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Original article

Toxicity of mycotoxins to honeybees and its amelioration by propolis*

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Abstract – Honeybees (*Apis mellifera*) and their resource-rich nests are hosts to a wide range of saprophytic fungi, including species that produce mycotoxins. The toxicity of aflatoxin B1 (AB1) and ochratoxin A (OTA), products of *Aspergillus* species often found in honeybee hives, was evaluated and LC_{50} values for both toxins were calculated. Workers can tolerate a wide range of concentrations of both OTA and AB1. At low concentrations, AB1 (1 μg/g and 2.5 μg/g diet) and OTA (1 μg/g) did not have any apparent toxic effects on bees. Enhancement of the toxicity of AB1 by piperonyl butoxide (PBO), a known inhibitor of cytochrome P450 monooxygenases, indicates a role for P450s in AB1 detoxification in honeybees. Extracts of propolis, a complex mixture of plant-derived chemicals, including many flavonoids and other phenolic compounds, similarly ameliorated aflatoxin toxicity and delayed the onset of mortality. Collectively, these results suggest that tolerance of AB1 by honeybees may be due to P450-mediated metabolic detoxification. Propolis may serve a hitherto unrecognized role in honey bee health by enhancing the activity of P450 enzymes involved in mycotoxin detoxification.

honeybee / aflatoxin B1 / ochratoxin A / cytochrome P450 monooxygenases / piperonyl butoxide / propolis

1. INTRODUCTION

Honeybees (*Apis mellifera*) and their resource-rich nests are hosts to a wide range of saprophytic fungi. Fungi in the genus *Aspergillus*, for example, can colonize stored products in the hive, including pollen (González et al., 2005) and honey (Jimenez et al., 1994; Martins et al., 2003). Such fungi present a challenge to honeybees not only because they compete with honeybees for the stored food but because under certain conditions the fungi can produce mycotoxins that can cause toxicity in bees. *Aspergillus ochraceus* growing on pollen, e.g, produces both ochratoxins and aflatoxins (Medina et al., 2004). Aflatoxins at concentrations as low as 5 ppm can cause premature death of workers (Hilldrup and Llewellyn, 1979). In colonies weakened by other stresses, *Aspergillus flavus* can infect the bees themselves, gaining access to brood through the midgut and to adults through cuticular abrasions. Infection by this fungus manifests itself as a disease called stonebrood, typified by mummification of the lasvae (James and Pitt-Singer, 2008). Colonies are generally capable of recovering from such infections, depending on hygienic behavior and genetic identity (Gilliam and Vandenberg, 1988; Gilliam et al., 1989).

Although honeybees are more sensitive to the toxic effects of the mycotoxin aflatoxin B1 than *Drosophila melanogaster* and *Musca domestica* (Hilldrup and Llewellyn, 1978), the fact that colonies are often able to fend off infections is indicative of some capacity for coping with these mycotoxins, which may be frequently encountered in the hive environment. In many insect species, aflatoxins are metabolized by cytochrome P450 monooxygenases

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(P450s), heme-based enzymes that generally catalyze oxidative reactions that reduce lipophilicity and hence toxicity of a wide range of both natural and synthetic xenobiotics (Li et al., 2007). Aflatoxins and several other mycotoxins are unusual, however, in that in many species P450-mediated metabolic reactions result in bioactivation, or an increase in toxicity, via conversion of the aflatoxin to the more toxic epoxide metabolite (Saner et al., 1996).

Species that naturally encounter aflatoxins in their environment display some degree of adaptation to mycotoxins in that they metabolize these compounds not to bioactivated epoxides but rather to nontoxic breakdown products. The corn earworm *Helicoverpa zea*, for example, frequently causes damage in its hostplants that leaves them vulnerable to opportunistic infection by *Aspergillus* spp. In the midgut, *H. zea* detoxifies aflatoxin B1 (AB1) via CYP321A1 (Niu et al., 2008). Even more tolerant of aflatoxins is the navel orangeworm *Amyelois transitella*. This species infests dried fruits and nuts, particularly almonds and pistachios, and appears to prefer fungus-infected fruit (Palumbo et al., 2008). This species can tolerate dietary levels of AB1 100-fold greater than levels that inhibit *H. zea* (Niu et al., 2009) and produces principally aflatoxicol as a metabolite. Neither this species nor the aflatoxin-tolerant codling moth *Cydia pomonella* produce AB1-8,9-epoxide, the principal bioactivated metabolite of AB1 (Lee and Campbell, 2000). Although P450-mediated metabolism has been implicated in this tolerance, the specific P450(s) responsible for this detoxification have not yet been identified in either species.

In this study, we set out to ascertain the degree to which *A. mellifera* can tolerate exposure to mycotoxins and to establish whether tolerance is associated with cytochrome P450-mediated metabolism by using a known inhibitor of honeybee P450s, piperonyl butoxide, and a known inducer of honeybee P450s, propolis, the resinous "bee glue" that is a ubiquitous component of the hive. Johnson (2008) demonstrated that extracts of propolis administered orally to honeybees effects upregulation of three CYP6AS P450 genes, at least one of which is known to metabolize flavonoids found in honey, pollen and propolis (Mao et al., 2009). Understanding the mechanisms of resistance to mycotoxins may shed light on how this managed pollinator copes with chemical stresses.

2. MATERIALS AND METHODS

2.1. Insects

Honeybees were obtained from colonies containing multiply mated queens at the University of Illinois Bee Research Facility at Urbana (Champaign County), IL. Frames of late-stage pupae were taken from hives and transferred to a dark humid incubator at 32–34 °C. Newly eclosed adults were brushed from frames every 24 h for immediate use in bioassays.

2.2. Chemicals

Aflatoxin B1 and ochratoxin A (OTA) were obtained from Sigma Co (St Louis, MO). Piperonyl butoxide (PBO) was purchased from Tokyo Kassie Kogyo (Tokyo, Japan). Analytical grade dimethyl sulfoxide (DMSO) was purchased from Fisher Scientific (Pittsburgh, PA). All solvents, including methanol, were of analytical reagent grade. AB1 and OTA were dissolved in DMSO as concentrated stock solutions and stored in a –20 °C freezer.

2.3. Substrate carrier

Chemicals to be tested were administered to bees by incorporation into "bee candy," made using equal weights of powdered sugar and concentrated sugar solution with a ratio of 2:1 sucrose to water (w/w). Granulated table sugar (sucrose) was ground in a blender for approximately 3 min to make powdered sugar. Approximately 5 g fresh candy was poured into a 2 oz (56 mL) plastic cup (Solo, Urbana, IL); mycotoxins or a vehicle control were incorporated into the wet candy, which was then allowed to harden for 30 min.

To test effects of propolis on aflatoxin toxicity to honeybees, propolis was collected from hives in a forested area at Phillips Tract Research Area (Champaign Co., Illinois). Propolis was frozen in liquid nitrogen and then ground with a mortar and pestle and stored at –20 °C. Approximately 3 g of ground propolis was dissolved in 50 mL of hot methanol. Wax was precipitated from the cooled

methanol extract and the remaining extract was concentrated to 5 mL by evaporation under a stream of air. Propolis solution was added to powdered sugar and the methanol was allowed to dry for 24 h before mixing with an equal mass of heavy sugar syrup to make candy with a final concentration of 50, 150 and 300 mg propolis/g candy.

2.4. Bioassays

Newly eclosed workers were used for bioassays. Stocks of 10 μg/μL or 20 μg/μL of AB1 and OTA were dissolved in DMSO and stored at –20 °C. Bee candy was used to test the oral toxicity of mycotoxins with and without synergists via bioassay. For all bioassays, approximately 3 g of unanaesthetized newly emerged bees were transferred quickly to a 6 oz (188 mL; Sweetheart, Owings Mills, MD) wax-coated paper cup into which the treated candy had been placed. The cups were then covered with cheesecloth and placed into a dark humid incubator at a temperature of 32–34 °C. The mortality of bees was recorded every six hours until all bees had died.

To determine the oral LC_{50} of AB1 to bees, AB1 was incorporated into candy to achieve levels of 0.5, 1, 2.5, 5, 7, 10, 15 or 20 μg AB1 per g candy; in an additional treatment, DMSO was added to bee candy as a control. To determine the LC_{50} of OTA to bees, candy containing 1, 5, 10, 20, 40, 60 or 80 μg/g OTA was prepared, along with control candy containing DMSO. To test the effects of the known P450 synergist piperonyl butoxide (PBO) on the toxicity of AB1 or OTA to bees, two concentrations of PBO (0.05% or 0.1% PBO) were tested in the presence of AB1 or OTA. For AB1 vs PBO bioassays, candy containing six different concentrations of chemicals was prepared: 0.1% DMSO, 10 μg/g AB1, 0.05% PBO, 0.1% PBO, 10 μg/g AB1 supplemented with 0.05% or 0.1% PBO. For the bioassay comparing OTA and PBO, two doses of OTA (10 and 40 μg/g) were tested and compared with corresponding doses of OTA supplemented with 0.05% or 0.1% PBO.

To determine the effects of propolis, which has been shown to induce a subset of cytochrome P450 monooxygenase enzymes involved in detoxification (Johnson, 2008), the toxicity of AB1 and OTA in propolis-treated candy was determined. Five μl of stock AB1 (20 μg/μL) was incorporated into 5 g of plain candy to prepare the candy containing 20 μg/g AB1 or into 5 g of candy containing 50, 150 and 300 mg/g of propolis and mixed until the toxin was homogeneously distributed in the fresh candy. Candy treated with DMSO only and candy containing 50, 150 or 300 mg/g propolis without AB1 were used as controls.

2.5. Statistics

The R statistical package (R Development Core Team, 2009) with MASS libraries (Venables and Ripley, 2002) was used for log-probit analysis. LC_{50} values with 95% confidence intervals were calculated using Fieller's method (Finney, 1971). Treatments with non-overlapping 95% confidence intervals for LC_{50} were considered significantly different. Survival data to determine effects of PBO or propolis on the toxicity of AB1 or OTA to bees were analyzed using SPSS statistical software (SPSS Inc., Chicago, IL). With the function of survival analysis in SPSS, the median time for survival was calculated for each treatment independently. To compare the data among groups in any given bioassay, the data from all treatments were compared with one-way analysis of variance (Tukey plot) using the function of survival analysis in the software package. In the bioassay testing the effects of PBO on the toxicity of AB1 or OTA to honeybees, the *P* value was calculated to compare effects of AB1 or OTA in the presence or absence of the inhibitor. In the bioassay testing the effects of propolis extracts on the toxicity of AB1, the *P* value was calculated to compare impacts of AB1 in the presence and absence of propolis. Comparisons yielding p values equal to or less than 0.05 were considered to indicate significant differences.

3. RESULTS

3.1. Toxicity of mycotoxins to *A. mellifera*

None of the treatments containing AB1, even at the highest dose of 20 μg/g, resulted in bee mortality in less than 24 h (Fig. 1). The low concentrations of AB1 (1 μg/g and 2.5 μg/g) did not have any apparent toxic effects on bees over the entire duration of the bioassays. The intermediate dose of AB1 (5 μg/g) caused less than 50% mortality after 72 h. The high doses of AB1 (10, 15 and 20 μg/g) caused over 90% mortality in 72 h (Fig. 1). The LC_{50} of AB1 (defined as the concentration causing 50% mortality of treated bees in 72 h) was 6.76 μg/g (95% conf. int.= 5.86–7.69; slope =

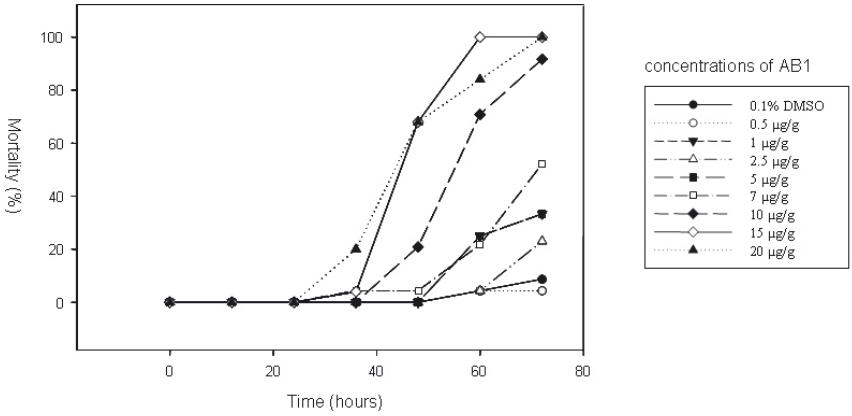

Figure 1. Mortality of *Apis mellifera* exposed to different concentrations of dietary aflatoxin B1 (AB1). Groups of approximately 30 newly eclosed workers were fed "bee candy" containing a range of concentrations of AB1 in DMSO and candy containing DMSO only. Each experiment was repeated three times (Tab. I); this figure graphically depicts the result of one replicate for visual clarity.

4.32+/–0.30; X^2 = 6.67; df = 4; n = 590) (Fig. 3).

Bees also displayed tolerance of a wide range of concentrations of OTA. The low concentration of OTA (1 µg/g) did not have any apparent toxicity to bees and the treated bees lived as long as the control bees. As with the AB1 treatments, no bee mortality was observed, even at the highest concentrations, in less than 24 h. Over time, mortality increased and concentrations equal to or greater than 10 µg/g caused 100% mortality after 72 h. Most of treated bees died during the final 24 hours (from 48 h to 72 h) (Fig. 2). The LC_{50} of OTA at 72 hours for bees was 5.04 µg/g (95% conf. int.=1.73–7.90; slope = 1.11+/-0.28; X^2 = 1.49; df = 2; n = 210) (Fig. 3).

While both OTA and AB1 exhibit similar toxicity to bees, the slope of the probit lines for AB1 is substantially steeper than is the slope of the probit line for OTA, indicating that the response to OTA is more heterogeneous than the response to AB1.

3.2. Effects of synergists on the toxicity of AB1 or OTA to bees

Enhancement of the toxicity of AB1 and OTA by administration of PBO, a known inhibitor of cytochrome P450 monooxygenases in bees (Johnson et al., 2006), indicates a role for P450s in mycotoxin metabolism in honeybees. Bees started to die earlier when consuming candy containing 10 µg/g AB1 supplemented either with 0.05% PBO or 0.1% PBO than did bees consuming candy supplemented with 10 µg/g AB1 alone (Tab. I). These results suggest that P450s contribute to enzymatic detoxification of AB1 in honeybees.

In contrast with AB1, PBO did not show any synergistic effects on the toxicity of OTA to bees. Two concentrations of PBO (0.05% and 0.1% PBO) were applied and two concentrations of OTA (10 and 40 µg/g) were used in the bioassays. There was no indication that PBO can accelerate or delay death of bees fed OTA (Tab. I).

3.3. Effects of propolis on the toxicity of AB1

To determine whether propolis, as an inducer of P450s, may contribute to tolerance of mycotoxins, bioassays of bees fed with AB1 alone or AB1 and propolis extract were conducted. Bees consuming candy containing 20 µg/g AB1 started to die after 24 h and all had died after 60 h, whereas bees consuming candy containing the same concentration

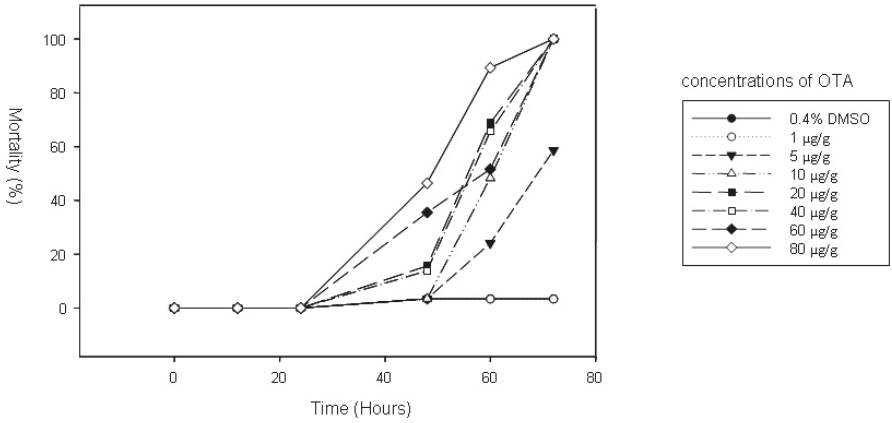

Figure 2. Mortality of *Apis mellifera* exposed to different concentrations of dietary ochratoxin A (OTA). Groups of approximately 30 newly eclosed workers were fed "bee candy" containing a range of concentrations of OTA in DMSO and control containing DMSO only. Each experiment was repeated three times (Tab. I); this figure graphically depicts the result of one replicate for visual clarity.

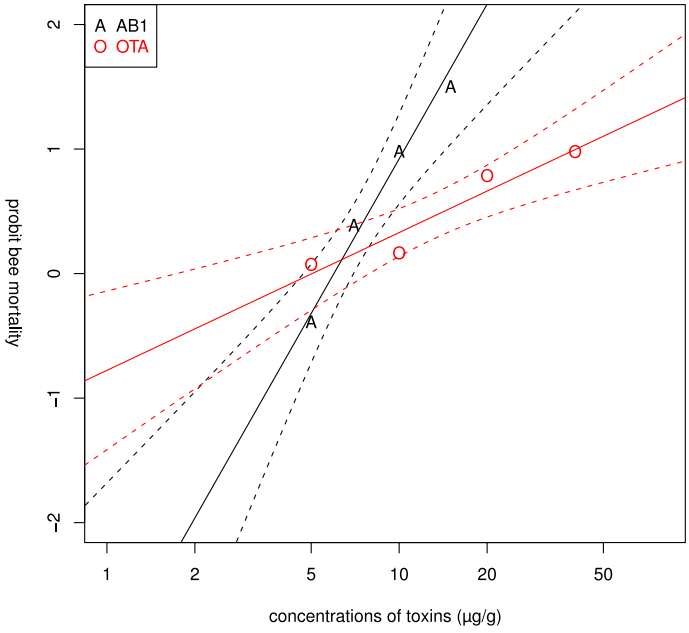

Figure 3. Comparison of LC_{50} of AB1 and OTA to bees. Plots of probits of bee mortality relative to different concentrations of AB1 or OTA. A (red) depicts AB1 treatments; O (black) depicts OTA treatments. LC_{50} is defined as the concentration of toxin that can cause 50% mortality in the tested bees after 72 h. Each experiment was repeated three times and the estimated LC_{50} is the average of three replicates. The calculated LC_{50} for AB1 and OTA are 6.79 µg/g and 5.04 µg/g.

Table I. Survival analysis of effects of piperonyl butoxide (PBO) on toxicity of AB1 (aflatoxin B1) or OTA (ochratoxin A) to bees. Bioassays were conducted to compare survival time of groups of bees fed mycotoxins in the presence and absence of PBO. Estimated median survival time of bees fed "candy" containing 10 μg/g AB1, 10 μg/g or 40 μg/g OTA, 0.05% PBO, 0.1% PBO, 10 μg/g AB1 supplemented with 0.05% PBO or 0.1% PBO, 10 μg/g or 40 μg/g OTA supplemented with 0.05% PBO or 0.1% PBO is the average of three replicates. Treatments of PBO plus toxin that differ significantly from toxin alone are indicated with "*" ($P < 0.05$).

Treatment	Median survival time (hours)	Error
0.1% DMSO	186	25
0.05% PBO	154	19
0.10% PBO	168	6
10 μg/g AB1	66	17
0.05% PBO + 10 μg/g AB1	39*	4
0.10% PBO + 10 μg/g AB1	39*	4
10 μg/g OTA	75	4
0.05% PBO + 10 μg/g OTA	72	0
0.10% PBO + 10 μg/g OTA	72	8
40 μg/g OTA	57	4
0.05% PBO + 40 μg/g OTA	60	0
0.10% PBO + 40 μg/g OTA	60	0

of AB1 supplemented with any amount of propolis lived longer than bees fed with AB1 alone; fewer than 10% of bees died in 60 h (data not shown). Survival analysis shows that the median survival time of bees fed with 20 μg/g AB1 is 44 h while that of bees fed with candy containing 20 μg/g AB1 plus 50, 150 or 300 mg/g propolis is 126, 150 and 154 h, respectively. There is a significant difference in survival time between bees treated with AB1 and those treated with AB1 and propolis (Tab. II). Longer median survival time with higher concentrations of propolis (150 mg/g and 300 mg/g) suggests a dosage-dependent ameliorative effect of propolis on AB1 toxicity.

4. DISCUSSION

As repositories of large quantities of food resources maintained under conditions of high relative humidities and temperatures, beehives are particularly vulnerable to opportunistic fungal infections. Given the frequency with which fungi such as *Aspergillus* colonize such environments, the relative tolerance that *A. mellifera* displays to mycotoxins such as AB1 and OTA is ecologically consistent with the likelihood of exposure. Similarly, *Helicoverpa zea*, the corn earworm, which only intermittently encounters aflatoxins in its preferred foodplants, displays substantially greater sensitivity to AB1 than does *Amyelois transitella*, the navel orangeworm, which routinely (and possibly preferentially) consumes fungus-contaminated plant food.

How *A. mellifera* tolerates mycotoxins such as aflatoxins and ochratoxins has not yet been determined definitively. In aflatoxin-tolerant species such as *H. zea* and *A. transitella*, metabolism of AB1 to non-toxic products is mediated by cytochrome P450 monooxygenases (Niu et al., 2008); in species that are less tolerant, such as *Drosophila melanogaster*, P450-mediated metabolism appears to result in epoxidation and hence bioactivation, or enhanced toxicity (Saner et al., 1995). That administration of PBO in the presence of aflatoxin increased its toxicity suggests that the honeybee, like the aflatoxin-tolerant *H. zea* and *A. transitella*, transforms these compounds to nontoxic metabolites via P450-mediated metabolism. Recent work by Johnson et al. (2009) indicates that the organophosphate coumaphos is also metabolized via P450s in bees to nontoxic products, rather than bioactivated to the more toxic oxon metabolite, as is the case for most insects.

Table II. Survival analysis of effects of propolis on toxicity of aflatoxin B1 (AB1) to bees. Bioassays were conducted to compare survival time of groups of bees fed mycotoxins in the presence of different concentrations of propolis. Estimated median survival time of bees fed "candy" containing 20 μg/g AB1, 50 mg/g, 150 mg/g and 300 mg/g propolis, and 20 μg/g AB1 supplemented with either of 50 mg/g, 150 mg/g or 300 mg/g propolis is the average of three replicates. Survival times for groups feeding on propolis and AB1 that are significantly different from survival time of groups consuming AB1 alone are indicated with "*".

Treatment	Median survival time (hours)	Error
0.1% DMSO	197	6
50 mg/g propolis	154	3
150 mg/g propolis	168	12
300 mg/g propolis	66	3
20 μg/g AB1	44	3
50 mg propolis + AB1	126*	21
150 mg propolis + AB1	150*	8
300 mg propolis + AB1	154*	18
0.10% PBO + 10 μg/g OTA	72	8

Ochratoxins are also produced by fungi that can be found in hive products (Gilliam et al., 1989). We found that, although workers can tolerate low levels of OTA (1 μg/g), concentrations higher than 5 μg/g will kill over 50% of exposed bees in 3 days under bioassay conditions. To date, no studies have been published on OTA metabolism by insects. Although metabolism of OTA in mammals is mediated primarily by P450s (Neal, 1995), the fact that, in our study, PBO has no effects on OTA toxicity suggests that this enzyme system might not be involved in the detoxification of OTA in honeybees. Studies with synergists must be interpreted cautiously, however, inasmuch as PBO does not necessarily inhibit all P450-mediated transformations in insects (Sanchez-Arroyo et al., 2001).

Propolis, prepared from resinous materials collected from plants by honeybees, is thought to function principally as a sealant for gaps within the hive to enhance structural stability (Burdock, 1998; Bankova, 2005). It also plays a key role in reducing decay of extraneous organic material within the hive and may also help bees fend off parasites and pathogens. Its antimicrobial activity is attributed to its high content of flavonoids and phenolics and in fact propolis extracts in vitro can inhibit the growth of fungi, including *Aspergillus versicolor*, A. *flavus, A. sulphureus* and *A. parasiticus*, and suppress production of mycotoxins, including AB1, OTA and sterigmatocystin (Aly and Elewa, 2007; Gómez-Caravaca et al., 2006; Ozcan, 2004; Pepeljnjak et al. 1982; Viuda-Martos et al., 2008). Simone et al. (2009) recently showed that exposure to propolis extracts downregulates expression of two honeybee immune-function genes concomitant with lowering the eubacterial loads, suggesting that propolis in the hive may reduce the need for immune gene expression by reducing bacterial loads.

That propolis may contribute to honeybee defense against fungi and their associated toxins not only by suppressing microbe growth but also by enhancing detoxification enzymes is a novel hypothesis consistent with the results of our study, at least with respect to AB1. Of the 46 P450s that are found in the honeybee genome, several in the CYP6AS subfamily are selectively induced by hive products such as honey, pollen and propolis, indicating a possible role in xenobiotic detoxification (Johnson, 2008). Indeed, Mao et al. (2009) demonstrated that CYP6AS3 is capable of metabolically transforming quercetin, a flavonoid found in propolis and in a wide variety of honeys. The ability of propolis to ameliorate the toxicity of AB1, as demonstrated in this study, suggests a function for propolis as an adjuvant for detoxification. Although the honeybee genome contains far fewer P450 genes than do other sequenced insect genomes, bees may compensate for reduced numbers by efficiently regulating the expression of a small number of

P450s for detoxification by induction with dietary phytochemicals. Individual P450s might be greatly induced by propolis extracts to biotransform AB1 into less toxic compounds, thereby reducing the risks of exposure to mycotoxins.

Propolis has long been used in traditional medicine and has been credited with immunomodulatory, anti-inflammatory and antimicrobial activity. At least some of the therapeutic activity of propolis in alternative medicine has been attributed to its function as an inducer of cytochrome P450 monooxygenase activity (Bhaudauria et al., 2007). Propolis treatment has been shown to increase activities of a range of P450s, including pentoxyresorufin depentylase, and ethoxycoumarin deethylase (Siess et al. 1996). More recently, Beltrán-Ramírez et al. (2008) reported that caffeic acid phenethyl ester, a common constituent of propolis, can modify expression of multiple P450s to inhibit the activation of diethylnitrosamine in rats. To some extent, it is remarkable that the body of knowledge relating to the biochemical activity of propolis in human health is substantially larger than that relating to the biochemical activity of propolis in honeybee health; a greater understanding of the multiple roles of propolis in the life of the honeybee can be gained by further study of the ways in which it is processed and metabolized.

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Toxicité des mycotoxines pour les abeilles et atténuation de cette toxicité grâce à la propolis.

Abeille / aflatoxine B1 / ochratoxine A / cytochrome P450 / monooxygénase / butoxide de piperonyle / propolis

Zusammenfassung – Toxizität von Mycotoxinen für Honigbienen und ihre Melioration durch Propolis. Honigbienen und ihre Ressourcen-reichen Nester, die bei hoher Temperatur und Luftfeuchtigkeit gehalten werden, sind eine geeignete Umgebung für das Wachstum einer großen Vielfalt von opportunistischen Mikroorganismen. Hierzu gehören mehrere Pilzarten, die toxische Substanzen produzieren (sogenannte Mycotoxine). Die Toxizität von Aflatoxin B1 und Ochratoxin A, Produkte von häufig in Bienenvölkern gefundenen Aspergillusarten, wurden in dieser Studie bestimmt und die Konzentrationen der jeweiligen Toxine wurden berechnet, bei denen im Mittel 50 % aller getesteten Bienen sterben. Arbeiterinnen können ein großes Konzentrationsspektrum beider Mycotoxine tolerieren. Bei geringen Konzentrationen waren weder Aflatoxin B1 noch Ochratoxin A giftig für die Bienen, aber höhere Konzentrationen waren nachweislich letal innerhalb von 48 h. Insgesamt wirkt Aflatoxin B1 stärker toxisch als Ochratoxin A. Die Bienen sind offensichtlich in der Lage, Aflatoxin B1 zum Teil mit ihrem Darmenzym, Cytochrom P450 –Monooxygenase, zu metabolisieren. Wir schliessen das aus dem Befund, dass die Toxizität dieses Mycotoxins durch die Anwesenheit von Piperonyl-butoxid, einem Inhibitor dieses Enzyms, erhöht ist. Extrakte von Propolis, einer Mischung von Pflanzenharzen, die von Bienen als allgemeiner Kitt im Nest eingesetzt werden, verringern die Toxizität von Aflatoxin B1 und verzögern den Sterbezeitpunkt von Bienen, die das Mycotoxin aufgenommen haben. Propolis könnte eine bislang unerkannte Rolle für die Bienengesundheit spielen, indem es die Aktivität solcher Enzyme steigert, die an der Mycotoxin-Entgiftung beteiligt sind.

Honigbiene / Aflatoxin B1 / Ochratoxin A / Cytochrom-P450-Monooxygenasen / Piperonyl-butoxid / Propolis

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