

# Safety of imidacloprid seed dressings to honey bees: a comprehensive overview and compilation of the current state of knowledge

Christian MAUS<sup>1</sup>, Gaëlle CURÉ<sup>2</sup>, Richard SCHMUCK<sup>1</sup>

<sup>1</sup>Bayer AG, Bayer Crop Science, Institute for Ecotoxicology, Monheim, Germany

<sup>2</sup>Bayer SA, Division AGRO, Puteaux, France

## Abstract

This paper reviews the available data on the risk potential posed by imidacloprid seed dressings to honey bees. Key studies are briefly described and their results are discussed; of particular interest for this topic are the numerous field and semifield trials which were carried out by different scientists from various institutions. From the reviewed studies a field-relevant NOAEC of 20 ppb is concluded for honey bees. Analytical studies on nectar and pollen samples of sunflower, rape and corn plants have shown that residue levels of imidacloprid and toxicologically relevant imidacloprid metabolites are typically well below 5 ppb. When comparing the NOAEC with the reported residue data, it becomes evident that imidacloprid seed-dressings will pose only a negligible risk to honey bees. This conclusion is supported by the findings of more than 30 semifield and field studies conducted in various regions of the world. Finally, the symptoms of the bee incidents in France, which were suspected to be related to imidacloprid seed-dressings, are described and factors potentially linked to these incidents are discussed.

**Key words:** honey bee, *Apis mellifera*, pesticide side effects, imidacloprid, seed dressings, ecotoxicology.

## Introduction

One of the issues related to pesticides and bees which has been discussed very intensively within the last years, concerns the question whether or not seed dressings containing the neonicotinoid insecticide imidacloprid may pose a risk to honey bees. Nearly ten years of intensive laboratory and field research by independent experts and Bayer scientists should allow to conduct a conclusive evaluation on this question. In this paper the history of this issue is briefly reviewed, followed by a summary of the key studies which were carried out to highlight any potential link between an imidacloprid seed dressing and bee hive damages. Finally, a conclusive evaluation on the risk potential of imidacloprid seed dressings for bees is drawn based on the current state of knowledge.

## Issue history

Gaicho<sup>®</sup>, a systemic insecticidal seed dressing (active ingredient: imidacloprid), was first used in sunflower crops in France in 1994. In response to a beekeeper's concern regarding possible adverse effects of this seed treatment on bees, Bayer in cooperation with external experts carried out several semifield and field studies to re-examine the bee safety of this treatment under the regionally prevailing use conditions. As in the pre-registration studies, no indications of an adverse effect potential were found. In 1997, severe bee colony and honey yield losses were reported by French beekeepers, and Gaicho<sup>®</sup> became accused again by some beekeepers to be responsible for these effects. These bee hive damages were reported to exhibit novel features such as e.g. depopulated bee hives due to a treatment-related disorientation of affected bees. The French Ministry of Agriculture initiated an expert review in 1997 which

concluded no apparent link between the reported symptoms and the Gaicho<sup>®</sup> seed treatment. However, in order to minimize any uncertainty in this risk evaluation the French experts recommended a replicated field study. This large-scale field study, performed in 1998, also showed no adverse effects of a Gaicho<sup>®</sup> seed treatment. Nevertheless, the authorization of Gaicho<sup>®</sup> in sunflower cultures was temporarily suspended by the Ministry of Agriculture in 1999 based on the "precautionary principle" since the causal factors of this seemingly novel bee malady were still obscure and a laboratory study reported very low effect concentrations of imidacloprid, the active ingredient of the seed dressing Gaicho<sup>®</sup> to bees (Decourtye, 1998). Further studies were requested from the notifier on rotational crops, and these studies were carried out and submitted in 1999. The study results showed that honey bees will not encounter relevant imidacloprid concentrations in rotational crops from previous soil treatments with imidacloprid. In addition to these rotational crop studies, the laboratory study of 1998 was repeated by an independent scientist who was not able to confirm the reported low effect concentrations of imidacloprid. In addition, the bee hive damages in sunflowers continued in spite of the suspension of Gaicho<sup>®</sup> in this crop since 1998.

## Materials and methods

### Biological studies

The reviewed key studies cover a large range of test designs from laboratory to complex field trials including a couple of novel test designs which were developed to investigate potential treatment-related effects of seed treatments on the behavior of bees and bee colonies. In standard laboratory studies the acute oral toxicity of

imidacloprid was investigated by feeding bees with spiked sugar solution. These studies were carried out according to the international testing guidelines EPPO 170 and OECD 213 and 214 (EPPO, 1992; OECD, 1998 a, b) and findings reported by Schmuck (1999) and Schmuck *et al.* (2001, 2003). Likewise, the chronic oral toxicity of imidacloprid was investigated by a dietary exposure of honeybees over 5 to 10 days (Decourtye, 1998; Belzunces *et al.*, 1998; Colin *et al.*, 1998; Kirchner, 1999, 2000; Pham-Delègue *et al.*, 2000; Suchail *et al.*, 2001; Schmuck, in prep.). Other endpoints investigated in the laboratory included the associative learning behavior of honeybees. In these tests an artificial chemical stimulus is linked to the proboscis extension reflex of the bees (Bitterman *et al.*, 1983).

Short- and long-term effects of imidacloprid residues in bee diets were also investigated under more natural conditions in semifield and field tests. More than 30 semifield and field tests were performed by scientists from the notifier as well as from universities and other institutions. In some of these tests, bee colonies were fed up to 39 days exclusively with an imidacloprid-spiked diet such as corn pollen, sunflower honey, or sugar solution. Likewise, chronic feeding tests with pollen harvested from sunflowers or corn grown from dressed seeds were conducted. For simulating most natural exposure conditions, bee colonies were placed next to sunflower or rape fields which had been drilled with imidacloprid dressed seeds. In all these trials, bee mortality, bee losses, foraging activity, bee behavior, colony development, brood status and changes in pollen and nectar stores were monitored (e.g. Belzunces *et al.*, 1998; Brasse, 1999; Schulz, 1999; Schmuck, 1999; Kirchner, 1999; Schmuck *et al.*, 2001; Scott-Dupree and Spivak, 2001; Maus and Schöning, 2001; Maus, 2002; Schulz, 2002) (figures 2 and 3). Furthermore, specifically designed field studies were conducted to investigate in detail the sublethal effect potential of imidacloprid. In these tests, antifeedant responses and treatment-related effects on the intraspecific communication in small bee hive nuclei were examined. For example, Kirchner (1999) examined the dancing behavior of bees in hive nuclei for accuracy of the communicated information on the food source location (direction and distance from the hive) after a dietary exposure to imidacloprid. In other feeding studies with spiked sugar solution the effects of dietary exposures on large bee hive colonies were investigated, with a particular focus on field orientation of the bees. In these studies, bees were individually marked and followed over the entire study period (Schmuck, 1999).

### Residue analyses

Intensive analyses of imidacloprid residues in various plant matrices were conducted to characterize the exposure of honeybees to imidacloprid residues in seed-treated crops. Investigated crop species included sunflower, rape, and corn. From each of these crops, nectar (except for corn) and pollen samples were taken from several field locations which differed in their soil and climatic characteristics, and subsequently analysed. In total, 18 residue analyses were performed on sunflower

pollen and nectar, 15 on rape nectar or pollen, and four on corn pollen (e.g. Schmuck, 1999; Schmuck *et al.*, 2001; Schöning and Schmuck, 2003). The limit of determination (LOD) for the parent compound in these plant materials was 1.5 ppb, with a limit of quantitation (LOQ) of 5.0 ppb. All nectar and pollen samples were not only analysed for the parent compound but also for insecticidally active plant metabolites of imidacloprid, i.e. olefine- and hydroxy-imidacloprid (Nauen *et al.*, 2001; Schmuck *et al.*, 2003). The LODs for these metabolites were 1.5 ppb and 3.0 for hydroxy-imidacloprid and olefine-imidacloprid, respectively (Schöning and Schmuck, 2003). For a description of the analytical methods applied, see Schmuck *et al.* (2001) and Schöning (2001). Likewise, the persistence of imidacloprid in soils and its potential for accumulation after repeated sowing of dressed seeds was investigated in long term field dissipation studies (Krohn and Hellpointner, 2002).

## Results and discussion

### Effect thresholds of imidacloprid to honey bees

The acute oral LD<sub>50</sub> of imidacloprid to honey bees as found in the majority of laboratory studies was between 40.9 and > 81 ng/bee with NOED values between 1 and 5 ng/bee (Schmuck, 1999; Schmuck *et al.*, 2001, 2003). In the literature, also some lower LD<sub>50</sub> values are published (Pflüger and Schmuck, 1991; Suchail *et al.*, 2000). These differences in the oral LD<sub>50</sub> values may be related to methodological aspects as discussed by Nauen *et al.* (2001). In chronic feeding studies conducted under laboratory conditions (5-10 day feeding studies with worker bees) no-observed lethal effect concentrations (NOEC) of > 20 ppb were reported (Colin *et al.*, 1998; Kirchner, 1999, 2000; Pham-Delègue *et al.*, 2000; Decourtye *et al.*, 2003) (figure 1). Two studies, however, reported lower NOEC values. A NOEC value of ≤ 4 ppb was published by Decourtye (1998) which, however, could not be reproduced in subsequent studies of the same research group (Decourtye *et al.*, 2003). Suchail *et al.* (2001) found 50% mortality rates after a dietary exposure of young honeybees to 0.1, 1, and 10 ppb over ten days. However, in this study all rates and all metabolites tested revealed the same chronic toxicity profile, irrespective of the presence or the absence of the toxophor. For this reason and since these findings are contradictory to all other reported values they need to be carefully re-considered as discussed by Schmuck (in prep.).

Regarding sublethal effects of imidacloprid, a laboratory NOEC of 10 ppb was found for associative learning by Kirchner (2000). Pham-Delègue *et al.* (2000) and Decourtye *et al.* (2003) claimed NOEC values between 6 and 48 ppb depending on the physiological state of the tested bees (summer and winter bees, respectively). However, the 6 ppb value is based on a single measurement recorded briefly after application and it is considered highly unlikely that such a very transient short-term effect will have any biological significance under field conditions.

Generally, results from laboratory trials have to be evaluated with particular care since honeybees are subjected to an artificial stress situation under the conditions prevailing in the laboratory. More relevant and reliable for an evaluation of compound-related risks are tests carried out under semifield (tents or tunnels) and field conditions where honeybees encounter their natural environment within the social context of their colony.

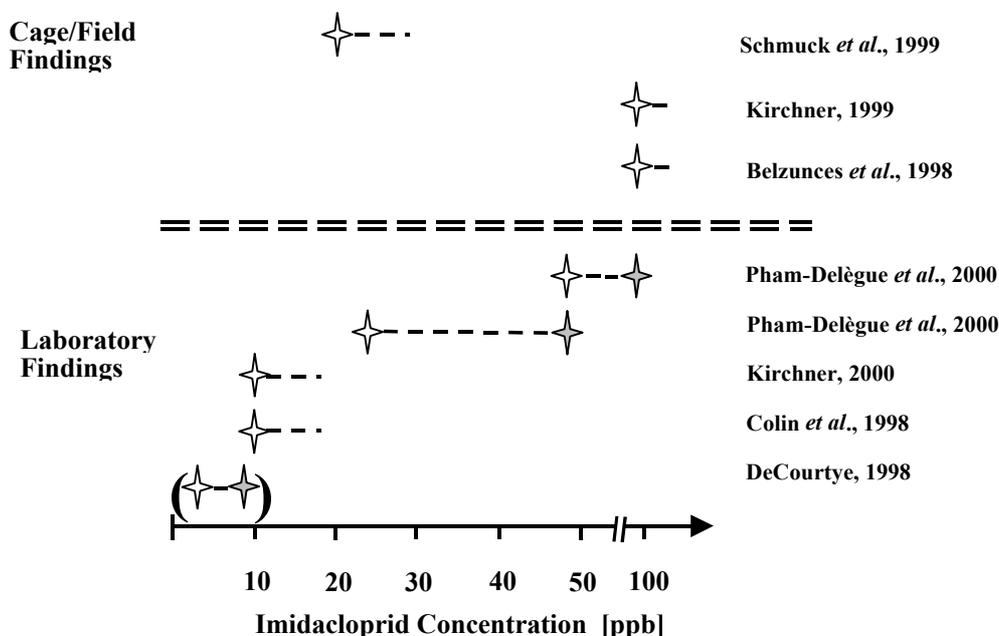
In field studies on the effects of imidacloprid-spiked sucrose solutions on the intraspecific communication of honey bees, Kirchner (1999) reported a NOEC of 10 ppb. At concentrations of 20 ppb bees perceived the contaminant and responded by rejection of that food. In response to this protective behavior, the recruitment activity of honeybees decreased resulting in an overall lower foraging activity at 50 ppb and higher. However, up to a concentration level of 100 ppb, foraging bees continued to sample spiked sucrose solution and had no problems in returning to their hives over distances of 500 m. At study termination, the number of surviving bees was not different between the treatment and the control group. In further feeding studies with spiked sucrose solution or pollen diets, no acute or chronic adverse effects were recorded on bee mortality, bee losses, foraging activity, bee behavior, colony development, brood status and changes in pollen and nectar stores for a dietary imidacloprid residue level of 20 ppb (e.g. Belzunces *et al.*, 1998; Brasse, 1999; Schulz, 1999; Schmuck, 1999; Kirchner, 1999; Schmuck *et al.*, 2001; Scott-Dupree and Spivak, 2001; Maus and Schöning, 2001; Maus, 2002; Schuld, 2002) (figures 2 and 3).

One study (Colin and Bonmatin, 2000) reports effects on foraging and feeding behavior at residue levels of as low as 1.6 ppb. Unfortunately, no analytical verification of the test concentration was made in this study, and therefore, this value has to be considered with reservation, considering the bulk of data (see above) showing much higher NOEC values for this endpoint.

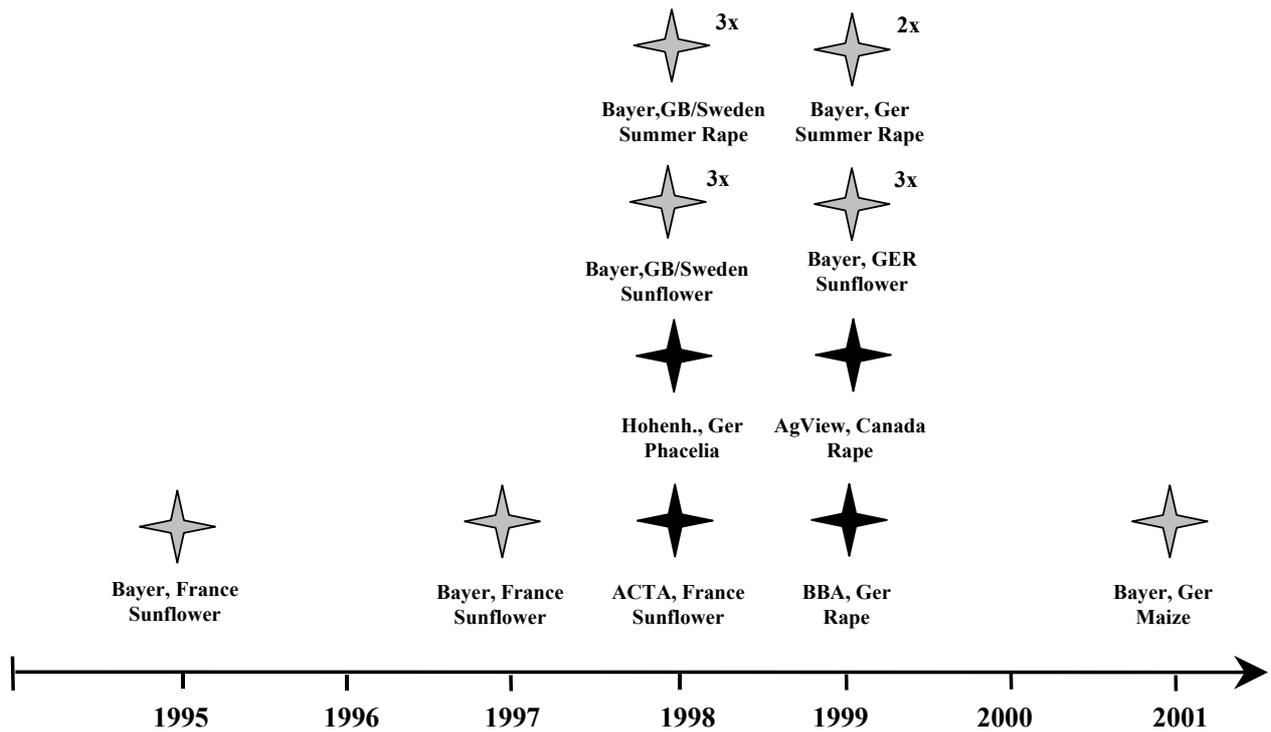
In support to a field-relevant NOEC of 20 ppb, no adverse effects were found in any semifield and field trials carried out on either seed-dressed crops or with pollen from seed-dressed plants.

From the results of the referenced key studies, it can be concluded that no adverse effects are expected under field conditions for imidacloprid residue levels of 20 ppb or less. The definition of this NOAEC is based on the following findings:

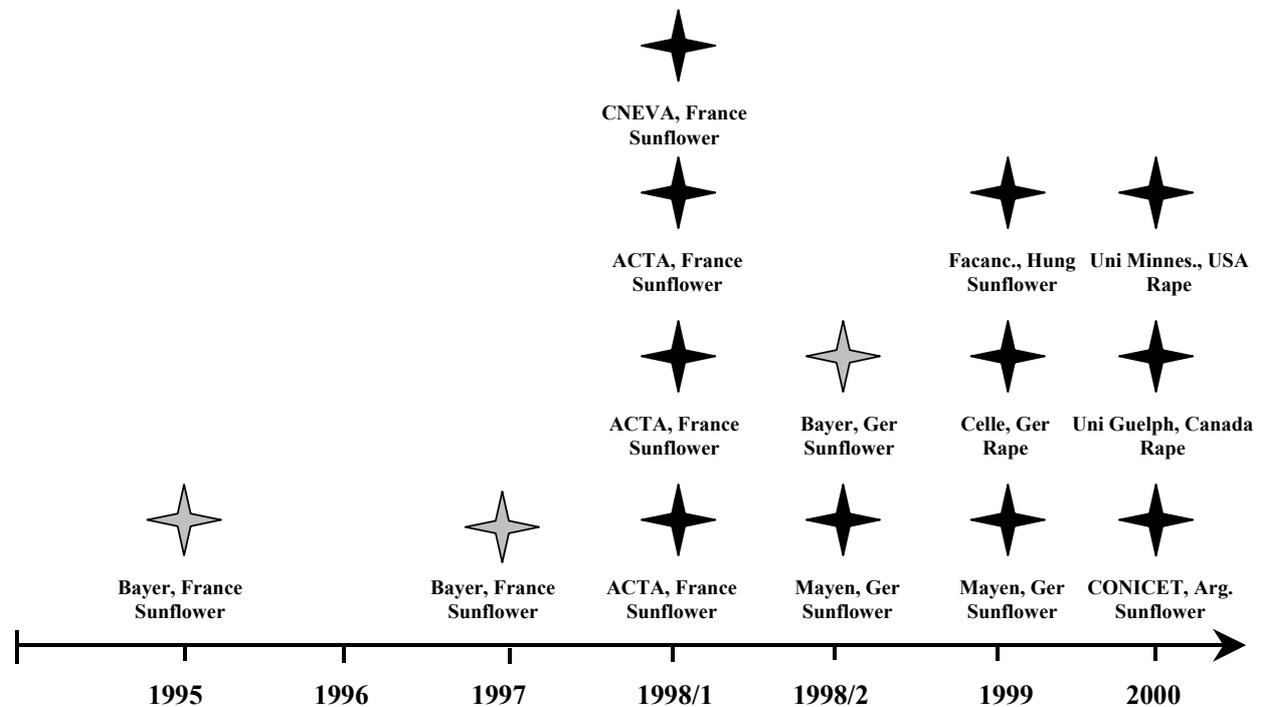
- No chronic mortality occurred at concentrations of < 20 ppb (reported lower NOEC values from laboratory trials were found to be not consistent with findings from other researchers and are not supported by findings from higher Tier studies)
- No antifeedant effects occurred at concentrations of ≤ 20 ppb (reported lower NOEC values were not supported by a weight-of-evidence approach)
- Short-term behavioral effects observed at concentrations < 20 ppb were very transitory (< 1 hr, Decourtye *et al.*, 2003)
- Hive performance was not affected at concentrations of ≤ 20 ppb over 39 days
- No loss of foraging bees was observed at concentrations of ≤ 100 ppb



**Figure 1.** Summary of the study results on honey bee mortality after chronic exposure to imidacloprid residues. The white stars mark the NOEC found in the respective study, the gray stars the LOEC. In cases where no LOEC is given, no effect was found in the respective study and the NOEC given corresponds to the highest dose tested.



**Figure 2.** Overview of the semifield studies carried out. Gray stars represent studies conducted by Bayer, black stars studies carried out by independent scientists. Studies were performed under various conditions regarding all relevant parameters (location, climatic conditions, soil conditions, crops, bee strains). No adverse effects were found under natural conditions.



**Figure 3.** Overview of the field studies carried out. Gray stars represent studies conducted by Bayer, black stars studies carried out by independent scientists. Studies were performed under various conditions regarding all relevant parameters (location, climatic conditions, soil conditions, crops, bee strains). No adverse effects were found under natural conditions.

## Exposure of honeybees to imidacloprid residues in the field

For an evaluation of the risk potential posed to honey bees by seed dressing applications of imidacloprid the residue levels encountered in the field is of particular interest; the crucial question here is to which residue levels of imidacloprid or imidacloprid metabolites foraging bees will be exposed in the field.

In sunflower crops, the imidacloprid residue levels in nectar and pollen were in all but one sample below the LOD of 1.5 ppb. Only one out of 18 analysed samples showed a higher residue level of 1.6 ppb. In rape, residue levels between < 1.5 and 5 ppb were recorded in the nectar and pollen samples. Only in one out of 15 analysed samples, a higher residue level was detected (7.8 ppb in a pollen sample). In corn pollen, the residue level detected was between < 1.5 and 5 ppb. (Schmuck, 1999; Schmuck *et al.*, 2001). The two insecticidally active plant metabolites, olefine- and hydroxy-imidacloprid were never detected in either nectar or pollen of any investigated crop species (Schmuck, 1999; Schmuck *et al.*, 2001; Schöning and Schmuck, 2003).

Field dissipation studies revealed that residues of imidacloprid will remain in the soil after harvest (Krohn and Hellpointner, 2002). However, no significant accumulation of imidacloprid in soils has been recorded. The maximum soil residue level after long-term and repeated use of imidacloprid on the same field will never exceed the soil residue levels resulting from a single application by more than 40%. In addition, plant bioavailability of aged soil residues is greatly reduced as indicated by the increasing Koc value with time (Krohn and Hellpointner, 2002). Thus, no relevant exposure of honey bees is expected in nectar or pollen of crops succeeding imidacloprid seed-dressed croppings. This assumption has been supported by residue analyses of nectar and pollen samples from rape, sunflower and corn plants grown in soils with residues up to a level of 18 ppb imidacloprid. In all nectar and pollen samples, residues were well below the LOQ (Schmuck *et al.*, 2001).

## Conclusions

The available results from numerous studies carried out by various scientists from different institutions strongly support the conclusion that crops grown from seeds dressed with imidacloprid do not pose any significant risks to honeybees under field conditions. Nectar and pollen residue levels from different locations and crops were all below the field-relevant NOAEC of 20 ppb as determined in various laboratory, tunnel and field studies. There is also no evidence of an adverse effect of imidacloprid seed-dressings from the numerous semi-field and field studies conducted in Argentina, Canada, France, Germany, Hungary, Italy, Sweden, UK, and the USA (figures 2 and 3). Additionally, although Gaucho® has now been suspended for almost five years, bee incidents in sunflower crops are still going on in France, which shows that the presumed link between the French bee damages and a imidacloprid seed treatment

in sunflower is not existing.

## The French bee incidents: possibly related factors

If no link exists between imidacloprid seed dressings and the French bee incidents, the question is raised which other factors may be involved in causing the reported “bee malady”. The French bee incidents have been described in detail by some of the concerned beekeepers (Aletru *et al.*, 1998). According to this description, the incidents are characterized by the occurrence of apathetic, immobilized bees, which tend to aggregate on the ground outside the bee hives. Furthermore, bees are trembling and bee hives gets depopulated due to disorientation of foraging bees. Affected bees are attacked by guardian bees at the hive entrance. Morphologically, the abdomina of affected bees appear blackish and shiny. Usually, approximately one third of the bees of a colony show these symptoms. On the colony level, the malady results in severe bee losses without apparent mortality, and a strongly decreased yield of honey; the syndrome commonly appears in July or August. It is worth to mention that this syndrome was occurring already long before the introduction of imidacloprid to the market (see also the website: <http://www.beekeeping.com/spmf/bd.htm>); and it likewise still continues after suspension of Gaucho® in sunflower cultures.

Considering the data at hand, it seems very likely that the “French Bee Malady” is caused by a complex of heterogenous factors - which nevertheless manifest themselves in similar symptoms - or in a combination of several factors (see e.g. Mackensen, 1951; Bailey, 1963; Oertel, 1965; Haydak, 1970; Atkins, 1975; Kauffeld *et al.*, 1976; Wilson and Menapace, 1979; Kulinkevic *et al.*, 1982, 1983, 1984; Bruce *et al.*, 1990; Boecking and Drescher 1991; Ritter, 1996; reviewed by Schmuck, 1999), which include:

- *Varroa* infestation
- Bacterial diseases (in particular spiroplasmas)
- Adverse climatic conditions (e.g.: mild winters which induce early beginning brood activity, followed by cold springs; very cold or very hot summers)
- Physiological incompatibilities (e.g.: attempts to establish bee strains in areas where inappropriate climatic conditions prevail, for instance establishing bees from New Zealand in Northern regions)
- Genetic incompatibilities (e.g. insemination of queens using drones with incompatible genetic material)
- Inappropriate bee management practices (e.g. inappropriate *Varroa* treatment, exaggerated pollination activity)
- Poisoning incidents (pesticide spray application, over-dosed *Varroa* treatment)

## References

- ALETRU F., CHAUVANCY F., CLEMENT H., MARY M., VEDRENNE Y., VERMANDERE Ph., 1998.- Observations autour du butinage du tournesol en 1998.- *Unpublished Report*.
- ATKINS E. L., 1975.- Injury to honey bees by poisoning.- In: *The hive and the honey bee*, Dadant and Son, Hamilton, Ill: 663-696.

- BAILEY, L. 1963.- *Infectious diseases of the honey bee*.- Land Books, London.
- BELZUNCES L. P., GUEZ D., SUCHAIL S., 1998.- Effets de l'imidaclopride chez l'abeille *Apis mellifera*.- *Unpublished Study Report*, INRA, Avignon.
- BITTERMAN M. E., MENZEL R., FIETZ A., Schäfer S., 1983.- Classical conditioning of proboscis extension in honeybees (*Apis mellifera*).- *Journal of Comparative Psychology*, 97: 107-119.
- BOECKING O., DRESCHER W., 1991.- Response of *Apis mellifera* L. colonies infested with *Varroa jacobsoni* Oud.- *Apidologie*, 22: 237-241.
- BRASSE D., 1999.- Preliminary report on a tunnel test with imidacloprid-treated summer rape.- *Unpublished Study Report*, 11 September 1999, Biologische Bundesanstalt (BBA), Braunschweig, Germany.
- BRUCE W. A., HACKETT K. J., SHIMANUKI H., HENEGAR R. B., 1990.- Bee mites: Vectors of honeybee pathogens.- In: *Proceedings of the International Symposium for Recent Research on Bee Pathology*, Gent, 180-182.
- COLIN, M. E., BONMATIN J. M., 2000.- Effets de très faibles concentrations d'imidaclopride et dérivés sur le butinage des abeilles en conditions semi-contrôlées.- *Unpublished Study Report*, INRA/CNRS/AFSSA.
- COLIN M. E., LE CONTE Y., DI PASQUALE S., BÉCARD J. M., VERMANDÈRE P., 1998.- Effet des tournesols issus de semences enrobées d'imidaclopride (Gaucho®) sur les capacités de butinage de colonies d'abeilles domestiques.- *Unpublished Study Report*, INRA Avignon.
- DECOURTYE A., 1998.- Etude des effets sublétaux de l'imidaclopride et de l'endosulfan sur l'apprentissage olfactif chez l'abeille domestique *Apis mellifera* L.- *Unpublished Study Report*, INRA Bures-sur-Yvette.
- DECOURTYE A., LACASSIE E., PHAM-DELÈGUE M. H., 2003.- Learning performances of honeybees (*Apis mellifera* L.) are differentially affected by imidacloprid according to the season.- *Pest Management Science*, 59: 269-278.
- EPPO, 1992.- Guideline on test methods for evaluating the side-effects of plant protection products on honey bees.- *EPPO Bulletin*, 22: 203-215.
- HAYDAK M. H., 1970.- Honey bee nutrition.- *Annual Review of Entomology*, 15: 143-156.
- KAUFFELD N. M., EVERITT J. H., TAYLOR E. A. 1976.- Honey bee problems in the Rio Grande Valley of Texas.- *American Bee Journal*, 116: 220-232.
- KIRCHNER W. H., 1999.- The effects of sublethal doses of imidacloprid on the foraging behaviour and orientation ability of honeybees.- *Unpublished Study Report*, University of Konstanz.
- KIRCHNER W. H., 2000.- The effects of sublethal doses of imidacloprid, hydroxy-imidacloprid and olefine-imidacloprid on the behaviour of honeybees.- *Unpublished Study Report*, University of Bochum.
- KROHN J., HELLPOINTNER E., 2002.- Environmental fate of imidacloprid.- *Pflanzenschutz-Nachrichten Bayer*, 55: 3-26
- KULINKEVIC J. M., ROTHENBUHLER W. C., RINDERER T. E. 1982.- Disappearing disease. Part I - Effects of protein sources given to honey-bee colonies in Florida.- *American Bee Journal*, 122: 189-191.
- KULINKEVIC J. M., ROTHENBUHLER W. C., RINDERER T. E. 1983.- Disappearing disease II. Effects of certain protein sources on brood rearing and length of life in the honey bee under laboratory conditions.- *American Bee Journal*, 123: 50-53.
- KULINKEVIC J. M., ROTHENBUHLER W. C., RINDERER T. E. 1984.- Disappearing disease III. A comparison of seven different stocks of the honey bee (*Apis mellifera*).- *The Ohio State University Research Bulletin*, 1160: 1-21.
- MACKENSEN O., 1951.- Viability and sex determination in the honey bee (*Apis mellifera*).- *Genetics*, 38: 500-509.
- MAUS C., 2002.- Evaluation of the effects of residues of imidacloprid FS 600 in maize pollen from dressed seeds on honeybees (*Apis mellifera*) in the semifielld.- *Unpublished study report*, MAUS/Am18, Bayer AG.
- MAUS C., SCHÖNING R., 2001.- Effects of residues of imidacloprid in maize pollen from dressed seeds on honey bees (*Apis mellifera*).- *Unpublished study report*, MAUS/Am12, Bayer AG.
- NAUEN R., EBBINGHAUS-KINTSCHER U., SCHMUCK R., 2001.- Toxicity and nicotinic acetylcholine receptor interaction of imidacloprid and its metabolites in *Apis mellifera* (Hymenoptera: Apidae).- *Pest Management Science*, 57: 577-586.
- OECD, 1998a.- Guideline 213: Honeybees, acute oral toxicity test.- *OECD Guidelines for the testing of chemicals*.
- OECD, 1998b.- Guideline 214: Honeybees, acute contact toxicity test.- *OECD Guidelines for the testing of chemicals*.
- OERTEL E., 1965.- Many bee colonies dead of an unknown cause.- *American Bee Journal*, 105: 48-49.
- PFLÜGER W., SCHMUCK R., 1991.- Ecotoxicological profile of imidacloprid.- *Pflanzenschutz-Nachrichten Bayer*, 44: 145-158.
- PHAM-DELÈGUE M. H., DECOURTYE A., LACASSIE E., 2000.- Etude en condition de laboratoire des effets létaux et sublétaux de l'imidaclopride et de ses principaux métabolites chez l'abeille domestique.- *Unpublished Study Report*, INRA LNCI Bures s/Yvette.
- RITTER W., 1996.- *Diagnostik und Bekämpfung der Bienenkrankheiten*.- Gustav Fischer Verlag, Jena.
- SCHMUCK R., 1999.- No causal relationship between Gaucho® seed dressing in sunflowers and the French bee malady.- *Pflanzenschutz-Nachrichten Bayer*, 52: 267-309.
- SCHMUCK R. (in prep.)- Effects of a chronic dietary exposure of the honeybee *Apis mellifera* to imidacloprid.
- SCHMUCK R., SCHÖNING R., STORK A., SCHRAMMEL O., 2001.- Risk posed to honeybees (*Apis mellifera* L., Hymenoptera) by an imidacloprid seed dressing of sunflowers.- *Pest Management Science*, 57:225-238.
- SCHMUCK R., NAUEN R., EBBINGHAUS-KINTSCHER U., 2003.- Effects of imidacloprid and common plant metabolites of imidacloprid in the honeybee: toxicological and biochemical considerations.- In: *Proceedings of the 8<sup>th</sup> International Symposium "Hazards of pesticides to bees"*, September 4-6, 2002, Bologna, Italy (PORRINI C., BORTOLOTTI L., Eds). *Bulletin of Insectology*, 56 (1): 27-34.
- SCHÖNING R., 2001.- Analytical method for the determination of residues of imidacloprid, NTN 33893-5-hydroxy, and NTN 33893-olefin by HPLC with electrospray MS/MS-detection in plant- and other materials.- *Pflanzenschutz-Nachrichten Bayer*, 54: 413-452.
- SCHÖNING R., SCHMUCK R., 2003.- Analytical determination of imidacloprid and relevant metabolite residues.- In: *Proceedings of the 8<sup>th</sup> International Symposium "Hazards of pesticides to bees"*, September 4-6, 2002, Bologna, Italy (PORRINI C., BORTOLOTTI L., Eds). *Bulletin of Insectology*, 56 (1): 41-50.
- SCHULD M., 2002.- Field test: Side effects of oil-seed rape grown from seeds dressed with imidacloprid and beta-cyfluthrin FS 500 on the honey bee (*Apis mellifera* L.).- *Unpublished Study Report 99398/01-BFEU*, GAB Biotechnologie & IFU Umweltanalytik.
- SCHULZ A., 1999.- Preliminary report on a field test with imidacloprid-treated sunflower plants.- *Unpublished Study Report*, 20 September 1999, Institute for Agriculture, Viticulture and Horticulture, Mayen.
- SCOTT-DUPREE C. D., SPIVAK M. S., 2001.- The impact of Gaucho and TI-435 seed-treated Canola on honey bees, *Apis mellifera* L.- *Unpublished Study Report 110405*, April 11

2001, University of Guelph/University of Minnesota.  
SUCHAIL S., GUEZ D., BELZUNCES L. P., 2000.- Characteristics of imidacloprid toxicity in two *Apis mellifera* subspecies.- *Environmental Toxicology and Chemistry*, 19: 1901-1905.  
SUCHAIL S., GUEZ D., BELZUNCES L. P. 2001.- Discrepancy between acute and chronic toxicity induced by imidacloprid and its metabolites in *Apis mellifera*.- *Environmental Toxicology and Chemistry*, 20: 2482-2486.

WILSON W. T., MENAPACE D. M., 1979.- Disappearing disease of honey bees: A survey of the United States.- *American Bee Journal*, 119: 184-217.

**Corresponding author:** Christian MAUS, Bayer AG, Bayer Crop Science, Institute for Ecotoxicology, Alfred-Nobel-Str. 50, D - 40789 Monheim, Germany.  
E-mail: christian.maus@bayercropscience.com