HOUSE OF COMMONS

ORAL EVIDENCE

TAKEN BEFORE THE

ENVIRONMENTAL AUDIT COMMITTEE

INSECTS AND INSECTICIDES

WEDNESDAY 28 NOVEMBER 2012

DR MIKE BUSHELL, DR FRASER LEWIS and DR JULIAN LITTLE

DR NIGEL RAINE, DR CHRIS CONNOLLY and PROFESSOR SIMON POTTS

GEORGINA DOWNS

Evidence heard in Public Questions 154 - 257

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Oral Evidence

Taken before the Environmental Audit Committee

on Wednesday 28 November 2012

Members present:

Joan Walley (Chair)
Peter Aldous
Neil Carmichael
Martin Caton
Katy Clark
Zac Goldsmith
Caroline Lucas
Caroline Nokes
Mr Mark Spencer
Dr Alan Whitehead
Simon Wright

Examination of Witnesses

Witnesses: **Dr Mike Bushell,** Principal Scientific Adviser, Syngenta, **Dr Fraser Lewis**, Division Head, Environmental Safety, Syngenta, and **Dr Julian Little**, Government Affairs, Bayer CropScience, gave evidence.

Q154 Chair: I would like to welcome each of you to our session this afternoon, an inquiry that is important to this Select Committee and one that I think is of great interest to many people. For your information, we are expecting votes at 4 pm and we have three separate sessions, so we have a number of detailed questions that we wish to ask of you, if we may.

I would like to start off with a question particularly for Dr Lewis and Dr Bushell. I am referring to today's *Farmers Weekly* and the quote, "Based on previous statements, we believe this committee"—the Environmental Audit Committee—"is in danger of pinpointing the bee colony decline on a single pesticide when there are other important factors at play." On what evidence did you feel compelled to make that statement?

Dr Bushell: That statement was made by one of our Basel corporate affairs people.

Q155 Chair: Do you have the evidence for it?

Dr Bushell: That we made the statement or that you are focusing only on insecticides?

Chair: I wondered what evidence you have to cause Syngenta to make that statement.

Dr Bushell: The issue about bee health is multifactorial, as I am sure you know, and focusing only on a single one is unlikely to get a good result for bee health.

Chair: Thank you very much indeed. We must move on to detailed questions.

Q156 Martin Caton: Do you accept the findings of the recent Gill, Henry and Whitehorn studies on the sub-lethal effects on bees of neonicotinoids?

Dr Bushell: Firstly, all of those studies, in common with a number of other studies in the literature implicating pesticides as a particular problem in bee decline, are purporting to be field-realistic when in reality they are laboratory studies, usually using doses that are very

unrealistic so that you are actually getting toxic effects on insects from insecticides. With the Henry study particularly, I heard him speak in Cambridge in September and he admitted himself that the rates he used were unrealistically high. Julian, perhaps you would like to address the Gill and Whitehorn data.

Dr Little: I think the Whitehorn study is interesting. When you first looked at the headline that came out of there it suggested that this was a field study. In reality it wasn't, it was a laboratory study in which essentially insects were force-fed high levels of neonicotinoids and then given some chance to be outside. It is very different from how a bumble bee would normally be and therefore it is very difficult to see how you come to a conclusion that as a result of this study there is clearly a problem. With all of these three studies the research in itself is not in question; the conclusions that can be drawn from them are very much in question. I think that is the key point that we need to point out. We are not rubbishing the research at all. What we are very concerned about is where conclusions are drawn from these sort of studies that, to be honest, were not designed to make those conclusions.

Q157 Martin Caton: Isn't this sort of Orwellian mantra of field study good, laboratory study bad just simplistic and very unscientific? We have taken evidence, and we will be taking some more evidence after you, from scientists who are saying all forms of research have their drawbacks and none more so than attempting to do field tests.

Dr Little: In the end fields are where bees and other pollinators are, so if you can do field trials or you can do field studies then clearly that is the best way of finding out whether a particular product has an impact. In the evidence that we submitted, we pointed out two very large field studies that go into a lot of detail about what is causing problems. This is using real bees and real beekeepers in real situations. In those situations they can see really key effects or key linkages between poor bee health and things like varroa destructor and very little, if any, correlation between poor bee health and insecticide use.

Q158 Martin Caton: Without getting into the "our research is better than yours" sort of argument, there are criticisms of the research that you have quoted in your submission to us. There are other scientific criticisms of them. Has either of your companies considered withdrawing your neonicotinoid products pending conclusive research to justify their continued use?

Dr Bushell: We believe that the body of evidence that supports safe use of neonicotinoids is very compelling. These products have been on the market for many years and the decline in bee population is due to other factors. That is what our overwhelming assessment of the data shows.

Dr Little: Just to add to that, I think you need to go back and understand why neonicotinoids are used. There are some very good reasons why farmers have looked at these sort of products and recognised them as being quite simply a better way of controlling the pests and diseases that they absolutely need to control at the very early stages of germination and early growth of a crop. When you look at things like the mammalian toxicity of these products, they are incredibly safe compared to some of the older products that used to be used many years ago. Traditionally insecticides were always an issue. When it comes to neonicotinoids, there isn't that issue. If you combine the ease of use, the fact that you don't have to spray nearly as often, the fact that they are much safer to use means that there are compelling reasons why farmers want to use these products and why we would continue to supply them.

Q159 Chair: Dr Bushell, could I press you a little bit more? You said just now that the evidence is compelling and it would be really helpful to the Committee if you could name which evidence you are referring to.

Dr Bushell: Fraser, perhaps you would like to talk about the regulatory studies, which are in the possession of CRD.

Q160 Chair: Sorry, I thought you were referring to academic studies.

Dr Bushell: We have provided in our written evidence a variety of different papers that show that the major influence on bee health comes from, as Julian has already said, the varroa mite, the viruses that they bring into the colonies, the weather and, very importantly, habitat availability and food resources.

Q161 Chair: It would be really helpful if you could name one of them so that we know which ones you are directing us to.

Dr Bushell: Schneider is one of those. I think you could look also at the Cresswell paper and look at the one on scientificbeekeeping.com. It is a very balanced view of bee health from beekeepers.

Chair: Thank you. I will hand you back to Mr Caton.

Q162 Martin Caton: It is interesting, we had Professor Cresswell here a few days ago and, although he endorses your position as far as honey bees are concerned, largely, he does identify a problem with bumble bees caused by this particular systemic pesticide. So even the scientists you are quoting are not confident that there is no contribution from this particular pesticide. In your written evidence both of you have recognised that the European Food Safety Authority will introduce a new pesticide testing regime and risk assessment next year, partly in recognition that the existing system does not adequately assess systemics. That new regime, as we understand it, will just be looking at new products and will not include existing neonicotinoids. Will your companies voluntarily submit your existing TMX and IMD products to the new tests next year?

Dr Lewis: As part of the reregistration programme that exists within Europe, those products will be evaluated under the new scheme. When we made the original submission we used a scheme that was 91/414, which was the original pesticide reregistration. That has now been updated to 1107. The new bee guideline that is being worked on at the moment and is in the draft phase will come into place and we will evaluate our compounds against that bee scheme. We are also already beginning to look at bumble bees for example. We have a study with bumble bees and oilseed rape planned for next season, flowering next year. We will do that and all of that data will be submitted to the appropriate regulatory authorities.

Q163 Peter Aldous: This is a question to Dr Bushell and Dr Lewis. The French Government were very much impressed by the Henry report, to such an extent they have actually withdrawn the registration for Cruiser oilseed rape. How do you feel about that? Do you think they made the right decision or got it wrong?

Dr Lewis: We think that they have got it wrong. The French decision was against all of the evidence and the actual recommendation of their own experts within ANSES, the French pesticide safety authority. It is also interesting to note that the French continue to allow the use of Cruiser on maize, which is a bee-relevant crop as well, as well as on sugar beet, which isn't. The other interesting point as well for the Committee is that the French authorities are still allowing their seed treatment companies to continue to treat seed for export, so we do think they have got it wrong.

Q164 Peter Aldous: Why do you think they made the decision they did?

Dr Lewis: That is something that we are trying to follow up at the moment. We are in discussion with the French authorities, and I think there is likely to be legal action to challenge that decision through the courts.

Q165 Peter Aldous: Do you want to speculate as to why they made that decision?

Dr Bushell: If you would like us to speculate, I am sure it was an entirely political decision. That is my speculation. We don't know.

Q166 Peter Aldous: Other than proceeding through the courts, are you doing anything else to seek to get them to reverse the decision; negotiation, perhaps, or discussion?

Dr Lewis: We are in discussion with them. We are working very much with ANSES who are the relevant government body we work opposite. We are talking to them and we are trying to talk to the Minister who made the decision somewhat in isolation. Clearly, given the situation we are in, we have no other action than to take some form of legal action.

Q167 Peter Aldous: Just to clarify, was this a decision made by the new French Government post-May?

Dr Lewis: We are not exactly sure.

Dr Bushell: We are not entirely sure when the date was. I thought it was in April.

Dr Lewis: Yes, it was the new Government.

Q168 Peter Aldous: There was no wind of it happening before the elections there?

Dr Lewis: No.

Q169 Mr Spencer: Is there an inspection regime in place to stop a French farmer importing UK or German seed treated by a chemical and drilling it within the French borders? The question is: how can the French Government enforce that ban?

Dr Lewis: I am sorry; I don't know the answer to that.

Dr Bushell: I don't either. We can find out for you. It would be clear that with oilseed rape, if someone came and inspected the field they could look for residues of those products and then the farmer would be acting illegally. We certainly wouldn't sell banned materials to farmers. We would always abide by the regulation and laws that are in force in any territory.

Q170 Simon Wright: I would like to talk about the evidence from Italy in relation to contaminated dust. This led to the suspension of three neonicotinoids as maize treatment, and the evidence suggested that the fine dust generated by the drilling of treated seed is lethal to bees. Do you accept the results of the Marzaro study, and what do you make of the policy response to it?

Dr Little: It is a very interesting area of work. We recognised that there was an issue with dust and maybe I should explain a little bit about what we are talking about here. If you are talking about a neonicotinoid seed treatment, essentially you are taking a seed and applying a product to that seed. The idea is that the farmer would drill that seed and as that seed germinated would take up that chemical and it would protect it at the very early stages of that plant's growth. One of the areas that was observed was that when a farmer was planting that seed there was the occasion where you could get a fine dust coming out of the drill, especially if it was a maize drill. The reason for that is it is a pneumatic drill, so they push the seed into the soil, and the way that those drills had been originally designed meant that they vented to the air. This was recognised as being an issue and companies have been working with the manufacturers for new machinery but also to retrofit machinery that was already in

the marketplace to make sure that that dust is vented to the ground, thus massively reducing the incidence of airborne particles. That appears to have been very successful over the last two years in absolutely minimising that sort of issue happening. That is a bit of background to that. In Italy itself they have had very clear restrictions on the use of a number of products for a few years now.

Dr Bushell: I would add one other point to this and that is looking at the formulation science to make sure that by the use of appropriate stickers and polymers you get a much better adherence of that material to the seed. All the companies work with the professional applicators. These materials are not generally applied by farmers themselves. They are applied by professional applicators using machines that are designed for the purpose. We have set very low levels of dust-off, the amount of dust that is coming out of a treated seed, and anything that fails that can't be sold without being re-treated.

Q171 Simon Wright: Are there any possible other effects relating to venting to the ground, for example on wildflowers? Is that something that is being looked at?

Dr Little: What you are looking for is a method of exposure. The way that most people saw dust as being an issue was airborne dust. The fact that it is on a flower or whatever doesn't necessarily mean to say that a pollinator is going to pick that up. In fact, the chances are it is not going to pick that up, because they are looking for something specific.

Dr Bushell: Studies in the literature seem to imply very much that this is airborne contamination of bees flying through the field as the major route of uptake and not taking this in from other areas. Of course, if you can stop the dust by minimising it, by making sure much less exits the field, then this keeps the risk very low.

Dr Lewis: I was just going to add one final point. Clearly, if you are aiming the dust from the pneumatic driller into the ground the ability of that dust to drift is significantly reduced, so it would also prevent movement. In modern fields there are very few wildflowers in the field itself.

Q172 Caroline Lucas: What research have you done on the effects of neonicotinoids on bees that has not been published or has not been submitted to the regulatory authorities?

Dr Lewis: I think it is fair to say all of our data has been submitted to the relevant regulatory authorities. In many cases we might wish to publish more of that data more freely, but the current regulatory system means that if we did that that data would be available for competitor companies to use to achieve their own registration, so as soon as we publish it we lose our data protection. Having said that, there is an intention, within Syngenta at least, to publish two of our main studies that we have relied on for registration, which have already been submitted to peer review journals and will hopefully be available at the end of this year or the beginning of next.

Q173 Caroline Lucas: We were going to ask if we could get access to those before you get it back from the assessors.

Dr Lewis: As long as it is kept confidential by the Committee such that it doesn't interfere with the scientific peer review, then yes, we can supply both of those papers to you.

Dr Bushell: But they would be in draft form and, of course, they may be changed during the peer review process.

Dr Lewis: I would like to add one point, though, that I think is important for the Committee to understand. All companies have a system where we are mandated to submit any adverse data to the regulatory authority as soon as it is generated. We have no option with that. It is currently enshrined in the EU at least in article 56 of 1107/2009, which I have brought with me and I could quote or leave with the Committee if you wish, but it says, "In

particular potentially harmful effects of that plant protection product or its residues, metabolites, etc, that have harmful effects on human or animal health or groundwater or their potentially unacceptable effects on plants or plant protection in the environment shall be notified." So it would be illegal for us to conduct any research and not submit it to the relevant regulatory authorities. That is not something that only happens in Europe. It happens elsewhere in the world as well. So we have no ability to hide any data. As soon as we generate adverse data, it needs to be immediately submitted to the authorities.

Q174 Caroline Lucas: How about more proactively sharing unpublished research in relation to risk assessments with the academic community, for example? We heard some evidence last week that suggested it can be quite difficult to get hold of some of the research.

Dr Lewis: As I said, one of the difficulties we work with at the moment within our current paradigm is we lose data protection.

Q175 Caroline Lucas: I appreciate the point that you are making, but can you envisage any way in which academics would be able to have access in a way that doesn't prejudice your competitive advantage?

Dr Lewis: In the revision of 91/414 that produced 1107 we made a submission that we should change the data protection rules such that we should make the system more transparent and allow our data to be published. That was rejected by the EU. The main way that we could do that is if the system that we currently work under was changed to give us some kind of data protection while still publishing our data. It is driven principally by freedom of information rules that cause the problem and some of these are currently antiquated in terms of the system, particularly in the electronic age we live in.

Q176 Caroline Lucas: In terms of following that up further, would you be able to send us a memo explaining what you think would need to change at EU level to enable you to do what you say you would like to be able to do?

Dr Lewis: Yes.

Q177 Caroline Lucas: That would be really helpful. I don't know if you would agree, but in a sense there is a bit of an imbalance. What we have heard from some of the academics is that they publish their material and you can challenge it, and yet sometimes it feels like it is not a very even balance, because some of the stuff that you have is not in the public domain for them to be able to challenge.

Dr Lewis: Yes, I think we can submit that. We have some ideas. Clearly it is for governments, not just this one, to look at that, but we would gladly put some thoughts together. As I said, we are publishing two of our key studies, one of our long-term four-year studies where we have continued to look at a set of hives over a number of years.

Caroline Lucas: We look forward to having that. That would be marvellous.

Q178 Zac Goldsmith: I would be interested to hear why you think the EU turned down your suggestion.

Dr Lewis: I am not sure, to be honest. I don't know the answer to that.

Q179 Zac Goldsmith: Is the view that you have put forward widely shared among industry?

Dr Little: I think there is also an element here that there are a lot of competing elements when this part of the rules was drawn up, thinking that making data more available without protection would result from these rules. Actually it is the reverse: less data comes

out. There was a view that if generics were able to come into the market earlier then that would bring down the price of crop protection products, but, as I said, all it has done is driven companies to being more careful about releasing information rather than less.

Q180 Zac Goldsmith: Who would be responsible for that decision? To whom did you submit your ideas; which department was it?

Dr Lewis: Sorry, I don't know the answer to that personally, but we can let you know. As part of the thoughts that we submit to you, we can put that in.

Q181 Zac Goldsmith: As far as you know, this was not rejected as a result of lobbying by your competitors? This is not something that industry itself has tried to do?

Dr Lewis: Not as far as I am aware, no.

Q182 Caroline Lucas: In the spirit of interrogating research, I wanted to look at the German study that was cited in the Bayer submission of evidence to us. You talk about the impressive size of the study of the Genersch research—1,200 colonies—but is it not the case that only 215 of those were screened for pesticides and of those, as I understand it, around 74 tested positive for at least one neonicotinoid? That does demonstrate beyond doubt that bees are regularly exposed to significant quantities of that insecticide, and crucially no data is presented on the survival of those particular colonies. The only analysis that is done is a very crude one comparing survival in colonies with high levels of pesticides very generally, which includes lots of fungicides that are not known to harm bees, with survival in those with low levels. In other words, the research that you have put forward and are citing, which I understand was funded entirely by industry as well, for what it is worth—

Dr Little: There are a few things on that. It was funded mainly from the German Government.[1] We helped out with a couple of the techniques that they needed help with in terms of identifying individual products. You are right that not all 1,200 colonies were assessed for individual content of products. They took what they saw as a rational sample, a reasonable sample.

Q183 Caroline Lucas: So, 215 out of 1,200?

Dr Little: What they did do, though, is look very much at the viability of all those colonies. I am not 100% au fait with each and every detail of it—the problem of not speaking German is always an issue—but what is very clear is that, if you go through the conclusions of that report and look at the correlation between either the problem with varroa or the diseases that they spread, or issues with climate, weather and habitat, those were overwhelmingly the issue in that particular case.

Q184 Caroline Lucas: But in that case there was no analysis done on colonies with neonicotinoids versus those without, so that comparison was not there.

Dr Little: But if you looked at those situations where those products were present you saw no correlation between either a healthy or an unhealthy colony.

Dr Bushell: I think a particularly interesting example would be to look at Australia when you are talking about the real issues associated with bee decline. Australia probably has the healthiest bees anywhere in the world because they don't have the varroa mite. They go to

[1] Note from witness: Dr Little has pointed out that the German study on bee health was funded 50% by the Ministry of Agriculture and 50% by the Federal Bee Institute, which are themselves funded by the German Government, from 2010 to 2013. Before 2010, the study was 50% funded by the Federal Bee Institute and 50% funded by the industry.

extreme efforts to keep the varroa mite out, as you may have seen in the press this week, and they, of course, use neonicotinoids very widely.

Q185 Caroline Lucas: Just to end up on that point, the trouble with that kind of statement—as indeed the statement in the Bayer evidence where it says, for example, "It is worthy of note that France has restricted the use of neonicotinoids for over 10 years. Despite that, bee health in France remains similar to or worse than here in the UK"—is that they purport to be scientific but you can't really interrogate them scientifically at all, because there could be a million other things going on in France or in Australia. When it comes to France, as I understand it, they still use neonicotinoids quite a bit on sunflowers, for example. I think that sometimes it can sound as if the evidence is supporting the case that you want to make, whereas when you challenge it a bit more that is not necessarily the case.

Dr Bushell: That is true also of the people who argue that insecticides are the real cause of bee decline. I think a very powerful example would be our Operation Pollinator, which I am sure many members of this Committee will be familiar with. We have more than 10 years' experience of planting up field margins as areas of high biodiversity that show a huge benefit and increase in numbers of all pollinators, bumble bees, honey bees, solitary bees, for example, including in the first studies bumble bee species that were thought to be extinct in those areas. This is an example of sustainable, intensive agriculture in action. You have a good high-yielding crop in the middle of the field, and a small area of land in intensive farming areas, managed well for biodiversity, will have a hugely positive impact on beneficial insects, birds and other biodiversity.

Q186 Martin Caton: Dr Lewis, in the quote you just gave us from the document from the EU on the things that you would have to report if you came across them in your research, one of the things you mentioned was groundwater pollution. What assessment of groundwater pollution has been done with regard to neonicotinoids?

Dr Lewis: I think it is fair to say that in Europe we have probably the most stringent regulatory regime, particularly when you apply it to groundwater, with the absolute cutoff of 0.1 micrograms per litre. The assessment that we have to go through for every use, not just every compound but every single use, against that criterion is by far the most stringent in the world. Half of my department spend their time looking at just that issue. It is a very rigorous assessment. Prior to approval of that use, whether you were using conservative models that assume all groundwater is at 1 metre below the surface, there is an extensive modelling and field study and lab study set of data to look at the parameters that would affect movement of a compound in soil. Also increasingly now throughout Europe post-registration we are seeing significantly more monitoring of groundwater going on across most countries in Europe. At the moment, all of those are showing that there is no contamination of groundwater by these types of compounds, and in fact it shows very clearly that the models used for registration are very conservative and significantly over-predict what would happen with groundwater.

Q187 Caroline Lucas: Could you tell us how long the chemicals persist in soil? How long are they active in terms of being a potentially toxic chemical?

Dr Little: It is a little bit of, "How long is a piece of string?" because it will depend on a huge number of different things, including soil type, climate, temperature, what has been grown in there, how many worms there are—everything will affect that figure. But if you are looking at something like imidacloprid or clothianidin you can be talking a half-life of anywhere between 16 and, say, 200 days.

Dr Lewis: For thiamethoxam we are slightly shorter than that longer figure. We are talking in the region of 20 to 80, 90, 100 days.

Q188 Caroline Lucas: There is a factsheet on one of the products that says that trials have shown 100% control of pests up to 11 months from application, so doesn't that suggest a longer activity?

Dr Little: I have no idea which product you are talking about in which crop.

Q189 Caroline Lucas: It is called Turf Merit, a granular formulation of neonicotinoid intended for spreading on amenity grasslands. The factsheet on a Bayer website says, "Trials have shown 100% control of pests for up to 11 months from application." I will send you the link.

Dr Little: Thank you.

Q190 Dr Whitehead: I will ask you some questions about your own procedures. DEFRA said early this autumn that they would need to see unequivocal evidence of harm to bee colonies before they acted on neonicotinoids. What sort of standard of proof do you require internally before advancing a new product for risk assessment and taking it externally?

Dr Little: Are you talking in terms of taking it to commercialisation?

Dr Whitehead: Yes. If you have developed a product, and presumably you are testing it and looking at it internally, what sort of checks and balances and degree of proof do you require before you would submit it for risk assessment subsequently? How does that work?

Dr Little: If you are talking about an insecticide, you will have a whole raft of different insects that you will want to control. In some cases it may be one particular insect that you will be interested in, so you have to be able to control that insect for a farmer to be interested in buying your product. That is a given: you have to have efficacy. In addition to that, there will also be a whole raft of indicator species that you really don't want to have a high level of control of and you won't be surprised that the honey bee is one of those. In the case of Bayer—and it will vary depending on individual companies—we will invariably test insecticides in the field, irrespective of what happens in the laboratory, because that is the most important of all the things that we have been talking about today in terms of understanding the effect of an insecticide in real conditions. So we will go through all of those processes, and if we get good efficacy and very little impact on bees and other indicator species, if these products are used properly, then we would look to advance that product, depending on other things as well. But from an environmental perspective that is how we would go through that process.

Dr Lewis: To build on that, at a very early stage in the development of a new product, while it is still in research and before you even know you have something that can be really commercialised, we look at its environmental profile. You can understand that. If we are going to invest something in the region of $250 million to $300 million in taking a product through to commercialisation, we want to be very sure that we have a good product that is safe, can be used safely and can be registered. So we conduct a lot of early tests. Coming back to the example earlier about groundwater where you have an absolute trigger, we have to be very sure that the compound can be used safely and not contaminate groundwater, because clearly that would mean that we would not be able to get a commercial licence in Europe. I think it is fair to say in all companies, and I can definitely speak for my own, we take two separate decisions. The first decision we take is we satisfy ourselves that it is safe before we satisfy ourselves we can commercialise it. To do that we do a lot of early stage testing, principally in the laboratory but also under field conditions, to take those decisions.

Q191 Dr Whitehead: If you are risk assessing a new product, particularly in terms of what might be its collateral damage over and above what it is intended to damage, what are the costs? Is that the whole cost you have just mentioned or is that a part of the costs?

Dr Lewis: If you were talking about the regulatory package, for example, depending upon how broad, how many crops, how many countries, you are talking somewhere in the region of $60 million to $100 million to do the regulatory and safety testing that is required these days. In Europe it is probably at the upper end of that because we are dealing with probably the most stringent regulatory regime in the world.

Q192 Dr Whitehead: Does that sort of cost deter you as companies from putting forward new products? Is it regarded as perhaps a brake on new and improved products?

Dr Bushell: At the industry-wide level it is a deterrent to innovation and investment. If you look at the case of herbicides, simply because I have the data on this, if we go back to when I started in the industry over 30 years ago there were the top 50 research-based companies that the Wood Mackenzie report would talk about. Now if you are looking at herbicides, there are probably three or four global companies researching for new herbicides, and this is principally because of the cost of registration.

Dr Little: In that internal process you reject so many so it becomes very difficult to find something that can do what a farmer needs in terms of the ability to grow safe, high-quality, affordable food but at the same time have minimal effect on the environment. It is very difficult to find a product that will work in those situations.

Dr Bushell: I should also add that if we have developed a product, let's say for a small number of crops, big crops like maize, wheat, maybe soya, you can't then just extrapolate from the data that we have and use it on all crops. Every application in every country at every rate will be subject to a risk assessment to convince our development committee that it is safe to release to sale.

Q193 Dr Whitehead: Would your risk assessments include or would you want them to include—let's say when you have decided that you really don't want your product to harm bees—all pollinators, or do you think all pollinators should be included as a category of harm in risk assessment, as opposed to just bees?

Dr Lewis: The current regulatory regime worldwide uses honey bees as a surrogate for all pollinators, and there are safety factors built into the risk assessment regime that account for extrapolation to other pollinators. As has been referred to already, the new work that has been done, the new research, is going to expand the number of species that we look at, but given the diversity that is out there, you could never look at every species. You just couldn't do it. Most of them would be untestable under the current science that we have, so it will still be necessary to focus on a number of them. We are just going to expand the number that we look at, and we agree with that. As I said earlier, we will begin testing on more species, particularly for products that we are inventing now that won't reach the market for another five to 10 years.

Dr Little: If you think about essentially there is one sort of honey bee, looking at bumble bees just in the UK you are talking 20, so which one would you choose? Then if you are looking at solitary bees, you might be going up to, say, 200. If you then go into non-bee pollinators you may be talking about 2,000. I don't know what the exact number is on the bigger value, but you can understand that to be able to test on every single pollinator doesn't make sense. The trick is to find the species that really are representing the rest of that pollination group. I think one of the key things that EFSA will have to come up with is a group that represents this whole class of pollinators much better than just a single pollinator.

Q194 Dr Whitehead: Forgive me, but how do you know what is a representative species under those circumstances?

Dr Little: That is when we go back to the entomologists and start looking at how that will work. If you are talking about a bumble bee and you are talking about European legislation, you are going to have to look for a bumble bee that is in all countries in Europe, otherwise it wouldn't make sense, would it? So it will take some time to identify those really good indicator species that genuinely improve the predictability of insecticides on pollinators over and above what we already have with the honey bee. As we have already said, the honey bee is currently the species of choice, but there are a lot of worst-case scenarios built into that to try to take into account the possibility that other species might be slightly more sensitive.

Q195 Zac Goldsmith: Are either of your companies represented on the EFSA committees that take a view on new chemicals?

Dr Little: No.

Dr Lewis: EFSA has a mandate that it will not involve commercial companies in those committees.

Q196 Zac Goldsmith: Why is the composition of those committees confidential? Why do you think that is?

Dr Lewis: I am not sure. I think some of their technical committees are not confidential and they are published, so I am not exactly sure what committees you are referring to.

Q197 Zac Goldsmith: I don't know what the technical term for the committees is, but the committees that are tasked with effectively providing a green light for new products entering the market.

Caroline Lucas: What about the Advisory Committee on Pesticides, the ACP?

Dr Lewis: That is in the UK and that membership is made public.

Zac Goldsmith: I am talking about the committees delegated by EFSA to take a view on new products. I forget the technical term. I am going to have to stop that line of questioning because I can't remember the name of the committee. I wish I was able to come back to you, and I may be able come back to you after the session.

Q198 Caroline Nokes: I want to move on to the issue of funding of the Chemicals Regulation Directorate. I want to try to get to the bottom of whether it is funded directly by yourselves or whether there is some other funding arrangement.

Dr Lewis: When I started working for Syngenta 24 years ago, when we made a submission to a regulatory authority, other than a minor nominal fee, we didn't pay for the service of registering that compound. About 10 years ago it was changed by Parliament such that it was deemed that, as we wanted the registration, we should pay, so we now pay a fee with every submission to CRD in the UK to process that and either decide for or against a registration. A good analogy for this, perhaps, is your driving licence. At the moment we pay an agency to give us a driving licence. When you pay that fee, it doesn't make any decision that you will or will not get a driving licence. I think it is the same with the regulatory system. We pay regardless of whether we achieve a registration, and there are many instances in the UK where we do not achieve a registration.

Q199 Caroline Nokes: Do you have any indication of what proportion of their funding comes direct from agrochemical companies?

Dr Lewis: I do not, no, I am sorry.

Q200 Caroline Nokes: Does it give you any influence over their deliberations or, as you indicated, is it just a straightforward you pay your fee, end of negotiation/discussion?

Dr Lewis: In my personal opinion, none at all. I find that CRD are one of the most challenging regulatory authorities we deal with, similar to the French and the Germans but more challenging than many others in Europe, and if anything they are becoming more challenging as the regulatory paradigm develops throughout Europe. So, no.

Q201 Caroline Nokes: On a different theme, do your companies have any contractual or financial relationships with research that is going on in universities and, if so, how does that work?

Dr Bushell: We have thousands of individual research collaborations with university groups throughout the world. In the UK we probably have more than 100 projects underway at any time, and they would cover all aspects of plant science, physical science, chemistry for example, and bioscience.

Q202 Caroline Nokes: As part of those relationships, do you take any steps to safeguard the findings of that research, for instance to prevent publication if it is in any way unhelpful to your objectives?

Dr Bushell: If we are talking about a piece of contract research where it might be that they have a particular machine that can do something, then that would be a confidential thing, but that is a very small number. We couldn't and wouldn't ban publication of studies except to protect, for example, confidentiality during the filing of a patent. But of course we realise that if you are working with students and academics, they have a need to publish, and banning them from publishing would mean that none of them would work with us.

Q203 Caroline Nokes: So it is in no way unusual for you to come to an arrangement with a researcher that they would get the specific ability to publish a view? Quite the reverse, it would be normal for you to expect them to publish?

Dr Lewis: Absolutely normal, and that applies whether we are working with them directly or through a scientific funding agency like BBSRC.

Dr Bushell: In some cases where, for example, in a chemistry project an interesting invention has been made, perhaps in formulation technology, then the contract would have in it a clause that would say, "We may require publication to be withheld but this will normally have a period of not more than six months", which would allow the filing of a patent and then, of course, you would be free to publish it even before the patent was published, because the filing date would be set.

Q204 Mr Spencer: It is a bit of an unfair question, but could you give us an idea of the cost to the taxpayer if the taxpayer were to pick the tab up for the funding of CRD and the sort of research that goes on at universities? What sort of bill would that amount to?

Dr Bushell: I think you would be better off asking CRD what their total budget is and how much of it they get from industry, because of course we don't know what other companies are paying. They are taking several years to review dossiers. In the old days when we used to print everything out as a dossier, it resembled the *Encyclopaedia Britannica*. Now it goes in on CD, so it's a little bit easier to carry.

Q205 Mr Spencer: If the Government turned around said, "We are taking the funding of the CRD off you and the public purse is going to pick that up," how sorry would you be?

Dr Bushell: We would be really delighted.

Q206 Mr Spencer: We heard evidence from the Soil Association, which is one of the largest organic farmers in the country. They don't grow oilseed rape at all. If we withdrew these sorts of chemicals, would that wipe oilseed rape out as a viable crop in the UK, or could we continue to grow it by other means?

Dr Bushell: Julian has a very interesting study talking to farmers, and I think we should ask him to answer this.

Dr Little: I mentioned it in our submission, but essentially we asked farmers what does it mean if you lose seed treatments, and it is worth going back and saying there is a reason why they use these things. In many cases it is about establishment of their crop, getting their crop going at the very early stages. In many cases it is actually to control not the pest itself, the insects themselves, but the diseases that they will spread, so, for example, in oilseed rape it might be turnip yellow virus, in wheat it might be barley yellow dwarf virus. Those viruses can have really crippling effects on your yield. With turnip yellow virus, for example, in a worst-case scenario you are looking at a 25% reduction in yields.

We asked farmers what would be the consequences: 87% of people said that it would severely impact their ability to grow oilseed rape; 72% suggested that it would definitely have an adverse environmental impact; 79% said their oilseed rape yields would probably decrease; 90% would need to increase the number of sprays on that crop, so increase their pesticide spraying; and 84% suggested that pest control would be a lot more expensive, so they are getting less yield and it is also costing them more to grow a crop. One that really threw us, to be absolutely honest, was that 47% of farmers said they would consider not growing oilseed rape, either some of their oilseed rape or oilseed rape altogether, which was a very big surprise to us. Oilseed rape is a major crop in the UK, and certainly in many cases is the crop of choice if you are not growing wheat. So, without that crop of choice the economics of UK agriculture start to look a bit less attractive.

Dr Bushell: In France, Cruiser on oilseed rape has been deregistered, and if you ask our French national company what are we recommending to farmers for this year, you find that the tools available to farmers for insect control are very limited. This is quite dangerous, because if you are relying on essentially a single set of tools like the pyrethroids, you are going to cause resistance in major pests very quickly without having the benefit of the other mode of action coming in from these simple seed treatments.

Q207 Mr Spencer: Could you send us the data?

Dr Little: Of course.

Q208 Mr Spencer: To focus on that, could you run through the argument where some of your opposition says that if you don't use this seed treatment, which is obviously a blanket application of pesticide, then you would only treat where there is an issue, where it becomes a pest problem? In what sort of percentage of occasions is there likely to be a pest problem? Are we talking 10%, 50%, or 90%? Are those chemicals better or worse for the environment?

Dr Little: It is another one of those "How long is a piece of string?" questions, but let's give it a go. What you are suggesting is that you go towards, "If there is a problem we will deal with it." The only slight problem with that is that frequently by the time you have spotted there is a problem the damage has already been done, and that is especially true with insect-borne diseases. Once you have spotted that you have a particular virus in your crop, the chances are you have lost that crop, or lost a lot of that crop, and basically the damage has been done. So you have to be able to take out those particular vectors almost as they are

happening, or even slightly before they are happening, rather than once you have seen that they are happening.

The other thing about seed treatments that is absolutely critical here is that they are a management tool for farmers. They don't have to look out the window and say, "Oh my God, I can't spray today," and then panic as to whether they are going to lose their crop or a significant yield from that crop. They already know that at that early stage—and frequently it is at the wettest times of the year when they are less likely to be able to spray—they have at least these particular problems under control. So it is a fantastic management tool for them to really make a difference when it comes to growing their crops, producing those high-quality affordable crops that we are all used to being able to go into the supermarket and buy at a reasonable price. Without that certainty, farmers will take whatever opportunity they have to spray. In a bad year that might be four extra sprays. I am not convinced that if you are an entomologist or an environmentalist you really want farmers to go out there and spray insecticides during those times. If it is early in the season, that is exactly when bees are coming out of hibernation, and it is not a good time to be spraying, so anything that you can do to reduce the number of sprays is a good thing, and seed treatment is a fantastic way of doing that.

Dr Bushell: When you are applying a seed treatment, of course by definition it is in the soil and so you are controlling soil pests that attack the germinating seedling as well as getting protection for the developing foliage from attack from leaf-feeding insects. So that is a really good help to farmers. Of course they could come in later on and spray against foliage pests. It would be very difficult to control those soil-dwelling pests because of the very limited range of chemicals available.

Q209 Mr Spencer: I may be completely wrong, but is it possible to fine-tune these chemicals to the degree where you could block the chemical reaching the flower, so it covered the rest of the plant but didn't get to the flower? You would then deal with the insects that were attacking the plant but not the pollinators that were landing on the flower. Is that technically possible? Have you done any research to look at that?

Dr Little: In a way that is exactly what imidacloprid, clothianidin and thiamethoxam do. They are present at a high enough concentration to control those insects they need to control at the early stages of that crop and yet are present, if at all, in tiny, tiny amounts once it gets to the flowering part of the plant. Quite simply, once it has got to that level it is not controlling any pests at all, let alone having any significant impact on a pollinator. It is certainly not controlling an aphid that is far more sensitive to a neonicotinoid than a bee will ever be.

Dr Bushell: You have created a zone of protection in the early stages of growth and by the time, as Julian said, you come to flower, when pollinators and bees are being attracted into the crop, the levels are very low. Of course, that will have all been taken into account, as Fraser said, during the risk assessment process to make sure that important pollinators are not being damaged by these products.

Q210 Chair: We are just about to get to the end of this part of our hearing this afternoon, but I would like to go back to my first point about the statement that Syngenta made to *Farmers Weekly* and the concern that you have that chemicals have little to do with the decline of the bees and that it is other factors. Are you doing research on what those other factors might be that are contributing, how are you testing that, and how is that in the public domain or not in the public domain?

Dr Little: Mike has already mentioned in a lot of detail Operation Pollinator, which essentially demonstrates that if you have more nesting sites, more foraging sites, you have

more pollinators. That is one area that we recognise that European agriculture has to do better at. It has to improve, quite simply, the amount of food that is out there. Specifically to Bayer, we have been a bee health company now for nigh on 30 years, producing products to control disease and pests in bees. We see that as a massive step forward. If you can control varroa, as they have managed to in Australia, you have the healthiest bees on the planet. We view that as being the best way of dealing with the problem with honey bees. Quite simply, blaming the nearest chemical doesn't make sense.

Q211 Chair: In respect of the tests that you would be doing, would that be part of the official licensing of products? Would that be included in the testing that you would be carrying out?

Dr Little: All of these are looking at the problem with bee health or pollinator health. They are not compound-specific; they are not product-specific. It is really about improving what we see as the big problems with bee health rather than, as we say, the use of insecticides per se.

Q212 Zac Goldsmith: We heard evidence last week that, for the last couple of decades at least, the amount of area that provides good habitat, in terms of both foraging and nesting, has if anything grown in the UK, but the total number of pollinators, not just bees but across the board, has plummeted. I don't think there is any argument about the numbers in relation to pollinators—maybe the details, but the general direction of travel is downwards and I think everyone accepts that. I would ask you to go back on the point that you made earlier about the need for greater habitat, greater foraging areas and so on. It cannot be the reason why we are seeing such catastrophic falls in the number of pollinators. If I can add to that question, I don't think anyone argues that chemicals are the only reason why we are seeing this decline, but I think a lot of people are arguing that it is a significant contributing factor. No one disputes the existence and the dangers of varroa, because it would be impossible to do so, but we have also heard concerns from previous scientists that we have spoken to that varroa is becoming more effective at destroying bee colonies, that something is weakening the general colony immune system and that that itself could be chemicals. I would be interested to hear your response to both of those points.

Chair: Very, very quickly.

Dr Little: A couple of things, very briefly. Norman Carreck down at Sussex—in fact I think he was with Rothamsted at the time—did some very good work looking at not just varroa but also the viruses that it carries. It showed that colony health was not particularly affected by varroa itself, but a combination of virus that it would be carrying plus the varroa itself had a catastrophic effect on the hive. What is very clear is that varroa mites are carrying more and more of these viruses and are causing more and more problems. So that is at least one area of research that we are looking into a lot.

It is also true that not all bees and not all pollinators are going down. Bombus hypnorum, for example, has just arrived in the UK but is spreading north very quickly. It arrived in my garden last year. It is no more sensitive to pesticides than any other bumble bee but it is thriving. So it is not as simple as one thing or another. I think right at the very beginning Mike said this is a multifactorial problem that is far more complicated than probably we know.

Dr Bushell: I would add two things. Again, you all will be aware of the massive amount of work done by John Beddington's Foresight project. The principal finding from that, as Charles Godfray said last week, the inescapable conclusion from the Foresight work is that the sustainable intensification of agriculture is a critical issue. That means, of course, growing more productively but getting better outcomes environmentally and using all the

resources and inputs that go into agricultural systems more efficiently. When we talk about bee health, again we have to look at the systems level. I think again, although varroa mite is getting more difficult to control because the acaricides that used to be used by beekeepers to control it are now much less effective, such that they are adding more to a point where they are almost damaging the bees themselves in some cases, there are very interesting themes of research in academia on biological control agents. There is also a company called Beelogics with their RNAi technology, which again we do not have time to go into today but looks a very interesting way of controlling those viruses.

Chair: Yes, we do need to move on, because I think what you have raised is when is the right time to act and when is it too late to act, but we do need to move on. Thank all three of you for coming along this afternoon. We will move very quickly to our next set of witnesses.

Examination of Witnesses

Witnesses: **Dr Nigel Raine**, Royal Holloway University of London, **Dr Chris Connolly**, University of Dundee, and **Professor Simon Potts**, University of Reading, gave evidence.

Q213 Chair: Dr Connolly, Dr Raine and Professor Potts, I think you have each sat through the previous session, and we do need to move on very quickly indeed. We have heard from the industry and from business, and we really want to look at the academic side of things. With no further ado, I will invite Mr Spencer to continue the questioning.

Mr Spencer: Before we start your areas of expertise, do you want to comment more generally on the transparency of the process of regulation and whether you feel that basically there is enough transparency for people like yourselves to get involved and access the knowledge that you require?

Professor Potts: We certainly welcome the industry move to make things more accessible. The point at the moment is it is not as accessible as it should be. I do not think I need to repeat the argument that all academic literature is there for peer review and for comments, but a lot of industry and some government research is completely inaccessible. Anything that moves us in the right direction is very welcome. At the moment, we are not anywhere near close enough.

Q214 Mr Spencer: Do you all agree with that?

Dr Raine: Yes, I think so.

Q215 Mr Spencer: In your view, are the risk assessments sufficiently broad to capture the full range of possible effects?

Dr Raine: I will come in on that. I think they are not, would be the short answer because we are focusing very much on one species in terms of bee health. We are focusing on the honey bee, which is a reasonably atypical bee with large colonies, so it would be very important to look at other species of bee, varying in terms of their ecology and their life histories, which differ a lot. We are also looking and focusing very much on what the lethal dosages are, lethal exposure levels. I think it is very important to consider that these chemicals are mostly affecting the nervous system and they will be affecting how information is processed by the nervous system and how it is transmitted through the nervous system. One output of the nervous system is obviously behaviour, so I think we should be looking at sub-lethal effects, and if we are looking at low levels of exposure, which we have heard is the levels of residue we are seeing in nectar and pollen that bees are consuming, then sub-lethal effects are really important.

I would also say that bees are typically foraging in an environment where they are visiting a crop that may have multiple pesticides or agricultural chemicals used on it, or they may be visiting multiple crop species, so they are really exposed to more than one chemical at any one time or over their lifetime. I think we need to be looking at combinational exposure as well.

Most of the exposure tests are looking at adult bees, and there is obviously another big phase of their life, which is larval development. That is happening inside the nest or inside a particular brood cell over a long period of time for solitary bees. That could be really important, because their environment is controlled by other individuals within the hive or their mother, who is provisioning that nest. They could be exposed to potentially much greater residues there, where they are being concentrated. We do not know what the levels are in nectar and honey in colonies. I think that would also be important.

Q216 Mr Spencer: Would you acknowledge, though, you have to be realistic in trying to achieve that?

Dr Raine: Yes.

Q217 Mr Spencer: Given the amount of agrochemicals that are on the market, given the amount of pollinators that are out there in the environment, to assess every combination that is possible would take us for infinity. So how—

Dr Raine: There would be huge time and cost implications, absolutely, so I think we have to be smart in the way we do this. We have to prioritise key combinations. I think we could either do that based on looking at the pharmacological properties of these pesticides and looking at how they act on the nervous system and/or we could look at what are commonly used as combinations in agriculture. It is not just pesticides; obviously we are talking about fungicides and miticides that beekeepers are using for honey bee control and for control of varroa.

Q218 Mr Spencer: Dr Connolly, I know you have been quite critical publicly. Do you want to come in?

Dr Connolly: Yes. I don't think a knowledge gap can just be ignored. This is an important knowledge gap. So many pesticides, so many species; this is a big technological challenge that we have to try to find a way to face.

The other thing I should like to add to this is the kind of super-chronic. Neonicotinoids are the nicotine for the humans equivalent. We know that nicotine is bad for us all eventually, but we know it is very quickly bad for pregnant mothers and for their offspring. There is a neuro-developmental consequence, and a lot of diseases have been associated—sudden death syndrome, ADHD and so on. These things may be happening on a bee as well, so we have to think more long term over multiple years about how the bees are surviving. Are they as intelligent when they grow up in this way as they would have been before? As you would expect from a scientist, it is not easy; it is a very complicated story.

Q219 Mr Spencer: But it is possible to extrapolate results to draw comparisons to other pollinators or combinations of chemicals?

Dr Connolly: It is possible, because we know where the targets are and it would not be too difficult to clone all the targets. It is more difficult to express them and work with them in the cell lines, but we can at least identify what the targets are. We can compare them, and using biophysics you can see where the neonicotinoid works in the receptor. You could probably identify which species are likely to be affected and which ones may get away with it.

Professor Potts: Just one quick point to add to that: I completely agree with you, Chris, but an additional part of that is the extent of exposure and the fact that the ecology of so many pollinators is so completely different that you get very localised pollinators that will only go in a very small area around their nest while other ones travel large distances. In addition to understanding the physiological biochemical part, we need to understand how they interact with the environment and we need to pick representative species or model species that can help us get into that. I don't think we need to have an excessive number of model species, but we need to cover those major functional groups.

Q220 Mr Spencer: The previous panel talked about funding and the fact that the chemicals industry was funding a lot of the research and testing. They were not over-enthused about having to do that. Do you view it cynically, frankly, that they are funding this research? Is it good science that they are pumping the money in, or should we read anything into the fact that they are funding it?

Dr Connolly: Is that a rhetorical question? It seems quite clear that it would be inappropriate. Not to say that they are fiddling any data or anything, but it is just naturally inappropriate to test the safety of your own compounds.

Q221 Mr Spencer: Would all three of you go as far as to say that the public purse should pick up the tab for that research?

Dr Connolly: Not necessarily. It can be organised so that if the product is licensed, then the user of the product could pay, but not directly by relationship with the academic. It could be siphoned via BBSRC, NERC and so on, and so it becomes truly independent but still answers the questions that need to be answered, and is fair to everybody. Then the industry can defend their statement data and say, "Look, it is totally independent. It is safe."

Dr Raine: Can I jump in on that? I think the key issue there is really about scrutiny of information, and Simon already alluded to this asymmetry of information. Dr Little was talking about levels of neonicotinoids in crops as it goes through and, if we could get access to data more readily, that sort of data would be fantastically useful for us to know what are the residues in pollen and nectar. These kinds of data are there, and it is very hard for us to see them, so we cannot really know how useful the data is until we can see it. In terms of the regulatory process, I think it does not matter necessarily that the industry is paying for it; it is who is doing the research. If you could divorce those two and it could be done by someone independently, that would presumably be better.

Q222 Zac Goldsmith: Just on this point before we move on, I think all three of you were here for the previous session so you heard the evidence that we had and you heard that both companies supported moves towards freeing up or opening up the research that they do and getting over the issues that we heard about commercial confidentiality and sensitivity and so on. They did not go into much detail about what that would mean, but the principle was clear. I am assuming that is something that you would support. If so, does that cover the concerns that you have just raised? If you had access to the data that is currently inaccessible, would that allow the level of safeguards that are necessary? Is it enough?

Dr Connolly: The biggest barrier that I have to make the research that we do relevant is to know what the concentration that we should be looking at is. At the moment we are relying on data from the US to do so, and I am sure we have the data here to address the issues. But still—

Q223 Chair: You say that you are sure that we have the data here. When you say "we", who do you mean?

Dr Connolly: I guess the agrochemical companies presumably have the data to say what the exposure—

Q224 Caroline Lucas: If that were released, I don't see why that would compromise their competitive issues, if you are just talking about the concentration of the dosage and so forth. It seems to me that what we are trying to get at is to what extent are the companies hiding behind the shield of commercial confidentiality and how much is it a genuine issue. If you are talking about things like the intensity and concentration of a dosage, then I cannot see why that being in the public domain would compromise commercial issues. Can you?

Dr Connolly: I agree—I cannot see.

Q225 Chair: Do you all agree?

Professor Potts: Yes. I can understand the need to protect the actual formulation specifics, but it is actually the impacts that we are interested in as academics.

Q226 Chair: Can I come back to you on that, because I think you were in the room when we had the evidence just now? The point is made all the time about commercial confidentiality to stop other competitors, but is that really a valid reason not to go ahead with that? What would the companies have to lose if other competitors came forward as a result of their work being in the public domain?

Professor Potts: I think if it is a novel formulation that would be something, but I do not see why the actual impacts would put them at a commercial disadvantage. But this is not particularly my area so I am not—

Q227 Zac Goldsmith: I want to try to be clear. I was very clumsy in my last question. Following on the question you had from Mr Spencer about the contribution and the fact that the industry is paying, effectively, for this work, on the one level I think you said that you thought that was inappropriate. My question to you is, if you had a system that was much more transparent, if you were able to ensure that the research that they paid for was publicly accessible except for those bits that might genuinely compromise commercial advantage, would that suffice? Is it really an issue of transparency, rather than of funding?

Professor Potts: Yes, I would say it is an issue of transparency.

Q228 Zac Goldsmith: Would you be happy with the status quo if the data collected and generated were more publicly available?

Professor Potts: Providing it is everything except the commercially sensitive aspects, and that needs defining very clearly so all data is put forward, that would be—

Q229 Zac Goldsmith: Is that something you also agree with?

Dr Connolly: More or less. Obviously it is not the gold standard.

Zac Goldsmith: My last question was very clumsy, and I wanted to—

Q230 Mr Spencer: I think this is really important. Clearly, the chemical formulation is the bit that drives the whole thing forward. I wondered how useful that data would be if it did not include that chemical formulation.

Chair: In terms of disclosure, you mean?

Q231 Mr Spencer: Yes. If you say, "Here is everything apart from the most important bit," how useful is that, frankly?

Dr Connolly: It is still quite useful. Obviously, in the case of the neonicotinoids, imidacloprid is a partial agonist on the receptor and clothianidin is a full agonist on the

receptor. These can have quite different effects. As it transpires from our research, they actually do the same thing, but it could be important. So, yes, there is some information that you could potentially lose, but maybe that could be released under some kind of confidentiality agreement so that work can be done.

Q232 Caroline Lucas: From the evidence you have given so far, you have been talking about the importance of understanding that some of the effects from neonics is long term, some of the issues are around the cocktail effect of several chemicals interacting together. Are you able to say anything from that perspective about the German study that Bayer was citing in the previous evidence session in terms of how rigorous you think that study was, bearing in mind your concerns around cocktail effects, long-term effects and so forth? Maybe you don't think it is a fair question.

Dr Raine: This is the Genersch study we are talking about?

Caroline Lucas: Yes.

Dr Raine: I would not feel confident to comment on that at the moment, sorry. I could have a look at it and send some comments if you prefer.

Q233 Caroline Lucas: That would be helpful if it is not a huge job I am asking you to do. I am not quite sure how big a job I have just asked you to do, but that would be very good.

Dr Connolly: I do think the combination is important. The flu virus does not kill people but it can kill weak people. It may be if you look at these neonicotinoids on fully well fed, nourished bees that are really strong, there may not be very significant effects, and that is good. But it may be if there is something else happening, like they have *Nosema* infection, a gut parasite, or the viruses from the varroa or the varroa themselves, they now may succumb to otherwise fairly innocuous exposure to pesticides. It is really fundamentally important.

Q234 Caroline Lucas: Going back to the Gill study, which I think you were involved in, Dr Raine, could you just say a little bit more about the importance of combinations of chemicals interacting together? I know you did start to answer as well the question about how practical it is to be able to recombine 100 or more different combinations. Could say a bit about maybe if computer modelling can help us with that or what other practical ways there are of actually being able to assess it?

Dr Raine: Briefly, yes. That study was done in my lab. We aimed to do a field-realistic trial with early stage bumble bee colonies and expose them to two pesticides. We chose the neonicotinoid imidacloprid and we chose a pyrethroid, lambda-cyhalothrin. What we were trying to do was make it as realistic as possible, with the bee colonies in the lab so we could monitor their development and growth there. We were monitoring their foraging behaviour, and they were able to forage outside and collect all their pollen and most of their nectar from real flowers. We had different colonies in different treatment groups that were exposed to either one chemical or the other or both together, and obviously controls that were not treated at all.

In terms of the neonicotinoid effects, we found that there were very strong effects at both the individual worker level and also the colony level. What we found was in terms of the colonies that were treated with the imidacloprid, they sent out many more workers to go out foraging and each of the foragers was much less effective at bringing back pollen. They seemed to be struggling to meet the pollen demands of the larvae, and that had a knock-on effect over a period of two to three weeks in terms of colony growth. We saw that was much reduced compared to the control colonies. In small bee colonies like bumble bees at the beginning of the season you can see that kind of feedback effect from individual behaviour to

colony level effects. That is much harder to see in something like a honey bee colony, which is much larger and there is much more redundancy in the system. In terms of combination effects, we found that with the pyrethroids and the neonicotinoids together, those colonies suffered the most and they performed the most poorly in all our measures of behaviour. Also, two of those 10 colonies failed completely, so there was a significant rise in colony failure rates.

In terms of combinations, it is a very significant problem. We have done a first step here looking at the combination of two pesticides. Clearly, it would be nice to do more and you build up to a very large number of colonies very quickly, which becomes unfeasible. I think we have to be clever about targeting and maybe using computer modelling along the lines of what Chris was talking about with biophysics of looking at different modes of action and saying, "Well, is there likely to be an active or a synergistic effect with the combination of these pesticides, and, if so, would that trigger a more extensive series of testing compared to a single pesticide, which may not?" I think the different approaches could be used in combination. Computer modelling is one approach; lab studies are really important as well for us to ground truth what is going on and then taking that to the field as well, so multiple-level testing is probably important.

Q235 Caroline Lucas: Thank you. Just one last question. DEFRA and indeed others, and maybe even the people who were on the panel just before you, have criticised recent scientific evidence or scientific studies saying that the doses of the neonics were not field-realistic, as they call it. How justified do you think that criticism is?

Dr Raine: Talking about the Gill study primarily, we are confident that they are field-realistic dosages. We have used published data on the levels of residue in nectar and pollen of crops as a guide to that, and for the pyrethroids we have used the guideline preparation instructions for application to crops. That is as close as we can get to field-realistic. If there are better data out there, that would be really useful, and we could do better with better data. I think the same is true for the Whitehorn study. They used published data to inform their exposure levels. But it is not just about the concentration. It is also about the amount of active ingredient that bees are exposed to. In our study, they had a small amount of sugar water with a neonicotinoid in it, but they were able to go and collect nectar from real flowers as well. They were not getting exposure all the time, so our effective level of exposure was probably much lower than our concentrations in the treatment solutions suggest. Similarly, in the Whitehorn study, that was the only thing they could choose for two weeks, but after that they were not exposed to it, whereas if they were going out into an oilseed rape field they would be exposed potentially for four to five weeks in a flowering period. It is about not just the amount you get but what period you get it over, and they may be different effects, absolutely.

Dr Connolly: Could I add on here to give you the concentrations? Bayer have published to say there is one to five parts per billion present in the nectar and the pollen. This has been supported by Bonmatin and Blacquiere. These are major studies, and we are now talking about the Henry one. They used 27 parts per billion, a bit higher. Nigel's lab used 10 parts per billion. Whitehorn used six parts per billion in the pollen plus 0.7 parts per billion in sugar. Our research looking at the electrophysiological function of a bee brain in a lab gives major effects at only 2.5 parts per billion. So these are the right ballpark relevant concentrations to be looking at. Whether these things all are relevant in a complicated ecological system, and over a long period of time, and whether it is causing the bee decline are quite different questions.

Q236 Martin Caton: Dr Connolly, you have moved from mammalian neurology to looking at bees. What made you make that fairly fundamental shift?

Dr Connolly: An important part of this issue is the neuro-pharmacological issue. The neonicotinoids target the nicotinic receptors in the central nervous system and peripheral nervous system of all animals. Currently, I have been working on humans and I am interested in how information flow gets disturbed, how it can lead to neuro-degenerative disease, epilepsy and so on. This is really the same science; it is just a different animal. It is not really a giant step, but it is an important step. We have an enormous, robust scientific base in this country and it is time that we applied that to deliver the impact into the areas that are really important now because we have the growing population. This is not a problem that is going to go away. This is a problem that is going to get bigger and bigger.

Q237 Martin Caton: When will your insect pollinator initiative study be published?

Dr Connolly: I have an early draft paper here, which you can have as evidence. It is really down to the review process. You might be interested to hear we got knocked back because our idea that two pesticides might work in the brain sequentially and add to the toxicity was classed as being rather obvious, so it is not such a long shot as people might consider. I have had a letter from the International Union of Basic and Clinical Pharmacology who say they are going to submit a letter to *Nature* to say that this is blindingly obvious basic pharmacology, that compounds that target pathways that converge will work together. Of course, on top of this we have what we call in pharmacology off-targets, and these are the unknowns that we cannot actually predict and they can only be determined empirically. So I am doing the same stuff I always did, really.

Q238 Martin Caton: If it is neonics contributing or causing neurological dysfunction in bees, might it be possible to fine tune this effect by changing the chemical nature of the neonicotinoids?

Dr Connolly: It is possible, and Simon has touched on the reason why this is difficult, because of the complex ecological systems in a number of species. I think it may be possible if you decided which species you were going to target and which ones you were going to protect and then ignored all the others. If you made those decisions, then it may be possible, but hard.

Q239 Martin Caton: Is there anybody actually working on this?

Dr Connolly: The only people I know working on this are trying to make them more and more effective on pest species but not less and less effective on the beneficial ones.

Q240 Zac Goldsmith: I have a question that will be very quick. You would have heard the evidence before where an enormous emphasis was put on, first of all, loss of habitat, which I think is a suspect reason, but also increasing strength of varroa. As far as I understand, that only applies to honey bees, does it not? How many of the other pollinators are affected by varroa, if any?

Dr Connolly: Well, they do affect bumble bees, don't they, Nigel?

Dr Raine: No.

Dr Connolly: Don't they? Oh.

Dr Raine: It is going to be very restricted, so it is not—

Q241 Zac Goldsmith: So it can't possibly be the reason, then, for the decline in the pollinators?

Dr Connolly: There may be different major drivers.

Professor Potts: I think one of the really telling things is, as for many other components of biodiversity where we have had declines, the scientific community has

actually managed to nail the drivers. The fact that there has been a lot of ongoing research and just now we are starting to pick it apart is quite indicative that there is probably more than one driver. Certainly, there is very good evidence that habitat loss, not only in the amount of habitat but whether that habitat is fragmented, and the general quality, so that might be in terms of falling resources and nesting opportunities for all pollinators, not just bumble bees but hoverflies and so on. Disentangling all those is difficult and we are just now starting to get the studies that are bringing together two and we have not even got to the stage of bringing together three. We have only done some pairs out of maybe the half dozen potential drivers of the losses that we are seeing. It is not surprising it is a tough one for the scientific community to crack, but we have very compelling evidence that certainly single drivers, as in habitat loss, are part of the story and potentially the combinations of habitat loss with potentially pesticides, potentially pathogens, could be a big part of the story as well.

Q242 Zac Goldsmith: I suppose that leads us to the next question. Given what we do know, given the scientific documents and research that already exists, do you think that if DEFRA was adhering to the precautionary principle it would at least put a moratorium if not a ban on neonicotinoids?

Dr Connolly: I would say that there is good evidence of clear negative impact on bees, so I guess the answer would be yes. But at the same time what would be the alternative, and that has to be weighed up in this answer. If the alternative is do not use them and, as Julian Little says, that it is a disaster for the crops, then that has to be weighed in. If the alternative is something like Fipronil—and we all know that is quite bad—which is worse? I prefer that we compared the chemical choices and made a choice.

Q243 Zac Goldsmith: Do you have a view as to whether or not such a moratorium or ban would lead to worse additional outcomes?

Dr Connolly: I don't know that the crops wouldn't do just fine, but then I am not an expert.

Professor Potts: In the short term a moratorium would have huge implications for farmer livelihoods, for food security. A moratorium obviously would be good for pure conservation reasons because there is no doubt that pesticides do cause harm. The question is, is that having a population level effect—is it actually meaning that populations are going down? Does that then feed into loss of pollination services both for crops and for wild flowers? Should there be a moratorium on conservation grounds alone? Probably; for the greater, wider issues, including food security and economics, probably no.

Q244 Caroline Lucas: Why are the two separate? Sorry, but why isn't food security connected to that wider point? It is not conservation over here and food security over there surely. Surely the two are connected.

Professor Potts: No, absolutely: clearly, they are interacting, and pollinators are part of the food security picture. They provide more than half a billion to the UK economy agricultural sector every year. However, the instantaneous loss of effective chemical control is certainly going to reduce crop productivity unless other chemicals can be brought in very quickly and we do not know whether they will be as safe or less safe than the current regime. The productivity would go down and that would have consequences for the overall economics. It might in the short term help the pollinators but what we need, in my opinion, is a longer-term phased reduction in all pesticides, not just neonicotinoids, and increasing uptake of more IPM strategies, things like biocontrol, better crop management and so on. A lot of those tools are out there and if we are going to get co-benefits of good production, food

security and good environmental quality, then we need to be a lot smarter about the way we intensively farm.

Q245 Mr Spencer: Some of those biotechnologies that are out there and available, are they licensed in the European Union or are they just available in other parts of the world?

Professor Potts: Can you be specific about the biotechnologies you are pointing to?

Mr Spencer: I am thinking that you talked about new technologies—I hesitate to use the word "GM" because it takes us in a direction I do not want to go in—that are available to us. There is a reluctance to license some of those technologies in the European Union when they seem to be available in other parts of the world. I wondered if it is realistic to say that we can use those technologies here if they are not licensed.

Professor Potts: There may be some additional cost to farming, so I am thinking along the lines of better dust control for neonic drilling and I think in Germany they have a more stringent set of criteria for what farmers have to do to reduce the dust. I am also thinking more about what I call soft biocontrol technologies, so capitalising on natural enemies, those natural predators of pests, and those are well established around the world. I think they are used in some systems in the UK, but I think we have a great potential to increasingly use those to try to reduce our reliance on synthetic inputs, which are going to increase in cost anyway because of energy costs and so on. It is going to be even more difficult for farmers to be able to afford to use all these treatments, so some sort of combination would probably be the way forward in the medium term.

Dr Connolly: Can I just say one thing on this? One thing that we should consider is: do we need to allow the pesticides to be used in gardens for recreational use? Apparently, golf courses use five times as much neonicotinoids than the farmers do—I got this information from *A Spring Without Bees* so I don't know if it is accurate or not—and that US householders put as much fertiliser on their lawns as the whole of India does to produce their crops. If we can take away pesticides from gardens, we create a nice nature reserve all over that would provide us a sink from which animals might be able to re-emerge. At the moment, if they are being poisoned everywhere, we do not have this opportunity. I don't know if Simon thinks that is—

Chair: I am very conscious of time.

Q246 Mr Spencer: You are going into an area where you are going to have to draw a line between what is a garden, what is an allotment, what is a smallholding and what is a farm. Could you draw that line for us?

Dr Connolly: I think that is your job, isn't it? I don't how to draw that line. That would be difficult.

Chair: All right, okay. Zac, have you finished?

Zac Goldsmith: Yes, thank you.

Chair: Okay, I am just going to ask Simon Wright, and then I think we will move very swiftly to our next session, if that is all right.

Q247 Simon Wright: Professor Potts, what in your view is the most accurate, useful way of measuring of the economic value brought by pollinators?

Professor Potts: There are multiple ways of valuing and there are lots of different figures floating around. I hope that I can try to clarify. I think there are three basic ways. The first way would be the direct contribution to UK agriculture. That is basically looking at how dependent some crops are on pollinators. If they are highly dependent, you know a proportion of that crop relies on pollination. Something like apples is almost 90%, whereas wheat would be zero. If you sum that all up for all the crops, the most recent published figure is £510

million per annum for 2009. The most recent unpublished is it has gone up to £603 million for 2010. There is a definite trend for increase because of more demand for biofuels, more demand for locally grown fruit and vegetables, which are very dependent on pollinators, and also there are new crops coming in, things like blueberries, which are very pollinator dependent. That would be the agricultural sector, but that is actually a very considerable underestimate because it does not take into account things like the contribution that pollinators make to forage crops like clovers that are very important for meat and cattle and dairying. It does not take into account ornamental flower growing. It does not take into account seeds. It does not take into account allotments and home-grown food, which have a value. We are talking £603 million per annum plus certainly some more. So that would be the agricultural sector.

A different way of valuing it is how does the public value pollinators in terms of their cultural value. That would be things like those iconic bits of nature like bumble bees. People like flowery gardens and they like to see flowery meadows. We did some work, and other people have done work as well, that would put the willingness to pay by the general public in the region of £1.3 billion to £1.8 billion per year. That is nothing to do with the agriculture. That is just valuing it because of its conservation, its aesthetic appeal. However, there is a big caveat with that. When you do this sort of research it is very easy in an interview or a survey to say, "I would pay so much per year to support pollinators because I believe in them". If you actually ask people to dip into their pocket, it is a very different matter. But that gives you an idea of the kind of figures we are talking about. It is even more than we estimate for agriculture alone.

Very quickly, the third valuation, which we do not have a figure for, is the fact that these wildflowers that pollinators are essential to maintain, contribute a lot to other, what we call ecosystem services. Having healthy plant communities in the UK means that we have healthy soils both in terms of fertility and its structural integrity. It helps with things indirectly like good quality water. These plants help purify some of the water. It also contributes to recreational and other kinds of services. Indirectly, pollinators make a huge contribution to these other ecosystem services, but to put a figure on that is just not possible. We do not have the data or the methods.

Q248 Simon Wright: How close are we to having the methods that we would need to inform a market-based approach that would capture the cost of the detrimental impact of pesticides and also reward choices that favour pollinators?

Professor Potts: That would be going along the lines of "the polluter pays" where we would need to quantify the impact of insecticides and then come up with what is the potential financial loss of a particular pesticide—in theory, that would be exceptionally doable. In terms of the datasets and the methods, I think we are a long way off that. It is something much discussed in Europe at the moment. It goes along with the payment for ecosystem services, which is something that is very much on the horizon for the UK as well. The problem is we have not quantified three steps. Exactly how much do pesticides impact on pollinators? How much do pollinators then deliver or reduce the amount of pollination they do? Then how much does that pollination impact on the economics? We are quite fuzzy on the last two, and we are only just starting to make headway on the first. It is a great idea in theory, but I think we are quite a long way off being able to do that, except for having a very simple tax or something equivalent to a tax on pesticides where it would go into a communal pot, but that is also probably not a good fiscal instrument. I cannot imagine many people buying into that.

Chair: There we must leave it. Thank you all very much for coming along this afternoon. We shall look very carefully when we get the written evidence of what you have

provided to us this afternoon. Thank you very much indeed. We would like to invite our next witness to come forward.

Examination of Witness

Witness: **Georgina Downs**, UK Pesticides Campaign, gave evidence.

Q249 Zac Goldsmith: Do you want me to get going with the first question? We have so little time before the vote, if there is a vote, so I am going to be very rapid here and ask you to be as quick as you can in your answer. I am really sorry about that, but time is not on our side. In the evidence that you provided to the Committee, you said, "Pesticides can cause a wide range of both acute and chronic adverse health effects." Can you briefly give us what hard evidence you have that pesticides have and do cause a wide range of adverse health effects?

Georgina Downs: Yes. Can I just clarify with the Chair first of all what happens? Do people go off to vote and then come back, or is it literally I have got three minutes?

Q250 Chair: No, we are expecting a vote in the House of Commons soon. What we are hoping is that we will be able to have sufficient time for the minimum of questions that we have and to try to establish that we will be able to have a quorum if we then have to come back after the vote. At this stage, let us assume that we perhaps have a good 10 minutes and then we will see where we are, if that is okay.

Georgina Downs: Okay. I think it is easiest to go through the acute and chronic effects separately. To start with acute effects, these are adverse effects that occur shortly after exposure. They can be local at the site of contact or systemic effects. I detailed at paragraph 2.15 in the written evidence the acute adverse health effects in residents and other members of the public that are recorded annually in the Government's own monitoring system as a result of exposure to agricultural pesticides in crop spraying. I won't have time to go through them all, but just to list a few, there are chemical burns including to the eyes and the skin; rashes; blistering; throat problems including damaged vocal chords; sinus pain; respiratory irritation; breathing problems; asthma attacks; and then headaches, dizziness, nausea, vomiting, stomach pains and so on. I reiterate that these are acute effects from the Government's own monitoring system, although it should be noted that the current system involves a very significant degree of under-reporting. For the last 11 years the campaign I run has received many reports of the exact same types of acute effects in residents and also children attending schools near sprayed fields as well. Just to point out about acute effects, the correlation between acute effects and exposure is usually quite straightforward because the manufacturers' own safety datasheets can have the listing for those types of effects, both the local and systemic effects.

In relation to chronic adverse health effects, there has now been over 60 years of scientific and medical evidence showing pesticides associated with a wide range of chronic adverse effects on human health. And just to give a couple of brief examples of such studies, one reputable study published in March 2009 found that exposure to just two pesticides within 500 metres of residents' homes increased the risk of Parkinson's disease by 75%. Another study—

Q251 Zac Goldsmith: Which were the chemicals?

Georgina Downs: Paraquat was one. I think Maneb was the other, but I can check that and get back to you. I think it was Paraquat and Maneb. Another study that involved nearly 700 Californian women showed that living within a mile of farms where certain pesticides are

sprayed during critical weeks in pregnancy increased by up to 120% the chance of losing the baby through birth defects. In fact, a comprehensive pesticides literature review from Canada in 2004 on the chronic effects of pesticide exposure reviewed at that time over 250 in-depth studies from around the world and found consistent evidence linking pesticide exposure to brain, kidney, prostate and pancreatic cancer, as well as leukaemia, non-Hodgkin's lymphoma, neurological damage, which is usually irreversible, Parkinson's disease and other serious illnesses and diseases. The review also found critically that children are particularly vulnerable to the effects of pesticide exposure and identified increased risks for a number of illnesses and diseases, including kidney cancer and acute leukaemia. It is important to point out that over the last 11 years the campaign that I run has received many, many reports of such illnesses and diseases in residents living in the locality of sprayed fields. Again, just the same as acute effects, in a number of these reports the individuals involved do have confirmation from either their doctor or other medical professional that the chronic effects are caused by pesticides, especially when the chronic effects are related to irreversible neurological damage and injury. If I could just make one more point on this question—

Chair: You have about eight more minutes.

Georgina Downs: Just one final point on it is that, as pointed out in the written evidence, the Government has repeatedly failed to take action when faced with, including in its own monitoring system in relation to acute effects—there is no monitoring system as such regarding chronic—they have failed to act on the evidence of harm as well as the risk of harm to human health from crop spraying under the current policy and approvals regime, yet European legislation requires that pesticides can only be authorised for use if it has been established that there will be no immediate or delayed harmful effect on human health. It also requires a proactive approach to reviewing authorisations after approval, including that authorisation shall be cancelled and pesticides prohibited where there is a risk of harm to human health. It is based on the risk. It does not even need to be that you have been damaged first. It is based on the risk of harm.

Q252 Mr Spencer: If any of that was true, surely anecdotally farmers' children and farm operators, that demographic, would demonstrate an enormous increase in those conditions.

Georgina Downs: Well, actually, going back to the British Medical Association report in 1990, that actually highlighted quite a lot of different studies back then, and this is going way back to that time, of different cancers, lymphomas and leukaemia in farming and operators. That is from the British Medical Association's own report. I should also point out I don't just get reports in the campaign of residents and other members of the public. Farmers, sprayers, ex-sprayers, ex-farm managers also contact the campaign with chronic health problems as well, particularly neurological problems and cancers.

Q253 Zac Goldsmith: Has there ever been a study into the health of farmers?

Georgina Downs: Oh, yes, there have been loads.

Q254 Zac Goldsmith: That would make the point that Mark Spencer has just—

Georgina Downs: Yes, there have. The British Medical Association's report highlighted all the various different studies at that time, and that was 20 years ago. There has been a load more since then, and I can obviously provide the Committee with information on that after the session.

Q255 Zac Goldsmith: We have heard from a number of different sources that if systemic neonicotinoid pesticides were removed, were banned, in the UK that one outcome

could be that farmers would opt for older and worse chemicals as a replacement. First of all, do you accept that and, secondly, do you believe that that would, therefore, represent a regressive step?

Georgina Downs: The first point to make is that the use of systemic neonicotinoid pesticides seed treatment does not currently preclude the spraying of other insecticides on such crops. Therefore, other insecticides are still being sprayed. In any event, the reality of crop spraying in the countryside is that there are already innumerable mixtures of pesticides being applied to crops, and not only insecticides but fungicides, herbicides and other agricultural chemicals, on a regular basis year after year. As I pointed out in the written evidence, approximately 80% of pesticides used in the UK per year is related to agricultural use. Therefore, in answer to the question, the campaign I run, the UK Pesticides Campaign, supports a ban on neonicotinoids but I would stress the fact that the problems with pesticides in general is obviously much wider, especially considering the risks of acute and chronic adverse impacts on human health from the innumerable mixtures of pesticides currently used in agriculture. Therefore, it is a complete paradigm shift that is needed to shift policy away from the dependence on using pesticides altogether to the utilisation and prioritisation of non-chemical methods. No toxic chemicals that have related risks and adverse impacts for any species, whether it is humans, bees or other, should be used to grow food.

Zac Goldsmith: Okay, we should move on.

Q256 Caroline Lucas: On the risk assessment, do you think the current risk assessment process for pesticides is sufficiently transparent?

Georgina Downs: Obviously, it is not just a case of whether it is transparent, but whether it is adequate. I know that others have commented on the non-transparency side so I would like to very briefly focus on the complete inadequacy of the Government's current approach to exposure and risk assessment regarding human health. I have briefly detailed in the written evidence how the Government's current short-term bystander model does not and cannot in any way cover the exposure scenario of residents who live—and the bell is ringing.

Zac Goldsmith: It is not our bell.

Georgina Downs: It is not? Okay. It cannot cover residents living in the locality of sprayed fields as residents' exposure is chronic, it is cumulative, it is to mixtures of different pesticides, all the different exposure factors involved in the air, in precipitation, immediate drift, volatilisation, and the exposure for residents can go on for decades, like my own situation that is nearly 30 years now. The fact is that the Government has been approving pesticides for years without having assessed the exposures and risks specifically for residents living in the locality to sprayed fields and which the Government is required to do under the European legislation. This includes the astonishing fact that there is currently no assessment at all for babies that are near sprayed fields, for children that are near sprayed fields, for pregnant women and for other vulnerable groups. Considering how many millions will be living in that situation, that is an extraordinary situation. The absence of any risk assessment for residents means that pesticides should never have been approved for use in the first place for spraying in the locality of residents' homes, schools, children's playgrounds, nurseries and other areas.

Also, very, very briefly I will highlight the fact that in the written evidence I highlighted that the regulators previously failed to act on their own findings of 82 exceedances of the EU limit set for exposure, which is called the AOEL for short. In some cases, the AOEL was exceeded by up to 20 to 30 times over, which is an order of magnitude higher. In one case, based on the regulator's own figures, there would have been an exceedance of 95 times above the AOEL at three metres from the sprayer, which would have been well over 100 times at one metre and, of course, obviously some residents' homes and gardens are a metre or less

away from where the sprayer would pass. Just to point out that this product that found this 95 times exceedance, calculating on the regulator's own figures, is a product that is still approved in the UK until 2021. No action was taken in relation to those findings, and yet under EU legislation any exceedance of the AOEL, even by one time over, is supposed to lead to immediate action of authorisations being refused or trigger revocation if already approved. [*Interruption.]*

Q257 Chair: I am afraid that we do have a Division now in the Commons.

Georgina Downs: Shall I wait?

Chair: I am afraid that in terms of getting a quorum it is going to be difficult because of the timing, but can I just say to you that we have the written evidence that you have provided us with, and that is part of the hearing. I think that in the interest of time we are just going to have to bring our session to a close this afternoon .