochen Proben von Arbeiterinnen und Drohnenbrut entnommen. Aus jeder infizierten Zelle wurden die Milben und ihre Nachkommen entnommen und das Alter der Puppen festgestellt. Der Grad der Nachkommenssterblichkeit wurde anhand von 601 Arbeiterinnenzellen und 142 Drohnenzellen bestimmt und folgte in beiden Zelltypen einem ähnlichen Muster: Die Nachkommenssterblichkeit nahm vom ersten bis zum letzten der gelegten Eier zu (Abb. 3). Über den Jahresverlauf schien die Nachkommenssterblichkeit von März bis Juni anzusteigen (Abb. 4). Eine einfache Regressionsanalyse zeigte, dass die Sterblichkeit des ersten (männlichen) und dritten (weiblichen) Nachkommen 67 % bzw. 53 % der Variation von *Wr* erklären können (Abb. 5). Eine deutliche Verminderung der mittleren Anzahl gelegter Eier wurde sowohl in Arbeiterinnenzellen als auch in Drohnenzellen beobachtet, wenn die Zellen mehrfach befallen waren (Tab. I). Die bei den untersuchten Völkern erhaltenen Ergebnisse zeigten zwei Mechanismen auf, die die jahreszyklische Fluktuation von *Wr* und damit die Resistenz der afrikanisierten Bienen gegen die Milben erklären könnten. Dies ist einmal die Sterblichkeit des Milbennachwuchses, zum anderen die dichteabhängige Verminderung der Reproduktionsfähigkeit in mehrfachbefallenen Drohnenzellen.

***Varroa destructor* / Honigbienen / Milbenreduktion / Resistenz**

REFERENCES

Anderson D.L., Trueman J.W.H. (2000) *Varroa jacobsoni* (Acari: Varroidae) is more than one species, Exp. Appl. Acarol. 24, 165–189.

Arechavaleta-Velasco M.E., Guzmán-Novoa E. (2001) Relative effect of four characteristics that restrain the population growth of the mite *Varroa destructor* in honey bee (*Apis mellifera*) colonies, Apidologie 32, 157–174.

Corrêa-Marques M.H., Medina L.M., Martin S.J., de Jong D. (2003) Comparing data on the reproduction of *Varroa destructor*, Genet. Mol. Res. 2, 1–6.

Donzé G., Herrmann M., Bachofen B., Guerin P.M. (1996) Effect of mating frequency and brood cell infestation rate on the reproductive success of the honeybee parasite *Varroa jacobsoni*, Ecol. Entomol. 21, 17–26.

Guzmán-Novoa E., Vandame R., Arechavaleta M.E. (1999) Susceptibility of European and Africanized honey bees (*Apis mellifera* L.) to *Varroa jacobsoni* Oud. in Mexico, Apidologie 30, 173–182.

Harris J.W., Harbo J.R. (1999) Low sperm counts and reduced fecundity of mites in colonies of honey bees (Hymenoptera: Apidae) resistant to *Varroa jacobsoni* (Mesostigmata: Varroidae), J. Apic. Res. 92, 83–90.

Harris J.W., Harbo J.R., Villa J.D., Danka R.G. (2003) Variable population growth of *Varroa destructor* (Mesostigmata: Varroidae) in colonies of honeybees (Hymenoptera: Apidae) during 10-year period, Environ. Entomol. 32, 1305–1312.

Ifantidis M.D. (1983) Ontogenesis of the mite *Varroa jacobsoni* in worker and drone brood cells, J. Apic. Res. 23, 227–233.

Martin S.J. (1994) Ontogenesis of the mite *Varroa jacobsoni* Oud. in worker brood of the honeybee *Apis mellifera* L. under natural conditions, Exp. Appl. Acarol. 18, 87–100.

Martin S.J. (1995a) Ontogenesis of the mite *Varroa jacobsoni* Oud. in drone brood of the honeybee *Apis mellifera* L. under natural conditions, Exp. Appl. Acarol. 19, 199–210.

Martin S.J. (1995b) Reproduction of *Varroa jacobsoni* in cells of *Apis mellifera* containing one or more mother mites and distribution of these cells, J. Apic. Res. 34, 187–196.

Martin S.J., Medina L.M. (2004) Africanized honey bees have unique tolerance to *Varroa* mites, Trends Parasitol. 20, 112–114.

Medina L.M., Martin S.J. (1999) A comparative study of *Varroa jacobsoni* reproduction in worker cells of honey bees (*Apis mellifera*) in England and Africanized bees in Yucatán, Mexico, Exp. Appl. Acarol. 23, 659–667.

Medina L.M., Martin S.J., Espinosa L.M., Ratnieks L.F.W. (2002) Reproduction of *Varroa destructor* in worker brood of Africanized honey bees (*Apis mellifera*), Exp. Appl. Acarol. 27, 79–88.

Mondragón L., Spivak M., Vandame R. (2005) A multifactorial study of the resistance of honeybees *Apis mellifera* to the mite *Varroa destructor* over one year in Mexico, Apidologie 36, 345–358.

Rinderer T.E., Buco S.M., Rubink W.L., Daly H.V., Stelzer J.A., Riggio R.M., Baptista F.C. (1993) Morphometric identification of Africanized and European honey bees using large reference populations, Apidologie 24, 569–585.

Ritter W., de Jong D. (1984) Reproduction of *Varroa jacobsoni* O. in Europe, the Middle East and tropical South America, Z. Angew. Entomol. 98, 55–57.

Rosenkranz P., Engels W. (1994) Infertility of *Varroa jacobsoni* females after invasion into *Apis mellifera* worker brood as tolerance factor against varroatosis, Apidologie 25, 402–411.

Spivak M., Reuter G.S. (1998) Performance of hygienic colonies in a commercial apiary, Apidologie 29, 285–296.

Vandame R., Colin M.E., Otero-Colina G. (1999) Africanized honeybees tolerance to Varroa in Mexico: mite infertility is not the main tolerance factor, Apiacta 34, 1–5.

Vandame R., Colin M.E., Morand S., Otero-Colina G. (2000) Levels of compatibility in a new host-parasite association: *Apis mellifera*/*Varroa jacobsoni*, Can. J. Zool. 78, 2037–2044.

Vandame R., Morand S., Colin M.E., Belzunces L.P. (2002) Parasitism in the social bee *Apis mellifera*: quantifying costs and benefits of behavioral resistance to *Varroa* mites, Apidologie 33, 433–455.
