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HONEYBEE NEWS

"The voice of the Beekeeper"

Volume 7 Number 1

JANUARY - FEBRUARY 2014

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CONTENTS

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PRESIDENT'S REPORT

Happy New Year and I hope everyone had a safe and happy holiday season. Unfortunately the beginning of 2014 did not bring any rain to speak of, which means this season is looking like it is going to be one of the worst we have seen for many years. The silver lining to this cloud is that there is an increase in honey prices, but it is a shame it only happens when honey is in such short supply.

SENATE INQUIRY
The Executive met in Sydney in early January this year to work on our submission to the Senate Inquiry, *Future of the beekeeping and pollination service industries in Australia*. Each of the Executive team had been discussing the issues with various NSWAA members, and we had also received various emails and letters from a number of Branches and individuals via our secretary. Thanks to all of you who provided input.

At this stage we have the first draft of the submission and are in the process of fine-tuning it. There has been an extension of the deadline for submissions until March 31, which means we will now have time to send a draft to branches for feedback before we submit the final version. It will still be a short turn around time, but I look forward to your further input.

Due to the importance of this submission, the short turnaround time, the breadth of topics and the other commitments of the Executives, we felt that it was in the best interest of the Association to engage consultants to help with preparation to make sure our submission is as powerful as possible. I am confident the NSWAA's submission will argue a strong case for the importance of our industry, and our need for security of access to floral resources, as well as the need for support in protecting industry from biosecurity risks.

SYDNEY SHOW
The Sydney Show is fast approaching; this year it runs from 10-23 April. Once again Bruce White is doing a fantastic job for us as the Show Coordinator. Your help as a volunteer at Honeyland would be very much appreciated. Volunteers sell honey, help promote the Association by just being there and talk to the adoring public about bees and honey – the city slickers who visit the Show and *Honeyland* are absolutely fascinated by bees and love meeting real beekeepers like you.

Please contact Bruce (02 9634 6792 or blwhite11@hotmail.com) if you are able to volunteer. Tickets for entrance to the Show and accommodation will be provided. And if there is anyone who can donate honey, other bee/honey/hive products or promotional material, Bruce would certainly like to hear from you.

2014 CONFERENCE
After the fantastic conference in 2013 we are working hard to ensure we put together a great meeting for members this year. The Conference will be held at the Crossing Theatre in Narrabri Thursday 8 and Friday 9 May. As I have flagged in previous reports I am very happy that this year we will be holding a workshop on Wednesday 7 May to discuss and plan the future direction of our Association.. The Branches will shortly be receiving an invitation to nominate two of their members to participate. These nominees will be asked to participate in the workshop and bring to the day feedback from their Branches on their vision for the future of the Association.

NSWAA WEBSITE
Although we currently have a website, which was developed through the generosity of Kieren Sunderland, the Executive feel that we need a site that is easier to manage and keep up to date. As website development has advanced so much in recent times, we are working on creating a new NSWAA website within a system that will allow much more flexibility and allow easy updates. We hope that this will grow as a communication tool, which will compliment other tools we have, like the fantastic Honeybee News.

FORESTRY POLICY
Although we have been waiting 15 months the Forestry Corporation of NSW (FCNSW) has still not released its new policy on beekeeping its managed lands. The Executive have been working with the Southern Region FCNSW management on a temporary beekeeping policy as an interim measure because the current lack of policy has meant that FCNSW in this region have not been issuing any site licences.

All branches will be sent a copy of this temporary policy once it is finalised. We are also hoping to meet with a FCNSW representative during our next Executive meeting to further push our case to ensure better security for NSW beekeepers around access to resources.

COTTON SPRAYING AND BEES
Once again NSW is seeing large areas planted with cotton, and although bees on cotton flowers can increases a farmer's yield by almost 20% many of them are unaware of the importance of bees for pollination services. But more importantly for us, many of them don't think about the impact of their pesticide spraying on our bees. We want to make sure that we don't have a repeat of the huge number of bee deaths through spraying of cotton crops that we saw last year. If you put bees anywhere near cotton (or for that matter other mono-cultural crops likely to be sprayed), make sure you let all the local farmers know where your hives are. Send them a letter, send them an email AND turn up on the doorstep with a bucket of honey to make sure they get the message. Then if anything unfortunate does happen it will be much harder for those responsible to get away with it. The Executive have been talking to the EPA to raise our concerns and our profile.

If anything untoward happens near your bees contact the EPA and the Executive right away.

Casey Cooper
State President

YOUR EXECUTIVE AT WORK

The Executive Committee met on the 10 January 2014. This was a short notice meeting called in order to brief engaged consultants to provide an industry response to the Senate Rural and Regional Affairs and Transport Reference's Committee inquiry into 'the future of the beekeeping and pollination service industries in Australia'.

The Executive Committee is currently providing feedback on the first draft which will be further discussed at their next formal meeting in February. Following this the final draft will be provided to the Branches for their comment.

A final position will be provided to the Senate Committee by the newly extended submission deadline of 31 March 2014.

The next formal meeting date of the Executive Committee will be Monday 17 February 2014.

A SNEAK PEEK AT CONFERENCE

- Using satellites to move beehives
- The Chinese Industry – An Australian beekeeper's perspective
- The way forward – what will our industry do next?
- Varroa mite in Papua New Guinea
- Inter species matings
- Lambplan and its applicability to bee breeding programs

CROP REPORT

Here we are into February and still with very dry conditions with very high fire dangers, and no honey production.

A lot of bees had been moved onto the River Red Gum in the Central West with next to no honey produced.

There has been reports that some hives had melted down while on the River Gum, and there has been another report a couple of beekeepers had their hives sprayed from cotton spraying while on River Gum. It appears that the EPA Authority still doesn't want to take any action against the cotton industry, so beekeepers should be a bit more proactive when placing hives near where cotton is being ground.

Some of the honey packers have increased their honey and wax prices due to the lack of honey production and honey on hand in beekeeper's sheds. There are a few trees putting bud on for autumn but with no rain in sight and still very hot conditions they could grow too quickly and flower early or drop bud.

This report doesn't seem very encouraging, but maybe by the end of February we may see the weather pattern start to change with some rain by the time I do my next report.

Mal Porter
Central Tablands

2014 SYDNEY SHOW

VOLUNTEERS NEEDED

Once again the Association will have *Honeyland* at the Sydney Royal Easter Show which runs from:

10 April - 23 April

In this edition there is a form for volunteers

If country volunteers need accommodation return your form no later than 28 February

If you would like to assist this year you would be very welcome and I'm sure you will enjoy the experience.

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BRUCE WHITE
SHOW COORDINATOR

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WHY THE HONEY BEE INDUSTRY NEEDS

THE NATIONAL BEE BIOSECURITY PROGRAM

AHBIC is currently reviewing the honey levy, with a view to reforming and increasing the honey levy from 2.3c/kg to 4.6c/kg which would apply to a producer selling over 1500kg of honey per year. AHBIC is consulting with all sectors of the industry about these proposed changes, with an industry-wide ballot on these proposed changes to be held throughout early-mid 2014. The purpose of this increase in the honey levy is to raise additional funds to contribute to additional biosecurity activities for beekeepers, with a focus on the management of established pests and diseases of honey bees in Australia.

AHBIC is proposing to develop a National Bee Biosecurity Program (funded from this levy increase) which would be underpinned by an Australian Beekeeping Code of Practice and would promote beekeeping best management practices for commercial beekeepers in Australia. The development of this program sets out the case for industry to take a leadership role in managing their own biosecurity issues for established pests and diseases. Where possible, the Code will be compliant with existing state and territory legislation but the objective is to achieve 'best practice' by taking the best elements of existing legislation, both in Australia and overseas (such as the AFB National Management Strategy in NZ).

Established pests and diseases cause significant economic and social harm and need strategic management to limit the impact to individual beekeepers and the broader industry and economy. Evidence shows that problems caused by pests, such as American Foulbrood (AFB), are only getting worse and the current state-based policies and systems are not working. Overseas experience also suggests that if major established pests such as AFB are not properly controlled when a pest such as Varroa mite arrives, the dual effect is worse than expected. For these reasons, greater national coordination and industry leadership is urgently needed. It is envisaged that the National Bee Biosecurity Program would deliver five major benefits to commercial beekeepers in Australia:

1) Greater industry communication, training and educational material will be produced and provided to beekeepers which will focus on surveillance, identification, prevention and control of honey bee pests and diseases;
2) Improved level of overall biosecurity of commercial beekeepers in Australia through the development of the Australian Beekeeping Code of Practice;
3) Reduced incidence of established pests and diseases, such as AFB, thereby lowering the economic losses presently experienced by beekeepers;
4) Improved surveillance for exotic pests (such as Varroa mite) as beekeepers will be required to inspect hives more frequently and have better knowledge of identification of pests and diseases;
5) Establish an effective working management and coordination structure between industry and government, which will help in the event of an incursion of an exotic pest (such as Varroa mite).

If you support the honey bee industry taking a leadership on biosecurity, and developing the National Bee Biosecurity Program which will deliver greater communication, training, awareness, education material and inspection services for beekeepers to deal with established pests and diseases, **please vote yes for the proposed reforms and increases in the honey levy.**

For more information about the proposed honey levy reform and increase, go to: http://honeybee.org.au/programs/honey-levy-reform-and-increase/

WHY THE HONEY BEE INDUSTRY NEEDS

THE NATIONAL BEE PEST SURVEILLANCE PROGRAM

AHBIC is currently reviewing the honey levy, with a view to reforming and increasing the honey levy from 2.3c/kg to 4.6c/kg which would apply to a producer selling over 1500kg of honey per year. AHBIC is consulting with all sectors of the industry about these proposed changes, with an industry ballot on these proposed changes to be held throughout early-mid 2014.

The purpose of this increase in the honey levy is to raise additional funds to contribute to additional biosecurity activities for beekeepers. Part of the money raised as part of this levy increase would go towards AHBIC's commitment to the National Bee Pest Surveillance Program (formerly known as the National Sentinel Hive Program). The National Bee Pest Surveillance Program is currently cost shared at a national level between AHBIC, pollination-reliant industries through Horticulture Australia Limited (HAL) and the Commonwealth Department of Agriculture.

The Australian mainland is currently free from some of the most significant pests of honey bees, namely the Varroa mites (*Varroa destructor* and *V. jacobsoni*), Tropilaelaps mite (*Tropilaelaps clareae* and *T. mercedesae*) and Tracheal mite (*Acarapis woodi*). The establishment of any of these pests in Australia would greatly increase the costs for the honey bee and pollination services industry.

The National Bee Pest Surveillance Program is a nationally cost shared and risk based surveillance program that is undertaken across Australia to deal with the risk posed by these exotic pests (such as Varroa mite). The Program involves a range of surveillance methods conducted at locations considered to be of most likely entry of bee pests and pest bees throughout Australia.

The NBPSP supports two objectives:

1. **Exotic bee pest and pest bee early warning:** to act as an early warning system to detect new incursions of exotic bee pests and pest bees. This greatly increases the possibility of eradicating an incursion, and limits the scale and cost of an eradication program.
2. **Trade support:** to facilitate the export of queen bees and packaged bees to countries sensitive to a range of bee pests and pest bees. This Program provides technical, evidence based, information to support Australia's pest free status claims during export negotiations and greatly assists exporters in meeting export certification requirements.

AHBIC's commitment of $75,000 per year is currently being accessed from industry reserves which are held in the industry Contingency Fund. However, this is not sustainable as this money is meant to be held in reserve for industry to contribute to an emergency response in the event of an incursion of an exotic pest (such as Varroa mite). If the proposed levy reforms and increases are not supported, AHBIC will not be able to contribute for their component, which will most likely lead to the discontinuation of the National Bee Pest Surveillance Program. If you support the honey bee industry contributing to a cost shared national surveillance program, which acts as an early warning system for bee pests and pest bees and also provides trade support for beekeepers who export queen bees and packaged bees, **please vote yes for the proposed reforms and increases in the honey levy.**

For more information about the proposed honey levy reform and increase, go to: http://honeybee.org.au/programs/honey-levy-reform-and-increase/

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BEE- TAG?

A drop of glue, tweezers, a micro-sensor and there it is - a bee that's also an e-tag

The sensors are being rolled out across the backs of four Tasmanian swarms this summer in world-first CSIRO research to track the movements of thousands of bees in the wild.

It is the first time such large numbers of any insect have been employed for environmental monitoring, and brings hope of disease control breakthrough at a critical time for the honey bee industry, the CSIRO said.

"This could just revolutionise things for us," Tasmanian beekeeper Peter Norris said. "It's just amazing what they've got inside that tiny chip."

Mr Norris, whose bees are part of the project, said the micro-sensors offered the potential to fight disease, work out the best placement of hives for honey return - even perhaps future GPS tracking.

CSIRO micro-sensor specialist Paulo de Souza said entomologists and beekeepers planned to fit 50 sensors a day to bees already calmed by refrigeration, release them, and watch where they fly.

The radio frequency identification sensors work like a car's toll-road e-tag, recording when the insect passes a checkpoint.

These bees will yield data from flights around apple and cherry orchards of Geeveston, south of Hobart, past monitoring points in their hives and feeding stations.

Data will then be assembled from about 5000 sensors to build a three-dimensional image of the insects' movements through the landscape. It is called "swarm sensing".

Honey bees are vital to crop pollination globally. But in the northern hemisphere, many are in trouble, hit by colony collapse disorder and varroa mite, which threaten to invade Australian hives.

Dr de Souza said the movements of the Tasmanian bees would be tracked in an attempt to work out the effect of pesticides used to protect bees from these diseases.

"Bees are social insects that return to the same point and operate on a very predictable schedule," Dr de Souza said. "Any change in their behaviour indicates a change in their environment.

"Worker bees live around two weeks in summer, and so we plan to keep going across six generations," Dr de Souza said.

He said the 2.5 mm by 2.5 mm sensors could potentially be made even smaller, so that a 1mm sensor could be glued to a mosquito.

Read more: http://www.smh.com.au/environment/animals/bee-etag-unlocks-swarm-secrets-20140115-30u8r.html#ixzz2qSx5Qf6Y

PLANT VIRUS JUMPS TO HONEY BEES

Courtesy: *Catch the Buzz*

A viral pathogen that typically infects plants has been found in honey bees and could help explain their decline. Researchers working in the US and Beijing, China report their findings in *mBio*, the online open-access journal of the American Society for Microbiology.

The routine screening of bees for frequent and rare viruses "resulted in the serendipitous detection of Tobacco Ringspot Virus, or TRSV, and prompted an investigation into whether this plant-infecting virus could also cause systemic infection in the bees," says Yan Ping Chen from the US Department of Agriculture's Agricultural Research Service (ARS) laboratory in Beltsville, Maryland, an author on the study.

"The results of our study provide the first evidence that honey bees exposed to virus-contaminated pollen can also be infected and that the infection becomes widespread in their bodies," says lead author Ji Lian Li, at the Chinese Academy of Agricultural Science in Beijing.

"We already know that honey bees, Apis mellifera, can transmit TRSV when they move from flower to flower, likely spreading the virus from one plant to another," Chen adds.

Notably, about 5% of known plant viruses are pollen-transmitted and thus potential sources of host-jumping viruses. RNA viruses tend to be particularly dangerous because they lack the 3'-5' proofreading function which edits out errors in replicated genomes. As a result, viruses such as TRSV generate a flood of variant copies with differing infective properties.

One consequence of such high replication rates are populations of RNA viruses thought to exist as "quasispecies," clouds of genetically related variants that appear to work together to determine the pathology of their hosts. These sources of genetic diversity, coupled with large population sizes, further facilitate the adaption of RNA viruses to new selective conditions such as those imposed by novel hosts. "Thus, RNA viruses are a likely source of emerging and re-emerging infectious diseases," explain these researchers.

Toxic viral cocktails appear to have a strong link with honey bee Colony Collapse Disorder (CCD), a mysterious malady that abruptly wiped out entire hives across the United States and was first reported in 2006. Israel Acute Paralysis Virus (IAPV), Acute Bee Paralysis Virus (ABPV), Chronic Paralysis Virus (CPV), Kashmir Bee Virus (KBV), Deformed Wing Bee Virus (DWV), Black Queen Cell Virus (BQCV) and Sacbrood Virus (SBV) are other known causes of honey bee viral disease.

When these researchers investigated bee colonies classified as "strong" or "weak," TRSV and other viruses were more common in the weak colonies than they were in the strong ones. Bee populations with high levels of multiple viral infections began failing in late fall and perished before February, these researchers report. In contrast, those in colonies with fewer viral assaults survived the entire cold winter months.

TRSV was also detected inside the bodies of Varroa mites, a parasite that transmits viruses between bees while feeding on their blood. However, unlike honey bees, the mite-associated TRSV was restricted to their gastric cecum indicating that the mites likely facilitate the horizontal spread of TRSV within the hive without becoming diseased themselves. The fact that infected queens lay infected eggs convinced these scientists that TRSV could also be transmitted vertically from the queen mother to her offspring.

"The increasing prevalence of TRSV in conjunction with other bee viruses is associated with a gradual decline of host populations and supports the view that viral infections have a significant negative impact on colony survival," these researchers conclude. Thus, they call for increased surveillance of potential host-jumping events as an integrated part of insect pollinator management programs.

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Technical Specialist, Honeybees - NSW Department of Primary Industries - Goulburn
doug.somerville@dpi.nsw.gov.au

SENATE INQUIRY

The Australian Federal Government recently announced a new Senate Inquiry into the beekeeping industry.

What does this mean?

From my perspective it's exciting, as it gives the beekeeping industry an opportunity to comprehensively inform government (Federal and State) as to what the core issues are that are affecting the beekeeping industry. Also, perhaps, how these problems can be fixed or helped by governments.

The perplexing thing is that this inquiry comes not long after a previous inquiry into beekeeping. In 2008 the "More than Honey" report was published with 25 recommendations as a result of the then Senate Inquiry. This inquiry was very much championed by Alby Schultz MP, with the assistance from Dick Adams MP, a bi-partisan approach. There were 92 written submissions to this inquiry and a lot of media coverage, particularly due to Alby. However, there has been a sporadic (at best) approach at communicating the actions that have occurred at various levels of government as a result of that inquiry. For a copy of the *More than Honey* report and copies of all the submissions to this report either Google "more than honey 2008'" or go to www.aph.gov.au and search from there.

No single agency took ownership over the 2008 report to ensure that the industry was kept up to speed with what the progress was on each of the recommendations. Thus, to the majority of industry observers, they could be easily excused in believing that very little has happened since the 2008 Senate Inquiry.

In this article I'm not going to spend time going through each of those recommendations and outline what has and hasn't happened, rather I want to concentrate on the current inquiry.

Apparently this current inquiry originates from Senator Nick Xenophon, from South Australia. The inquiry was announced just before Christmas with submissions due by 7 February and a reporting date of 26 March. Fortunately, the submission date has been extended until the end of March, 2014.

Thus you, as a reader of this article, have the opportunity to have your say or input. This does not need to be an encyclopaedia, but rather a page or two of the issues you see as relevant and what you believe needs to happen to fix them. Of particularly importance would be an outline of what you believe governments can feasibly and sensibly do.

The title of the current inquiry is "Future of the beekeeping and pollination service industries in Australia" with six terms of reference. Briefly, these terms of reference are:

a. the importance of these industries
b. current challenges facing the beekeeping industry and its future sustainability
c. adequacy of current biosecurity arrangements
d. Australia's food labelling requirements
e. recommendations from the 2008 "More than Honey" report
f. any related matters

I interpreted the terms of reference of the inquiry to be very broad, essentially allowing any subject to be covered. On the 10 January I was involved with the NSW Apiarists' Association executive workshopping the main issues of importance to the beekeeping industry and what could be accomplished by governments to alleviate some of these problems.

The association executive is doing a lot of work to compile a thoughtful and meaningful submission on behalf of the NSW beekeeping industry.

So what are my thoughts on the issues?

I have copies of past submissions to the 2008 inquiry to refer back to, plus I have the insight into what was discussed at the NSW Apiarists' Association executive meeting on 10 January.

Let's start with a thumb-nail sketch of the industry. The NSW beekeeping industry is the largest within Australia, accounting for approximately 40% of the beehives. As of January 2014 we had 3,461 registered beekeepers, 214,296 registered beehives and 489 beekeepers operating greater than 50 hives.

The primary income source for beekeeping has been, and remains, honey production. In 1999 the average honey yield per hive was 90kg per hive. At this time only 20% of the commercial beekeepers derived any income from the provision of pollination services. The proportion of beekeepers providing paid pollination services has probably risen to 50-60% of commercial beekeepers, as a result of the growth in the almond industry.

Products produced are primarily: specific floral origin honeys; beeswax - as a result of honey production; and some comb honey. NSW is the national centre for package bees, queen bees and nucleus hive production, which are produced by specialist beekeepers for the domestic and export markets. Pollination services are provided for a growing range of horticultural and agricultural crops with almonds being the biggest receiver of beehives.

Horticultural industries are increasingly paying for the provision of pollination services and this is assisting in the diversification of beekeeping businesses.

Business models are primarily family based units with perhaps occasional casual assistance or outsourcing of services. Traditionally commercial beekeepers used to carry out all functions, repairs and maintenance. Construction of new hive components is increasingly being conducted by specialist service businesses.

There has also been a trend in the last 10-15 years for some beekeeping enterprises to expand the number of hives managed from 400 - 500 up to 1,000 - 3,000 hives. This has necessitated the employment of labour, often relying on the 457 visa scheme to find suitable persons. Over the last ten years records within the NSW Department of Primary Industries beekeeping registration system indicate that we have seen a decline in the number of commercial beekeepers but the number of hives for NSW has remained static.
The NSW beekeeping industry is serviced very well from its core beekeeping organisation, the NSW Apiarists' Association. This association has branches strategically placed across the state. It conducts an annual two-day conference and publishes a journal for members and subscribers six times per year.

The recreational beekeeping industry appears to be growing with the peak body, the Amateur Beekeepers' Association, increasing the number of affiliated branches. This organisation

used to have its major focus in the Sydney basin, but in recent years several organisations have been created in regional NSW.

The services provided by the NSW government include bee site permits in State Forests, National Parks and Travelling Stock Routes. A compliance service is managed by the Biosecurity Division of Department of Primary Industries, primarily focused on the bacterial disease American foulbrood, plus abandoned and neglected apiaries and the provision to deal with beehives creating a nuisance to the public. Microbiological services are available from the Elizabeth Macarthur Agricultural Institute to assist in the diagnosis of disease. The NSW Department of Primary Industries two beekeeping specialist staff manage educational and industry development programs.

Core issues affecting the NSW beekeeping industry include:

1. Increasing threats in the biosecurity area.
2. Access to floral resources to maintain healthy and productive bees.
3. Continuing sustainable business models.

A bit more info on the pests and diseases area, consider the following *Nosema apis*, wax moth and American foulbrood have been in Australia for several decades. European foulbrood was identified in 1977 and quickly spread through NSW, causing serious ongoing losses of bee colonies. Chalkbrood was identified in 1993 – a major production disease. Small hive beetles were identified in Sydney in 2002 and have now become one of the major pests of beehives. *Nosema ceranae* is believed to have entered Australia in the last 15 years and is a major disease of adult bees.

Regionally, European wasps (1978) and cane toads are major pests of honey bees. Recently (2007) Asian bees were found in Cairns and eventually became established. Evidence in other countries suggests that if this insect reaches NSW it will also be a major competitor to honey bees.

So whilst the wish list to such inquiries could be extensive, I have focussed on issues I believe either require ongoing government support or could do with further government support. It is often a mistake by many industry members to presume that just because a service or function is currently being supplied by government, to believe that this will continue. This is often not the case and it is always worthwhile emphasising what is already in place and why it should stay.

Please note the following points (recommendations) are my thoughts and not those of the NSW DPI or NSW government.

Recommendation

A review of honey bees in the Australian landscape with particular reference to public lands

Recommendation

For continued government support for the National Sentinel Hive Program

Recommendation
That the Federal government assist in further development of suitable training packages for the beekeeping industry

Recommendation

The Federal Government to continue to support Research and Development for the beekeeping industry

Sub-recommendation

That the Federal Government consider changing the legislation to allow a R&D levy to be collected on the provision of pollination services.

Recommendation

Increase the testing of imported honey

Recommendation

That the decision to relocate the honey bee quarantine facility from Sydney to Melbourne is reviewed and another location in Sydney is considered – the logical location to build a new facility

Recommendation

That labelling of Australian product is clear and consistent to the consumer

In my submission to the inquiry I have added explanatory notes for each of these recommendations. Thus the points as listed may not be 100% clear to the reader. My point in writing this article is to suggest to you that whether you agree or disagree with my recommendations this is the opportunity right now to have your say. Even one page is sufficient.

NICK's NEWS

from DPI NSW

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NATIONAL RESIDUE SURVEY

For the past ten plus years I have been responsible for the collection of honey samples from across NSW for the National Residue Survey (NRS). In this article I just want to advise what the NRS is, and discuss an issue that arises every year in the results, and question, should we be concerned.

So what is the National Residue Survey and why is it done????

To give a thorough clear explanation I have lifted the words from the federal Department of Agriculture web site. They coordinate the program nationally for a broad range of foods.

The NRS is a vital part of the Australian system for managing the risk of chemical residues and environmental contaminants in Australian food products. NRS supports Australia's food industry and primary producers by facilitating access to key export markets and confirming Australia's status as a producer of clean food. NRS programs encourage good agricultural practices, help to identify potential problems and indicate where follow-up action is needed.

The NRS was established by the Australian Government in the early 1960s following concerns about pesticide residues in exported meat. Since then, the NRS has expanded to test other animal, grain, horticulture and fish products for residues of pesticides and veterinary medicines, as well as for other contaminants. The NRS became an industry-funded activity in 1992 and relevant legislation was established.

The core work of the NRS is to facilitate the testing of animal and plant products for pesticide and veterinary medicine residues, and environmental contaminants. Product testing is done through either random or specifically designed sampling protocols. Other programs within the NRS, such as laboratory evaluation and business activities, support the core work of residue testing.

Residue monitoring aims to:

- provide an estimate of the occurrence of residues in products (using systems based on sampling and statistical probability)
- confirm (or otherwise) that residues in products are below set limits
- alert responsible government authorities and industry if, and when, limits are exceeded, so that corrective action can be taken.

Money to conduct the honey testing program is obtained from the beekeeping industry via the honey levy. The Australian honey producer levies is currently set at 2.3c/kg for annual honey sales greater than 600kg (this is currently undergoing reform). The breakup of the levies fund is:

1) Research and Development (R&D) – a levy of 1.5c/kg
2) Emergency Animal Disease Response Agreement (EADRA) – a levy of 0.7c/kg
3) National Residue Survey (NRS) – a levy of 0.1c/kg

For NSW this year (2013-14) 57 honey samples are required.

For NSW honey the sampling technique is random with honey collected from packers, beekeepers, markets, road side stalls and shops around the state. The samples collected are to test honey produced in NSW, not imported honey. The program is about producing evidence that the honey produced in NSW and Australia is suitable for human consumption and meets market expectations both here and overseas. It also provides a means to detect and act quickly on issues that do arise ensuring we maintain our good market reputation.

An example of this was when Paradichlorobenzene (PDB), known better for its use in moth balls or urinal deodorant, had approval for use for wax moth management. In 2007 PDB's were banned across Australia for controlling wax moth, with Qld the last state to implement the ban. An incident arose where honey tested by the European Authorities detected PDB. Information about the ban of PDB's and the associated contamination issues were communicated within the industry to minimise the chance of a recurrence. Since this incident, to provide assurance to our honey markets, a portion of the honey samples collected are tested for PDB.

Other contaminants that are tested for in the honey include heavy metals, anti-microbials, chloramphenicols (broad spectrum anti-biotics), nitrofurans (another type of antibiotics), and pesticides.

Over the years of NRS sampling very few issues have arisen. But one thing that has been observed and has come to the attention of the NRS is the occasional high zinc readings from the heavy metal analysis. There is currently no Maximum Residue Level (MRL) for zinc in honey and as such is not considered a human health issue, so there is no major problems or restrictions on honey with high zinc levels. However this high zinc level maybe considered a food quality issue in the future that beekeepers may need to consider.

From my observations the high zinc levels in honey appear to closely mirror the samples collected out of galvanised drums, which would be understandable. Zinc levels as high as 59 mg/kg in honey have been detected with the norm being around 0.5 mg/kg. Whether long term storage in galvanised drums increases zinc levels, I am unsure but suspect it would. I have not seen any literature that has examined this. Perhaps it is time to consider no longer using the old galvanised drums and galvanised storage tanks or extracting equipment and move to plastic or stainless steel for these purposes to reduce this potential food quality issue. Remaining ahead of the game as beekeepers and addressing such issues will help develop and increase your returns, and provide confidence to consumers of the delicious clean pure product you produce, Australian honey.

So over the next few months I will be out and about collecting honey samples for this years NRS. Please help me out if asked by providing a sample of honey so we can show consumers of Australian honey what great stuff it is.

HONEY LEVY REFORM & INCREASE

The Australian Honey Bee Industry Council (AHBIC) proposes that changes be made to the existing honey levy which is collected by the Levies Revenue Service (LRS) of the Department of Agriculture.

- **What is the honey levy?**

Australian honey producer levies are set at 2.3c/kg for annual honey sales greater than 600kg. These levies fund:

1) Research and Development (R&D) – a levy of 1.5c/kg is matched by the Australian Government and managed by the Rural Industries Research and Development Corporation (RIRDC).
2) EADRA Biosecurity – a levy of 0.7c/kg provides resources for the Emergency Animal Disease Response Agreement (EADRA) and is also used to meet industry's contribution to the National Bee Pest Surveillance Program.
3) National Residue Survey (NRS) – a levy of 0.1c/kg manages the risk of chemical residues and environmental contaminants in Australian food products including honey. This is a requirement for Australian to be exported to the European Union.

- **Who pays and submits the returns?**

The producer, or the person who owned the honey immediately before sale, or the person who uses the honey in the production of other goods is liable to pay the levy. Where the producer sells the honey to a buying/selling agent (e.g. Capilano), processor or shopkeeper, it is the buyer's responsibility to lodge a quarterly return, on behalf of the producer.

Where the producer sells the honey directly via markets or other retail opportunities, it is their responsibility to pay the levy via an annual return.

- **Are there exemptions in the levy payments?**

Exemption from payment of the honey levy only applies when the producer sells less than the 600kg of honey per year. Any producer selling over 600kg annually for honey must pay the honey levy. No other exemptions apply.

- **What changes do AHBIC recommend?**

AHBIC are proposing to raise the honey levy from the current 2.3c/kg to 4.6c/kg to pay for improved industry biosecurity – endemic pest and disease management and surveillance of exotic bee pests and pest bees.

The current R&D levy and the NRS levy will not be changed.

AHBIC promotes the following administrative changes:

- Changing the Emergency Animal Disease Response Agreement (EADRA) biosecurity component into an Emergency Plan Pest Response Deed (EPPRD) biosecurity component.
- Increasing the newly established EPPRD biosecurity component from 0.7c/kg to 3.0c/kg to help industry fund established and exotic pest and disease biosecurity activities.
- Establishing a Plant Health Australia levy of 0.1c/kg to pay for AHBIC annual subscription fees. This 0.1c/kg PHA levy will be established by reducing the newly established EPPRD biosecurity component by 0.1c/kg from 3.0c/kg to 2.9c/kg.
- Changing the management of the AHBIC Contingency Fund from Animal Health Australia to Plant Health Australia
- AHBIC are also proposing to raise the threshold of honey produced from which the levy applies from 600kg to 1,500kg per annum

- **How is the consultation being carried out?**

Consultation with the honey bee industry will be carried out over an 8 month period from between December 2013 – July 2014 in a variety of industry and association newsletters, journals, websites, popular media articles, and state department of agriculture mail outs. This consultation will be inclusive of all beekeepers which could be affected by the proposed changes.

Presentations on the proposed levy changes, with an open floor discussion on the proposed changes are scheduled for each of the six state beekeeping association conferences in 2014. At each of these conferences, voting on the proposed changes will also be undertaken.

- **Why is there a need for the proposed increase to the honey levy?**

The proposed increase in the honey levy will fund endemic pest and disease management and provide industry's contribution to exotic pest and pest bee surveillance.

Established pests cause significant financial and emotional harm to beekeepers. In particular American Foulbrood (AFB) is present in all Australian states and territories and is the most fatal and costly established pest. Evidence shows that problems caused by pests, such as AFB, are only getting worse and the current state based policies and systems are not working. Overseas experience also suggests that if major established pests such as AFB are not properly controlled when an exotic pest such as Varroa mite arrives, the dual effect is worse than expected. For these reasons, greater national coordination, industry leadership and funding are urgently needed.

Australia is currently free of some of the most significant pests of honey bees, namely the Varroa mite and Tropilaelaps mite. The establishment of these pests in Australia would be catastrophic for the honey bee industry causing huge losses in production. An industry – Government partnership known as the National Bee Pest Surveillance Program is in place to provide an early means of detection of exotic bee pests and pest bees. A sustainable source of funding is required to meet industry's contribution to its partnership agreement with Government. The National Bee Pest Surveillance Program also provides valuable trade support for exports of queen bees and packaged bees from Australia.

- **How will the increased levy benefit levy payers?**

The proposed honey levy increase will be spent on two national biosecurity programs that will both bring numerous benefits to beekeepers.

The National Honey Bee & Pollination Industry Biosecurity Management Strategy (The National Biosecurity Strategy) has a vision of increased productivity and profitability in the Australian honey bee industry through the control of endemic bee pests and diseases (National Bee Biosecurity Program), and improved surveillance and preparedness for exotic pests and diseases (National Bee Pest Surveillance Program). The 5 major benefits for the establishment of the National Bee Biosecurity Program are for:

1) Greater industry communication, training and educational material to be produced and provided to beekeepers which will focus on surveillance, identification, prevention and control of honey bee pests and diseases;
2) Improved level of overall biosecurity of commercial beekeepers in Australia through the development of the Australian Beekeeping Code of Practice;
3) Reduced incidence of established pests and diseases, such as AFB, thereby lowering the economic losses presently experienced by beekeepers;
4) Improve surveillance for exotic pests (such as Varroa mite) as beekeepers will be required to inspect hives more frequently and have better knowledge of identification of pests and diseases;

5) Establish an effective working management and coordination structure between industry and government, which will help in the event of an incursion of exotic bee pests (such as Varroa mite)

The National Bee Pest Surveillance Program (NBPSP) is an early warning system to detect new incursions of exotic bee pests and pest bees. The Program involves a range of surveillance methods conducted at locations considered to be of most likely entry of bee pests and pest bees throughout Australia. The NBPSP benefits beekeepers in 2 critical areas:

- The NBPSP acts as an exotic bee pest and pest bee early warning program
- The NBPSP provides critical trade support data to facilitate the export of queen bees and packaged bees

- **How much levy is needed? How will it be spent?**

Around $460,000 per annum is needed to help fund industry biosecurity activities. This includes:

- AHBIC's estimate for their contribution to the National Biosecurity Strategy is approximately $385,000 per annum. For more information, go to www.honeybee.org.au (under the Programs tab)
- AHBIC's contribution to the National Bee Pest Surveillance Program which is $75,000 per annum. For more information about this program, go to www.nbpsp.com.au

- **Why are you raising the threshold?**

AHBIC are proposing to raise the current threshold of 600kg to 1,500kg. Therefore, producers would be exempt from paying the honey levy if they sold less than 1,500kg of honey per annum. The reason for raising the current honey levy threshold from 600kg to 1,500kg per annum is because the costs of collecting the levy in these lower ranges are far exceeding the revenue raised. These proposed changes are an effort by AHBIC to make the honey levy more cost efficient.

- **What am I paying more levy and hobby beekeepers are not paying anything?**

The simple reality is that research funded with the honey levy and managed by RIRDC has not been able to identify a cost effective or legal mechanism for collecting levy from very small producers including amateurs. See for instance Granger and Woodburn (2010) and Ryan (2013).

Currently, the only model available for the honey bee industry to raise funds is through an increase in the honey levy.

The majority of the funds raised as part of this proposed levy increase will be spent on the proposed National Bee Biosecurity Program. Therefore, Stage 1 of the proposed National Bee Biosecurity Program will be targeted at commercial beekeepers which are registered for more than 50 hives. Therefore, this commercial honey levy will be spent directly on commercial producers with a direct benefit.

Stage 2 of the proposed National Bee Biosecurity Program will focus on raising additional funds from hobby beekeepers, where the benefit from this separate source of funds will be spent directly on hobby beekeepers.

- **Why should I pay more levy?**

AHBIC is mindful of low honey prices, high production costs and the perilous state of the industry's profitability. The proposed levy increase has been carefully costed, will be directed at biosecurity and is at the request of Australian beekeepers and beekeeping associations. The National Bee Biosecurity Program and the National Bee Pest Surveillance Program will be industry driven and reviewed on a regular basis to ensure they are meeting industry's aims.

Currently, the only model available for the honey bee industry to raise funds is through an increase in the honey levy. Since the raising of the honey levy will be paid for by commercial beekeeper, stage 1 of the proposed National Bee Biosecurity Program will be targeted at commercial beekeepers which are registered for more than 50 hives. Therefore, honey producers and levy payers will receive a direct benefit from the levy.

Stage 2 of the proposed National Bee Biosecurity Program will focus on raising additional funds from hobby beekeepers, where the benefit from this separate source of funds will be spent directly on hobby beekeepers.

- **Who is eligible to vote?**

Every beekeeper in Australia who is registered for more than 11 hives, and is therefore considered a levy payer is eligible to vote on the proposed levy changes. Because of the current levy threshold of 600kg, it is estimated that using the average production of 54kg from each hive (ABARES 2008), only beekeepers that are registered for 11 hives or more would be producing the current 600kg per year.

- **How can I vote?**

A formal ballot will be held at each of the six state beekeeping conferences held between May – July in 2014. At these conferences, every registered beekeeper owning more than 11 hives will be provided with the opportunity to vote. The vote will be tallied on both a yes/no basis, as well as using a weighted production basis.

- **How can I vote if I can't attend the state beekeeping conference to vote in person?**

If you are unable to attend the ballot held at the state beekeeping conferences in 2014, you are able to submit a postal vote. **The postal votes open Australia-wide on 1 March 2014**. You can download a ballot form and post it to your relevant state department of agriculture representative for counting when the ballot is tallied. Each state department of agriculture has nominated an independent voting scrutineer for the ballot. For more details, and to print out a ballot form and vote, go to www.honeybee.org.au (under the Programs tab). If you are unable to access the internet, contact AHBIC about how to receive a postal ballot form on (07) 5467 2265

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- **How do I get more information about these proposed changes?**

More detailed information about the proposed levy reforms and changes are contained on the AHBIC website. Go to www.honeybee.org.au (under the Programs tab).

For more information about specific changes contact AHBIC on (07) 5467 2265 or by email on ahbic@honeybee.org.au.

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Australian Government
Rural Industries Research and Development Corporation

Honeybee R&D News

Chairman's Foreword

Michael Hornitzky, Chairman, RIRDC Honeybee Advisory Committee

On the 19th November the Honeybee Advisory Committee (HAC) held a meeting in Melbourne at which 16 preliminary research proposals (PRPs) and 4 other proposals, outside the open call process, were considered for funding. Full research proposals have been requested for 4 of the 16 PRPs. Funding was recommended and RIRDC approved the following 3 proposals.

- *National Honeybee and Pollination Industry Biosecurity Management Strategy - Code of Practice.* This will involve developing a draft Australian Beekeeping Code of Practice and draft National Honeybee Biosecurity Program for government and industry endorsement by the end of the 2014 financial year. The development of this code of practice and national program, which will include the framework for a national American foulbrood control strategy, will lay the foundations for a greater degree of industry self reliance for the management of established, and exotic pests and diseases in Australia.

- *Symposium: Growers and beekeepers working together – protecting crops and their pollinators.* In August 2012 the Australian Pesticides and Veterinary Medicines Authority (APVMA) announced that it would conduct a review to look at the use of neonicotinoid insecticides in Australia to access whether they present more of a risk to honeybee health than other pesticides. The aim of this symposium is to provide a forum at which APVMA can (i) release their review into neonicotinoid use in Australia and (ii) outline and discuss the broad issues surrounding honeybees in pollinator landscapes, implementing pollination best practice management, research and stewardship of neonicotinoids and regulation of neonicotinoids.

- *International Union for the Study of Social Insects Conference – Cairns 13th to 18th July 2014.* RIRDC is sponsoring this Conference to support bringing a renowned international speaker (Dr Jay Evans) to Australia. Dr Evans will also be attending at least one State Beekeeping Conference in Australia. The Conference will also include a session on honeybee related topics for a beekeeper audience.

RIRDC is in the process of streamlining its management processes and moving to recover costs of its administration of the Honeybee and Pollination programs. Gerald Martin, Chair of the Pollination Advisory Committee and I have submitted a letter to the RIRDC Board requesting that the program management fee be capped at 10% of program expenditure. In our submission we also flagged a number of cost saving measures to be implemented which include the amalgamation of the Honeybee and Pollination Advisory Committees.

Current R&D Committee

Dr Michael Hornitzky (Chair)	(02) 4651 2729
Prof. Ben Oldroyd	(02) 9351 7501
Prof. Boris Baer	(08) 6488 4495
Ben Hooper	0407 820 474
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The Australian Pesticides and Veterinary Authority registered the APITHOR Hive Beetle Harbourage Insecticide on the 9th December. This was made possible largely due to the field trials conducted by Dr Garry Levot from 2006-2013. These APITHOR harbourages have provided very popular and very effective control of the small hive beetle. They also attract a royalty which the HAC uses to fund research.

I encourage those with an interest in the honeybee industry to apply for a RIRDC Horizon Scholarship. The Scholarship has been developed to support the next generation of agricultural leaders who will take up the challenge of farming for the future. It is for young people who are passionate and with a keen interest in the future of agriculture. The Horizon Scholarship is an initiative of RIRDC that in partnership with industry sponsors, supports undergraduates studying agriculture. The Honeybee Program is sponsoring an Horizon Scholarship from 2014 ($10,000 per year for four years including a bursary of $5,000 per year). To be eligible for the Horizon Scholarship students must be an Australian citizen or permanent resident entering their first year of university. This is a great opportunity for a young person interested in a career in agriculture. More information is available in the following link: http://www.rirdc.gov.au/researchprograms/rural-people-issues/horizon-scholarship

Bees attending honey comb they have formed on the exposed surface of an APITHOR™ hive bettle harbourage.

Three projects sponsored by the Honeybee Program have recently been completed:

(i) **Risk Assessment of ports for bee pests and pest bees (released for official use) to the National Bee Pest Surveillance Program managed by Plant Health Australia to inform where surveillance effort across Australia's ports should be targeted – Authors: Peter Caley, Daniel Heersink, Dean Paini and Simon Barry.** Exotic bee pests and pest bees have the potential to impact directly on the Australian honeybee industry through reduced productivity and on agricultural industries dependent on both managed and feral honeybee populations for plant pollination. This report is about identifying ports of high risk for the entry and establishment of exotic bees and/or bee pests based on the best available information. This will enable improved allocation of surveillance resources to reduce the likelihood of exotic pest bees and bee pests establishing. The report finds order of magnitude differences among Australia's maritime ports of first call for the hazard of exotic pest bee entry and establishment. These differences are driven by differing approach rates to ports arising from the port-specific combinations of shipping traffic, voyage duration, and country of origin interacting with the suitability of port surroundings for bee establishment and persistence. The results form a robust starting point for planning and prioritizing port surveillance activities for pest bees and bee pests.

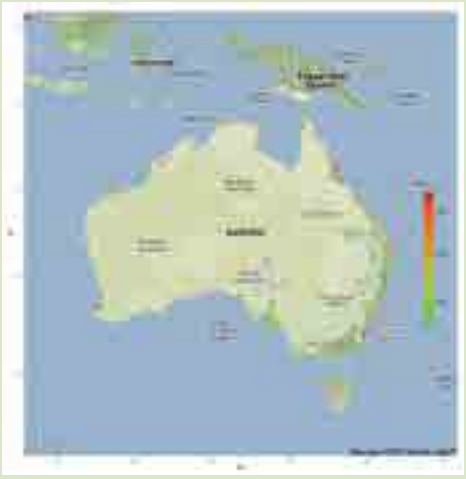


Figure. Estimated hazard of entry and establishment of *A. cerana* excluding random effects for ports. Estimates are rescaled such that they sum to 100.

(ii) Value Adding to Honey (coming soon) - Authors: Dr Joan Dawes and Dr David Dall. At present the commercial value of Australian honeys relates primarily to taste quality, but stronger health awareness by consumers has created scope for adding value to Australian honeys by exploiting any properties of the honeys that convey health benefits. Anecdotal evidence has identified three such potential functional properties of Australian eucalypt honeys: Glycaemic Index (GI), prebiotic properties and therapeutic activity.

Some key findings of the project are:

(a) No measured physical or chemical characteristic of the honeys contributed usefully to the assignment of floral source for Australian eucalypt honeys.

(b) Australian eucalypt honeys are probably low to medium GI foods when consumed by the majority of individuals, but not necessarily of lower GI value than honeys from other floral sources.

(c) Most of the Australian eucalypt honeys had significant prebiotic potential when tested *in vitro*.

(d) In *in vitro* studies most of the honeys, and all the Jarrah honey samples, elevated the levels of butyric acid, which at high concentrations is linked to a lowered risk of colon cancer.

A key recommendation is that industry should focus on prebiotic potential as the health-related property of Australian eucalypt honeys that is most likely to generate premium prices.

(iii) Genetic variation of *Varroa jacobsoni* and pathology of microbial pathogens (coming soon) - Authors: Dr John Roberts, Dr Denis Anderson and Dr Wee Tek Tay. *Varroa jacobsoni* is a parasitic mite that has emerged as a serious pest of European honeybees *(Apis mellifera)* following a recent jump from its natural host, the Asian honeybee *(Apis cerana)*. In 2008, a bee pathogen survey in Papua New Guinea (PNG) found populations of *V. jacobsoni* (of the Java haplotype) reproducing for the first time on the drone and worker brood of the local *A. mellifera* and causing colony losses. The research in this report was a genetic study of *V. jacobsoni* in PNG and potential viruses associated with this mite. It compared the genetic relatedness of mite populations on different hosts and examined *V. jacobsoni* on *A. mellifera* in PNG for known and unknown honeybee viruses. Mites now reproducing on *A. mellifera* in PNG appear unable to still reproduce on *A. cerana*. The adaptations required of mites to parasitise *A. mellifera* appear to be permanent and prevent reproduction on *A. cerana*. Multiple mite populations appear to have switched hosts to *A. mellifera* and new populations may have started to switch. Two genetically different populations were found on *A. mellifera* in PNG and the small numbers of mites found in Papua and Solomon Islands reproducing only on *A. mellifera* drone brood were also genetically different to mites in PNG. Mites reproducing on *A. mellifera* in PNG were not carrying any known honeybee viruses, but three new viruses were discovered. It is not yet known whether they infect honeybees, but one of these viruses appears distantly related to other honeybee viruses.

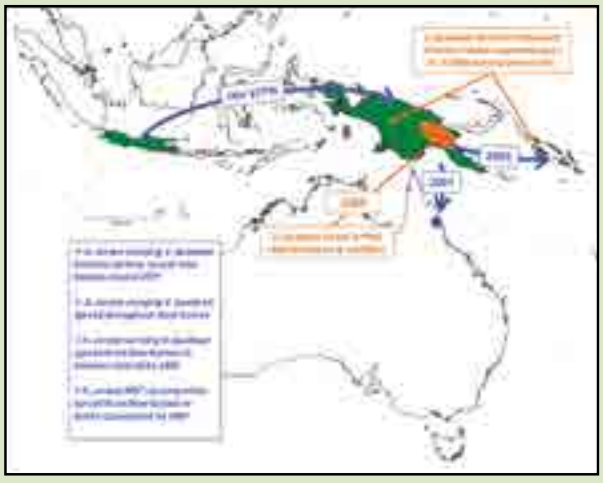


Figure. Distribution of Varroa jacobsoni (Java haplotype) on *Apis cerana* is highlighted in green. Spread of *A. cerana* from Java to New Guinea and then to Solomon Islands and Australia is highlighted in blue. *Varroa jacobsoni* reproducing on *A. mellifera* is highlighted in orange.

SICK BEES

PART 17 C

NOSEMA - THE SMOLDERING EPIDEMIC

by Randy Oliver - ScientificBeekeeping.com

I'm suspecting that we have been misled by our reliance upon spore counts as the measure of the degree of infection by *Nosema ceranae*. It may well be that nosema can take a considerable bite out of colony productivity, or even chance at survival, at even very low levels of infection, and perhaps even before it produces spores.

THE SCIENTIFIC METHOD

Woods, W.A., Heinrich, B., Stevenson, R.D., (2005) Honeybee flight metabolic rate: does it depend upon air temperature? Journal of Experimental Biology 208, 1161–1173.

The bees, therefore, transport more than 259 kg (83%/40% × 125 kg) nectar to the hive. Each bee carries only 40–80 mg nectar per trip, so about 4 million foraging trips must be made. If each trip is assumed to average 3 km, then the bees fly about 12 million km to collect the nectar for the 125 kg of honey. The energy input for bee flight is about 1.1 cal/km (or 4.6 joules/km) (Dade 1977, Frisch 1967, Schaffer et al. 1979, Tucker 1975, Wells and Giacchino 1968). Therefore, the total energetic input is about 13,200 kcal for collecting 259 kg nectar that has a food energy value of about 380,000 kcal (or a 29:1 ratio of return).

In addition to the nectar that provides the bees with food calories, the bees collect about 24 kg (18–30 kg) pollen per hive, providing them with protein and other nutrients. On a fresh weight basis, this pollen contains about 26% (8–40%) crude protein (Dietz 1978, Free 1970, Herbert and Shimanuki 1978, Standifer 1971, 1977). To collect the pollen supply, an estimated 1.2 million pollen-foraging trips are made during the summer season by bees, with each carrying 8–29 mg (avg. 20 mg) of pollen on each trip (Gary 1978, Ribbands 1964, Todd and Bishop 1941). The total distance in this instance is about 3.6 million km. Assuming the same flight energy per kilometer as before, the energy input is about 3960 kcal for the 24 kg pollen with an estimated food energy value of 81,000 kcal (or a 20:1 ratio of return). (For energy calculations, we used average pollen fractions of 26% protein, 29% carbohydrate, and 11% lipid).

Energy Efficiency of Honey Production by Bees

E. E. Southwick and D. Pimentel

BioScience

Vol. 31, No. 10 (Nov., 1981), pp. 730-732

Metabolic cost of forager per day 1073J
Net gain per forager per day 2167J

You may have noticed that I'm doing a sort of "about face" in my assessment of the impact of *Nosema ceranae* upon colony health, I feel that I owe the reader an explanation. I can probably do so by simply sharing with you the sort of inquiry and analysis that I'm in the middle of.

Science is not about microscopes and laboratories; it is about the thought process that you use to make sense of observations of the world around you. One can think in a scientific manner just as well while wearing (dirty) white coveralls as when wearing a (clean) white lab coat!

The good scientist frees himself of beliefs, holds no positions, and avoids any prejudices or biases toward any particular point of view. His only firm conviction is to remain completely open minded in the never ending quest to understand why things are the way they are.

Science is based upon the free sharing of data (accurate observations) and its interpretation. Not every scientist will interpret the data in the same way. That is why we have "peer reviewed" journals as an editorial check that good scientific method was followed; in an ideal world there is nothing to prevent the publication of highly controversial interpretations, and indeed good scientists relish having an established paradigm challenged.

As an aside, please realize that the editorial filtration process of scientific publication is hardly perfect. I personally am dismayed by the poor quality of peer review of late, and have serious criticisms of both the methodology and interpretation of a number of recently published papers.

Warning: unless you are willing to research more deeply, I caution you to take any new scientific findings that get splashed across the headlines with a grain of salt, especially when a "hot" topic, such as CCD, pesticides, the environment, or human health is involved!

(Back to the Scientific Method): any new interpretation as to why something is the way it is, or works the way it does, is subject to testing by proposing a "falsifiable" hypothesis. You can't scientifically prove that anything is true (or that anything is "safe"); you can only prove that a hypothesis is not supported by results from putting it to actual test in a well-designed experiment. The best that you can do toward seeking truth is to find that the results of multiple experiments "support" your hypothesis. When a hypothesis has eventually been supported by enough robust data, then it is accepted as a scientific "theory"—a word that has much stronger meaning in science than it does in common language. A scientific theory becomes the paradigm by which the scientific community "understands" things—of course subject to revision should any new data come to light that falsify it.

When Dr. Mariano Higes found that *Nosema ceranae* was highly associated with the collapsing colonies that he observed

in Spain, he proposed the hypothesis that the parasite was the cause. He further tested that hypothesis in various experiments by inoculating healthy colonies with spores, and then applying fumagillin or not, and tracking the buildup of nosema and various colony health metrics. He found that his results supported his hypothesis.

Other researchers, including myself, found his hypothesis extremely plausible—it appeared to reflect the typical high mortality associated with the invasion of a naive host population by a novel parasite. However, when we sought to replicate his results in our own bees, we simply didn't see a compelling cause and effect relationship, so questioned the validity of Dr. Higes' hypothesis.

I myself fell into this camp, but I go out of my way to truly understand alternative viewpoints; to that end I have maintained a friendly ongoing conversation with Dr. Higes for the past five years—constantly challenging and questioning him. Such frank discussions are the best method to arrive at the actual truth of the matter.

I want to be clear at this point that in this series I'm doing a lot of thinking aloud. I will try to be clear as to which conclusions (always subject to reevaluation) are based upon hard data and actual experimental testing; and which ideas or opinions are inferential—based upon suggestive data or observation. I also want to emphatically state that the evidence to date does not suggest to me that *Nosema ceranae* is directly responsible for either CCD or major colony losses, but appears to often be associated with them, and may be a contributor in some way. I'll return to the subject of colony collapse soon.

Effect of the Invasion

So, is the invasion of *Nosema ceranae* is having any substantial negative effect upon the health of our colonies. *N. ceranae* invaded East Coast apiaries as early as the mid 1980's without anyone even noticing it, until it was discovered twenty years later by researchers investigating CCD. But then again, it was discovered in colonies suffering from CCD!

The effects of infection by the new nosema seem, in general, pretty similar to those of its cousin, although it appears to cause somewhat more gut damage, and to be a bit more resistant to fumagillin. The most notable aspect that is different about *N. ceranae* is that it apparently "has better mechanisms to evade host immunity to allow for faster growth and reproductive capacity than *N. apis"* (Chen et al., 2009a). I suspect that it also has better mechanisms for transmission. Antúnez (2009) found that it up- and down regulates bee immune response genes differently than its cousin. Plus it is able to thrive over a wider range of temperature (Martin-Hernandez 2009), so it exerts its negative influence over a wider period each year. All the above differences make it a more virulent pathogen (in the sense that it reproduces more efficiently).

Why Would *Nosema ceranae* Not Cause Problems?

With the majority of U.S. bee samples currently being infected by Nosema (presumably *Nosema ceranae*), it seems to me that perhaps the question that we should be asking is, "Why wouldn't we expect it to be causing problems?"

There is a vast body of "classical" research on the fundamental negative effects of *Nosema apis* infection upon colony health and productivity. Nosema is an age-old nemesis of beekeepers. Why would we not expect similar effects due to the new nosema, which is even more successful at infecting bees?

Understanding Nosema

Nosema is adapted to turn a bee into a spore-producing factory; there is no benefit to the parasite in killing the bee. And therein lays the problem, because it makes nosema so insidious. But a widespread increase in the prevalence of such an insidious infection can have a major effect upon the honey bee population.

Understanding the Honey Bee Superorganism

In order to understand the effect of nosema upon the colony, one must stop thinking of the honey bee as merely an insect. Rather, we must think of it at the level of the superorganism, similar to an intelligent, warm-blooded, fast-growing ten-pound animal. But not just any animal; rather one ravenous for energy and protein to fuel its rapid growth—exactly the precious commodities that nosema steals from the colony.

In order to appreciate just how fast growing the bee superorganism is relative to other members of the animal kingdom, I'll compare it to the fastest growing animal that I could think of—the modern day broiler chicken.

I downloaded data for the growth rates of package bees and broilers, and transformed it into graphical form (Fig. 1).

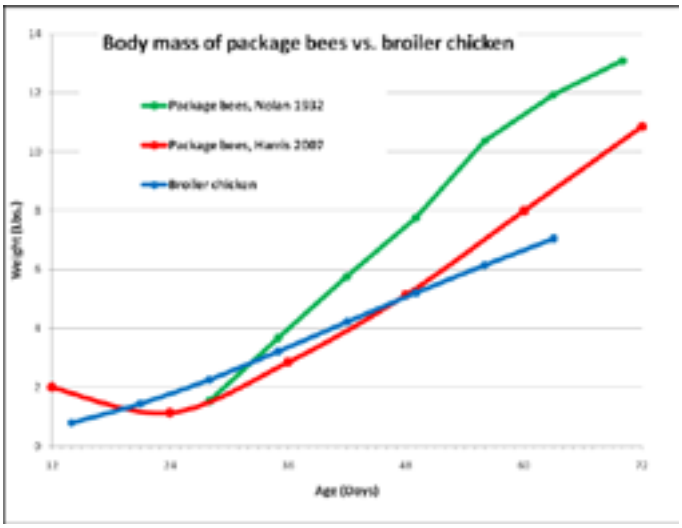


Figure 1. Comparison between the weight gain of package bees vs. that of a broiler chicken. The red curve shows how a package loses population until the first brood emerges. After that point, packages grow considerably faster than even a broiler chicken bred for rapid growth! Data for 3-lb packages calculated from Nolan (1932) and Harris (2008); for broilers, Jacob (2011).

Package bees gain weight even faster than a broiler chicken of similar age and body mass (age starting at emergence of the first brood)! But that ain't the half of it!

The chicken is penned in a warm room and provided with optimally-formulated chow. On the other hand, the industrious bees have to forage for themselves over a dozen square miles, spending a tremendous amount of energy in the process, as well as wasting a vast amount of body heat while individually foraging.

But I'm not done yet! If the chicken manages to store any excess energy or protein, it puts it on as fat or muscle—which then adds to its weight. The bees, on the other hand, store any excess as honey and beebread in the combs, and the above graph doesn't take that into account (the bees' stores are legitimately analogous to the fat that an animal stores to get it through lean times). So let's adjust the graph to take into account the bees' stores (Fig. 1).

lethargic

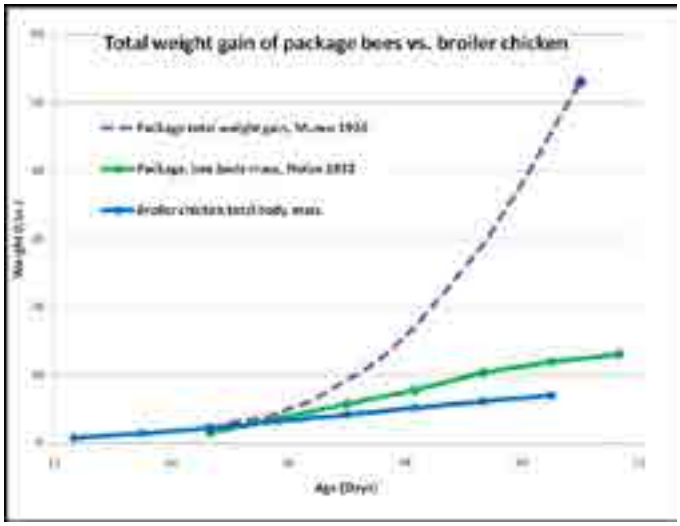


Figure 2. Total weight gain (including honey and beebread) of a 3-lb package installed two weeks before the main flow, compared to the gain in body mass alone of bees or a broiler chicken. When we measure total colony weight gain, the bees leave the broiler in the dust! The end points of the dotted line are actual data; I estimated the intermediate curve based upon measurements by Nolan. Added data from Nolan (1932).

By the end of the above graph, the broiler was essentially done growing; the colony was approaching its maximum population, but it was hardly done "growing." In the next two months, it gained yet another 218 pounds! It is not unheard of for a colony with a bee body mass of 12 lbs to put away its body weight in honey each day!

So what's my point? It's that we beekeepers expect our bees to perform a feat rapid growth beyond the ability of perhaps any other animal! The bee superorganism can only pull this off by being a prodigious consumer of protein and energy. That is why it is completely dependent upon two of the richest foods in nature—pollen and nectar (colonies only build well on the most nutritious pollens—at least 20-25% protein). A bee colony would starve to death on the sorts of diets that most organisms are adapted to.

Energy and Protein Metabolism

Table 1
Energetics of 'typical foraging' for *Apis mellifera* at an air temperature of 30 ºC

Nectar load	30 µl
Nectar energetic content	9 J µl $^{-1}$ (50% sugar)
Energetic reward per trip	270 J
Flight metabolic rate (30 ºC)	2.5 J (bee min) $^{-1}$
Trip duration	30 min
Cost per trip	75 J
Net gain per trip	195 J, 6.5 J min $^{-1}$
Trips per day	12
Reward per day	3240 J
Cost per day during flight	900 J
In-hive metabolic rate (30 ºC)	0.16 J (bee min) $^{-1}$
Daily in-hive metabolism of forager	173 J day $^{-1}$
Metabolic cost per forager day	1073 J day $^{-1}$
Net gain rate per forager day	2167 J day $^{-1}$
Hive bees fed per forager	9.4
% of bees which forage	10
% of total colony energy spent foraging	30

Foraging rewards and trip durations are derived from Winston (1987); within-colony metabolic rates were calculated from Kronenberg and Heller (1982), and metabolic rates during flight were taken from Roberts and Harrison (1999). Within-hive metabolic rates were considered identical for hive bees and foragers.

The excellent analysis by Harrison was a great starting point for me to try to gage the effect of nosema infection upon colony weight gain. So I created a spreadsheet to look at the energy dynamics for a colony of 40,000 bees (about 23 frames of bees), assuming that a quarter of them were foragers.

Using Harrison's numbers, colony weight loss and gain were a bit greater than what I observe in the field. So I compared his bee metabolic rate measurements with those of other authors, and tweaked the equations until they gave realistic results-- the colony would lose about a pound of weight a day if there was no bee flight, but gain about 5 lbs a day of fully cured honey under good conditions (with no adjustment of the forager force).

I got some interesting results. In warm weather, when there is only enough of a nectar flow such that the colony was just holding its own (neither gaining nor losing weight), one forager is essentially gathering enough nectar to feed about three house bees, which kinda makes sense if a quarter of the bees are foragers!

However, given the exact same colony, with the same nectar income, ***but on a cool day***, the colony will lose over a pound of weight a day, due mainly to the increased metabolic cost of foraging at lower air temperature.

OK, now let's go back to warm weather, with enough of a light nectar flow on for the colony is just holding its weight. Let's add a nosema infection to the picture, such that half the field force is infected, and guesstimate that the cost of infection causes an increased metabolic demand on the infected foragers of 50% for six hours a day. Without changing anything about the foraging trips or bloom, and without any bee mortality, the cost of the infection would result in about a half pound weight loss for the colony a day!

The infection above would be completely invisible to the beekeeper—the bee and brood population would be exactly the same, the number of foragers and the nectar income would be exactly the same, but the added metabolic cost of the nosema infection to only half the foragers (1/8th of the colony population) would cause that colony to lose significant weight rather than holding its own.

It gets even worse in cool weather. Everything else remaining the same except for the greater heat loss from the foragers to the cool air (I'm ignoring any additional heat loss by the cluster), the colony would now lose over a pound a day—more than it would if the foragers were simply kept in by poor weather!

Going back to ideal conditions in warm weather, with a healthy colony gaining 5 lbs per day. Adding in the metabolic cost of nosema infection to half the foragers only decreases the weight gain by 2/10 of a pound a day. The model indicates that the drag from nosema infection would be less noticeable during good flows in warm weather. So the model suggests that the observed decreased honey crops from colonies infected by nosema in spring are likely due to poorer buildup, rather than direct metabolic drain.

So let's look at the buildup. Colony buildup, given enough available honey, is limited by the income of protein in pollen. In order for a bee colony to build up at the amazing rate that we take for granted, it must consume and efficiently process a minimum of 2-3 pounds of high-protein pollen a week, and several (perhaps 10-15) pounds of sugar. By comparison, a rapidly-growing broiler eats only about 2-3 lbs. of ration; a similar sized growing cat a growing cat only about a pound of dry chow a week.

It may be that the main problem with nosema infection is its impact on the protein dynamics of the hive. Not only do the foragers have a more difficult time energetically in foraging for pollen, but the colony may "starve" for protein despite its being brought in, due to the inability of infected nurse bees to convert it to jelly.

Porrini and other researchers have found that infected bees can live nearly as long as uninfected workers provided that they are given plenty of protein. But that additional protein must be stolen from the overall protein economy—those depending upon the nurse bees to produce jelly (the queen, the brood, the drones, and the foragers) all get a little bit less. So let's look at the cost to colony buildup. In the graph above, those pre-varroa colonies build up damn quick! The multiplied their populations fivefold in two brood cycles! Their daily intrinsic rate of increase (r) was about 1.04 (1.04 times itself 42 times equals 5 x increases). At that rate a 5-frame nuc would cover 25 frames in six weeks (granted, I don't usually see that fast a growth rate these days).

So let's factor in the damage and drain to colony protein dynamics due to nosema. Say that a quarter of the bees in the hive were infected, and that such infection knocked by the intrinsic rate of increase by a quarter, from 1.04 to 1.03 (increase only applies to the amount in excess of 1). At that rate, the same colony, instead of growing into a 25-frame honey-producing monster, would cover only 17 frames.

And yet again, the colony would appear to be perfectly healthy, with no brood mortality nor dead bees--it would just seem a bit sluggish in buildup. This is why nosema is called the "invisible disease." And about half of all US colonies now test positive for *Nosema ceranae* to some degree! It sure makes me wonder if we haven't been paying enough attention to this new parasite.

Fundamental concept: honey bee colonies are by necessity voracious consumers of high-protein, high-energy food. Anything that affects the digestion and utilization of that food will negatively affect colony buildup and survival. Nosema siphons off a share of that protein and energy.

And how about those "fragile" bees that we keep hearing about, that no longer recover from pesticides the way that they used to? Could nosema be involved? I've seen the spore counts from a number of commercial beekeepers who complain loudly about certain pesticides. It's no surprise that their colonies don't rebound well.

OTHER ANIMAL Models

At this point, it makes sense to again look at other animal models. So I investigated the well-studied effects of other gut parasites.

However, infective larvae ingested in late summer-early autumn may become arrested until the following spring … A sharp rise in worm egg output from wild grouse in spring suggests that the delayed maturation of arrested larvae is synchronous. Resumed development of arrested larvae may therefore be linked with disease, as the most outbreaks usually occur in spring and early summer. High worm burdens have been associated with dramatic population crashes.

developing into adults. The authors concluded that developing burdens of *T. tenuis* are more pathogenic than patent infections and the simultaneous resumed development of many arrested larvae in the spring might be the main cause of disease. This would also explain why outbreaks of disease are not observed in autumn even when autumn burdens are much greater than spring burdens of adult worms associated with disease (Moss *et al*. 1993).

Two aspects of timing are crucial to the Shaw & Moss (1990) hypothesis. First, the greatest pathological effect to the host is related to the developmental stage of the parasite and its habits. Second the importance of this pathology to the host depends on the timing and duration of parasite development relative to the host's annual cycle. This study describes the timing of the greatest pathological effects of a developing infection of *T. tenuis* in red grouse.

Reduced growth rates and poor energy metabolism have often been associated with gastrointestinal worm infections in domestic animals (see MacRae, 1993). In contrast, the energetic consequences of parasitic infection in wild animals have been little studied, despite the possibility that even relatively small effects upon energy acquisition could significantly reduce survival and fecundity. Little is known about the mechanisms which may link parasite infection to ecological consequences for the host. One possibility is that the parasite may affect components of the host's energy budget, leading it into energy imbalance. This would reduce the energy available for reproduction, intraspecific competition and/or

Any physiological dysfunction is likely to have energetic consequences. Hence, the pathological effects of *T. tenuis* infection in red grouse have been assessed in this study by their subsequent energetic consequences. This approach allowed a measure of

Delahay, RJ, JR Speakman, and R Moss (1995) The energetic consequences of parasitism: effects of a developing infection of *Trichostrongylus tenuis* (Nematoda) on red grouse (*Lagopus lagopus scoticus*) energy balance, body weight and condition. *Parasitology* 110: 473-482.

MacRae, JC (1993) Metabolic consequences of intestinal parasitism. Proceedings of the Nutrition Society 52: 121-130.

Early findings on subclinical *T.* ***colubriformis*** infections (animals given 2500 larvaeld for 15 weeks) indicated little effect on digestibility but a reduction in the efficiency of utilization of both energy and nitrogen (Sykes & **Coop,** 1976). Subsequent studies, however, using respiration chambers to determine energy expenditures in infected and pair-fed control animals indicated a significant reduction in the digestibility of the ration and, hence, in the metabolizable energy (ME) available to the infected animal The reduction in metabolizability of the ration was concomitant with a much more serious problem relating to the N and P metabolism of the infected animals.

To test whether this was the result of either a reduced capacity to digest and absorb protein within the small intestines or enhanced secretion of endogenous N components into the lumen, Poppi *et* ***af.*** (1986) infused 35S-labelled microbial protein into the abomasum and 51CrC13-labelled plasma protein into the circulation. They concluded that the major factor was probably sloughed epithelial cells, and mucin secretion, which was estimated as elevated 42-150% depending on the resorbability of these desquamated and secreted proteins; the findings showed no change in absorption of 35S-labelled amino acids and although plasma leakage was elevated by 1-2 g N/d this was assumed to be largely resorbed before the terminal ileum. [in cattle] whereas skeletal muscle, which represents **45%** of protein mass, accounts for only 1622% of total whole-body protein synthesis, the GI tract, which comprises only 5% of total protein, contributes 2545% to total synthesis.

degraded and/or secreted

The representation in Fig. 3 of the degraded GI tract protein recycling directly back to the body amino acid pool is, therefore, over simplistic and the structure **of** the tissue needs to be considered. The proteins of the GI tract comprise two distinct types, those of the serosa involved in propulsion of digesta through the tract and those **of** the mucosal epithelia which assist in nutrient absorption. The mucosal cells are continuously differentiated in the crypt regions of the epithelium and then migrate to the tips of the villi over a period of **36-48 h** where they are desquamated into the lumen. Whilst the simple recycling concept illustrated in Fig. 3 could apply to the serosal proteins, a considerable proportion of the mucosal proteins, along with the digestive secretions and mucins require redigestion to amino acids and resorption of these before they are again available to the body. The efficiency of this salvage process is difficult to determine, but is not qualitative since Bown *et* ***al.*** (1986) estimated resorption of plasma proteins at *85%,* while only 70% of 35S-labelled microbial protein introduced into the tract is recovered (Poppi ***et al.*** 1986). The unresorbed residues will enter the large intestine and be excreted either in the faeces or, if sufficient energy is available for secondary microbial fermentation, via the urine as urea following ammonia release in the hind-gut, absorption and hepatic ureogenesis. Either way, this loss is detrimental to the N economy of the animal.

Increased protein and amino acid loss via the GI tract will reduce the amount available for other tissues. Thus, the overall metabolic consequence of the intestinal infection seems to involve the diverting of protein synthesis away from muscle and bone (the 'storage' tissues) and towards the repair, replacement and reaction to damage of the gut wall, to mucus production and to plasma or whole blood loss.

The aforementioned penalties may be exacerbated by alterations in the supply of individual amino acids. The amino acid composition of material absorbed from the small intestine is similar to mixed muscle protein, but is different from digestive secretions such as proteolytic enzymes and mucin, especially for valine, threonine, serine and proline (MacRae & Lobley, 1991). Thus, any elevation in digestive secretion losses will decrease the availability of these amino acids for protein synthesis and gain in other tissues. Most serious is the high concentration of threonine (280 mmoVmol), serine (130 mmoVmo1) and proline (130 mrnoUmol) in intestinal mucus (Neutra & Forstner, 1987), which is reported to be resistant to proteolytic degradation and poorly resorbed from the small intestine (Lindsay ***et*** *al.* 1980). It follows from the previously described considerations that

amino acid supply for protein gain in the storage organs, such as muscle, bone and skin (wool), is severely restricted during the primary infection phase of parasitism and this may continue for specific amino acids in the immunologically-resistant animal. It is not surprising, therefore, that where animals have received supplemental protein, either by increasing the protein: energy value of the diet (Abbott ***et al.*** 1988), or by infusion of protein into the abomasum (Bown ***et al.*** 1986), this has reduced faecal output and worm burdens, enhanced the onset of immunity, and offset the growth check associated with ***Huemonchus*** **contortus** (Abbott **et al.** 1988) and **T. colubriformis** (Bown et **al.** 1986) infections.

Conclusion

Intestinal parasitism diverts amino acids towards protein metabolism in the GI tract and this has major consequences for other organs and tissues. The inefficient absorption of enhanced digestive secretions, particularly mucus, and sloughed mucosal proteins represents a net drain on the N economy of the host animal and may cause amino acid imbalances. Impaired P absorption inhibits bone mineralization and may induce inappetence in infected animals. Supplementation of diets with protein and P appears to offset the growth check and may also enhance the onset of the immunological resistance in infected animals.

"However, parasitic infection is likely to exert its most important impact at the very first step of the alimentary process, by adversely affecting the intake of food through any of a variety of mechanisms."

[6]. The nutritional impact and the benefits of treatment are likely to be greatest for those with heavy parasite burdens and preexisting marginal intakes of protein and/or energy.

tease action. Thus, damage to the intestinal brush border surface results in inability to digest lactose, sucrose, to some extent maltose derived from starch, and the peptide products of protein digestion, in addition to the inability to absorb amino acids, sugars, vitamins, and minerals [22]. Dam-

Rosenberg, IH and BB Bowman (1982) Intestinal Physiology and Parasitic Diseases. Clin Infect Dis. 4 (4): 763-767.

Nosema's Effect Upon Colony Protein Dynamics

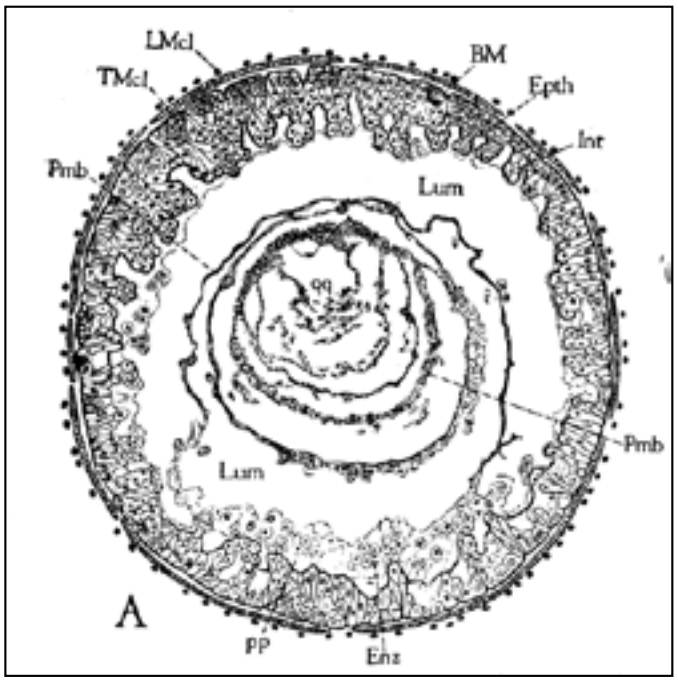
It's Not About the Spores!

The most limiting nutrient for bees is generally protein. A growing colony, due to the high turnover rate of the adult population, requires a huge amount of protein, which must be efficiently digested by nurse bees and converted to jelly—the currency of protein within the hive. Nosema steals a share that protein, and converts it into spores.

What share? Let's just do a little math: this spring, I had colonies that ran 30M spores per forager on average. So let's say that a colony consisted of 30,000 bees, of which a third were foragers. Multiply 30M x 10,000 and the total spore production of that single colony for the foragers alone would be 300 BILLION spores! OK, that is an inconceivable number, so I did the math again. I did a rough calculation of the volume of a single spore, and know that the spores are more dense than water. That many spores would weigh in the ballpark of 5g, about the same nickel coin.

Well, 5g of spores isn't all that much—so the problem with nosema clearly isn't because potential bee protein is being diverted into spore protein. The effect of nosema infection likely has more to do with protein digestion and utilization.

So at this point perhaps I should explain what we know about how bee digestion works. Pollen and nectar both pass from the crop into the foregut (also called the stomach or ventriculus), where digestive enzymes are secreted in order to break down the carbohydrates, proteins, and fats so that they can be absorbed by the epithelial cells that line the gut (see Fig. X). It is this critical interface that is what we need to focus upon.



Cross section of the bee midgut. In the center are the filmy, layered peritrophic membrane (Pmb) which shrinks around the food bolus, protects against abrasion and microorganism invasion, and assists in digestive enzyme circulation. The membrane is formed from the inner epithelial surface (detail of it forming at the lower portion of this drawing), the cells of which slough off and break down (lyse), thus releasing their contained digestive enzymes. Drawing from Snodgrass, RE (1910) The Anatomy of the honey bee. US Government Printing Office.

In order to increase the total absorptive area of the gut lining, the epithelial cells grow into fingerlike projections called "villi," which are covered by a protective and absorptive mucus layer, secreted by the epithelial cells. In humans, there exists a complex community of microorganisms that form a biofilm lining the gut. These endosymbionts have recently been found to be important in disease resistance, digestion, and nutrition. Surprisingly, even though gut endosymbionts have been well studied in some other insects, little investigation has been done in honey bees, although they clearly appear to be involved in resistance to chalkbrood, AFB, and possibly viruses and nosema.

The innermost layer of epithelial cells continually sloughs off, and do a neat trick—they bind their proteins with the mucus, and form a tube within the gut called the peritrophic membrane. Pollen and other solid food remain within the peritrophic membrane, and are pushed by waves of muscular contraction from the crop to the rectum—taking about a day for nurse bees, which have the ability to digest pollen, or as little as a few hours in foragers, which lack the enzymes to properly digest the grains.

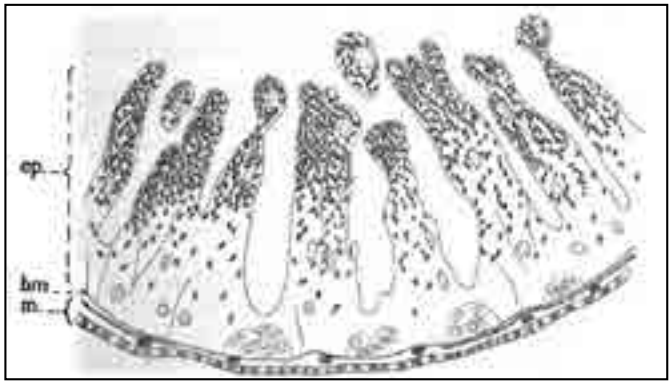
Those enzymes are produced from the epithelial cells, and are secreted into the mucus layer, and then pass through the peritrophic membrane to do their work. The digestion products (simple sugars, amino acids, fatty acids, etc.) then diffuse back out through the peritrophic membrane and through the mucus layer, and then are absorbed by the epithelial cells in the villi, eventually to be transferred into the bee hemolymph (blood) in the body cavity for transport to the rest of the body. Whew!

You may be wondering what this all has to do with nosema—I'm getting there! Nosema spores respond in some unclear manner to gut fluids, and discharge their long polar bodies, which look like incredibly long tiny hoses. It is not entirely clear whether the polar bodies "harpoon" their way through the peritrophic membrane and mucus layer, or whether they "dissolve" their way through, but the lucky ones finally hit their target—an epithelial cell.

Once within the cell, the spore discharges its "embryo" (not the proper term) into the cell, where it attaches to the cell wall and

begins to grow in what is commonly called the "vegetative" stage (again, not the proper term). The nosema organism then divides and grows, eventually producing "reproductive spores," which then send out their own polar bodies to infect adjacent cells. At this stage, nosema is undetectable by common microscopy, but is detectable by a fancy lab technique called PCR.

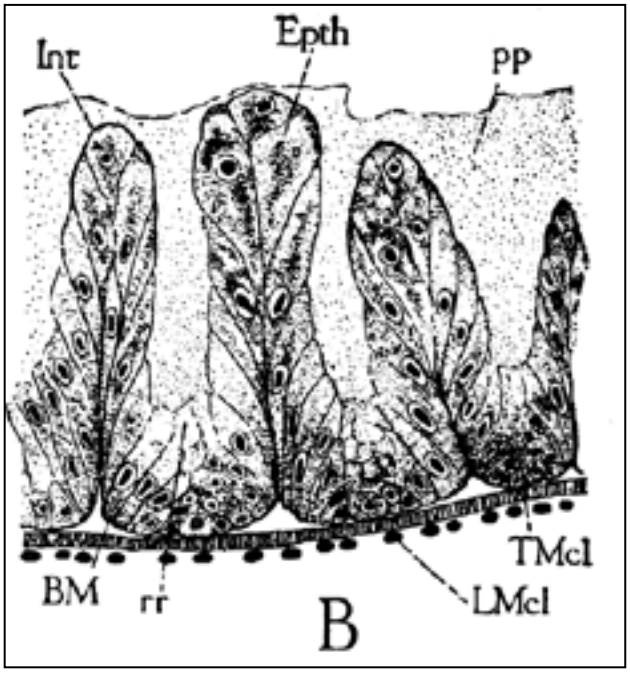
Finally, when the cell becomes crowded with the vegetative stages, they start to form hard-walled "environmental spores." These are the spores that we see by microscopy.



A bee midgut infected with nosema (dark ovals). The parasite infects the epithelials cells (ep) which form the intestinal villi (finger-shaped projections into the gut for nutrient absorption). Nosema generally infects the cells at the tips, and then spreads toward the basal membrane (bm), which lies just under the outer muscular layer (m). The epithelial cells naturally break off (loose cell at top) and break open (lyse) in order to release digestive enzyes, thus releasing the mature spores into the gut. If the bee is unable to generate fresh cells fast enough, then the infection can move into the basal cells, causing death. Drawing by G.F. White (1919) Nosema-Disease. USDA Bulletin No. 780.

New epithelial cells are continually being created by the basal cells, and grow inward to replace the older cells that are sloughed off (when bees eat toxic substances, they simply shed the entire gut lining).

The digestion of pollen, with its tough exine (outer shell) is metabolically costly, especially in the case of pollens containing toxins, such as the amygdalin in almond pollen. This may be the reason that there is a social division of pollen digestion within the hive—allocated to the nurse bees, whose guts are unlikely, under normal conditions, to be ravaged by nosema infection (which takes a number of days, if it occurs at, all to spread through the gut). The foragers and queen then, which, due to their receiving a rich diet of predigested protein (from the nurses) and readily-digested sugar (from nectar and honey) are better able to direct their energies (respectively) toward foraging and egglaying.

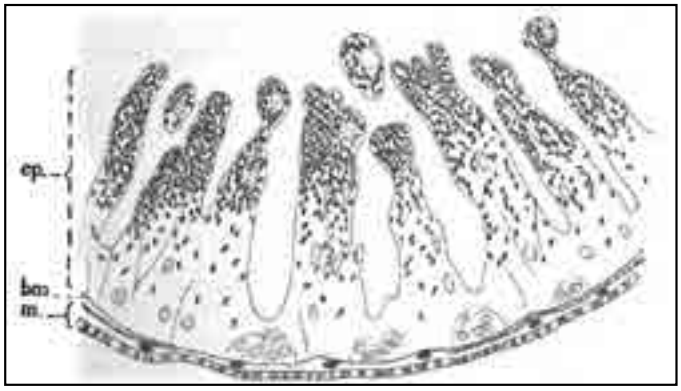


Enlargement of the intestinal villi in an uninfected bee. Note the individual epithelial cells (Epth), each containing a nucleus (oval with dark center). The cells are generated at the basal membrane (BM) and push their way up as the cells at the tip are sloughed off. The epithelium is covered by a thin membrane (Int) and a gelatinous matrix (pp). TMcl and LMcl are muscle layers; rr is where enzymatic cells are being produced.

The hyperplastic growth of the gut has a nutritional penalty for the animal, since the need to renew the gut surface more quickly than normal means that more of the dietary protein and energy are used up to maintain the faster turnover (18). However, this may be offset by the gain in absorption efficiency of the fresh gut surface after the removal of the lectin from the diet.

Since infected nurse bees must divert protein into gut cell replacement, their jelly-producing hypopharyngeal glands "dry up"—this causes a "recession" in the colony due to lack of that critical currency of protein. Broodrearing is curtailed, and Infected foragers, who no longer produce the enzymes necessary for protein digestion, must beg additional jelly from those stressed nurses. Since the colony's ability to process protein is handicapped by nosema infection, it must then forage for additional pollen to make up the difference.

The colony is forced to shift the allocation of its workforce toward pollen collection and processing, which reduces the number of foragers that can focus upon nectar collection. Plus, nosema infected bees begin foraging at an earlier age, which has the effect of decreasing average worker longevity, which seriously impacts the ability of the colony to increase its population. The net result is a reduction in colony buildup rate, its ultimate population, and the ability to gather, process, and store honey.



A bee midgut infected with nosema (dark ovals). The parasite infects the epithelials cells (ep) which form the intestinal villi (finger-shaped projections into the gut for nutrient absorption). Nosema generally infects the cells at the tips, and then spreads toward the basal membrane (bm), which lies just under the outer muscular layer (m). The epithelial cells naturally break off (loose cell at top) and break open (lyse) in order to release digestive enzyes, thus releasing the mature spores into the gut. If the bee is unable to generate fresh cells fast enough, then the infection can move into the basal cells, causing death. Drawing by G.F. White (1919) Nosema-Disease. USDA Bulletin No. 780.

Effect Upon Energy and Honey Production

Nosema is a highly specialized fungus that has lost the ability to process sugar into energy by itself. It is completely dependent upon the bee to convert sugar to the metabolic "battery" ATP. The cell membrane of the vegetative (growing) stage of nosema steals ATP from the bee cell. As a result, the blood sugar (trehalose) level of infected bees drops. Such a bee can no longer fly as far, heat the colony as well, produce as much beeswax, nor share as much food with other bees.

I previously mentioned that the colony superorganism is intelligent. Intelligence requires energy. The brain is one of the most energy-hungry tissues—a bee's ability to learn is suppressed by hunger. Also, at the larger colony level, the "thinking" of the hive is dependent upon pheromone production, trophallactic transfer of those pheromones and food, and the patrolling behavior of mid aged bees—all of which require

energy. When nosema steals a share of that energy, the ability of the colony to efficiently manage its economy may suffer.

The flow of energy within the colony is dependent upon food exchange (trophallaxis) between bees. Dr. Dhruba Naug (2009) investigated the effect of *N. ceranae* infection upon such food transfer. He found that infected bees were not only more likely to beg for food, but once they obtained it, they were averse to sharing it with their cohorts. Such behavior may aid in slowing the transmission of nosema within the colony, but would also put a brake on the flow of sugars from foragers to stored honey. It appears that each infected bee becomes a "sink" that drains energy from the colony and converts it into nosema spores rather than into honey.

Naug, D and A Gibbs (2009) Behavioral changes mediated by hunger in honeybees infected with *Nosema ceranae.* Apidologie 40(6): 595–599.

then sets out on the next collecting trip. Thus, in acquiring its food, a colony of honey bees functions as a large, diffuse, amoeboid entity that can extend itself over great distances and in multiple directions simultaneously to tap a vast array of food sources. If it is to forage efficiently, it must wisely deploy its foragers

We must realize how marginal the colony's ability to produce honey is. Dr. Rob Currie (pers comm) tells me that in Saskatchewan, where colonies put on large honey crops in a short period of time, that an infestation by varroa as low as one mite per hundred bees infestation will reduce honey production. Infection by nosema would have a similar effect.

Nosema Affect Upon Foragers

Bee "aging" does not appear to begin until the bee begins foraging. From that point on, they have a short life expectancy, due to predation and other hazards, wear and tear, and lack of critical mechanisms for wing muscle maintenance (see Old Bees, Cold Bees). Foragers typically survive for about a week (2-17 days); few make it past 70 foraging trips or 50 hours of flight (Visscher 1997).

Hungry bees start foraging earlier in life, which makes evolutionary sense, since a hungry colony would want to direct its efforts toward foraging rather than broodrearing. The downside of this is that the earlier in life that a bee starts foraging, the shorter it lives as both a forager, and in total lifespan. An unfortunate side effect of this is that foragers don't hit their stride as far as nectar-gathering efficiency until they near their normal expected lifespan (Dukas 1994), so shorter-lived foragers may be less efficient foragers. This premise is supported by data from Dr. Frank Eischen (2010), who found that in almonds, lightly infected colonies collected twice as much pollen as those that were heavily infected.

What's interesting is that foragers highly infected by *N. ceranae* do not necessarily altruistically fly off to die away from the hive, as sick bees normally do. They continue to forage, while their body just keeps cranking out nosema spores, with individual foragers sometimes containing half a billion or more spores! So the question arises, are those infected bees actually contributing to colony buildup, or are they simply going about trying to stay alive, inadvertently squirting millions of nosema spores all over the place?

Using a metabolic rate of about 700 mW/g at 20 8C or 450 mW/g at a more ideal environmental temperature of 35 8C (Woods et al., 2005), an infected forager can be estimated to have the ability to fly about only two-thirds the distance compared to an uninfected forager on any given day.

This reduced flight range can really hurt the colony, since:

1. If a healthy colony can normally forage over 12 square miles (2 mile radius), nosema infected bees could only cover 5½ square miles—less than half the potential foraging area!

2. Pollen foragers are even worse off, since they don't necessarily carry fuel for the return trip, and can simply "run out of gas" on the way home, and perish in the field.

Neither of the above effects of nosema infection would jump out at you, other than that the infected colony might appear lethargic in buildup.

Mayack, C and D Naug (2010) Parasitic infection leads to decline in hemolymph sugar levels in honeybee foragers. J. Insect Physiol. 56(11):1572-1575.

/Adjectives Lagging lethargic languishing hobbled constrained sluggish /Therefore, the number of hive bees fed per forager with the nectar intake described in Table 1 increases from 4 to 12 as air temperature rises from 20 to 40 8C. These results suggest that triple the foraging effort would be required for a colony to subsist at 20 8C relative to 40 8C. Also, at the same level of foraging intake, colonial honey accumulation should triple as air temperatures rise from 20 to 40 °C.

Harrison (2002), carefully measured bee metabolic rates, and mathematically modeled the energetic efficiency of foraging at different temperatures.

Pop age structure changes in warm summer months—the proportion of bees old enough to be foragers increases greatly.

During late winter and spring population turnover (old winter bees being replaced by new bees), a very large proportion of the population consists of old, potentially nosema- and virus-infected bees.

I'm having trouble finding accurate data as far as what percent of the worker population acts as foragers. but when I analyzed Lloyd Harris' data (pers comm) for colonies in Manitoba, in June and July, 40-50% of the workforce was over 24 days old

The key component of the honey bee colony as a superorganism is the forager force. In comparison to our coddled broiler chicken, which has food brought to its feet, a portion of the honey bee colony "explodes" each day over several square miles in its quest for food. When temperatures are cool, or if it is windy, such foraging is extremely energy expensive, to the point that at temperatures below 55°F, body heat is simply lost to the air faster than the bee can generate it.

Using a metabolic rate of about 700 mW/g at 20 8C or 450 mW/g at a more ideal environmental temperature of 35 8C (Woods et al., 2005), an infected forager can be estimated to have the ability to fly about only two-thirds the distance compared to an uninfected forager on any given day.

The degree of population growth of the colony is based upon the average longevity of the individual adult bees. If even a couple of days are knocked off the average lifespan of the foragers, the colony will be unable to grow in population. Nosema infection typically reduces forager lifespan (as well as brood survivability). So when you find lethargic colonies, you might want to check them for nosema infection.

Any number of studies in the past hundred years (reviewed by Hornitzky 2005) have demonstrated that even relatively moderate *Nosema apis* infection reduces honey yield. The same appears to be the case for *N. ceranae.* However, you may not notice the effect; in my own operation, colonies with spore counts in the 5M range are productive and appear to thrive. But I haven't run a controlled trial that measured nosema infection vs. honey production.

Effect of Environmental Temperature

We would expect to see the most negative effect of nosema infection during cool weather, when there is increased energy demand for colony thermoregulation. But especially noticeable at cool temperatures is the effect upon foragers. Not only does infection decrease their lifespan, but it robs them of the ability to fly as far, to carry as heavy a load, and results in a less efficient the net energy gain per forager flight Harrison (2002). It is not unusual to see exhausted returning nosema-infected foragers in the grass in front of the hive.

Nosema and Colony Population Dynamics

Premature foraging

/Khoury DS, Myerscough MR, Barron AB (2011) A Quantitative Model of Honey Bee Colony Population Dynamics. PLoS ONE 6(4): e18491. doi:10.1371/journal.pone.0018491

"The model predicts a critical threshold forager death rate beneath which colonies regulate a stable population size. If death rates are sustained higher than this threshold rapid population decline is predicted and colony failure is inevitable."

"When forager death rate is high, nurse bees begin foraging precociously (Fig. 6). While this restores the proportion of foragers in the population, it shortens the overall lifespan of adult bees (Fig. 6) and reduces the time each bee can contribute to colony growth and brood production. This reduces the brood-rearing capacity of the colony. Since precocious foragers are less effective and resilient than normal foragers [25], [26] forager death rate increases further, the pressure on colony population is compounded and the rate of colony decline is increased"

/On insidious effect of nosema infection is that it changes the population dynamics of the hive. During the spring and summer, average worker longevity is about 5 weeks under good conditions—about three days as a "cleaner," then 12 days processing pollen as a "nurse," then an indefinite period (usually about a week) as a mid-aged bee until "called" to graduate into foraging. From that point, it normally only lives for about another two weeks (read my "The Economy of the Hive").

It has long been known that nosema-infected bees transition to foraging behavior earlier in life. Such a precocious shift in task allocation has a huge effect upon overall colony buildup rate and productivity, since:

1. There are then fewer nurses to process protein for the colony.

2. There would be a smaller "reserve" of the mid-aged bees so critical for colony population buildup. These are the general-purpose workers that receive and process nectar, build comb, and store honey.

3. And perhaps most importantly, the clock on bee "aging" only starts ticking once the worker begins foraging behavior. So the initiation of premature foraging cuts a worker bee's potential productive lifespan off short. This abbreviation of worker longevity can bring colony buildup to a screeching halt, curtail honey production, ***and greatly decrease the ability of the colony to recover from "normal" virus infections.***

Practical application: colony growth and productivity, as well as its ability to deal with viruses, all depend upon long-lived workers. Nosema causes premature worker "aging."

Dr. Jim Frazier recently created a colony population model which suggested that induced premature foraging has a far greater negative effect upon colony population than does major (90%) brood mortality!

So does the impact upon worker longevity differ between the two nosema cousins? Dr. Zachary Huang compared the onset of foraging for young bees infected with either or both of the two nosemas:

- Control (uninfected) bees: fewer than 1 out of 10 were foraging by Day 7.
- Infected by *N. apis*: about 1 out of 8 foraging by Day 7.
- Infected by *N. ceranae*: 1/3rd had already shifted to foraging by Day 7.
- Coinfected by both nosemas: 6 out of 10 foraging by Day 7!

Even the mathematically challenged should be able to recognize that reducing bee longevity from the expected five weeks to perhaps three weeks can put the colony into a downhill spiral.

Nosema and Immune Response

Texier (2012) It is noteworthy that D. melanogaster infected with microsporidia develops a fundamentally different response by known immunity related genes as compared to the other microorganisms used for immune challenge (virus, bacteria and fungus). Nevertheless, these defence responses do not prevent the progression of infections which can either be chronic or cause host mortality. Moreover, several data suggest that some microsporidia may possess survival mechanisms and are able to module/suppress the host immunity [52,53,56].

Macrophage destruction by Nosema!!!

The ramping up of the bee immune response to nosema infection, and the need to regenerate damaged gut cells is costly in both energy and protein. In addition, immune stimulated bees have decreased memory formation/recall abilities (Tyler 2006). Such degradation of recall ability could explain the premature mortality of infected foragers.

The Nosema/Virus Connection

Not only are some bee viruses (such as Black Queen Cell Virus) nearly always associated with nosema infection, but the main mechanism for the colony to fight viruses is for bees with overt virus infections to abandon ship, sacrificing themselves for the good of the colony. But this strategy to try to purge the virus epidemic from the hive only works if the colony can maintain recruitment of replacement bees. Nosema hampers the colony in that effort. So even a nosema infection that does not directly cause bee mortality can precipitate colony dwindling from what would otherwise have been a "routine" virus infection (see my model in "Sick Bees 2").

Practical application: Call me bold, but I strongly suspect that this evolutionary "working out" of the new varroa/virus/nosema parasitism that our bees are suffering from may explain much of the increased colony mortality that has been plaguing our poor bees.

So Why are the Effects of Nosema Infection Not Obvious?

This question has bedeviled me since Dr. Mariano Higes (2007) scared the bejesus out of us with the finding that this parasite killed 100% of infected (caged) bees within 8 days! My operation was suffering from CCD at the time, and sure enough, I found that many of my hives were infected with *N. ceranae.* However, as Paxton (2010) observes, "Of course, the association between *N. ceranae* and poor health of *A. mellifera* colonies may simply represent reporting bias; a novel disease organism in moribund colonies will be understandably publicized whereas it may go unsampled and unnoticed if it does not have a marked pathological effect on its novel host."

It is frustratingly difficult to compare the various field studies, since samples of bees are taken from different parts of the hives, analyzed differently, fumagillin treatments are applied in different manners, to name some of the confounding variables. I suspect, however, that our main problem in understanding the true degree of impact of nosema upon colony health and productivity may be due to our relying spore counts, as opposed to the prevalence (proportion of bees infected in a sample) being used as the measure of the degree of infection at the colony level.

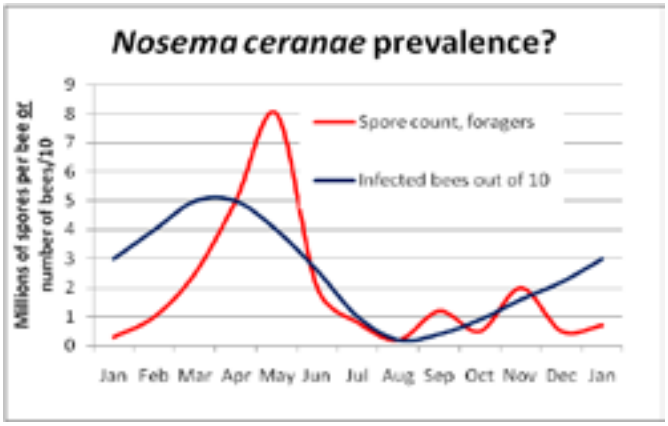
Nosema have been referred to as "the no-see-um" disease, "the silent killer," and "the invisible disease" since there are no overt symptoms of infection. At this point, please allow me to put a common misconception to rest: **Nosema infection does not cause dysentery.**

This was reported by Dr. White in 1919, and has been confirmed by every major bee pathologist since (Moeller, Farrar, Bailey, Fries, Gochnauer, Furgala, Shimanuki, Higes). I have confirmed by personal microscopic observation that dysentery occurs independent of nosema. That's not to say that bees with dysentery aren't infected with nosema, just that dysentery is not a diagnostic sign.

The robberies of energy and protein from the hive economy by nosema infection may go unnoticed when colonies are flush with nectar and pollen. I've been vocally skeptical (until recently) of *Nosema ceranae* causing any significant problems with colony health. However, I'm starting to think that I was a bit too hasty in drawing that conclusion.

I have continually asked myself, as well as Dr. Higes, why had I not noticed obvious problems due to *Nosema ceranae* in my California operation. I know my colonies are often infected, but I could never nail down any relationship between spore counts and colony health or productivity. Part of the problem may have been due to relying upon spore counts as the measure of degree of infection, as I've detailed previously. But there appears to be a more likely explanation.

High spore counts may be more of a function of colony nutrition, rather Porrini



A comparison of infection rate vs. nosema DNA abundance as determined by qPCR. Try to "smooth out" the blue curve in your mind's eye, and ignore the dip in the green line in May. The blue line estimates the estimated infection rate in 5-bee samples. The green line indicates the number of DNA transcripts in positive samples. **Note that infection rate rose during winter, and dropped during spring, whereas transcript abundance roughly reflected the normal high spore counts in spring.** Graph calculated from data in Traver, Williams, and Fell (2011b); see text for details.

Moeller (1978—a free download and good read) observed, "When the disease is acute, colonies may become depleted in population and eventually dwindle down to a handful of bees and a queen…"Oldtimers" called this stage "spring dwindling"…In colonies not so severely affected, brood emergence eventually allows the colony to recover and produce a normal honey crop. How much honey is annually lost because of such subacute or endemic Nosema infection is impossible to estimate, but the loss must be substantial."

Moeller (1978, citing his 1972 paper) also observed, "Because a primary natural defense against nosema is the emergence of brood—allowing replacement of infected bees with healthy young bees—any disruption or break in brood rearing and emergence of bees will make the colony a candidate for nosema disease." It appears that many commercial beekeepers today avoid major nosema problems by feeding pollen supplement when there is inadequate natural pollen available.

Moeller also states that "The best defense against nosema is to winter strong colonies with plenty of honey in the proper position, feed pollen supplement in the spring, and then divide the bees early to make colony increase. A two-queen colony that is properly overwintered is seldom lost or weakened enough to become a candidate for severe nosema disease."

And a very recent study by the Higes team (Botias 2011) found that *N. ceranae* infection can be largely suppressed by yearly requeening.

Another thing to consider is that colony winter mortality picked up from historical 5-10% levels to 20-25% after the establishment of varroa, and the evolution of associated viruses. Not to say that nosema can't cause problems by its own right, but it appears to me that there is a morbid synergy between *Nosema ceranae* and some viruses. My Australian friends have not noticed any serious problems since the invasion of *N. ceranae* (Hornitzky 2011), but they don't have the added stress due to varroa and viruses that we do. I will discuss this further in an upcoming article.

Data presented recently by Dr. Frank Eischen indicates that even at low infection intensity (0.5 million spores/bee), as measured by mean spore counts for a 100-bee sample from under the lid, that there was substantial decrease in performance. He's also found that controlling nosema helps greatly in bringing stronger colonies to almond pollination. But note that even a single infected bee containing a "normal" 50 million spores would put the mean infection level for the entire 100-bee sample at 0.5M spores, even if not a single other bee were infected at all! Hey, I'm as much at a loss for explanation as you are!

I'm also not entirely convinced that *Nosema ceranae* is really that much of a problem as a sole infection. As I've said before, colonies in my operation certainly appear to thrive while carrying average forager spore loads in the range of 5M. But when I inoculated such colonies with purified virus, they started collapsing. I strongly suspect that nosema/virus synergy is the more common problem. Luckily, the same management methods (young queens, good nutrition) that help colonies deal with nosema also likely help them to hold their own against viruses. There are also no proven antiviral treatments for bees on the market to date, whereas there are several effective or potential treatments against nosema.

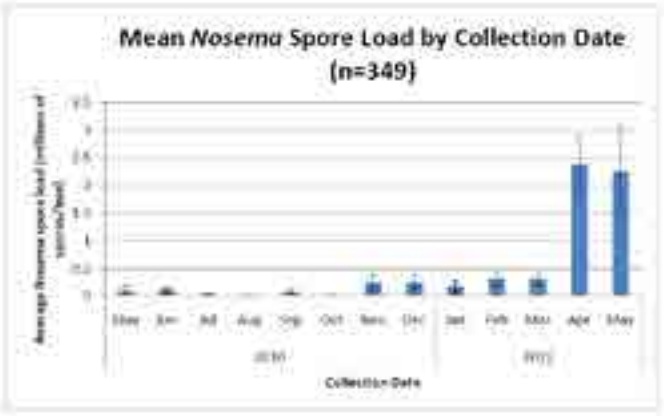
The Weslaco lab This spring we are planning large field Nosema study in TX that will hopefully answer most of the questions you are asking.

But before you go out and start dumping in fumagillin to keep nosema at bay, Eischen also reports that treatment temporarily sets colonies back, so it appears that it should be used judiciously. He didn't find a significant difference in colony mortality due to treatment (but I did in a trial that I will soon report). It's also worthwhile to note that Eischen, and other researchers as well, generally don't necessarily find any correlation between fall and spring spore counts—so it is very difficult to make any treatment recommendations based upon spore counts alone. Sorry!

Bottom line: Under good conditions, or with current methods of measuring the degree of nosema infection, you may not

notice any effect of (apparently) moderate infection by *Nosema ceranae*. However, should the colony suffer from any of the four horsemen of bee apocalypse—chill, poor nutrition, toxins, or other parasites (especially varroa or viruses)--nosema can exert a major drag upon colony buildup and production, or even be the straw that breaks the camel's back, leading to collapse.

I don't want to be alarmist about *Nosema ceranae*. I have plenty of it in my own operation, have not treated (until spot treating dinks this month), and have not experienced excessive colony losses. It is clear to me that high winter mortality and colony collapse can be caused by different combinations of factors. But nosema and viruses appear to be common factors to all.



Rennich, K, et al (2011) **2010-2011 National Honey Bee Pests and Diseases Survey Report**

60% of this year's samples contain Nosema (Karen Rennich, 2012 ABF Convention).

Recent studies: at the 2010 AHPA meeting, presentations by Drs. Pettis, Eischen, Rinderer, Aronstein, and vanEngelsdorp all strongly suggest that *Nosema ceranae* impacts colony health or productivity—sometimes at relatively low infection rates. Research from others (Pernal, Nasr, and myself) also confirms this.

The Hidden Cost of Nosema

Infections by many human pathogens may be "subclinical" or "asymptomatic"—meaning that your body is fighting them off, but you don't come down with the full complement of symptoms. Your body may eventually "clear" itself of the pathogen, or find that that it is most cost effective to simply tolerate a low level of infection (as in the case of parasitic worms, viruses, and gut microbes), sometimes for the rest of your life, without causing serious disease.

Nevertheless, fighting (and perhaps winning against) the pathogen comes at a cost—you may lack "energy," lose weight, or become more susceptible to other pathogens. Such a chronic infection may go unnoticed, unless the doctor takes a blood test or stool sample.@@@

If all we focus upon is spore counts, we will likely overlook a substantial "hidden" cost of the new nosema. Traver (2011) analyzed samples of bees from some 300 hives from across the state of Virginia. Using state-of-the-art qPCR analysis of samples of 5-bee samples, they estimated that 70% of the hives were infected by *Nosema ceranae* (had they analyzed larger sample sizes, my guess would be that they would likely have detected nosema infection in virtually every hive).

So here's my point: qPCR indicates that spore counts don't begin to tell the story—we only detect spores when the colony immune response fails to keep nosema from producing large quantities of "environmental spores"—the final product of a serious nosema infection. The rest of the time, *Nosema ceranae* smolders as a largely "vegetative" infection—invisible to standard microscopy. It is the "cost" to the colony of keeping that smoldering vegetative infection in check that I wonder about.

Texier (2010), who details a number of different insect immune responses against microporidians), explains:

"Whatever the host is, the resolution of [microsporidian] infection strongly depends on the efficiency of the [induced] immunity…, which may depend not only on the inner characteristics of the host but also on the parasite manipulation and evasion capacities...host resistance towards microsporidia has a fitness cost at the population scale…, illustrating the… necessary trade-offs between resistance and other biological functions."

In other words, it is a zero-sum game for the bees—if they want to resist nosema infection, they have to give up something else, such as honey production. Let me use a parable to illustrate:

A Parable of the Cost of Protection

Once upon a time, there was a prosperous town of 200 folk, with an average income of 100 gold coins a year. The town's prosperity drew thieves, and soon it got so bad that you could see them everywhere, and if a homeowner let down his guard, he might find his home entirely looted.

The mayor, in response, levied a tax to enable him to hire two very effective private security contractors, at a cost of 500 gold coins each per year. Within a week, there was not a thief to be seen! The townsfolk were happy.

Some months later, the mayor bragged, "As anyone can see, there are no longer any thieves about, so there is no cost to our community from thievery anymore!"

But the town's tax collector wryly notes, "True that there is no longer any direct cost due to thievery. But the hidden cost of vigilance comes at a price of 5% of our former income, so our protection from thievery makes us all just a little bit poorer."

The moral: just because you don't see any thieves, that doesn't mean that you aren't paying a price for constantly eradicating them. The only time that you'd actually notice any actual thieves is when security breaks down.

The point: just because you don't see nosema spores, doesn't mean that the parasite isn't exacting a hidden cost in buildup or production. This "invisible" cost of immune response to *Nosema ceranae* could be having a significant effect upon bee productivity and survival, without any necessary indication by spore counts! The only time that you'd see spore counts rise is if overall colony immunocompetence had already tipped into the failure zone.

And even if the colony immune system fails, and a nosema epidemic takes hold, in most cases the colony will rally in late spring, and recover by summer. But a "recovered" colony may be a far cry from a "productive" colony. Since we're still not clear on the major mode of transmission of *N. ceranae*, we really don't know if there is a "legacy" effect from nosema buildup. Such a residual effect could plausibly effect colony winter survival, either directly due to nosema, or in synergy with viruses and other factors (more on this later).

Allow me again to quote Texier: "Microsporidian infections in immunocompetent mammals are often chronic and asymptomatic whilst immunocompromised hosts develop lethal disease." The same likely applies to bees. So long as a colony is able to maintain its immunocompetence, it can keep a lid on nosema. But the moment that its immunocompetence falters, nosema and viruses are just smoldering there, ready to explode.

Texier, C, et al (2010) Microsporidia: a model for minimal parasite–host interactions. Current Opinion in Microbiology 2010, 13:443–449

It appears that the rules of the nosema game have changed. Is the apparent explosion of nosema prevalence since the year 2000 associated with the higher winter mortality that we have been experiencing? I don't know, but it sure appears guilty by association. I guess that the take home message is that our bees now have a chronic, constantly mutating, year-round infection, albeit generally at low levels. This smoldering epidemic exacts a hidden cost upon colony health and productivity, and threatens to burst into flame any time that colony immunocompetence is compromised by poor nutrition, chilling, high mite/virus levels, or pesticides.

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HONEY BEES DECIDE!

Honey Bees demonstrate decision making process to avoid difficult choices.

A recent study on the metacognitive ability of honey bees suggests that they, like humans, avoid difficult decisions when they lack sufficient information to solve a problem.

Researchers from Macquarie University in Australia tested honey bees with a series of trials involving visual discrimination between targets inside a two-chamber apparatus. The bees had to learn a rule to match a combination of shapes with nectar. A correct identification was rewarded with sweet nectar, but an incorrect decision resulted in a bitter tasting solution. Bees could also choose not to take the test at all and 'opt out'.

Researcher Dr Andrew Barron says the results showed that the more difficult the challenge, the more likely the bees were to 'opt out'.

"It's a highly debated topic, whether non-humans have the same abilities to gauge their level of certainty about a choice before taking action."

Co-author Dr Clint Perry says, "Similar metacognitive testing has been conducted with dolphins, dogs, and rats. However this study is the first to demonstrates that even insects are capable of making complex and adaptive decisions.

"The honey bees' assessment of the certainty of a predicted outcome was comparable to that of primates in a similar paradigm."

The size, shape, color and positions of the targets were constantly changed during training so the bees had to learn a geometric rule to solve the task correctly. The bees demonstrated a high level of learning ability to solve the tasks, but when the discrimination of the targets was made harder the bees' behavior changed.

"As we made it harder for the bees to assess the correct shape combination, the bees' uncertainty about the correct choice grew, and we observed an increase in the decision to exit the chamber and not take the test to avoid the chance of getting it wrong," said Dr Barron.

"This suggests that the bees were only taking the test when they were confident of getting it right."

The full study *Honey bees selectively avoid difficult choices they lack the information to solve* has been published in full by the National Academy of Sciences.

Clint J Perry, Andrew B Barron *Honey bees selectively avoid difficult choices*. National Academy of Sciences of the United States of America.

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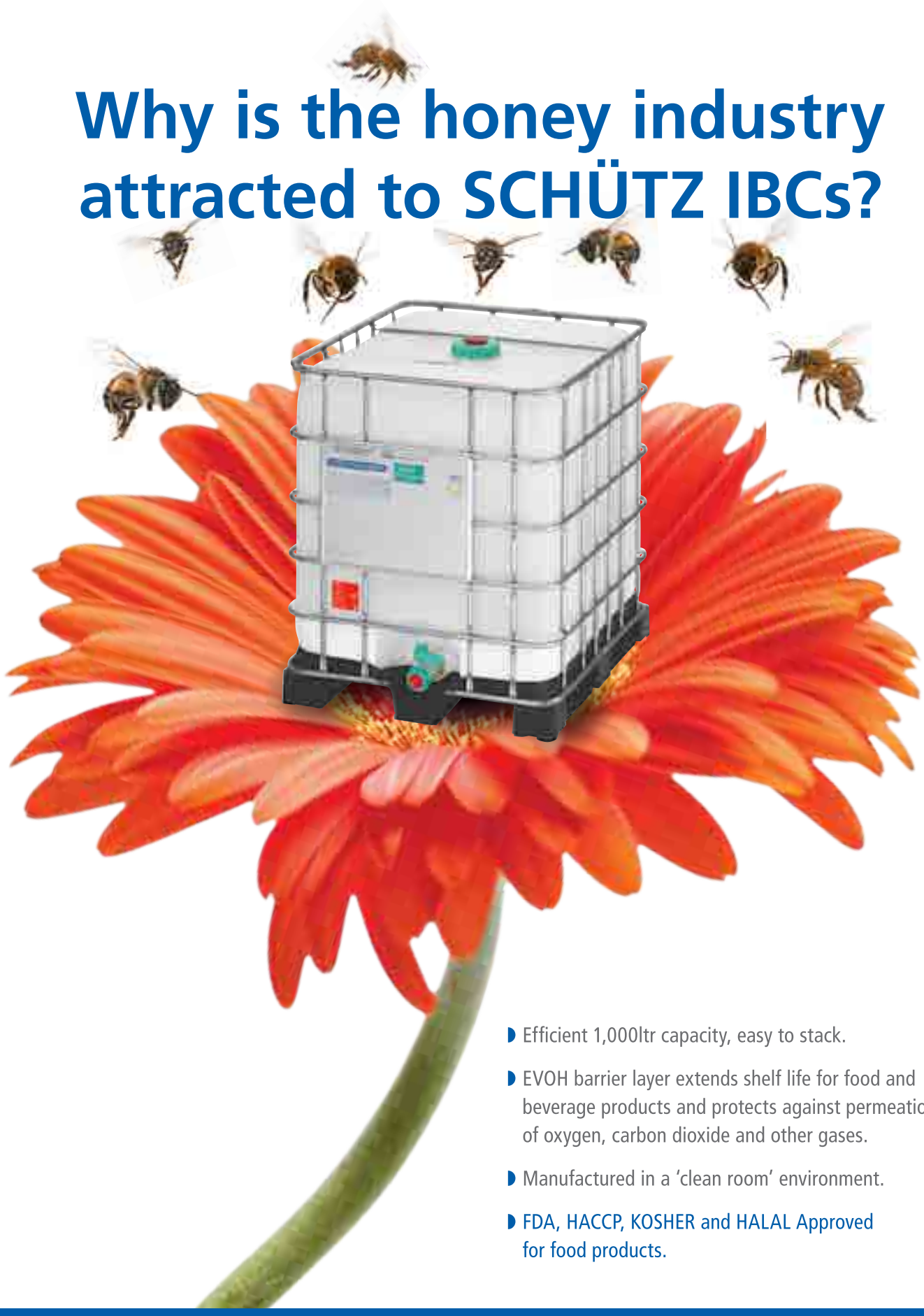

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