Interview with a varroa mite

Gabrielle Almecija
APINOV and IRBI, University of Tours, France

The varroa mite (*Varroa destructor*) is one of the greatest threats to honey bees and has caused colony losses across almost the entire world. Small and secretive, it hides on bees and in brood cells, mostly out of sight of the beekeeper. To control the mite, miticides have been used since the earliest days of its discovery, but the mite fights back. I asked a varroa mite how they do it.

Gilles San Martin & Sam Droege CC BY-SA 2.0

Since your arrival in *Apis mellifera* colonies, beekeepers have tried many methods to keep you at bay. How do you combat these actions?

It's hard! Beekeepers' methods have variable efficacy, and we need to adapt constantly to survive. We hope that, like so many of our cousins, we can develop ways to resist and make treatments ineffective.[1] We can resist in several ways: by changing our physiology (thickening our cuticle to create a physical barrier), through metabolism (excreting the miticide) and by mutating (modifying the miticide target so that the treatment is less effective). Some ways of resisting have a high physiological cost for us,[2,3] so we develop resistance only if we have to.

What did you develop against the tau-fluvalinate miticide that is the active ingredient in products such as Apistan?

Well, we used different strategies depending on where we were located. Varroa in Europe and USA, for example, developed resistance in different ways. To understand how we developed resistance, you need to know how tau-fluvalinate kills us. Tau-fluvalinate is an insecticide from the pyrethroid II family. This group of insecticides is neurotoxic. When tau-fluvalinate enters us, it targets the sodium channel.[4] When it finds its target, it binds with it and causes our nervous system to malfunction. A few hours later, we become paralysed and die.

To avoid this, we develop two types of resistance.

The first is metabolic.[5] We increase our detoxifying enzymes (oxygenase, Cytrochompe P450).

The second is a mutation of the miticide target (we modify or increase the target).[6] Mutations often make the target too difficult to recognize, so the miticide becomes ineffective. In Europe, it seems that we have developed the mutation L925V.[7,8] However, some mites in the USA have developed other mutations.[9] Both these resistance methods have a cost to us and, after few years without being exposed to tau-fluvalinate, we lower our guard and become susceptible to it again.[10,11]

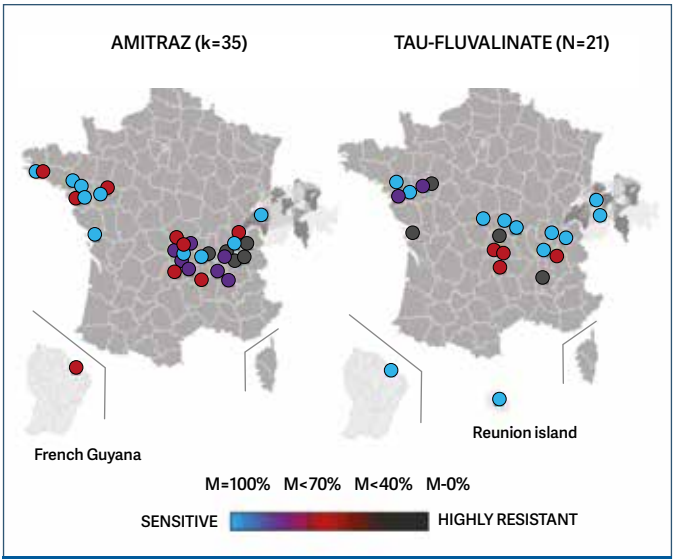
Beekeepers have used amitraz for many years. Have you found a way to resist that?

We have lived with amitraz for more than 40 years but it is hard to develop resistance to it. However, some of my cousin mites (*Rhipicephalus* spp. and *Tetranychus* spp.) have developed resistance to amitraz.[12–15] Mutations of the target and metabolic resistance have been described by researchers.[12,13,16–19] These cousins have to survive several treatments of amitraz each year.[20]

In beekeeping, it's different. We must survive only a single Amitraz treatment each year, but it's a long treatment. Today, some of us can survive it.[21,22] At the moment no one fully understands how we do it. We survive it better now than we did a few years ago when beekeepers used the same miticide year after year. Since then we have reduced our vulnerability to the treatment slowly, but many beekeepers haven't noticed that yet.

Can beekeepers use a strategy to force you to stay sensitive to a miticide?

We have no interest in developing or staying resistant to a miticide if we are not exposed to it. In some cases, some of us that have developed resistance to a miticide have fewer offspring than those of us who haven't.[2] Today, several miticides with different active ingredients are available. If beekeepers used a different miticide from one year to the next, we are forced to develop resistance to the latest miticide used.[23,24] We find it very difficult to maintain different types of resistance, so we need to lose one



Mapping susceptibility to amitraz and tau-fluvalinate

type of resistance to be able to develop another.[25] Alternating miticides reduces our resistance. But if beekeepers don't use this strategy, our resistance to amitraz is likely to increase, just as it happened with tau-fluvalinate.[24,26]

Evaluating mite resistance to tau-fluvalinate and amitraz

In France, APINOV, a scientific beekeeping and training centre, is working with Vita Bee Health on this project. Mite susceptibility is evaluated with a bioassay. Mites are contaminated with different concentrations of amitraz and tau-fluvalinate. By applying different concentrations, the susceptibility to amitraz and tau-fluvalinate can be categorized in three classes:

- susceptible: mortality more than 75%
- moderately resistant: mortality between 40% and 75%
- highly resistant: mortality less than 40%.

The trial was conducted from 2018 to 2020. A total of 41 mite populations were tested for resistance to amitraz and 33 to tau-fluvalinate. Mite susceptibility has been mapped for tau-fluvalinate and amitraz.[27]

In France, mite populations show a high level of susceptibility to amitraz and tau-fluvalinate (as confirmed in field trials). The variations result from the differing practices of individual beekeepers. For example, organic beekeepers have mites that are mostly susceptible to amitraz and tau-fluvalinate. Beekeepers who have used amitraz for many years have mites susceptible to tau-fluvalinate. These differences result because of the cost to the mite of developing resistance – if the miticide is not applied, there is no need to develop resistance.

However, the disappearance of the resistance trait can take many years (four years for tau-fluvalinate; unknown for amitraz). The high level of susceptibility to amitraz and tau-fluvalinate observed in France is good news in the fight against the mite: susceptible mites are still present in the general population and can reduce the speed of the development of resistance in other populations.

The aim of this work is to determine ways to reduce varroa's resistance to miticide by:

- treatment strategies that can be used to reduced resistance to miticide
- altering the treatment parameters (eg duration and timing of treatments).

REFERENCES

1. Vanoosthuyse, F et al. La résistance des insectes et acariens aux produits antiparasitaires pour les espèces agricoles présentes au Quebec. *Inst Rech Dév En Agroenvironnement* 108 (2018)

2. Kliot, A and Ghanim, M. Fitness costs associated with insecticide resistance. *Pest Manag Sci* 68, 1431–1437 (2012)

3. Bajda, S et al. Fitness costs of key point mutations that underlie acaricide target-site resistance in the two-spotted spider mite *Tetranychus urticae*. *Evol Appl* 11, 1540–1553 (2018)

4. Davies, TGE, Field, LM, Usherwood, PNR and Williamson, MS. DDT, pyrethrins, pyrethroids and insect sodium channels. IUBMB Lif 59, 151–162 (2007)

5. Hillesheim, et al. First data on resistance mechanisms of *Varroa jacobsoni* (OUD) against tau-fluvalinate. *Exp Appl Acarol* 20, 283–296 (1996)

6. Hubert, J et al. Point mutations in the sodium channel gene conferring tau-fluvalinate resistance in *Varroa destructor*. *Pest Manag Sci* 70, 889–894 (2014)

7. González-Cabrera, J et al. An amino acid substitution (L925V) Associated with Resistance to Pyrethroids in *Varroa destructor*. *PLOS ONE* 8, e82941 (2013)

8. González-Cabrera, J et al. A single mutation is driving resistance to pyrethroids in European populations of the parasitic mite, *Varroa destructor*. *J Pest Sci* 91, 1137–1144 (2018)

9. González-Cabrera, J et al. Novel nutations in the voltage-gated sodium channel of pyrethroid-resistant *Varroa destructor* populations from the Southeastern USA. *PLOS ONE* 11, e0155332 (2016)

10. Milani, N and Vedova, G. Decline in the proportion of mites resistant to fluvalinate in a population of Varroa destructor not treated with pyrethroids. *Apidologie* 33, 417–422 (2002)

11. Panini, M et al. Pyrethroid resistance in Italian populations of the mite *Varroa destructor*: a focus on the Lombardy region. *Bull Insectology* 72, 227–232 (2019)

12. Chevillon, C et al. Accumulation of acaricide resistance mechanisms in Rhipicephalus (Boophilus) microplus (Acari: Ixodidae) populations from New Caledonia Island. *Vet Parasitol* 147, 276–288 (2007)

13. Chen, AC, et al. Mutations in a putative octopamine receptor gene in amitraz-resistant cattle ticks. *Vet Parasitol* 148, 379–383 (2007)

14. Jonsson, NN and Hope, M. Progress in the epidemiology and diagnosis of amitraz resistance in the cattle tick *Boophilus microplus*. *Vet Parasitol* 146, 193–198 (2007)

15. Li, AY et al. Detection and ccharacterization of amitraz resistance in the southern cattle tick, *Boophilus microplus* (*Acari: Ixodidae*). *J Med Entomol* 41, 193–200 (2004)

16. Baron, S et al. Differentially expressed genes in response to amitraz treatment suggests a proposed model of resistance to amitraz in *R. decoloratus* ticks. *Int J Parasitol Drugs Drug Resist* 8, 361–371 (2018)

17. Corley, S W et al. Mutation in the Rm AOR gene is associated with amitraz resistance in the cattle tick *Rhipicephalus microplus*. Proc Natl Acad Sci 110, 16772–16777 (2013)

18. Sammataro, D et al. The resistance of varroa mites (Acari: Varroidae) to acaricides and the presence of esterase. *Int J Acarol* 31, 67–74 (2005)

19. Monteiro, HR et al. Amitraz toxicity to the midge *Chironomus riparius*: Life-history and biochemical responses. *Chemosphere* 221, 324–332 (2019)

20. Bianchi, MW et al. Factors related to cattle infestation level and resistance to acaricides in *Boophilus microplus* tick populations in New Caledonia. *Vet Parasitol* 112, 75–89 (2003)

21. Kamler, M et al. Comparison of tau-fluvalinate, acrinathrin, and amitraz effects on susceptible and resistant populations of *Varroa destructor* in a vial test. *Exp Appl Acarol* 69, 1–9 (2016)

22. Rinkevich, FD. Detection of amitraz resistance and reduced treatment efficacy in the varroa mite, *Varroa destructor*, within commercial beekeeping operations. *PLOS ONE* 15, e0227264 (2020)

23. Thullner, F et al. Acaricide Rotation Strategy for Managing Resistance in the Tick *Rhipicephalus* (Boophilus) microplus (Acarina: Ixodidae): Laboratory Experiment with a Field Strain from Costa Rica. *J Med Entomol* 44, 5 (2007)

24. Sudo, M et al. Optimal management strategy of insecticide resistance under various insect life histories: Heterogeneous timing of selection and interpatch dispersal. *Evol Appl* 11, 271–283 (2017)

25. Cloyd, RA. Pesticide mixtures and rotations: Are these viable resistance mitigating strategies. *Pest Technol* 4, 14 18 (2010)

26. Denholm, I. Challenges with managing insecticide resistance in agricultural pests, exemplified by the whitefly *Bemisia tabaci*. *Philos Trans R Soc B Biol Sci* 353, 1757–1767 (1998)

27. Almecija, G, Poirot, B et al. Inventory of *Varroa destructor* susceptibility to amitraz and tau-fluvalinate in France. *Exp Appl Acarol* 82, 1–16 (2020)