AUSTRALIA'S

HONEYBEE NEWS

"The voice of the Beekeeper"

Volume 5 Number 1
JANUARY - FEBRUARY 2012

- Sydney Easter Show - 5-18 April
- 2012Conference - Coffs Harbour 24/25 May

AUSTRALIA'S HONEYBEE NEWS

The Journal of the NSW Apiarists' Association Inc. (NSWAA)

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PRESIDENT'S REPORT

Season
Generally the season has been good with good rains sporadically over most of the State, then flooding rains in recent weeks both on the North Coast and Northern inland.

Southern NSW was relatively dry early in the season then gaining substantial rains later in the season.

Most beekeepers had problems with bees wanting to swarm, this resulted in many queenless hives failed to get a queen of their own.

Honey production has been varied, however most beekeepers have gathered near to average production.

Honey stocks are low, as most production has gone straight to processors or market.

Prices are remaining firm, with most packers paying $3.40 - $3.60 and some looking for further supplies.

Sydney Show
Bruce White has agreed to be the coordinator for our Honeyland Stand at the Sydney Royal Easter Show. Bruce has had a long relationship with the Show as a regular volunteer along with his Judging duties. As Bruce lives in Sydney it makes this a very good appointment.

Preparations are well in hand and volunteers are required. If you have a group who could assist at the Show please contact Bruce White on 02 9634 6792 or return the volunteer form included in this edition.

Conference
We have invited a good selection of speakers, on pertinent subjects so we are expecting an interesting array of presentations.

We would like to invite sponsors for the conference. If you would like to sponsor Morning Tea or place an Ad in the Conference Papers please contact the Association Secretary on Phone: (02) 6373 1435 or Email: nswapiaristsassociation@gmail.com. This is a great opportunity to promote your business and at the same time help the Association.

It is time now to consider carefully who you would like on the Executive Committee of NSWAA. Conference will come around quickly and thought should be given to who represents your views with the Association.

The Conference is on 24/25 May at the Coffs Harbour Ex-Services Club. Followed by a Field day on Saturday 26 May. We look forward to seeing you there.

New Secretary
A warm welcome is extended to our new Secretary.

Her name is Kate McGilvray from Mudgee. Kate has had experience in agricultural associations through her work as Secretary of a Dairy Association.

It is expected the she will begin her duties at the Executive Meeting to be held in Orange on Thursday 23 February and the BICC Meeting on Friday 24 February. Kate's contact details are on Page 4.

VALE
On behalf of the Association I would like to extend sincere condolences to Kae Klingner, Craig Klingner and families on the passing of husband and father Monte Klingner and also to Jeff Smith and family on the passing of his mother Myrtle Smith.

Bill Weiss
State President

BEE TRADE SHOW 2012

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In conjunction with

NSWAA AGM / Conference

Further Information to Register
Therese Kershaw
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ltkershaw@bigpond.com

ECROYD WINE & CHEESE EVENING
Thursday 24th May
6pm

NEW MEMBERS

A warm welcome to the following new Members:

Tracey Hargans	Sydney
David Lord	Bathurst
Mark McClelland	Stonehenge
Michael Standen	Moss Vale

SYDNEY ROYAL EASTER SHOW 2012

VOLUNTEERS NEEDED 5 - 18 April 2012

There is a buzz around the Showground as we approach the 2012 Sydney Royal Easter Show which runs from Thursday 5 April to Wednesday 18 April.

We have a new Show Coordinator – Mr Bruce White, who is working very hard to make the Show a success for the Association.

Experiencing the Show as a volunteer is a truly rewarding experience. Sure they are long days, your feet hurt, some customers ask the same questions over and over again but you are promoting HONEY and what better place to do it? So come along this year and join in. If you haven't volunteered before this is the year to do it.

Accommodation is available for Country Members at Ashfield - just a short train ride to the Showground.

Enclosed is a form for volunteers. Members can choose the day/days they wish to work but there is a limit to the number on each day so you are asked to give your second preference.

Please return your form no later than 21 March.

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DOUG'S COLUMN

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O H & S

This acronym, which most people are very familiar with, conjures up an image of rules, regulations and red tape. OH&S of course means Occupational Health and Safety.

The primary reason for a focus on OH&S is to prevent injury and death incidents in the workplace.

On the 13 December 2011, NSW Farmers conducted a Work Health and Safety Seminar at Queanbeyan specifically for the beekeeping industry. The session was relatively brief, starting at 10.30 and finishing by lunch time with more than 35 present.

There was some apprehension and resentment by the audience, which was predictable to any imposition of rules and regulations. While the presentation was directed at companies with multiple levels of employees, there were some very clear take home messages.

Effective as of 1 January, 2012 a new Work Health and Safety Act replaced the old NSW OHS Act. There are 9 separate OH&S Acts covering Australia, the intent of the changes was to align all jurisdictions with one model Work Health Safety Act.

As I listened to the presentation some of the main points were:

- The OHS Act applies to everyone on the worksite; managers, workers, visitors and the general public. This includes contractors, delivery persons, metre readers, government personal etc. "All" have an obligation under the Act to exercise a duty of care.
- In the case of a beekeeping business, have you as the owners, operators taken all 'reasonable' steps to prevent injury.
- In the event of an accident, the authorities investigating the incident will take into account any industry codes of practice, industry guidelines or what is considered reasonable industry practice.

In the past businesses with multiple numbers of employees usually have a designated employee who has as part of their duties to be the OH&S officer. This has implications with the rest of the staff and perhaps the management that if 's__t' happens, then the OH&S officer may wear some of the repercussions. The new legislation clearly indicates that 'everyone' has a duty of care. Anyone in the workplace, workers, visitors or managers that identify a significant risk has an obligation under the Act to do something about it. In the case of a visitor, it many be informing the manager (beekeeper) that having a six foot trench in the front drive without adequate signage posses a significant risk.

In preventing injury, are all the electrical devices used in the extracting room safe? Have they been tested by a qualified electrician? In the event that someone is electrocuted on your premises the investigating officers will presumably ask for evidence of the equipment being safe. Having appliances tested and a record kept of such tests would be vital information for you to demonstrate that you have taken reasonable steps to mitigate this risk.

Bees' sting! This is stating the bl__dy obvious, but this is also something that many beekeepers take for granted and a degree of complacency can evolve in many beekeeping operations.

In NSW, State Forests and National Parks through their terms and conditions for beekeepers to place apiaries on their land have stated that they require adequate signage of the presence of bee hives. While any beekeeper will be able to identify bee hives, particularly ones belonging to another beekeeper a kilometre or more away, this is not a skill that the average person possesses. Thus it is reasonable for us as beekeepers to assume that bees may pose a risk, to non-beekeepers (the majority of the population). Given that bees sting and pose a risk to the public they should be given sufficient warning of the presence of bees.

A very small percentage of the population are likely to have a life threatening reaction to bee stings. The majority of the population will have an uncomfortable and painful experience if stung. Thus there should be serious attempt to warn people of the risk. If you take all reasonable steps to warn of the risk of bees in the area and a visitor (for example) was stung, then you are not automatically at fault. If you haven't provided reasonable warning of the potential risk, then you have a harder position to defend.

Codes of practice, rules and regulations, plus guidelines involving OH&S matters can be very useful for the beekeeper to use as a reasonable guide to determine if they are meeting their obligations under the Act. While such documents are not intrinsically part of the Act, they will certainly be referred to by the authorities investigating an accident on your work place.

For instance, State Forests and National Parks have a list of conditions for beekeepers to adhere to when placing apiaries on their lands.

If either of the agencies observe you not adhering to these 'rules' they may refuse to allow you access in the future. The risk of loosing sites to the individual beekeeper is very real and the loss of any site is a major issue to a beekeeping business. Thus the primary incentive to behave and comply with the rules is obvious.

No such 'rules' exist with private property sites. If an incident occurred that a visitor, or worker on the private property was stung in close proximity to your apiary and the event was considered serious by the authorities, then what is likely to happen?

Even though there are no conditions provided by the property owner, an investigation would reveal conditions accepted by the beekeeping industry on public lands. Through default they would be potentially considered as an industry standard if no such standard is stated anywhere else. Thus the need to place adequate signage, warning of the presence of bee hives would be regarded as the industry standard.

A similar situation about warning signage arose at the seminar.

The question was asked about a visitor to a honey extracting shed. This visitor may be the local council metre reader. The visitor is stung, but the beekeeper has no bee hives within 100km of his shed. Obviously the bee did not belong to the beekeeper. Who then is responsible? Is it not common place for bees (not yours) to hang around your shed most of the time? The answer is yes in most cases.

As stated, bee stings can be classified as a risk to many people. We have thus identified a risk. What do we do about it? We can't stop people coming onto your property, thus we need to adequately warn people of a potential risk. A sign at all entry points to the property on which your honey shed is situated may significantly address the risk factor and thus visitors are now informed and if they choose to enter the premises will do so with the knowledge of the potential hazard. This concept has been around for many years in the building game. There are a number of specific warning signs around building sites communicating the potential risks.

Trucks transporting bee hives are another potential risk as far as stings are concerned. If you are transporting hazardous chemicals, inflammable material you are obliged by law to provide safety signs on the truck, warning people of the nature of the cargo. If you assess that transporting bee hives is a potential risk to police or the RTA who may have cause to inspect your truck, then what means do you take to mitigate this risk? A warning sign at the front and back of the truck should do the trick. Many beekeepers are currently practising this by placing warning signs on trucks when transporting bee hives.

Another example of managing risk was discussed by one of the participants at the seminar. The question asked was, what would be necessary for employees travelling and working in apiaries in remote locations. While the audience was hoping for clear defined guidelines, alas they were not going to receive them.

One of my favourite responses is that "it depends". Does the person know their way around the region? Is there mobile phone coverage at the site or destination? Is the person carrying a fully charged mobile phone? Is there a property residence close to the apiary that the person can obtain help from in the case of an emergency?

In an extreme situation it may be necessary to ensure that two persons are required to work in remote locations. If you have identified all the reasonable risks and are satisfied that they are adequately dealt with, then sending a single employee to an apiary may be justified.

The principle issue is that there are no hard and fast rules, rather you as the manager (beekeeper) have identified the risks and have taken steps to address those risks.

There are standards to follow with work hazards in specific fields. There are defined rules for working with chemicals; these include fumigants such as phosphine gas used to manage wax moth. There are set safety precautions to follow with no room for interpretation.

If you have a beekeeping operation where you employ someone on a part-time, casual or permanent basis, it is important to have policies and procedures in place for your workers in matters involving risk. This may, for instance, cover PPE (Personal Protective Equipment). As a manager you have provided gloves, jacket and veil to protect and reduce sting events. Instruct employees how to use this equipment and record in your diary or notes when this instruction was provided. Ideally the employee signs a document to indicate that they have received such instruction. In the event that the employee chooses not to wear all or some of the protective equipment then it could be argued that the employee has taken the bulk of the responsibility associated with the identified risk. In the event of employing foreign workers it may require extra attention to the delivery of the original training to ensure detail of the issue is not lost in translation.

Around 2003 the NSW Apiarists' Association Inc. produced an Occupational Health and Safety Manual which was described as a practical tool to help your apiary business implement an OHS Management System. Issues such as manual handling, long distance driving, working alone in isolated locations and bee stings were identified as potential hazards.

Much of the manual I would regard as common sense, but as has been stated to me in the past, often risks are taken, shortcuts made and accidents that should have been avoided occur with sometimes deadly results.

Perhaps it is time to revise the manual and provide the NSW beekeeping industry a clear pathway to assist in their obligations with the new Act.

Like all laws they are open to interpretation, the new Act will need to be tested in court before its full significance is understood even so ignorance is not a defence in law.

NSW DPI – COMPLIANCE OPERATIONS – APIARIES

Mick Rankmore, Regulatory Specialist, Apiaries, Gunnedah mobile: 0402 078 963 email: michael.rankmore@dpi.nsw.gov.au

Compliance operations are operations where NSW Department of Primary Industries Regulatory Officers (apiary inspectors) from the Agricultural Compliance Unit conduct compliance operations in an area. There could be up to six inspectors involved.

The objectives of these operations are twofold:

1. To provide advisory material to beekeepers on registration requirements and disease notification/ eradication and
2. To investigate compliance with, and obtain evidence of breaches, of the provisions under section 6(1) – unregistered beekeepers, section 15 – unidentified brood boxes and section 22(1) – fail to notify AFB and take appropriate regulatory action where offences are detected

The major objective is to heighten future compliance

Three operations were conducted between September and November 2011. They were conducted in the Grafton area, Leeton/Griffith area and in the Orange/Bathurst area.

The outcomes of these operations are summarised below.

Grafton
Ten apiary sites representing nine beekeepers were inspected. The owner of one site of 30 deadout hives was directed to burn all the deadout hives due to AFB being confirmed in samples taken from the dead hives. A follow up inspection revealed that the owner had moved the hives back to his residence and had started to burn the hive material.

Griffith
Seven sites representing seven beekeepers were inspected. Two beekeepers were unregistered. Written cautions were issued to each beekeeper and were provided with an application to registerer as a beekeeper. One other beekeeper is still to be located, most likely unregistered. If the owner can not be located the hives will be seized and disposed of.

Two beekeepers were issued with a written caution for failing to correctly identity their brood boxes with their registration number.

At another site, AFB was confirmed in samples taken from dead out hives. The equivalent of about 200 dead out hives were stacked up on the property. Three or four colonies (swarms that had established themselves in the dead out material) were sighted. This material may have been a source of AFB infection around the Griffith area and due to previous AFB history, the beekeeper being unregistered and his complete lack of responsibility, all the material was burnt under supervision by an apiary inspector.

Leeton
A report of neglected hives at Leeton resulted in the detection of two stolen hives amongst the five hives at the site. The local NSW Police Detectives supervised the seizure and removal of the hives to a secure location. The owner of the stolen hives confirmed ownership and he reclaimed his material. The owner of the other hives has not been established.

Orange
Five sites were inspected representing one owner. AFB was confirmed in 24 of the hives over three sites. Due to the owner's age and ill health, the hives were burnt on the owner's property under supervision of the Apiary Inspectors and the Rural Fire Service.

Bathurst
One site was inspected representing two owners. One owner had died recently and the family relocated the hives back to the family property at Gosford. There were some dead hives but no sign of AFB.

The other owner had four hives on site. The brood boxes were not correctly identified and a written caution was issued. No AFB in these hives.

Beekeepers assisted NSW DPI apiary inspectors during some of these operations with information about abandoned, neglected and/or diseased hives and with the removal of such hives for destruction.

Thank you to those who were involved.

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VALE - GRETCHEN WHEEN
1929 - 2012

Gretchen Wheen was one of Australia's best known commercial beekeepers. Early in her career, the physical demands of manipulating hives heavy with honey persuaded Gretchen to act more strategically. She soon found her niche as a supplier of quality queen bees for local and overseas markets. The work was still arduous but producing queens better engaged her sharp mind and manual dexterity.

Gretchen pioneered instrumental insemination of queen bees to complement her efforts in stock improvement, having learnt the rudiments of queen bee insemination at Hawkesbury Agricultural College from gifted apiculture lecturer, Neville Cutts. Gretchen reciprocated by teaching Neville another of her skills - how to become a potter which served him well as a professional potter in retirement.

Eager to improve her insemination skills and apply these to stock improvement, Gretchen developed strong links with leading bee researchers in Europe, including Frederich Ruttner and Tilly Kuhnert.

Recognising the need to import breeding stock from overseas, Gretchen played a key role in the establishment of the Honeybee Quarantine Facility at Eastern Creek in 1980. She was bitterly disappointed when the Howard Government sold the whole national quarantine complex for a pittance in 2001. Gretchen was prepared to fight the battle all over again for a replacement bee quarantine facility, even offering her own property for this purpose. That battle continues through the Wheen Bee Foundation she established in 2009 using her own funds and portion of the estate of her cousin and working partner, Frank Wheen.

Gretchen was deeply conscious of the link between honeybees, nature's premier pollinator, and food security. She was greatly concerned about the huge problems facing the small, but strategically important, beekeeping industry. Her commitment was such that she made a living bequest by donating her farm and other resources to establish the Foundation to promote honeybee research and protect our food supply. She was a tireless and fearless letter writer and phone caller; and she got responses from luminaries like David Attenborough, David Suzuki, Ministers, bureaucrats, researchers and journalists.

Visionary beekeeper championed food security

Gretchen early in her queen rearing days

Gretchen, the art connoisseur

Indeed, it was only days before her death that Gretchen received a reply from DAFF, on behalf of Dr Mike Kelly, Parliamentary Secretary for Agriculture, Fisheries and Forestry, defending the Department from claims of incompetence and corruption; and possibly flow-on effects over poor management of the Asian Bee Incursion at Cairns and the closure of the Eastern Creek Facilities. Gretchen's letter had been prompted by an article in the Sydney Morning Herald, citing a report on DAFF's operations from the Auditor General. Gretchen had not been impressed; nor was she with DAFF's response to her complaint.

Gretchen was a strong advocate for a Cooperative Research Centre on Honeybees and Pollination Services. She provided funds to prepare a bid for the 2012 round for CRCs. The Foundation is committed to supporting the CRC financially for 8 years if the bid is successful.

Born in London in October 1929, Gretchen was the daughter of Arthur Wheen and Aldwyth Lewers (sister of sculptor, Gerald Lewers). Both parents were Australian but lived their adult lives in the Quaker village of Jordans in Buckinghamshire. Gretchen's mother, who maintained a number of bee hives at home, shaped Gretchen's basic understanding of beekeeping.

Gretchen's father, Arthur, was a highly decorated soldier in WWI, and the 1919 NSW Rhodes Scholar. Arthur eventually became Keeper of the Library at the Victoria and Albert Museum. Arthur was responsible for the much acclaimed English translation of Erich Remarque's *All Quiet on the Western Front.*

Having been shipped to relatives in Australia for 'safe keeping' during WWII, Gretchen rejoined her parents in Jordans in 1945. But tensions caused by her mercurial mother made family life difficult. In 1951, Gretchen returned to Australia, obtaining a Bachelor of Arts Degree from Sydney University and later a Diploma of Horticulture. Collecting a swarm of bees whilst on a homeward journey from university confirmed Gretchen's interest and set her on the path to beekeeping and horticulture.

Moving from Sydney to Richmond in 1978 allowed Gretchen to expand her

beekeeping and horticultural activities. Her Hawkesbury River farm provided the ideal setting for a pecan orchard, a nursery with well over 300 old-fashioned roses and near perfect conditions for bees and a research laboratory.

Perhaps as a result of long absences from her parents, Gretchen valued her independence and guarded her privacy. Nevertheless, she responded to friends and relatives in need by offering them a place to live or work for extended periods. With the sale of artworks, she also helped others financially.

Inherited from her parents, was her appreciation of the visual arts and passionate interest in the small miracles of the natural world. The arc of a spider's web or seeds suspended in thistle down could be a source of continuing fascination, as were her collections of pods, seeds, twigs and bark twisted into sculptural shapes by the vagaries of nature.

Gretchen could hold her own with her beekeeper colleagues without compromising her charm and femininity. In recent years Gretchen's aging body could not keep up with her alert and enquiring mind. This frustrated her enormously. Although a strong supporter of Nietzche and euthanasia, she died peacefully from breathing complications in Hawkesbury Hospital on 6 January 2012.

Letters to Gretchen from her erudite father form a large portion of a recently published book: *We talked of other things: the Life and Letters of Arthur Wheen*. To Gretchen's delight, the NSW Governor, Professor Marie Bashir, used the occasion of the book launch to emphasise the importance of food security, bees and the role of the Wheen Bee Foundation.

The ABC 7.30 reporter, Rebecca Baillie interviewed Gretchen about the letters from her father: http://www.abc.net.au/7.30/content/2011/s3368372.htm
Gretchen, separated from her parents most of her life concluded the ABC 7.30 interview (16 November 2011) with the comment "The letters are a reality that I wouldn't have had otherwise".

Gretchen never married but leaves behind many cousins, friends and admirers, especially in beekeeping, horticulture and the local community. Her Foundation's motto reflects Gretchen Wheen's enduring legacy to mankind "Food Security Needs Bee Security".

Gretchen and Frank Wheen, a familiar duo in the Industry

Many thanks to Dr Max Whitten for supplying this information about Gretchen's life.

NICK'S NEWS
from DPI NSW

Nick Annand
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This article focuses on the second of five trials I researched on SHB over recent years. But first some more observations on SHB. Please let me know of your own success or failures with SHB management so these can be shared with the readers.

General SHB info

- Following on from the last article, I placed a few chux clothes in various locations within my hives. The hives did not have many SHB but yes some SHB did get caught in them, but still some SHB wondering the hive after 2-3 weeks. Other beekeepers have been reporting success reducing SHB numbers with the chux wipes. There is no need to used old clothes as mention previously as the bees readily fluff them out entangling the beetle.

Chux cloth from a Frank Malfroy hive with lots of entangled SHB

- It was noticed in my wax capping that had been left draining of honey for several weeks that some small pockets of SHB larvae infestation had developed. I had heard how the SHB larvae ball together when placed in coolrooms and appear to be able to survive. When scooping out the wax capping with my hand it was found the temperature was noticeable warmer (guessing possibly as high as 5ºC) around the infestation. This ability by SHB larvae to create heat would definitely assist their survival in cool conditions.
- When using Apithor traps be aware the bees can seal the entrances with propolis. I had one completely seal off so no SHB could enter. Yes this will prevent them working.
- Reports of some big losses west of the range where two wet summers in a row seem to have amplified the SHB problems.

Trial 2

My report here is an abbreviated version. For more detail please go to the RIRDC web site https://rirdc.infoservices.com.au/items/11-044 to get the complete project 'Small Hive Beetle Biology – Producing control options'.

Aim

To compare the attractiveness and susceptibility of strong, weak and queenless hives to SHBs.

Anecdotal reports from beekeepers have often referred to queenless hives and weak hives being very susceptible to the ravages of SHBs. This part of the study was to examine if this is indeed the case. Knowing this would inform beekeepers if preventative management strategies to reduce such situations are necessary.

Methodology

Hive preparation commenced in spring 2007. All colonies were requeened with marked sister queens and located to Bathurst where favourable conditions enable good hive build up. Low SHB numbers at Bathurst minimised the opportunity for SHB infestation. 27 hives were used for the trial with 9 hives being randomly allocated for each treatment (strong, queenless or weak). All the hives had a brood box, queen excluder and one honey super throughout the trial.

Two weeks prior to the trial beginning, to reduce the strength of the hives allocated to be weak, hives were split in half. Any capped honey in the supers of these hives was also extracted prior to the trial. One week prior to the trial beginning the hives selected to be queenless had their queens caged and returned back to the hive. This was to minimise attempts to replace her, as her pheromones would still be present. Eight days later during the first trial inspection the queens were then removed from the colonies and any queen cells found during the inspection were removed. The strong hives were left untouched

3.3-2: Trial activities

The hives were inspected and assessed at 3 stages (start, midway and end) over the 10 week trial which start 29/1/08 and ran to 16/4/08. To establish the differences between the hives allocated to each the three categories of strong, weak or queenless, several key indicators of hive health were recorded throughout the trial interval. These included:

- areas of worker brood,
- drone brood,
- honey and pollen,
- frame weights for the brood box and super
- returning flying bee counts and the
- weight of extracted honey.

During the initial examination, hives were inspected for SHBs and any found were removed. SHB numbers were very low. Immediately after the first inspection all hives were relocated to the apiary yard at the University of Western Sydney, Hawkesbury campus where a high endemic SHB population existed. Hives were randomly placed into two rows in a predominantly shaded area (Figure 1).

Figure 3.1. Strong, queenless and weak hives in location at University of Western Sydney's, Hawkesbury Campus

The same data was again collected midway and at the end of the trial. The only difference was that for the mid-way assessment SHBs were collected, counted and returned under the lid of the

same colony no earlier than two hours after the inspection. This provided time for the bees to re-establish some order within the colony.

Using a pooter, (a devise to suck up and capture the beetles) SHBs were aspirated up from each hive. The collected SHB from each hive were kept separate in ventilated labelled containers and later that day chilled to facilitate counting before being returned to their hive of origin.

To calculate the areas of brood, pollen and honey in the brood box digital photos of every frame were taken. Using a computer the photos of each comb were then divided into the components (pollen, capped honey, drone brood etc) and the areas calculated.

Much of the data collected and analysed was to establish differences between the strong, weak and queenless hives and to determine whether any hive condition was associated with increased SHB susceptibility.

Results

Changes in seasonal conditions need consideration while looking at the results. The start of the trial was very wet with 180.4 mm recorded in the first 10 days of February 2008 at the University campus. The bees came off high-quality breeding conditions at Bathurst with good pollen loads. For the first half of the experiment surplus nectar was available. In the second half conditions deteriorated with limited nectar available for the bees. As a result, at the end of the trial during hive examinations bees were observed robbing the opened colonies.

At the mid-point examination, three of the "queenless" colonies had re-queened. The data from these hives was removed, leaving only six queenless colonies in the experiment.

Many records were taken but for this article (to conserve space) only two graphs are used that clearly show and represent the hive differences, those being capped worker brood area and returning bee counts. This allows focus on the impact the varying hive conditions have on SHBs and not on the difference between the hives.

The main observation from the graph below is the absence of worker brood in the queenless colony at the midway point of inspections and at the end. However the drone brood area (graph not shown) for the queenless colonies was over 6 times greater than that of the other hives midway due to laying worker bees developing.

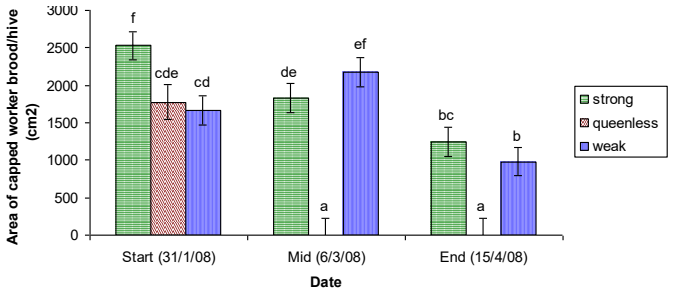

Figure 3.2. The predicted mean area/hive (cm^2) of capped worker brood in strong, queenless and weak colonies as observed at the start (31 January 2008), mid (6 March 2008) and the end (15 April 2008) of the trial. Bars represent the standard errors of the means and LSD (P ≤ 0.05) ranking is shown by the letters above the bars.

On four different occasions the returning bees were counted through observation. Counting the returning bees was done without disturbing the hive allowing for more regular assessments. Poor weather was experienced for the counts conducted on the 6 February 2008. Apart from the initial count, the strong hives had more returning bees, while numbers for the queenless colonies declined relative to the other hives.

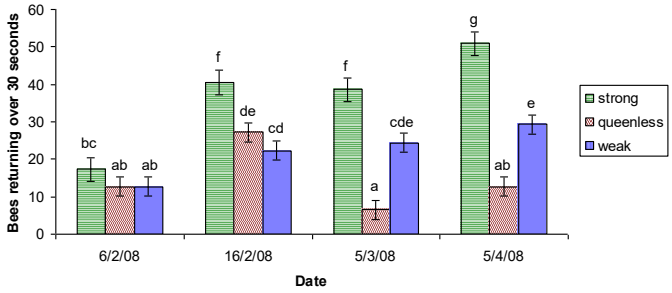

Figure 3.3. The predicted mean number of bees/hive counted returning into strong, queenless and weak colonies over one to three 30 sec periods on the 6 February 2008, 15-16 February 2008, 5 March 2008 and 5 April 2008. Bars represent the standard errors of the means and a LSD (0.05) ranking is shown by the letters.

As the experiment proceeded significantly more SHB were being found in all the hives. The *hive condition* showed that the strong hives had significantly more SHBs than the weak hives at both the mid and end inspections. The position of the hive in the apiary did not significantly affect SHB numbers in the hives

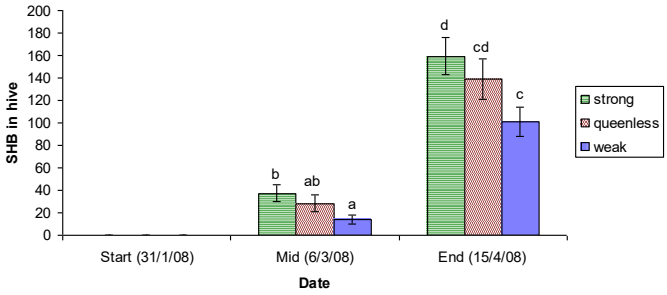

Figure 3.4. The predicted mean number of small hive beetle/ hive counted in strong, queenless and weak colonies at the start (31 January 2008), mid (6 March 2008) and the end (15 April 2008) of the trial. Bars represent the standard errors of the means and a LSD (0.05) ranking is shown by the letters.

Some SHB larvae were found during the mid inspection (5 March 2008) between two full frames of honey in the honey super of a strong colony. An area of approximately 7 x 7 cm was affected and showed signs of 'sliming' (Figure 3.5). Only 15 adult SHBs were within that colony at the time when the damage was observed.

Figure 3.5. Area infested and slimed by SHB larvae at the mid point inspection when two honey frames were close preventing bee access in a strong colony.

At the final examination, all six queenless hives had extremely low bee populations, as shown by the counts of returning bees, but all still had bees living and working in them. Queenless hives were the only ones to have SHB larval infestation at the trial end. Out of the six hives, four had signs of early stages of larval infestation. For three of these hives, larvae were still small, bees were still present and no damage to the combs or fermenting honey was evident. In the other infested hive, larval damage

was more advanced with larger larvae present and bubbles in the uncapped honey cells indicating that fermentation was occurring (Figure 3.6). The few remaining bees had yet to be driven out of the hive by the larval damage.

Figure 3.6. One of the queenless hives at the end (15 April 2008) infested with SHB larvae and honey bubbling in the cells.

Conclusion

The results indicated significant differences between the strong, queenless and weak hives throughout the trial period. It was found that:

- There were significantly more SHBs in the strong hives than the weak hives. This suggests that strong hives are more attractive to SHBs for some, as yet undetermined, reason. Other researchers have found no observable differences in SHB numbers in hives as a result of variation in phenotype (number of bees and amount of stores) but large, unexplainable variation in SHB numbers between colonies alongside each other have been observed (Spiewok *et al.*, 2007; Neumann *et al.*, 2010). There has been speculation that an aggregation pheromone is involved (Spiewok *et al.*, 2007; Neumann *et al.*, 2010) but none has been identified

- Contrary to this, however, SHB numbers in the queenless colonies were similar to those in the strong hives at both the mid- and final observations when the former hives were quite weak. The queenless colonies developed large pollen reserves because they had no bee larvae to feed. It is possible that with limited bees to keep guard SHBs were able to access the pollen stores possibly contaminating them with *Kodamaea ohmeri,* a yeast SHB carry and appear to have a symbiotic relationship with. Should this have been the case (which was not confirmed), the release of isopentyl acetate (IPA), a volatile produced by the yeast which has been found to be attractive to SHB, may have attracted more SHBs (Torto *et al.*, 2007).

- Results from this trial support the conclusion of Spiewok *et al*. (2008) that the queen status of a colony does not innately make it more attractive to SHB adults or more vulnerable to larval damage. However the data does contradict anecdotal evidence from beekeepers who suggest that queenless colonies are much more susceptible to the ravages of SHBs.

- The results suggest that when SHB populations in a hive are low to moderate, colonies become vulnerable to SHB larval damage once there are insufficient bees present to protect the colony. Four out of six queenless colonies had larval infestations at the end of the trial when bee numbers were extremely low. If beekeepers are proactive and check regularly for queen activity and respond immediately when required by removing, joining or compressing the high risk colonies and requeening as soon as possible, colony losses to SHBs might be reduced. These management strategies may also help curtail SHB pressure on remaining colonies.

- SHB damage essentially only occurred in queenless hives. The exception was in one of the strong hives at the trial mid-point where bee space had been lost between two frames of bulging combs. This gave the SHBs a bee-free area for their eggs to hatch and larvae to develop even though only 15 SHBs were in the hive at that time. Care when returning these combs ensured bee access over the whole frame. When extracting honey on the 18 March 2008, no evidence of SHB larvae or honey spoilage was found in that hive, demonstrating that strong colonies are capable of cleaning and repairing small areas of SHB damage, provided they can access them. This demonstrates the opportunistic breeding habit of SHBs when suitable situations arise. When requeening, for example, queen cages placed between frames can create such an environment for SHB. Anecdotal reports tell of beekeepers having new replacement queens die in the cage as a result of such SHB activity.

- Across all colonies, SHB numbers increased during the trial. This trend is consistent with the observations of other researchers that SHB numbers increase in colonies throughout summer and into autumn and peak around the end of autumn (de Guzman *et al.*, 2010; Frake *et al.*, 2009). Similar findings were observed in trials 3 and 4 that will be presented in later Nick's News articles.

Implications

Data obtained from this trial did not provide conclusive evidence that SHBs are more attracted to queenless or weak colonies but indicated that inadequate bee numbers or bee protection can expose the colony to SHB larval damage. The implications are that beekeepers need to act as soon as they identify a problem of this nature. By being proactive and removing, joining or compressing the high risk colonies as soon as possible colony losses to SHB can be reduced. This will allow time and monetary saving for the beekeeper. It also helps curtail the SHB population therefore reducing the SHB pressure on remaining colonies.

However, there are many other factors suspected of making colonies susceptible to SHB damage. These include hive manipulation, disease, making up nucleus hives, requeening, hot weather and excess full honey supers left on colonies.

These factors need to be fully investigated to obtain a better understanding of what makes hives vulnerable to SHBs. These factors need to be fully investigated to obtain a better understanding of what makes hives vulnerable to SHBs. Hopefully, then we can develop management approaches to minimise SHB-related colony losses.

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IS THE IMADICLOPRID THE CAUSE OF COLONY COLLAPSE DISORDER?

ApiNews from Canada - 1 February 2012

This article explains the investigation made by specialist Jeff Pettis

Honeybees are sensitive creatures. From time to time, a hive simply gives up the ghost and vanishes. Colony collapse disorder, as this phenomenon is known, has been getting worse since 2006. Some beekeepers worry that it may make their trade impossible and could even have an effect on agriculture since many crops rely on bees to pollinate them.

Climate change, habitat destruction, pesticides and disease have all been suggested as possible causes. Nothing, though, has been proved. But the latest idea, reported in Naturwissenschaften by Jeff Pettis of the Bee Research Laboratory in Beltsville, Md., suggests this may be because more than one factor is involved.

Pettis and his colleagues knew from previous reports that exposure to a pesticide called imidacloprid has a bad effect on honeybees' ability to learn things and wondered whether it might be causing other, less noticeable, damage.

Since one thing common to colonies that go on to collapse seems to be a greater variety and higher load of parasites and pathogens than other colonies, they wondered in particular whether it might be weakening the insects' immune systems, and thus allowing infections to spread through a hive.

To find out, they gave 20 hives protein food (a substitute for pollen, which is fed to developing larvae) that had been spiked with imidacloprid. In 10 cases the dose was five parts per billion; in the other 10 it was 20 parts per billion. Previous experiments have shown that neither dose perceptibly harms bees.

A further 10 hives were given unspiked food as a control. Then, when the young bees emerged a few weeks later, Pettis collected them and fed them with spores of a fungal parasite called Nosema. Twelve days later, he killed them and estimated the extent of their infestation.

Both of the groups that had been exposed to imidacloprid harboured an average of 700,000 parasite spores in each bee. Bees from the control colonies, by contrast, harboured fewer than 200,000 spores in their bodies. The insecticide, in other words, was exposing bees to infestation, and thus to a much greater chance of dying prematurely.

Whether this is actually the reason for colonies collapsing remains to be determined. But it is a plausible hypothesis and is likely to get beekeepers buzzing with interest.

IS THIS PARASITIC FLY RESPONSIBLE FOR BEE DEATHS IN USA?

An *Apocephalus borealis* fly implants its eggs into the abdomen of a honey bee *Photo: AP/San Francisco State University*

Scientists in Northern California believe they have found a possible explanation for the honey bee die-off: a parasitic fly that hijacks the bees' bodies and causes them to abandon hives.

The symptoms mirror colony collapse disorder, in which all the adult honey bees in a colony suddenly disappear. The disorder continues to decimate hives in the US and overseas.

The disease is of great concern, because bees pollinate about a third of the United States' food supply. Its presence is especially alarming in California, the nation's top producer of fruits and vegetables, where bees play an essential role in the $US2 billion ($1.9 billion) almond industry and other crops.

The latest study, published in the science journal *PLoS ONE* on 3 January, points to the *Apocephalus borealis* fly as the new threat to honey bees. It is another step in continuing research to find the cause of the disease.

Researchers have not been able to pin down an exact cause of colony collapse or to find a way to prevent it. Research so far points to a combination of factors including pesticide contamination, a lack of blooms - and hence nutrition - and mites, fungi, viruses and parasites.

Interaction among the parasite and multiple pathogens could be one possible factor in colony collapse, according to the latest study by researchers at San Francisco State University. It says the phorid fly, or apocephalus borealis, was found in bees from three-quarters of the 31 hives surveyed in the San Francisco Bay area.

Scientists say the fly deposits its eggs into the bee's abdomen, causing the insect to walk around in circles with no apparent sense of direction. The bee exhibits zombie-like behaviour, said lead investigator John Hafernik. The infected bee leaves the hive at night and dies shortly thereafter.

The combination of a parasite, pathogens and other stressors could cause die-off, Professor Hafernik said. The parasitic fly serves as a reservoir that harbours pathogens - honey bees from parasite-infected hives tested positive for deformed wing virus and other pathogens, the study found.

"We don't fully understand the web of interactions," Professor Hafernik said. "The parasite could be another stressor, enough to push the bee over the tipping point. Or it could play a primary role in causing the disease."

Professor Hafernik stumbled on to the parasitic fly by accident. Three years ago, the biology professor looked for something to feed a praying mantis. He found some bees outside his classroom, placed them in a vial and forgot about them. When he looked at the vial a week later, he found dead bees surrounded by small fly pupae. A parasitic fly was feeding on the bees and had killed them, he said.

The fly is a known parasite in bumble bees. Scientists used DNA barcoding to confirm the parasite in the honey bees and bumble bees was the same species.

The fly might have recently expanded its host presence from bumble bees to honey bees, Professor Hafernik said, making it an emerging threat to agricultural pollinators.

The fact that honey bees live in large colonies placed in close proximity to one another and beekeepers frequently move the hives throughout the country could lead to an explosion of the fly population, he said.

The fly, which is found all over North America, could also become a threat to native bees.

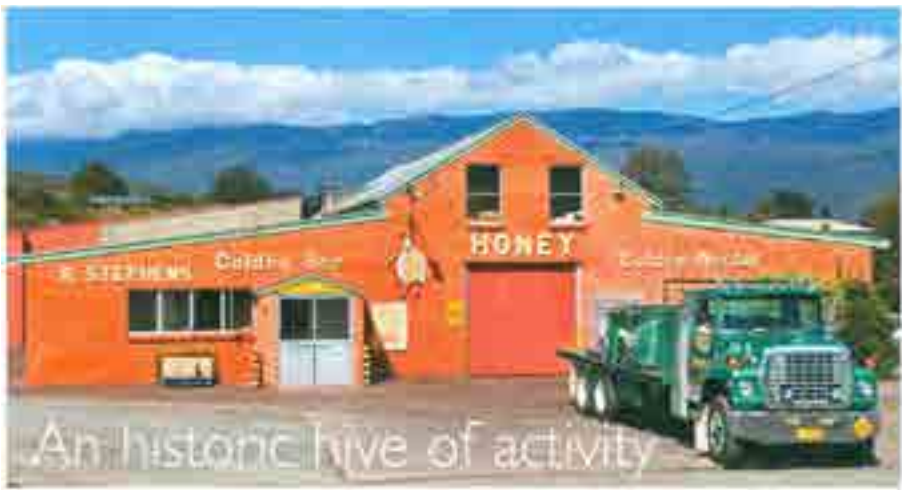
Professor Hafernik plans to expand his research to other parts of the country and to study the parasite's impact on agriculture in California's Central Valley.

Since it was recognised in 2006, colony collapse has destroyed colonies at a rate of about 30 per cent a year, according to the US Department of Agriculture. Before that, losses were about 15 per cent per year from a variety of pests and diseases.

R STEPHENS HONEY FACTORY GAINS HERITAGE LISTING IN TASMANIA

The R Stephens Honey Factory was recentlyenteredinthe Tasmanian Heritage Register because of its extraordinary history.

Chairperson of the Tasmanian Heritage Council Michael Lynch says that the listing was certainly one of the more unusual ones he had seen, but it was an important and fascinating part of the Tasmania's historic heritage.



In 1930 the iconic 'Golden Bee' label was born. In the summer of 1934 bees were transported to the Mt. Arrowsmith area of the West Coast to gather the delicious honey of the native Leatherwood flower, found only in the Tasmanian wilderness. Owing to bad roads, poor transportation and the Second World War, Robert did not return to that area until 1951.

"It's terrific to think that one Tasmanian family exports this internationally renowned honey to the world, from a site occupied since 1920," Mr Lynch says.

The registration includes the honey factory, apiary, nuclei (where the queen bees are bred) and residence.

"The place is like a living museum. Everything is done on site - the breeding of the bees, maintenance of the meticulous records, packaging of the honey and marketing the product. The original beekeeping paraphernalia is still there.

"The Heritage Council included the Stephens family home in the listing too, as it was designed by notable Australian architect lliffe Gordon Anderson who was a friend of the family. He also designed the Golden Nectar Real Leatherwood Honey label in the 1950s, which is still in use today;' Mr Lynch says.

The original Golden Bee Honey label launched in the 1930s is also still used, and Edith Stephens (Robert's wife) assisted with its design.

Mr Stephens says he's delighted that Golden Bee Honey Factory is now permanently entered in the Heritage Register. "It's a great honour. Our history is now recorded forever, and that is just wonderful."

The R. Stephens Apiary at Mole Creek was established by the late Robert Stephens in 1920, and has grown from a pre-World War 1 hobby of a few hives to 2400 hives today, producing approximately 35 per cent of Tasmania's honey crop.

The history and growth of this family business, now into its third generation, is very much a part of Mole Creek's progress.

Robert Stephens was the eldest son of the local Methodist Missionary, who tended the original hobby hives at the back of the Church House while his son served in the First World War. Upon returning in 1919 Robert turned the hobby into a living, purchasing the two acres where the factory and apiary now stand in the picturesque village.

In 1923 fifty hives were moved to neighbouring Caveside by horse and cart. In 1929-30 an A Model Ford truck was purchased and two men were employed to work 450 hives. This produced a bumper crop, only to result in very poor sales because of the onset of the Great Depression.

Retail outlets were then established for this unique honey and its distinctive 'Golden Nectar' and the famous 'Real Leatherwood Honey' label was designed. Like the Golden Bee label, it is still in use.

In 1966 Robert's son Ian took over the business and updating and expansion began The process continues today alongside his wife Shirley plus their three sons Ewan, Neal and Kenneth, who are all multi skilled, fully qualified apiarists. Ian remains administration /sales manager, Shirley is in sales, Ewan is production manager, Neal apiarist and wax production specialist, and Ken the Queen breeder and sales.

Both Ian and Shirley have been extremely active and influential in the Tasmanian honey industry for decades, and like Robert before them have been made Life Members of the Tasmanian Beekeepers Association.

Today the company specialises in two varieties of Tasmanian honey: Golden Bee Clover/ Blackberry & Ground Flora Honey and Golden Nectar Real Leatherwood Honey. The latter is now certified organic and is sold around Australia in Woolworth's supermarkets and various health, gourmet and natural food outlets, as well as tourist shops and supermarkets in Tasmania. Ever expanding shipments are made to the USA.

Seventy per cent of the company's leatherwood is harvested from the wilderness catchment area of the Franklin River, which the Stephens family visits every summer via rough bush tracks. These high rainfall forests are entirely free from insecticides and artificial fertilisers, ane men work the bees during the honey flow. The honey is returned by a fleet of distinctive green Ford trucks to Mole Creek where another crew uses three 70-frame radial extractors and an automatic uncapping machine to extract the honey, extracting five to six tonnes of honey per day.

The all stainless steel factory equipment, storage tanks and honey bottling and labelling machines are the most up to date and efficient for a factory of its size in Australia.

But some of the equipment hand-made last century by Robert Stephens is still in use, such as nuclei, hive boxes, drawers and stools, much of it constructed from old petrol boxes.

For more information visit:
www.leatherwoodhoney.com.au

SICK BEES

by Randy Oliver - ScientificBeekeeping.com
First published in American Bee Journal September 2010

PART 6

INFECTIONS BY MULTIPLE VIRUSES

We beekeepers hear from researchers that our sick bees are full of viruses. Understandably, we want to know what we can do about it. But to most of us, virus infections are a "black box"—a generally invisible, mysterious phenomenon about which we can do little other than to control one of the modes of transmission (via varroa). Our questions to those very researchers are often frustratingly met by more questions. I can assure you that researchers have been working hard to come up with the answers—we gave gained a great deal of understanding these past few years. As Marie Curie once observed: "One never notices what has been done; one can only see what remains to be done."

Our understanding of viruses and disease has expanded greatly within the lifetime of modern bee researchers. In 1925, the great cell biologist E.B. Wilson wrote: "The key to every biological problem must finally be sought in the cell." But when Wilson wrote those words the world inside the cell was largely inaccessible. The primary instrument for investigation at the time--the light microscope--was physically incapable of resolving a cell's finer interior details. The situation encountered by Wilson and his contemporaries could be compared to that faced by astronomers, "who were permitted to see the objects of their interest, but not to touch them; the cell was as distant from us as the stars and galaxies....[1]

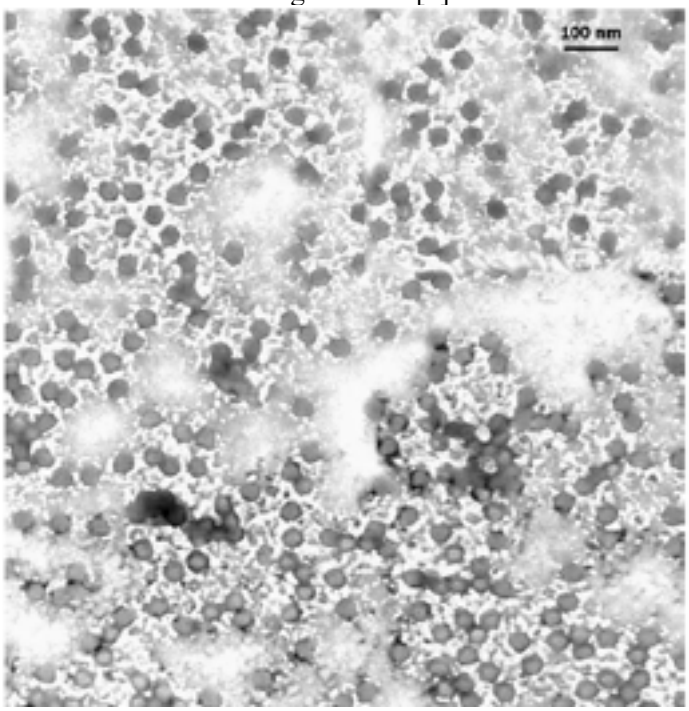


Figure 1. An electron micrograph of a mixed infection of four different bee viruses, which are essentially indistinguishable. What you see are the round virions, which are the end product of an infection. From Chen, et al 2004.

The development of the electron microscope finally allowed scientists to look deep into the cell—the first virus was directly viewed in 1939 (Fig.1). It wasn't until 1952 that viruses were first grown in cell culture, finally enabling scientists to isolate strains and observe the cellular effects of infection.

Another huge leap forward occurred in the 1980's when researchers learned how to amplify DNA by polymerase chain reaction (PCR). This set the stage for in-depth study of the genome; concurrently, the field of epigenetics--the study of the cellular and environmental factors that affect how the genome is expressed--took off. ***In a mere fifty years, the study of virology went from the macro, to the micro, to the molecular level!***

The scope of advancement is apparent in bee research. Most known bee viruses were first isolated and identified (beginning in 1963) by the great bee pathologists Leslie Bailey, Brenda Ball, and RD Woods. They used crude electron microscopy and antisera painstakingly made by injecting viruses into rabbits. Brenda Ball is still active in the field of bee virology, but today has at her disposal such new technologies as real-time PCR, mass spectrometry, and rapid microarray probes. It is amazing to grasp the sheer scope of advances that have taken place in a single career!

Infection by Viruses

Author's note: I apologize in advance for the rest of this article lacking direct practical applications. However, it is material that I need to cover in order to give you the necessary background before I move on to my next subject, which I suspect will be of more practical interest to beekeepers. That will be an explanation of why we are seeing so many problems in our bees since the arrival of varroa, and in what ways varroa completely changed the dynamics between viruses and bees.

In my previous article in this series, I gave a brief overview of the bee immune response to viruses (you may wish to review the November ABJ before going on). When a virus infects a bee, one of the first things that it does is to trick the bee into producing proteins that suppress various aspects of that bee's immune response. These immune suppressors may be specific for that virus species, or they may be more generic. So the question then, is what happens in a multiple infection, with each virus producing different immune suppressors? One might assume that the combined effect would invariably lead to virus synergy, but that doesn't appear to always be the case.

The dynamics of virus-virus interactions are complex and poorly understood. Certain bee viruses may indeed enhance the virulence of other viruses [2, 3]. On the other hand,

however, some bee viruses competitively suppress the multiplication of others--KBV suppresses the replication SBV and BQCV [4]; ABPV interferes with replication of CBPV [5]; and Bromenshenk's [6] data indicate that the iridovirus suppressed DWV (KBV = Kashmir Bee Virus; SBV = Sacbrood Virus; BQCV = Black Queen Cell Virus; CBPV = Chronic Bee Paralysis Virus).

Thus, once a bee is infected by a virus, and that virus's immune suppression proteins take effect, then that may either open the door for other viruses, or inhibit them. Seeing as how bees in collapsing colonies are often infected by multiple viruses [7], it looks as though viruses tend to gang up on stressed bees and create serious mayhem.

In some cases an infection by a second virus may cause a dormant virus to start replicating, similar to the way in which a human infected by the flu virus may experience an outbreak of cold sores--virtually every adult human carries herpes virus as an inapparent (not exhibiting symptoms) infection, but you don't get cold sores unless you become stressed, or contract another virus infection.

I've mentioned the word "stress," which is a rather generic term. In bees, "stress" may be due to chilling, nutritional deficiency, environmental toxins, or infection. The most insidious form of stress is the targeted suppression of the normal bee immune response by pathogens, notably viruses. This is what makes Human Immunodeficiency Virus (HIV) so devastating—it directly targets one's immune response, eventually allowing opportunistic pathogens to run rampant, resulting in the syndrome called AIDS (Acquired Immune Deficiency Syndrome). CCD is remarkably like AIDS (CCD could just as well have been called Bee AIDS). And as in human AIDS, in bee collapse events several normally suppressed viruses may explode into active infections.

A number of researchers have found that the mere action of a varroa mite feeding upon a bee may induce or activate the replication of inapparent and normally non pathological virus infections. This physical insult, along with the injection of immune suppressants by the mite [8, 9], and the resultant depletion of bee body protein all contribute to the sort of "stress" that makes varroa-parasitized bees much more susceptible to overt virus infections.

So it may well be that the combination of varroa immune suppression, coupled with additional immune suppression by viruses could throw the bees' immune response for a loop. Add to that nutritional stress and the energy- and nutrient-robbing effects of a nosema infection, and ***you could have colonies at the tip point for the initiation of collapse, even though a cursory inspection of the hives would not indicate that anything was amiss!***

The bottom line is that we still have a great deal to learn about virus/virus interactions, and more importantly, the complex effects of virus suppression of the bee immune system, especially when more than one virus (and varroa) are involved. Allow me to digress for a moment, after which I will return to this subject.

Adaptive Evolution

Adaptation is the heart and soul of evolution. *Niles Eldredge*

Adaptation is the ongoing process of adjustment of the fitness of an organism to its environment.

A trait is ***adaptive*** if it enhances the probability of an organism surviving and reproducing.

The evolution of life forms generally progresses at a relatively sedate pace—over thousands and millions of years. To understand why, imagine taking a perfectly-running complex machine, and randomly changing one part. In general, any random change is going to make the machine run less efficiently. A similar situation exists for all complex organisms—they are the culmination of millions of years of fine tuning (by adaptation) that has resulted in a bunch of parts, elaborate mechanisms, and complex chemistry that must all work in perfect harmony. Any change in any part or process is likely to be nonadaptive. And if a change turns out to indeed be adaptive, then generally a number of other parts or processes must then also change in order to fine tune the modified system. As a result of this restraint on random change, organisms are "cautious" about passing genetic or epigenetic changes to their offspring, as such changes, if nonadaptive, could result in the parent's germline not surviving into the next generation. This scenario can be applied to all organisms, including parasites.

Viruses, on the other hand, are unlike any other bee parasite, in that they are not really living organisms. Rather, they are (very successful) mindless packets of instructions that depend upon the bee ribosomes to act as copy machines. But that very copying process is very unlike that of a an office copier—any copy may well contain a rewriting of those virus instructions in the form of insertions, deletions, or recombinations of specific instructions, or "collating" errors in that the "pages" (genetic information) of different documents might get randomly inserted into another document (the virion being the final "document").

RNA viruses are notable for not having any correction mechanism for such random copying (or collating) errors. They can afford to be frivolous in this aspect of their reproduction, since any successful virion, rather than producing only a few offspring, may produce ***millions***! And if some of those offspring are nonadaptive, little is lost. However, if the rare virus mutation happens to be more adaptive, then it may immediately start competing with its "normal" brethren, even in the same cell and same bee! (I strongly suggest that the interested reader download (free) Mike Carter and Elke Genersch's review [10] of the genetics of bee viruses).

The viruses themselves are mindless, and have no plan, strategy, or agenda. Those instructions that are successful at getting replicated into the next generation of bees are perpetuated; those that are either unsuccessful at infecting a bee, ***as well as those that are so virulent that they kill the bee before being transmitted to the next bee (or colony) go into nature's wastebasket*** (remember this very important point).

The error-prone virus replication process has major implications, as it endows the viruses with enormous genetic "plasticity," and therefore, the unique ability to quickly adapt to new situations (such as the arrival of varroa, or to any stressor that affects the host bee). We normally think of evolution of a species as occurring over the timescale of tens of thousands of years. Not so with RNA viruses—***within mere days, within a single bee pupa, an RNA virus can mutate into a vast pool of novel forms.*** What then exists in that pupa is a "quasispecies" or "viral swarm" of genetically distinct, although closely related, variants of the virus. So any RNA virus "species" can be more accurately described as a "cloud" of mutants distributed around a generic, well-adapted infectious form (***that normally doesn't kill the bee***)(Fig. 2).

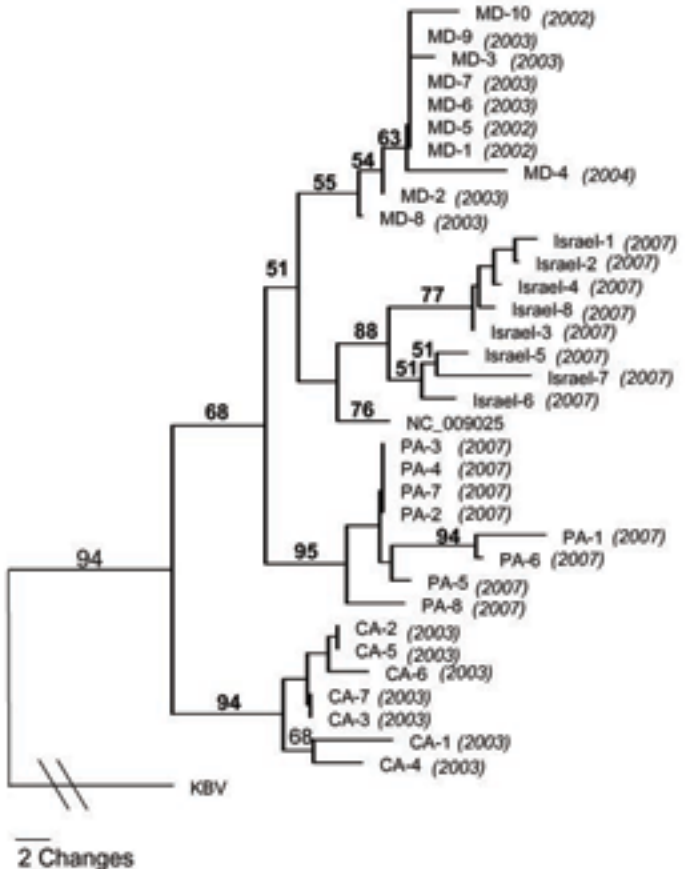


Figure 2. A phylogenetic tree illustrating the evolutionary divergence of strains of Israeli Acute Paralysis Virus from CA, MD, PA, and Israel. Note the different strains in each geographical region, and IAPV's evolution from a KBV ancestor. Recent research indicates that there were two main introductions of IAPV into the U.S. (East/Canada and West/ Australia), and that the Israeli strain is different than either [20]. Figure from Y. Chen & J. Evans, USDA ARS [19].

Now multiply that rapid evolution by every infected bee in the hive! Any particular mutant might be better at infecting a certain bee tissue, avoiding the bee immune response, or be more successful at being vectored by, or reproducing in, varroa mites. Thus, any one of the myriad new forms of the virus has the potential of starting the next virus epidemic in the local bee population! ***Since the "local" bee population nowadays is homogenized via the migration to almond pollination in California, it is no wonder that new virus epidemics can soon reach every corner of the continent*** (and also why beekeepers are often cautious about who they set down next to).

Viruses allow us an opportunity to observe the equivalent of "evolution on steroids." Even during a single season, there is a progression of factors that favor or handicap any of the myriad viral mutations—temperature, nutritional abundance or stress within the colony, rate of growth of the bee population, and mite levels. During spring buildup, for instance, more virulent virus forms are favored, due to the rapid turnover of the bee population. I will return soon to how this affects the dynamics of virus epidemics in bee populations.

Virus Infections are Complex

Virus replication, as I've explained, is not merely about cloning an original set of genetic instructions; rather RNA viruses are successful by virtue of their constant evolution in ways that you wouldn't have dreamed of!

In an infected cell, some parts of the virus RNA strand may replicate into incomplete virus forms. These "defective" virus strands may not be infective in their own right, but may still be able to sneak into newly forming virus capsids (the protective "shell" of a virus) and thus be transmitted to new hosts [11]. In the new host, the defective viruses may be able to continue to replicate by utilizing proteins created by "intact" viruses. These "defective interfering" virus segments may actually suppress the virulence of intact viruses, or may flood the host cells with virus proteins. This makes me wonder what role varroa plays in transmitting not only intact bee viruses, but in also transmitting these defective elements.

Or maybe something else is also taking place: "Co–infection by multiple viruses affords opportunities for the evolution of cheating strategies to use intracellular resources" [12]. The "defective interfering" virus strands can evolve into what are known as "cheater" or "satellite" viruses—viruses that cannot infect or transmit on their own, but are able to do so only when another "helper" virus is present, by "stealing" some of the helper virus's proteins. An example of the above is Chronic Paralysis Virus Associate (CPVA), which requires the presence of CBPV in order to replicate [13]. These defective viruses can then alter the susceptibility of the host to the intact virus by activating or inactivating the host immune response.

So viruses can act as vectors and accomplices for rogue self-replicating proteins (ever looked into prions?) or defective RNA segments. Can it get any weirder than this?

It Gets Even Weirder

This whole discussion is starting to sound like the Twilight Zone, but I just can't stop! ***An intriguing possibility is that a mixture of several defective viruses in combination could mutually complement each others' defects, and thereby productively infect cells even in the absence of an intact virus*** [14]. Indeed, López-Ferber [15] found that a co infection of both a defective virus and an intact virus can be more pathogenetic that the intact virus alone!

Or one virus's transmission proteins (that allow it to infect additional cells) may act as "chaperones" for the RNA of another virus. ***This transmission function is of great interest in understanding Deformed Wing Virus (DWV), especially why it normally appears to be relatively benign, but sometimes multiplies wildly in the wings and other tissues*** [16, 17] (Fig. 3).

Figure 3. Most bees today test positive for Deformed Wing Virus (DWV), but show no symptoms. However, when varroa infestations get out of hand, as evidenced by the white guanine deposits in the tops of these cells, DWV can explode into a devastating epidemic, as evidenced by bees with shriveled wings, or simply too weak to emerge from their cells. Photo by the author.

Virus Hybrids

The bee picornavirus genomes are composed of "functional modules," some encoding "structural proteins" (those used to build the virus "shell"—the capsid), and "nonstructural proteins" (which include the enzymes, micro RNA's, and other proteins involved in the hijacking of the host cell ribosomal and immune functions). What has been recently discovered, is that these functional modules are able to evolve independently of each other, and even more important, ***are interchangeable between closely-related viruses!*** Moore/Ryabov [18] found that in a co-infection of DWV and Varroa Destructor Virus-1 (VDV-1) that ***over the course of infection that the main forms of virus produced in the bees were hybrids ("recombinants") between the two viruses!***

So depending upon the primers used by researchers to identify viruses in bees, they may not even recognize that they are dealing with a virulent hybrid! The Moore/Ryabov team's paper deserves further elaboration, since it, along with groundbreaking work by the great Israeli virologist Ilan Sela, and intriguing studies by Galacias and Genersche, may have opened a window for us to better understand the dynamics of bee viruses and varroa.

Why would hybrid forms of viruses be more successful at infecting a bee? It appears to boil down to the role of varroa in virus transmission. In order for a virus to multiply in a bee, it must first "recognize" the tissues of certain cells to attempt to gain entry, then avoid or suppress the cellular antiviral response, and finally to manage to be transported (viruses can't move of their own accord) to uninfected bees. Sometimes a hybrid appears to do some aspect of the above more effectively.

What the authors suggest is that the VDV-1 hybrid may be better at being vectored from bee to bee by the mite, and then the DWV component is then more successful at replicating within the bee. Alternatively, by incorporating VDV-1 RNA, the hybrid may be able to escape the bee RNAi defense against DWV.

This brings up another whole subject—some bee viruses (DWV, KBV) are able to infect other species of bees and wasps, and possibly varroa gut cells. The viruses will quickly evolve to better suit the new host. But what happens when those viruses then reinfect a honey bee? Will the adaptation to the other host then make them (initially) more pathogenic to the bee? And does this go back and forth with each mite-to-bee and bee-to-mite transmission of the virus (assuming that a virus replicates in the mite [16])?

Bee-Virus Co evolution

If you're still with me, I commend you! I'd like to return now to the adaptive evolution of bee viruses, as I feel that the study of this will help us to understand why bees are having such a hard time these days.

Professor Ilan Sela of Israel has been on the cutting edge of this field of research, first in plants, and lately in honey bees. He surprised bee virologists when he first discovered that honey bees in Israel had incorporated IAPV sequences into their own genome, thus conferring them with resistance to the virus [11].

Professor Sela explained to me:

"What we have found is that about 3-10% of bees infected with IAPV are acquiring viral sequences…into their genome. Some of these become virus resistant. However, when we did a field survey (in Israel) we found that about 30% of the bees carried integrated viral sequences, and I suspect that the proportion is increasing. We also found that over 70% of bees in a large USA apiary which suffered significantly from CCD in the last 2-3 years carry viral sequences.

"As to the general question, integrated sequences that are not stabilized within the genome (these are the majority of occurrences) will gradually disappear, and at most (only if you look specifically for them) will be categorized as "junk" DNA, with no evolutionary importance. However, the infrequent occurrences in which the viral sequences have been stabilized (and we have parameters for these) may bring about new phenotypes and might have a great effect on evolution."

These bee/virus combinations are called "chimeras" (after the mythical lion/goat/serpent creature). But that's only the half of it; ***the sequence exchange is reciprocal--Sela also found that viruses can incorporate portions of the bee genome (which may confer the virus resistance to the bee immune response)***:

Sela continued: "The resultant virus–host chimeras, stabilized, and possibly replicative as "parasites" of the native virus, or even encapsidated within viral particles, can now be transmitted horizontally to other host individuals, to other host species, or even acquire a new host range."

This phenomenon has stunning implications for our understanding of bee/virus co evolution. Eyal Maori explained to me that the viral capsids can function as "genetic spaceships" that can deliver non-viral DNA or RNA from one organism to another other. Should such a "spaceship's" contents manage to get into a queen or drone germ cell, then the new genetic material could be passed to the next generation.

In other words, viruses can act as effective vectors of genetic material between different organisms, and can (albeit rarely) transfer genetic material, say, from the varroa mite, bumblebee, or yellowjacket into the honey bee! Their point is that all organisms are in reality "transgenic," and that viruses play a critical role in vectoring genetic material that can accelerate the evolutionary process!

NEXT EDITION

Varroa Changed Everything! Why have our bee problems so increased these past several years? And a look into the future.

References and Suggested Reading
The following three papers are free downloads:

Aubert, M (2008) Virology and the Honey Bee. (Google the title) ***This recent review contains a wealth of information—I highly recommend it for the serious beekeeper.***

Obbard, DJ, et al (2009) The evolution of RNAi as a defence against viruses and transposable elements. *Phil. Trans. R. Soc. B* **364**: 99-115.

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Free download: http:www.ask-force.org/web/Bees/Palacios-Genetic-Analysis-2008.pdf

"These articles were originally published in the American Bee Journal. All of Randy's bee articles may be found at: www.Scientificbeekeeping.com

If you find these articles of use, Randy appreciates donations to fund his efforts."

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The following is an update of recent activities of AHBIC if you should seek further clarification please do not hesitate to contact the AHBIC office.

AHBIC NEWS Jan - Feb 2012

On behalf of AHBIC I would like to wish all a Happy New Year.

Already it has been a busy year, developments include:

A further incursion of Apis cerana has occurred in Queensland and in this edition of the newsletter we include details. One important issue that came out of the incursion was the ability or non-ability of the *Varroa jacobsoni* mite to be harmful to the European honeybee. Until 2008 the Java type of *Varroa jacobsoni* was regarded as harmless to *Apis mellifera* (the European honeybee) as it lacked the ability to reproduce on the brood of that bee (Anderson, 1994). In 2008 mites belonging to the Java type of *Varroa jacobsoni* were found to be producing offspring on *Apis mellifera* brood in Papua New Guinea, even though mites of this type still did not show this ability in Java, Irian Jaya or the Solomon Islands (Anderson, 2008). To clarify this situation we invited Dr Denis Anderson to write in respect of the biosecurity risks to industry.

In respect of Apis cerana, Industry has also been involved in further discussion in respect of the containment strategy for the current operation in Cairns Queensland. A Scientific Advisory Panel including Industry representatives: Trevor Weatherhead and Max Whitton have forwarded a report recommending certain projects be undertaken. These are currently being evaluated by RIRDC to cost their implementation but have, in principle, been supported by the Committee overseeing the containment strategy.

It is with deepest sympathy we acknowledge the death of two of Industry's well known and respected figures, namely Gretchen Wheen and Monte Klingner. We extend our condolences to their family and friends. They were an inspiration the Industry and all who knew them.

The AHBIC Executive is meeting on Monday 27 February in Melbourne. Importantly they will consider an updated Five Year Business Plan which will be circularised to member bodies and delegates prior to the Annual General Meeting. On behalf of AHBIC thank you to those who have already contributed to this process.

Training.gov.au has now been launched, replacing the National Training Information Service (ntis.gov.au) as the official National Register of information on Training Packages, Qualifications, Courses, Units of Competency and Registered Training Organisations (RTOs). The new-look website has been developed to improve navigation for users and all of AgriFood's Training Packages are now available to download from the website.

Please note that some data is yet to be uploaded to the website, including superseded Training Packages. It is anticipated that this information will be loaded progressively. For more information, please visit:
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DETECTION OF ASIAN HONEY BEES AT TOWNSVILLE PORT

Monday 9 January 2012

Biosecurity officers from the Department of Agriculture, Fisheries and Forestry (DAFF) have taken immediate action to contain and exterminate Asian honey bees detected on a cargo vessel that arrived at Townsville from Port Moresby on Friday.

Dead and alive bees were detected as the vessel was being unloaded.

Shipping containers and the vessel were carefully inspected by DAFF Biosecurity officers. A pest controller was engaged to exterminate the bees.

Nearly 300 dead bees were collected and these have been examined by DAFF Biosecurity entomologists. To date, no queen bee or hive has been identified.

DAFF Biosecurity is working closely with the Queensland Government's Department of Employment, Economic Development and Innovation in an ongoing response to the detection.
Biosecurity officers are continuing surveillance of the area, but so far no additional bees have been found.

The vessel has since left Townsville.

The initial detection and notification by the stevedores, the swift response by DAFF officials, and the ongoing surveillance and analysis being undertaken by DAFF and Queensland officials is an example of Australia's biosecurity system at work — where the Australian and state governments, and the community, each play a role.

Statement by First Assistant Secretary, Tim Chapman Biosecurity Quarantine Operations, DAFF

INFORMATION ON THE "JAVA TYPE OF *VARROA JACOBSONI*"

Dr Denis Anderson
CSIRO Ecosystem Sciences
24 January 2012

Identification

- Its native host bee is the 'Java type of *Apis cerana*' from Java Indonesia (Anderson & Trueman 2000).
- It is distinguishable from *Varroa destructor* by its smaller body size. It is also morphologically distinct from *Varroa rindereri* and *Varroa underwoodi* (Anderson & Trueman 2000)
- It is distinguishable from all other varroa mite types using molecular markers (Anderson & Fuchs, 1998; Anderson 2000; Anderson & Trueman 2000; Navajas et al 2010).

Invasive Biology

- It is native to Java (on the Java type of *Apis cerana*) (Oudemans 1904)
- It was introduced to New Guinea during the 1970's on *Apis cerana* that were introduced by Indonesians into Irian Jaya (or Papua) from Java (Anderson, 1994).
- It first arrived in Papua New Guinea (PNG) in 1987 on *Apis cerana* that had spread from neighbouring Irian Jaya (Delfinado-Baker and Aggawal, 1987).
- By 1995 it had spread throughout the entire Island of New Guinea and onto offshore islands, including Biak and Yapan (Irian Jaya) and Boigu, Dauan and Sabai Islands off the southern PNG coast (which are part of Australian territory) (Anderson, 2006).
- It arrived in the Solomon Islands shortly before 2003 on the Java type of *Apis cerana* (which probably spread from New Guinea). *Apis cerana* first arrived in the far eastern parts of the Solomon Islands (San Cristobal, Guadalcanal and Savo Islands), so it did not 'island-hop' from New Guinea into the Solomon Islands. It is thought that forestry activities were responsible for introducing the bee into the Solomon Islands (Anderson 2010).
- By 2008 it had spread into New Britain, Bougainville and Manus Island on *Apis cerana* that had spread from mainland PNG.
- Since 1998 there have been two incursions of *Apis cerana* on to mainland Australia (Darwin 1998 and the current incursion at Cairns). No Varroa mites were found associated with either incursion and the Darwin incursion was successfully eradicated (Barry et al 2010).
- Since 1999 the Java type of *Varroa jacobsoni* has been found on 5 of 8 (62%) colonies of *Apis cerana* intercepted on vessels arriving at Australian seaports from the New Guinea region (Barry et al 2010; and the recent interception at Townsville).

Biosecurity Threat

- Until 2008 the Java type of *Varroa jacobsoni* was regarded as harmless to *Apis mellifera* (the European honeybee) as it lacked the ability to reproduce on the brood of that bee (Anderson, 1994).
- In 2008 mites belonging to the Java type of *Varroa jacobsoni* were found to be producing offspring on *Apis mellifera* brood in Papua New Guinea, even though mites of this type still did not show this ability in Java, Irian Jaya or the Solomon Islands (Anderson, 2008).

Current Research

- CSIRO is currently carrying out research to show:

(a) Whether the *Varroa jacobsoni* mites now reproducing on *Apis mellifera* in PNG originated from a single mother mite (similar to what happened for *Varroa destructor* on *Apis mellifera* – Solignac et al 2005) or from several different mother mites;

(b) Whether the *Varroa jacobsoni* mites now reproducing on *Apis mellifera* in PNG can also reproduce on *Apis cerana* (they may not now be able to reproduce on *Apis cerana*, as they had to change to be able to reproduce on *Apis mellifera*).

(c) The types of microbial pathogens associated with the *Varroa jacobsoni* on both *Apis cerana* and *Apis mellifera* in PNG.

Current Biosecurity Risk

Until more information is obtained on the reproducing *Varroa jacobsoni* mites in PNG (from current research), any *Varroa jacobsoni* mites arriving from PNG present a biosecurity risk to Australia (precautionary principal), although evidence suggests that this risk is not as great as that for *Varroa destructor*.

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WORK IN CANADA

August/September 2012

I am a small stock producer in the Shuswap Lake region or British Columbia Canada. We run a few hundred colonies, sell nuc's, queens, honey and some pollination. I also teach beginner bee courses and queen rearing courses. But this year I am trying to put forth a working holiday. I'll be looking for a small crew in Aug./ Sept to help pull the honey, do some extracting and processing and haul them around to see some local organic farms and breweries. I was thinking the other day it would be pretty neat to have some people from outside our country to join in on the fun!

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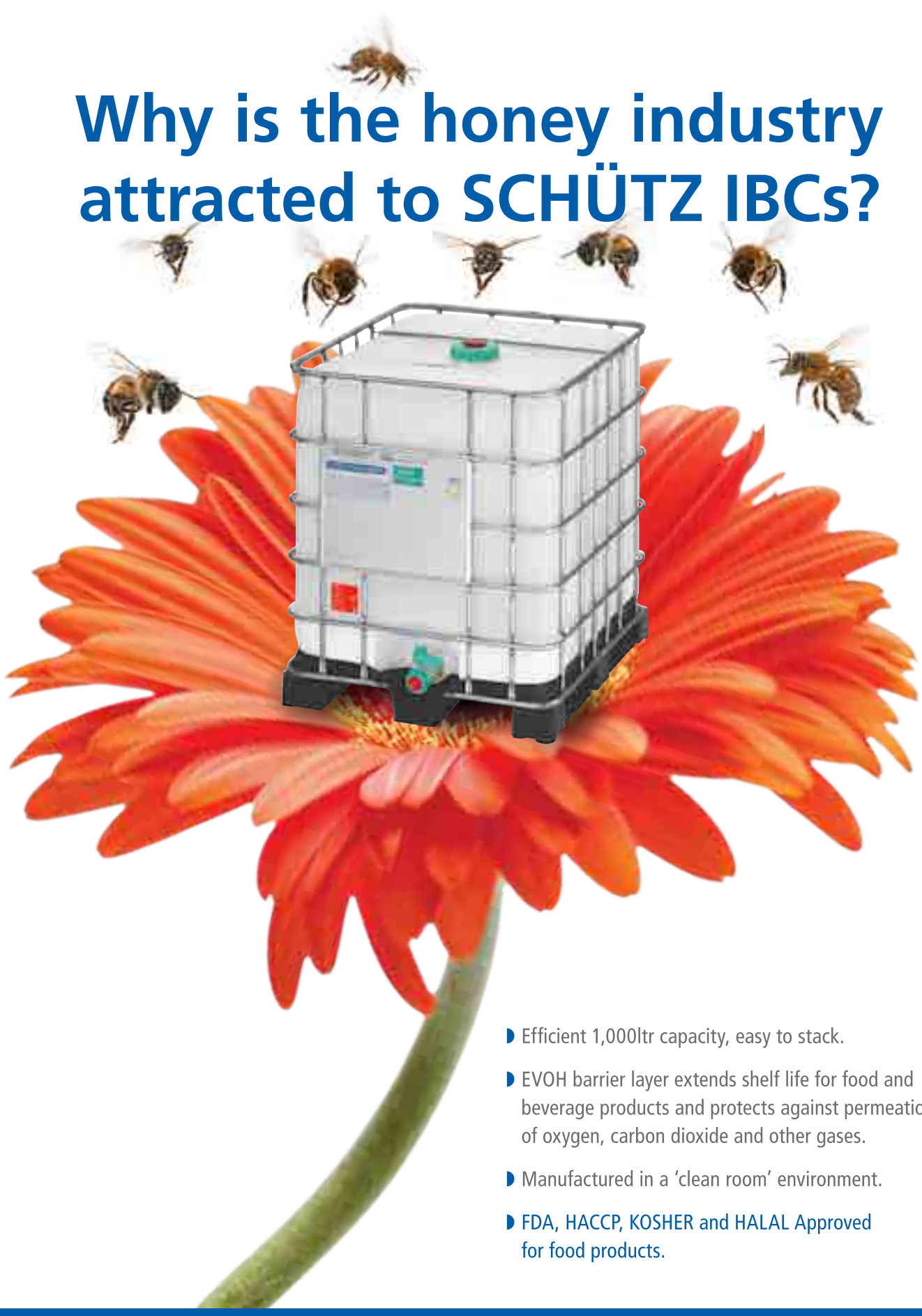

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