

The truth about the neonicotinoid insecticides

The pesticides industry stands accused of failure to investigate the hazards of systemic neonicotinoids fully and of failure to establish standard tests and protocols. The protection agencies stand accused of failing to protect human health and the environment.

With reference to the Executive Summary of the Workshop on Pesticide Risk Assessment for Pollinators January 15-21 2011 SETAC Pellston Florida

Authors: David Fischer from Bayer CropScience and Thomas Moriarty from the US EPA Office of Pesticide Programs.

http://www.setac.org/sites/default/files/executivesummarypollinators_20sep2011.pdf

This summary proves that the pesticides industry and all of the environmental protection agencies were aware of the following, which up until now, they had consistently denied.

- a) **That the systemic neonicotinoid pesticides are harmful to bees.**
- b) **That the tests and protocols that had allowed registration of the systemic pesticides were not adapted to assess potential hazard and risk from this type of pesticide.**
- c) **Despite knowing all this, the Protection Agencies have allowed the pesticides industry to keep neonicotinoids on the market.**
- d) **Many of the projects suggested for the future have already been done by independent scientists (See page 39 under Research and Recommendations).**

In addition, we have found that when *clothianidin* was conditionally registered in 2003, the US EPA knew it was highly toxic to honey bees on an acute contact basis and it had the potential for toxic chronic exposure to honey bees, as well as other non-target pollinators, through the translocation of *clothianidin* in pollen and nectar. They also knew it was persistent in soil and had the potential to leach into ground water, as well as surface water. There was also evidence of effects on the rat immune system and that juvenile rats appeared to be more susceptible to these effects (see Page 6).

The Executive Summary contradicts the replies to us. On **February 8th 2011**, Dr Steven Bradbury, Director, Office of Pesticide Programs wrote on behalf of Lisa P. Jackson, EPA Administrator: *"At this time we are not aware of any data that reasonably demonstrates that bee colonies are subject to elevated losses due to chronic exposure to this pesticide"*.

The European and UK Agencies sent similar replies. They denied absolutely that systemic neonicotinoids had toxic effects on honey bees. On **25th January 2011**, John Dalli, European Commissioner wrote: *"on the basis of current knowledge a ban would not be justified"*. On **15th February 2011** the UK Chemical Regulation Directorate, on behalf of Lord Henley, wrote to a UK MP: *"the data have not raised any cause for concern"*.

Admission on Page 12 *"Many who are familiar with pesticide risk assessment recognize that the methodology and testing scheme for foliar application products (where exposure may be primarily through surface contact) is not adapted to assess potential hazard and risk from systemic pesticides"*.

Admission on Page 10 [For many years, the systemic pesticide risk assessments have only involved a basic Tier 1 analysis]. “A *Tier 1 analysis is a conservative screen that efficiently separates those compounds that will not present a potential risk from those compounds that may present a potential risk*”.

Admission on Page 18 The report admits that these Tier 1 tests are only suitable for foliar pesticides, since they are based on “*the determination of the length of time between application and when bees could be safely exposed to residues on leaves and flowers of a treated crop*”. (The systemic pesticides are on the seeds, but there are neonicotinoid sprays).

Admission on Page 18: “*Higher tiered semi field or tunnel tests are recommended to refine the oral exposure assessment, at the colony level to both systemic and non-systemic sprayed on foliage*”.

Admission on Page 20: “*...but development of tiered species-specific tests requires significant effort and is seen as a high priority for future research*”

Admission on Page 20/21: The authors of the report also admitted that they still had no suitable standard tests for chronic toxicity to either adult honey bees or their larvae. Chronic toxicity tests on adult and larval bees “*require further development*” (**Page 19**). Conference members agreed that when these were developed they should be required as part of Tier 1 testing, as soon as test methodologies can be verified (**Page 21**).

Admission on Page 22: “*The sub-lethal impacts of pesticides on honey bee learning, behavior, and physiology have been well documented in the scientific literature*”. Instead of accepting this as a reason to suspend them urgently, delegates apparently agreed that further research was required. “*Additional work is needed in both laboratory and field test scenarios.*”

The delegates and authors were clearly aware of the work on sub-lethal doses affecting honey bee foraging by independent researchers, who were excluded from the workshop.

[Colin M.E., Bonmatin, I., Moineau, C. *et al* (2004) A Method to Quantify and Analyze the Foraging Activity of Honey Bees: Relevance to the Sublethal Effects Induced by Systemic Insecticides. *Arch. Environ. Contam. Toxicol.* 47: 387-395. In this paper, scientists from Montpellier, Orléans and Avignon Universities did **Tunnel Tests** in which they demonstrated that sub-lethal doses of 6 ppb *imidacloprid* or 2 ppb *fipronil* were enough to disrupt feeding. These were precisely the effects that Bayer itself had advertised for its use in termite control. In addition, the bees also exhibited signs of intoxication.]

[Yang, E.C., Chuang, Y.C., Chen, Y.L., Chong, L.H., (2008) Abnormal Foraging Behavior Induced by Sublethal Dosage of Imidacloprid in the Honey Bee. *Journal of Economic Entomology* 101 (6): 1743-1748. In this study **field tests** were done. These showed that treatment with sublethal doses of *imidacloprid* (as low as 50 ppb) delayed the return visit of a bee, and the time delay was dose-dependent. They found that the higher the dose to which they were exposed, the more likelihood that the bees would show abnormalities in revisiting the site, or even to go missing.]

[In 2003, in a 108-page document, the *Comité Scientifique and Technique* in France reviewed **all the independent scientific evidence** on systemic pesticides. Their findings were that “*the treatment of sunflowers is a significant risk to bees in several stages of life*”.]

Admission on page 16: “*Unique potential exposure sources for systemic pesticides include dust from seed treatment, consumption of aphid honey dew, or possible consumption of guttation water*”. [There is a reference to a paper on guttation drops at the foot of this page: V. Girolami, L. Mazzoni, A.Squartini *et al* (2009) Translocation of Neonicotinoid Insecticide from Coated Seeds to Seedling Guttation Drops: A Novel Way of Intoxication for Bees. *Journal of Economic Entomology* 102 (5) 1808-15. Guttation drops are a physiological

exudate from the xylem of plants, which bees often drink. It is a particularly valuable source of water for bees in spring when the plants are small. This paper showed that leaf guttation drops of all the corn plants germinated from neonicotinoid-coated seed contained amounts of insecticide constantly higher than 10 mg/l, with maxima up to 100 mg/l for *thiamethoxam* and *clothianidin*, and up to 200 mg/l for *imidacloprid*. The conclusion of the authors was that “When bees consume guttation drops, collected from plants grown from neonicotinoid-coated seed, they encounter death within a few minutes”. Indeed, if you enter “guttation drops” into Google, there are several Youtube recordings of these lethal events taking place between the corn seedlings.]

The fact that the authors, Fischer from Bayer CropScience and Moriarty from the EPA OPP, quoted this paper showing the high levels of insecticides measured in guttation drops from corn seedlings, and the rapidly lethal effects on bees after consuming them, suggests that they had read it, but decided to ignore it. Instead, on page 39 they wrote “Workshop participants recognized the uncertainty around guttation drops as a source of systemic pesticide exposure. The workshop participants recommended that research be conducted that would allow a more informed analysis of whether this route of exposure should be considered in pesticide risk assessment for pollinators”.

Also on Page 16: with reference to “*exposure to dust from seed treatment*”. There have been several incidents of mass deaths of bees during the process of planting maize in the sowing period from mid-March to May. In the Baden-Württemberg region of Germany in 2008, two thirds of the region’s honey bees died following the application of *clothianidin* (under the trade name Poncho). According to the German reports, tests on dead bees showed that almost all those examined had a build-up of *clothianidin*. The company said an application error by the seed company, which failed to use the glue-like substance “stickers” that sticks the pesticide to the seed, led to chemicals being dispersed in the air. Marzaro *et al* in Italy performed a **field trial** with two sets of bees in tulle cages. The cages were placed on the edges of the field, exposed to the dust of the drilling machine for 30 min, after which they were taken to the laboratory.

The 10 bees that were up-wind all survived, whilst the bees that were down-wind all died within 5-10 h. (Marzaro, M., Vivian, L., Targa, A. *et al* (2011) Lethal aerial powdering of honey bees with neonicotinoids from fragments of maize seed coat. *Bulletin of Insectology* 64 (1): 119-126.).

SETAC invited delegates.

The majority of invited “world experts” from Europe were members of the International Commission for Plant-Bee Relationships. The ICPBR appears to be self-appointed body. On closer examination it is clear that it represent the voices of the Pesticides Industry and the Crop Production Industry. At the 10th International Symposium of the ICPBR Bee Protection Group (2008), in his forward, the Chairman, Dr Peter G Kevan (University of Guelph, Canada) said that “for three decades it has provided an important forum for representatives from **industry**, national and international regulatory agencies, government and academic research bodies....divergent interests of **crop production**, etc.” “Natural ecosystems” was the last to be mentioned. It was “sponsored by the pesticides industry”. Many of the research presentations were headed by scientists from the industry. One paper: *The Proposal of the ICPBR Bee Brood Group for testing and assessing potential side effects from the use of plant protection products on honey bee brood* featured Roland Becker (BASF) Christian Maus (Bayer CS), Jens Pretorius (JKI), Ingo Tornier (Eurofins GAB). Authors of other papers included Mike Coulson (Syngenta) Mark Miles (Dow) Ed Pilling (Syngenta) and Dick Rogers (now working for Bayer CS US).

At the SETAC meeting, the UK was represented by Mark Clook (Chemical Regulation Directorate) and Helen Thompson (Food & Environment Research Agency, Fera). Helen Thompson had worked closely with three scientists from Bayer, Syngenta and Dow on the ICPBR Bee Protection Group (she became the Group's secretary). The same three had also helped with the UK Defra Research SID5A (2007-2009) Systemic Pesticide Risk Assessment, which, incidentally, only got as far as protocols for Tier 1 tests. The conclusions of the ICPBR working group presenting at the Bucharest meeting in 2008 were that protocols for the second and higher tier (Tunnel Tests and Field Tests) **were still to be developed**. So, members of the ICPBR must have known for **at least 3 years** that the science underpinning protocols for risk assessment for systemic pesticides was inadequate. The ICPBR have 17 members on their three bee working groups. Seven are from the pesticides industry, some of whom service two groups. This may explain why the CRD, Fera, Defra and the AFSSA (French equivalent of Fera) have repeatedly advised UK and European Ministers and informed us, the public, that there was no evidence that the neonicotinoid pesticides are harmful to honey bees.

The SETAC conference, as with the ICPBR, was also heavily sponsored by the pesticides industry. Thus, they were well represented; three from Bayer, two from Syngenta, two from BASF (one of whom had boasted on the net about BASF's financial contribution), one from Monsanto and one from DuPont. At least 12 of the delegates from Europe were members of the ICPBR or on the ICPBR Council. Independent researchers were excluded.

Letter to the UK Chemical Regulation Directorate

On January 6th 2011 we wrote to the CRD. *“The main point of the documentation we sent to the Minister concerned new work done in Holland by a Dutch toxicologist, Dr Henk Tennekes (**The neonicotinoid insecticides: a disaster in the making**)¹ in which he reports contamination of surface water by the neonicotinoid imidacloprid. The reply you have sent us in which you explain the functions of the Chemical Regulation Directorate seems to contain only a defence of the neonicotinoids in relation to claims that they might be connected to honey bee disappearances. In fact, your letter did not even once mention the subject we had raised with the Minister. In his own words, Dr Tennekes says that his book:*

“catalogues a tragedy of monumental proportions regarding the loss of invertebrates and subsequent losses of the insect-feeding (invertebrate-dependent) bird populations in all environments in the Netherlands. The disappearance can be related to agriculture in general, and to the neonicotinoid insecticide imidacloprid in particular, which is a major contaminant of Dutch surface water since 2004. The relationship exists because there are two crucial (and catastrophic) disadvantages of the neonicotinoid insecticides:

- They cause damage to the central nervous system of insects that is virtually irreversible and cumulative. There is no safe level of exposure, and even minute quantities can have devastating effects in the long term;*
- They leach into groundwater and contaminate surface water and persist in soil and water chronically exposing aquatic and terrestrial organisms to these insecticides. So, what, in effect, is happening is that these insecticides are creating a toxic landscape, in which many beneficial organisms are killed off.”*

In this letter (to the CRD) we will highlight findings related to the devastating effects on the environment and biodiversity caused by the neonicotinoid insecticides.”.....Page 4.

“Further work on imidacloprid contamination of surface water has recently come from Utrecht University in a PhD thesis (September 2010) from Teresa C. van Dijk.²

Effects of neonicotinoid pesticide pollution of Dutch surface water on non-target species.
The Maximum Tolerable Risk (MTR) norm is an ecotoxicological standard for general environmental quality and the minimum quality level that is desirable for all surface water in the Netherlands. The MTR-value for a substance is the environmental concentration of that substance, at which the species in an ecosystem are safe from effects caused by the substance. From Figure 2, page 14, the six maps show the locations at which the surface water concentrations of imidacloprid were measured, and where the MTR was exceeded. They have increased from 2003 to 2008, in some areas sometimes up to more than five times the MTR, and this was most noticeable in the regions where horti- and agriculture are concentrated, as shown from the four maps on Fig 1, page 13. On page 25-26, plots of log imidacloprid concentrations (ng/l) against square root numbers of organisms (for flying insects of the order Diptera) show decreasing abundance of Diptera with increasing imidacloprid concentration. Page 22 shows that as species are exposed to neonicotinoids longer, even very low concentrations will affect them. On p 8, “only a small part of the pesticide doses used reaches its intended target, while the major part of it ends up in the environment outside the field, where it can cause difficulties through toxicity to non-target species and accumulation can occur (Tisler 2009) especially if a pesticide is persistent. The author pointed out that the neonicotinoid pesticides are now registered in 120 countries. They have 25% of the world insecticide market.” We sent pdf files of Dr Tennekes’ book and Dr van Dijk’s thesis. There was no reply from the CRD to our questions.

Communications with the US EPA, Mrs Claire Gesalman, Communications Branch

We sent the above evidence about Dutch surface water and invertebrate declines to the US EPA, again with supporting evidence.

Mrs Gesalman wrote on 15th April 2011: *“With regard to potential effects on non-target invertebrates and surface water contamination, EPA is not aware at this time of any data demonstrating an imminent hazard from clothianidin..... If you are aware of reliable data that demonstrate an imminent hazard as defined by federal pesticide law, please forward to me the author’s name, publication name (peer-reviewed publications are preferred).*

We replied on 24th April 2011.

“Dear Ms Gesalman

You do not need science to see what is happening to the environment. Just stand in the middle of a field of oil-seed rape. Where are all the insects? Twenty years ago if you drove 200 miles in the UK you would have to stop to clean insects from your windscreen and headlights. In 2004, in June, 40,000 drivers found, using a device attached to their number plates, there was just 1 insect per 5 miles. Probably, 7 years later, there are even fewer. Perhaps nobody cares much for insects? But there are other sinister events that signify that the environment is acutely sick; catastrophic (but little publicised) declines in a wide variety of species in the US (and later in Europe); honey bees, frogs, bats, bumblebees and birds.”

The Global Environmental Protection Agencies. What is going on behind the scenes?

In January 2011, on the US EPA Home Page, one of Administrator Lisa Jackson’s mission statements was: ***“We have greater opportunity to protect human health and the environment than before”***. Yet, on December 13th 2010 her Office of Pesticide Programs had run a workshop: ***Streamlining the Risk Assessment Process***. Robert Schulz had designed an electronic programme (e-Builder Dossier) to facilitate the registration of pesticides by the applicants. According to slide 18, the prime benefits were *“reduced cost to the EPA”*, and *“quicker processing”*. There was no mention of human health or the environment on any of

the 67 power point slides. On looking on the SETAC website it became apparent that the relationship between US SETAC, the EPA OPP and the pesticides industry was unhealthily close. One Ralph.G.Stahl of USA DuPont heads the most important of the three work groups on SETAC's Ecological Risk Assessment branch, the EcoValuation group.

On May 30, 2003, Daniel C Kenny of the US EPA Registration Division granted conditional registration for *clothianidin* to be used for seed treatment use on corn and canola (oil seed rape). The EPA scientists had assessed the risks as: ***Clothianidin is highly toxic to honey bees on an acute contact basis. It has the potential for toxic chronic exposure to honey bees, as well as other non-target pollinators, through the translocation of clothianidin residues in nectar and pollen. In honey bees, the effects of this toxic chronic exposure may include lethal and/or sub-lethal effects in the larvae and reproductive effects in the queen. The fate and disposition of clothianidin in the environment suggest a compound that is a systemic insecticide that is persistent and mobile, stable to hydrolysis, and has potential to leach into ground water, as well as run-off to surface waters. There is evidence of effects on the rat immune system and juvenile rats appear to be more susceptible to these effects.*** The Summary of Data Gaps included the following: *Developmental immunotoxicity study. Environmental fate data: on Aerobic aquatic metabolism and Seed leaching study. Ecological Effects Data: Whole sediment acute toxicity to freshwater invertebrates; Field test for pollinators.*

Australia: Clothianidin obtained registration with the Australian Pesticides and Veterinary Medicines Authority (APVMA) in 2007. On one of the registration documents it quoted the US EPA. "Guidelines with honey bees indicated that clothianidin is very highly toxic to adult worker bees, ranking among the most highly toxic insecticide to bees." The APVMA also admitted that "clothianidin degradation in soil is slow under terrestrial field conditions (when sprayed on), with a half-life of one to more than two years." We suspect that they had been provided with the US EPA documentation by the industry. Beekeepers claim that there is no evidence that the APVMA did field tests for bees under Australian weather conditions.

In February 2009, Australian bee exporters had lucrative businesses. They were flying large packages of honey bees to the US to help with the Californian almond harvest. According to Dr Denis Anderson, who was in charge of biosecurity, Australia had no Colony Collapse Disorder and no *varroa* mite. By 2010 they were losing hives; by 2011 they had CCD. According to one beekeeper, agriculture has gone from using only small amounts of neonicotinoid pesticides to the current 85% on crops, in less than 12 years. He said that beekeepers used to love putting their hives on canola; now there have been so many disasters with disappearing or dying bees, that many have taken their hives as far away as possible. One beekeeper said "the last couple of years we have stayed away from canola and we've had the best bees for years".

The public has no idea of the extent of their exposure to the neonicotinoid insecticides

We have discovered that they are the active ingredient in many products, all with different names and are used in numerous situations. Now the patent has expired on *imidacloprid*, a large number of firms have "cashed in" on their success and are producing their own brands. In addition, aided by the environmental protection agencies, the agrochemical industry has added new uses. Now, without having been made aware of it, the public can be exposed to the neonicotinoids in almost every environment, in addition to farmland; in the house, the garden, golf course, playing fields, amenity areas and conservation areas. In January 2011,

we looked on the UK Chemical Regulation Directorate's website at their Approved Pesticides Database. Here we discovered 33 different *imidacloprid* preparations. They could be applied to a total of six different seeds; oil seed rape, fodder beet, sugar beet, barley, oats and wheat. We are also aware of seed firms who apply it to linseed and sunflower. In addition, they are used commercially as spray preparations on greenhouse-grown salad crops and vegetables, plant bulbs, and container-grown ornamental/house plant production for indoor and outdoor use and also pet products for fleas. There are at least four products for domestic garden use. Bayer Garden Products include Provado® Ultimate Bugkiller Concentrate (*thiacloprid*), Provado® Lawn Grub Killer (*imidacloprid*), Ultimate Bugkiller Ready to Use (*thiacloprid*) and Provado® Vine Weevil Killer (*thiacloprid*). They carry warnings on product labels about hazards to bees, toxicity, conditions and usage, but these are in extremely small print. *Clothianidin* is the active chemical in seven different products and can be applied to the seeds of 12 crops; barley, durum wheat, oats, rye, wheat, triticale, oil seed rape, forage maize, grain maize, sweet corn, fodder beet and sugar beet.

Laws have been shaped by the industry such that it difficult for the public to find out

In the UK, if a farmer does not wish to tell you whether or not his crop has been grown with neonicotinoid-coated seed, you will have to apply to a third party to find out. In addition, those selling the chemicals are not obliged to keep records for more than three years, so the trail is difficult to follow. It is possible that all the spring bulbs grown in Holland, which we are urged to plant to provide pollen for early bumblebees, are coated in pesticide. We do not know. Presumably, if you see an oil seed rape field with no insects in sight, it is probable that the oil seed rape is coated. But we cannot find out for certain.

The neonicotinoid insecticides are toxic to all taxa, not just invertebrates. Is our hypothesis of immune deficiency disease in wildlife correct after all?

There is increasing evidence from basic neuroscience research that that the neonicotinoids have effects on mammalian neurons as well as on invertebrate ones and that the effects on the nicotinic acetylcholine receptors (nAChRs) may be more significant in vertebrates than was initially suggested by the pesticide companies. We found that Bayer and the US EPA knew when *clothianidin* was conditionally registered in 2003 that there were effects on the rat immune system and that juvenile rats appeared to be more susceptible to these effects. In fact, one of the Data Gaps was a “Developmental immunotoxicity study”. We do not know whether or not the Data Gaps were ever filled, but two recent independent studies were done in rats^{3,4} and another on a preparation of human tissue⁵. All of the authors suggested that the neonicotinoids might have adverse effects on human health, and especially on the developmental brain⁴. We have also found a paper showing connections between the nicotinic acetylcholine receptors and the immune system in humans via the vagus nerve. In the process of trying to treat severe inflammatory responses in sepsis and haemorrhage (which are a major cause of death in patients in Critical Care), a specific anatomical and physiological connection was proved between the nicotinic acetylcholine anti-inflammatory receptors in the central nervous system and the innate immune system, which protects humans against infection and tissue injury⁶. This paper accords with evidence of the effects of *clothianidin* on the rat immune system noted in the registration document. Perhaps the epidemics causing massive declines in a wide variety of species in the US (and later in Europe, now in Australia) due to infections (honey bees, amphibians, bats, bumblebees and birds) gives added weight to our hypothesis of immune deficiency in wildlife (which was rejected by Defra Ministers in the UK).

What is happening to humans in the UK? Are there subtle effects on vulnerable foetal tissues?

In March 2009, the charity Brain Tumour UK reported that 40,000 brain tumour patients each year were missing from the official statistics ⁷. In the May/June 2010 issue of *Oncology News*, Dr Colin Watts, neurosurgeon from Cambridge, wrote a Report “*Brain Cancer: An Unrecognised Clinical Problem*”. He said that Office of National Statistics figures for the UK showed that the number of children dying from brain tumour in 2007 was 33% higher than in 2001; in contrast, child deaths from leukaemia were 39% lower than in 2001. In fact, brain tumours have now replaced leukaemia as the commonest cause of childhood death ⁸. In July 2010 Gwynne Lyons and Professor Andrew Watterson published the CHEM Trust Report ⁹. *A review of the role pesticides play in some cancers: children, farmers and pesticide users at risk?* In it, pesticide exposure of pregnant women is linked to childhood cancer. In the last 35 years; non-Hodgkin’s lymphoma has more than doubled; testicular cancer has doubled; breast cancer in women has increased by two thirds and in men has quadrupled; prostate cancer has tripled.

No evidence of monitoring of neonicotinoids in water in US, Europe, UK or Australia

The systemic neonicotinoid insecticides were (and still are) “*beneath the radar*”, since they do not feature in the 2009 US Geological Survey (USGS) National Water-Quality Assessment Program (NAWQA) Report: *Pesticide Trends in Corn Belt Streams and Rivers (1996-2006)* ¹⁰. The USGS authors of the Report said: “*The declines in pesticide concentrations closely followed the declines in their annual applications, indicating that reduced pesticide use is an effective and reliable strategy for reducing pesticides contamination in streams.*” One of the first national studies on the presence of pesticides in ground-water had been published in 2008 ¹¹. Laura Bexfield who conducted the data analysis said: “*The results of this study are encouraging for the future state of the nation’s ground-water quality with respect to pesticides*”. “*Despite sustained use of many popular pesticides and the introduction of new ones, results did not indicate increasing detection rates or concentrations in shallow drinking water resources over the 10 years studied*”. The authors of both reports expressed satisfaction with the results because they were (mistakenly) under the impression that pesticide use was decreasing and that the reduction in pesticides in ground-water was commendable. However, the chemicals that NAWQA were measuring were only those that they knew about. In 1991, the first of a group of novel insecticides was introduced; the systemic neonicotinoids. They started to replace many of the older insecticides. Their sales escalated, such that now, in 2011, they occupy a dominant position in the global pesticide market. *Imidacloprid*, *thiomethoxam* and *clothianidin* do not appear in the USGS list of monitored pesticides. The US Environmental Protection Agency is not measuring neonicotinoid levels either. This is extremely surprising since they knew the persistent properties of *clothianidin*, including its potential to leach into ground water, as well as the runoff into surface water. It is no wonder that everyone ignored Dr Henk Tennekes’ book and said it was “not peer-reviewed” and the two Bayer Scientists (Maus & Nauen) attempted to destroy Dr Tennekes’ paper in *Toxicology* 2010 (which was peer-reviewed).

The fate of neonicotinoids in water; what happens during flooding?

In 2001, in response to claims in a pesticide fact sheet, Bayer experts from different scientific fields issued a “position paper” on *imidacloprid*: “*The use of imidacloprid in agriculture does not entail unacceptable harmful effects for the environment as the substance will*

*disappear under all circumstances from the compartments soil, water and air”. “Although the substance is stable in sterile water in the dark, it decomposes readily under the influence of light. Biotic processes under the influence of microbes present in natural water and its sediments present **another mechanism** for the elimination of imidacloprid”.*

No-one told the Bayer experts from different scientific fields that microbes **are** invertebrates. They will be poisoned just as readily as the target organisms, non-target invertebrates (pollinators) and the organisms that break down the soil. In fact Bayer must suddenly have realised they were wrong, since we found warnings on the US EPA & the APVMA websites for *clothianidin*. **This product is highly toxic to aquatic invertebrates. Do not discharge effluent containing this product into lakes, streams, ponds, estuaries, oceans, or other waters. Do not apply directly to water or to areas where surface water is present or to intertidal areas below the mean high-water mark.** In fact, the conditional registration document for *clothianidin* in 2003 stated that it was “*persistent and mobile, stable to hydrolysis, and has a potential to leach into ground water, as well as runoff to surface waters.*”

In 2011, Australia (New South Wales and Queensland) had disastrous floods. The Darling River area had suffered prolonged drought followed by heavy rain and flooding. On March 11th Bourke Township experienced a massive fish kill. An eye witness said: “*It was phenomenal; you couldn’t see the water, there were carp gasping for breath and crayfish crawling onto the bank*”. Counting the dead fish passing Bourke Weir at 100/sec. Geoff Wise estimated 8 million per day and the event continued for 5 days; 40 million dead fish was said to be an underestimate. It was described as a ‘Black Water’ event and attributed to lack of oxygen from organic material being washed down the river following flooding of a plain. But beekeepers suspected otherwise: “*why were the crayfish trying to escape the water if it was only due to lack of oxygen?* Agricultural land borders 2,500 km of the Darling River. Cotton is grown in the area; more than 95% is seed-treated GMO and 96% is *imidacloprid* treated. Two further ecological disasters have occurred down the Queensland Coast after the floods in December 2010 and January 2011. In July it was reported that “*the northern coast of Queensland has become littered with sick and dying turtles and dugongs (sea cows)*”. It was attributed to run-off of nutrients into the ocean “*potentially killing the sea grass that both turtles and dugongs feed on*”. On September 19th in Gladstone Harbour, many sick fish were discovered; barramundi and bream were found with sores, skin rashes and infected eyes. Capricorn Conservation Council suspected industrial pollution and fishing was prohibited. According to beekeeping sources, Gladstone and the entire Queensland Coast above it are the biggest areas for sugar cane in Australia and *clothianidin* (Sumitomo Shield Systemic insecticide) has been granted registration for use on these very low-lying sugar cane farms.

Loss of biodiversity and a future without invertebrates

In the US in 1980, Professor E.O. Wilson, the eminent Harvard entomologist, was asked to identify the most important problem facing the world in the next decade. To quote his words: “*The one process on-going in the 1980s that will take millions of years to correct is the loss of genetic species diversity by the destruction of natural habitats. This is the folly our descendants are least likely to forgive us.*”

In 2007 Sir David Attenborough said: “*If we and the rest of the backboneed animals were to disappear overnight, the rest of the world would get on pretty well. But if the invertebrates were to disappear, the world’s ecosystems would collapse*”.

Bayer CropScience has used the environment as a huge, private experimental laboratory. In the early 1990s Bayer scientists launched onto the world the chemical weapon from hell; a powerful neurotoxin that, as Dr Tennekes demonstrated (and others have subsequently confirmed), causes a virtually irreversible blockage of postsynaptic nicotinic acetylcholine receptors in the central nervous system of all invertebrates from pollinators down to soil and aquatic organisms. When the patent ran out, the rest of industry followed Bayer. The US EPA scientists had found evidence that *clothianidin* affected the rat immune system. It confirms the suspicions of many: that the neonicotinoids are associated with immune deficiency in other wildlife. Please help to save the world's ecosystems by circulating this document to as many people as possible.

Watch Veteran US Reporter Dan Rather's documentary about pesticides and the US EPA.

<http://vimeo.com/29419200>

Please support the following organisations.

<http://asmallbluemarble.org> A charity for independent pesticide research.

www.buglife.org.uk The Invertebrate Conservation Trust. *Conserving the small things that run the world.*

www.bumblebeeconservation.org.uk *Saving the Sounds of Summer*

www.nrdc.org The Natural Resources Defense Council. A US Environmental Organisation. *Its purpose is to safeguard the Earth: its plants and animals and the natural systems on which life depends. "Knowledge isn't power. Only Power is Power" Bob Sass.*

References

¹ Tennekes, H.A. (2010) The systemic insecticides: a disaster in the making. EBook or Hardcover from <http://www.adisasterinthemaking.com> or in the UK from Northern Bee Books; sales@recordermail.demon.co.uk

² Van Dijk, T.C. (2010). Effects of neonicotinoid pesticide pollution of Dutch surface water on non-target species abundance. MSc. Thesis, 2 July 2010, updated 2 September 2010. *Sustainable Development Track Land Use, Environment and Biodiversity*. Utrecht University, Netherlands.

³ Duzguner, V., Edogaan, S. (2010) Acute oxidant and inflammatory effects of *imidacloprid* on the mammalian central nervous system and liver in rats. *Pesticide Biochemistry and Physiology* 97: 13-18.

⁴ Junko Kimura-Kuroda, Masaharu Hayashi, Hitoshi Kawano. (2011) Nicotine-like effects of neonicotinoids on rat cerebellar neurons. *Neuroscience Research*, Volume 71; suppl, September.

⁵ Ping Li, Jason Ann & Gustav Akk. (2011) Activation and Modulation of Human $\alpha 4\beta 2$ Nicotinic Acetylcholine Receptors by the Neonicotinoids Clothianidin and Imidacloprid. *Journal of Neuroscience Research* DOI:10.1002/jnr.22644.

⁶ Cai, B., Deitch, E.A., Ulloa, L. (2010) Novel insights for systemic inflammation in sepsis and haemorrhage. *Mediators of Inflammation* 2010 ID 642462.

⁷ Sullivan, D.J., Vecchia, A.V., Lorenz, D.L., Gilliom, R.J., and Martin, J.D. (2009) Trends in pesticide concentrations in corn-belt streams, 1996-2006: *U.S. Geological Survey*.

⁸ Bexfield, Laura M. (2008) Decadal-scale changes of pesticides in ground water of the United States, 1993-2003: *Journal of Environmental Quality* 37: S226-S239.

⁹ Brain tumour UK: 40,000 brain tumour patients missing from the official statistics. March 2009. www.braintumour.uk

¹⁰ Watts, C. Brain Cancer: An Unrecognised Clinical Problem. *Oncology News*, Volume 5 Issue 2 May/June 2010. www.oncologynews.biz

¹¹ A CHEM Trust Report by Gwynne Lyons and Professor Andrew Watterson. July 2010. A review of the role pesticides play in some cancers: children, farmers and pesticide users at risk? www.chemtrust.org.uk