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Newsletter E-mailed to You

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Synergism

Since years of study on colony collapse disorder (CCD) of honey bees have not produced “the smoking gun” (a single cause) for the malady, scientists are turning to potential multiple causes. The studies are designed to try to find synergistic interactions of chemicals in the hive that may be damaging the bees. The dictionary definition of synergism is: interaction of discrete agencies or agents such that the total effect is greater than the sum of the individual effects. In other words, one plus one equals more than two. The question is, “Can pesticide residues, infectious agents, and/or malnutrition combine to be much worse for the bees than simply the additive effect of each alone?”

Remember, I am not a biochemist or toxicologist, so this is just a synthesis of

things I have put together from various sources. It may or may not be the way things really are happening.

Most pesticides (insecticides, fungicides, and herbicides for this discussion) are formulated to disrupt vital biochemical pathways in the target organism enough to stop it from growing or to kill it. In some cases, the biochemical pathways are specific to the targeted pest. But, many of the disrupted pathways are common to life, in general, and the toxicants are not very specific. When we talk about those disruptions, we are discussing “modes of action.”

Many pesticides of animals are designed to damage some portion of the nervous system. Some damage the nerve cells, directly, but most of them interfere in one way or another with the flow of electric impulses from nerve to nerve. Although the dendrites (like rootlets on a plant) of nerve cells are extremely close together, they don't touch. So, a chemical mediator, acetylcholine, is released from one cell. If enough accumulates, the impulse moves along. Then an enzyme quickly breaks down the acetylcholine, so that the nerve does not “fire” excessively. The insecticides that are acetylcholine esterase inhibitors do not let the breakdown enzyme work, so the nerve cells in the body keep firing, muscles contract permanently, and the insect dies. Insecticides relying on this mode of action are the organophosphates (coumaphos) and the carbamates.

When nerve cells fire, there are some subtle, but extremely important, changes in permeability of portion of the cells. Calcium, sodium, and chloride ions can move in and out of cells through “channels” (act more like pores with valves, as I see it). So, keeping the channels permanently open

or closed inhibits normal nerve function. The pyrethroids (fluvalinate), abamectin, and many of the “older” chemicals like DDT use this mode of action.

Recently, insecticides have been formulated that function by combining, physically, with receptors for acetylcholine right on the nerve cells. They either can block the receptors (antagonists) or keep the receptors acting as though they are receiving acetylcholine even though they are not (agonists). The neonicotinoids and spinosad belong here.

Another group of acaricides and insecticides works in the mitochondria (small, but critically important cell organelles) where adenosine diphosphate and adenosine triphosphate interact to produce energy for the cells and organs to function. These pesticides usually interfere one way or another with oxidative phosphorylation.

There are inhibitors of chitin formation. Chitin is the meshwork exoskeleton of insects. There are hormone disruptors so that molts do not take place normally. Now, there are inhibitors of lipid synthesis (spiromesifen) and muscle paralyzers (flubendiamide).

Amitraz is in its own class of octopaminergic agonists. Octopamine is involved in many functions in insects, but the nervous tissue functions are targeted for insect control.

Journal articles have been published that report synergism between certain fungicides and certain insecticides. Early reports on residues of agrichemicals in beeswax and stored pollens (bee bread) from CCD colonies listed fungicides as one of the commonly occurring group of pesticides. While we don't usually think of fungicides

as being toxic to adult honey bees, we know we have problems with some of them with immature bees. Modes of action of fungicides fall into specific groups compiled by the Fungicide Resistance Action Committee: A. Nucleic Acid Synthesis; B. Mitosis and Cell Division; C. Respiration; D. Amino Acid and Protein Synthesis; E. Signal Transduction; F. Lipid Membrane Synthesis, Cell Wall Deposition; G. Sterol Biosynthesis Inhibitors (SBI fungicides); H. Glucan Synthesis; I. Melanin Synthesis in Cell Wall; M. Multi Site Action; P. Host Defense Inducer; and U. Recent Molecules.

As the names imply, some of these groups target fungal cell walls and probably would not affect bees. However, many other targeted metabolic pathways are shared with bees. Documented losses have occurred with captan (Group M – phthalamides), ziram (Group M – dithiocarbamates and relatives) and iprodione (Group E3 – dicarboximides).

It appears to me that there should be more research on the two largest groups of fungicides: the ones that affect respiration and the sterol biosynthesis inhibitors. At molecular levels, respiration is pretty similar in plants, fungi, and bees. At the molecular level plants, fungi, and bees wind up with lanosterol as a precursor to ergosterol (fungi) or cholesterol (bees). The fungicides act by preventing demethylation of lanosterol-related precursors of essential sterols, which eventually become critical cell membrane components and hormones of bees. A common problem with exposure to fungicides is loss of brood, as larvae, or as pupae that cannot molt properly to adults. That sounds to me like a hormone problem. We have a lot of work ahead of us to prove or disprove possible synergisms.

In late breaking news (Journal of Economic Entomology, 102(2); 474-479, 2009) R. Johnson, H. Pollock and M. Berenbaum from the University of Illinois published “Synergistic Interactions Between In-hive Miticides in *Apis mellifera*.” The miticides studied were fluvalinate and coumaphos. Their studies suggest that both chemicals are detoxified through biochemical pathways requiring cytochrome P450 monooxygenase enzymes (P450s). Similar to the relative paucity of genes for producing immunological peptides, honey bees seem to be pretty low in producing P450s. So, too high a dose of either chemical, or especially both together, can be problematic.

In these studies the researchers showed that two of three enzyme inhibitors made coumaphos more toxic to bees than normal, while all three made fluvalinate more toxic. Next, by pre-treating four day old bees with coumaphos or fluvalinate, it took less than normal amounts of the other chemical to kill the bees (in one case 32.1 times less).

I still think it would be a good idea to get rid of the heavily fluvalinate and coumaphos-contaminated (and whatever else may have been used) brood nest combs that have served through the *Varroa* wars.

Another imidacloprid study

Also in that recent issue of the Journal of Economic Entomology (Vol. 102 (2): 616-623, 2009) is a study sponsored by the Agricultural Department of the Walloon Region of Belgium. Beekeepers in that region have been experiencing abnormally high winter losses of colonies. Although no sunflowers or canola are grown there, they

do have bees around fields of corn, some of which are planted with seed treated with imidacloprid.

So, a set of ten researchers, from government agencies, the University of Liege, and the Institute of Tropical Medicine joined forces to look at the question: “Does Imidacloprid Seed-treated Maize Have an Impact on Honey Bee Mortality?”

The researchers analyzed a total of 48 honey, bee, and beeswax samples for pesticide residues and recorded colony mortality from apiaries located in ever increasing diameter circles from corn fields that were or were not treated with imidacloprid.

The residue results were fairly similar to those reported from our CCD studies in the U.S. The most common residue in the Belgium honey was rotenone, an “organic” miticide/insecticide that isn’t registered for use in hives in that country. The next most common detection was flusilazole, an azole fungicide not registered in the U.S. Next was methiocarb sulfoxide, a carbamate fungicide not registered in the U.S. Triflxystrobin, a fungicide produced by Bayer and sold in the U.S. as Flint[®] or Stratego[®], was detected six times, while imidacloprid was detected four times. But, it is important to state that rotenone, at 15.2 ppb was at much higher concentrations than the later mentioned chemicals. The later chemicals were between the limit of detection (LOD) and limit of quantification (LOQ), or very low levels. Coumaphos topped the list in amount of residue in honey – 128 ppb. One bee sample contained lindane.

The beeswax samples contained many residues with flusilazole most frequent, four *Varroa* control products next,

then many agrichemicals, but no imidacloprid.

Colony mortality, interestingly, was not related to imidacloprid exposure in the manner that might be expected. The colonies with the most exposure to pollen from imidacloprid seed-treated corn fields survived the best. In fact, there was a very strong correlation: very limited exposure to imidacloprid-treated corn acreage resulted in colony mortalities up to nearly 60%, while, with one exception, colonies exposed to nearby, large areas of treated corn suffered practically no losses.

There was another interesting correlation – simply the proximity to corn plantings was a favorable aspect of the study. The farther away from corn plantings, the less well the colonies survived, suggesting a beneficial effect of consuming corn pollen, despite the residual chemicals.

A third interesting correlation existed between winter loss and number of colonies in the apiary. No colonies were loss in apiaries of three to six colonies. Losses picked up at eight colonies per apiary and averaged around 50% for 14 to 42 colonies.

John Muir Views the Central Valley

Kathy Kellison, a relentless advocate for native and honey bees, as well as the founder of Partners for Sustainable Pollination, was able to obtain permission to reprint Chapter 16 of John Muir’s 1894 book “The Mountains of California.” Kathy hopes to sell this 36 page paperback booklet as a fund raiser for her non-profit organization. Following is a description of portions of the valley floor, as John Muir had seen it, before agriculture “made sad havoc in these

glorious pastures, destroying tens of thousands of flowery acres like a fire, ...”

“The Great Central Plain of California, during the months of March, April, and May, was one smooth, continuous bed of honey-bloom, so marvelously rich that, in walking from one end of it to the other, a distance of more than 400 miles, your foot would press about a hundred flowers at every step. Mints, gilies, nemophilas, castilleias, and innumerable compositae were so crowded together that, had ninety-nine percent of them been taken away, the plain would still have seemed to any but Californians extravagantly flowery. The radiant, honey-filled corollas, touching and overlapping, and rising above one another, glowed in the living light like a sunset sky – one sheet of purple and gold, with the bright Sacramento pouring through the midst of it from the north, the San Joaquin from the south, and their many tributaries sweeping in at right angles from the mountains, dividing the plain into sections fringed with trees.”

“Descending the eastern slopes of the Coast Range through the beds of gilies and lupines, and around many breezy hillock and bush-crowned headland, I at length waded out into the midst of it. All the ground was covered, not with grass and green leaves, but with radiant corollas, about ankle-deep next the foothills, knee-deep or more five or six miles out. Here were bahia, madia, burrelia, chrysopsis, corethrogyne, grindelia, etc., growing in close social congregations of various shades of yellow, blending finely with the purples of clarkia, orthocarpus, and oenothera, whose delicate petals were drinking the vital sunbeams without giving back any sparkling glow.”

It is abundantly clear that California beekeepers in the late 1800’s and early

1900’s had access to huge expanses of wildflowers that produced substantial honey crops. Now, in most places, the bees are lucky to eke out a living. Adapting to 20th and 21st Century changes can be difficult.

Open Mouth – Insert Foot

In case it hasn’t been brought to your attention, yet, I had quite a quote on CCD in the March 19th edition of the on-line publication Petaluma360.com. Jay Gamel quoted me as saying, “At this particular moment we can’t put our finger on what causes that problem. Many of the things that have been studied — trucking long distances, monoculture, pesticides — are all stresses, but don’t hurt the bees. I’m open to the possibility of an unidentified microbe out there,” he said.

So, how did that happen? Of course those stresses “hurt” bees. I wrote a published research article on the negative effects of some fungicides on larval honey bees.

It happened when we had spent a good deal of time going individually over all the possible causes of CCD. I had previously stated that all the discussed topics had negative impacts on honey bee populations. But, we headed into a sort of quick review mode, leading to a statement about still undiscovered possibilities. My mind went to the University of Montana virus work. So, instead of repeating, again, that the listed causes were not individually the cause of the problem, I summed up with “don’t hurt the bees.” It is obvious that “... don’t, individually, seem to be the cause of CCD” would have been a lot better, but a lot longer.

Did I hear my self say it? Yes. Did I think that out of all that was said and recorded, that would be the one statement to be used? No! I was wrong.

I will continue to give interviews and do my best to disseminate accurate and useful information. I hope I don't do something like that, again, for a long time. But, it happens!

Time to Prepare for WAS 2009

The officers of WAS, the Sonoma County Beekeepers and the Marin County Beekeepers are attempting to cover quite a few areas of interest for beekeepers and those interested in native pollinators at the next WAS Conference. Details of conference dates and times, daily speaker schedules, registration, etc. can be found at the Web location given at the end of this article.

Areas of interest are separated so that people living nearby, with special interests, may choose which sessions to attend. Monday afternoon will be "set up" time for vendors and the Silent Auction. A Delegates' and Directors' Meeting is scheduled for 3:00 pm in an out-of-the-way corner of the Conference room. Registration will begin around 3:00 pm, also, in the Krug Event Center.

Monday evening, August 17th, will start things off with a honey and wine tasting from 7 – 9 pm in the Krug Center at the Dry Creek Inn. The tasting will be led by WAS Treasurer, Mark Pitcher. A native of nearby Napa Valley, Mark teaches classes, annually, in wine making in northern California. Silent auction items will be available for bidding that evening, also.

Tuesday morning, August 18th, we are going to learn the latest information coming from university studies on honey bee diseases and honey bee health. Jerry Bromenshenk will describe the seasonality of virus titers in the samples they have processed. He also is likely to describe and display his hand-held, acoustic monitoring device that "listens" to the health of a honey bee colony. (For fun, look up Wood's Apidictor in your browser to see its predecessor.) Following Jerry, Häagen Dazs postdoctoral fellow Michelle Flenniken will describe her efforts to determine how honey bees respond to being infected with viruses. She also will tell us how things are coming along with the bee disease microarray diagnostic chip. Then, Steve Sheppard will describe the changes in pathogen and parasite loads over the season in commercial beekeeping operations in the State of Washington. Jerry and Michelle may share similar baseline data on other commercial and hobby beekeeping operations with which they are involved.

Tuesday noon many of us we will carpool a short distance to April Lance's property on West Dry Creek Road for a riverside BBQ (Dry Creek is too shallow for swimming). A map to the site will be provided in the green Healdsburg shopping bag handed out at registration. April is coordinating with a meat company to provide a great spread. A vegetarian entrée will be available upon previous request. Following the BBQ, participants can have a free afternoon or they can attend an afternoon session with Dr. Larry Connor dealing with topics of interest suggested by area club members. Dr. Connor's sessions have an additional fee, beyond the Conference registration free. (Don't abandon your carpool passengers!).

On Wednesday morning, August 19th, many of us will be leaving the Krug Event Center for a visit to Serge Labesque's apiary in Glen Ellen (about a 45 minute drive). Among a number of other items of interest, we should have a Hungarian motorized, rotating circular combs hive, and a number of different top bar hives and apiary watering devices. Carpooling is essential for this trip. A map to the White Barn will be provided in the green shopping bag handed out at registration. In Glen Ellen we will be meeting in the White Barn, for an introduction from Anne Teller, the owner/manager of the organic farm and produce center (Red Barn) on the other side of the hill, before visiting the apiary.

For others, Dr. Larry Connor will be conducting a second, additional fee, half day session, dealing with a set of beekeeping topics different from the previous afternoon session in the Krug Center. Following the morning activities, lunch will be on your own. A list of eateries, nearby the Krug Center, will be provided in the registration materials

Wednesday afternoon, we shift to native pollinators. UC Davis Emeritus Professor Robbin Thorp will introduce us to the fascinating world of non-*Apis* bees. He will be followed by two other professors, Neal Williams and Claire Kremen, who will describe their studies on the contributions of native bees to commercial crop production east and west of the Rockies, respectively.

Thursday, August 20th, the last day of the Conference, is a very full day. The morning session opens with Ron Fessenden, MD, MPH; founder of The Committee for the Promotion of Honey and Health, Inc., and co-author of the books: "The Hibernation Diet" and "The Honey Revolution." Following Ron's introduction to the topic,

we hope to have Liz Applegate continue the topic of honey and human physiology by sharing her findings on the effects of honey on athletic performance. Then, we have the Annual Business Meeting that will bring us to lunch time. Lunch is on your own and there are a number of interesting places to eat nearby.

Following lunch, Dewey Caron, Emeritus Professor, author of the textbook "Honey Bee Biology and Beekeeping" and next year's WAS President, will be describing the natural history of a honey bee colony. That will be followed by sideline beekeeper; part time researcher; part time school teacher; and bee journal author Randy Oliver. Randy will share with us his knowledge of many topics determined in many ways. After Randy, the Silent Auction ends and there is a Delegates' and Directors' Meeting at 3:30 pm.

At 6:00 pm we will have a no host social, then proceed to our Buffet Banquet and Awards Presentations in the Krug Center beginning around 6:30 pm. That evening, or the next day, everyone should enjoy a safe trip home or to wherever the next destination is.

Travel Tips

Healdsburg does not have much other than local transportation and taxis. There is a Charles M. Schultz (Sonoma County) Airport. Currently, Horizon Air [(800) 547-9308] has one flight in and out to Las Vegas, daily; two flights in and out to Los Angeles, daily; one flight in and out to Portland, OR, daily, and two flights out and one flight in from Seattle, daily. Avis, Enterprise, and Hertz rent cars at the airport, but it is only an eleven mile trip to Healdsburg, so a taxi might be less

expensive. There is an Airport Express that busses folks from the Sonoma County Airport to either the San Francisco or Oakland airport for \$32 one way (only save \$2 on round trip ticket). Greyhound does not stop close to Healdsburg, although it must go right through the town, as it has stops in Santa Rosa and Ukiah along highway 101.

Program details and registration information are available on the WAS Web site: www.groups.ucanr.org/WAS/.

Sincerely,

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