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HOUSE OF COMMONS

ORAL EVIDENCE

TAKEN BEFORE THE

ENVIRONMENTAL AUDIT COMMITTEE

INSECTS AND INSECTICIDES

WEDNESDAY 30 JANUARY 2013

DR JULIAN LITTLE and DR CHRISTINA GARSIDE

PROFESSOR VYVYAN HOWARD

Evidence heard in Public Questions 372 - 480

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Oral Evidence

Taken before the Environmental Audit Committee

on Wednesday 30 January 2013

Members present:

Joan Walley (Chair)
Peter Aldous
Martin Caton
Zac Goldsmith
Mark Lazarowicz
Caroline Lucas
Dr Matthew Offord
Mr Mark Spencer
Dr Alan Whitehead
Simon Wright

Examination of Witnesses

Witnesses: **Dr Julian Little**, Government Affairs, Bayer CropScience, and **Dr Christina Garside**, Environmental Safety Manager, Bayer CropScience, gave evidence.

Q372 Chair: I formally welcome you both—and I welcome you back a second time, Dr Little. We appreciate your coming back, and thank you and your colleague, Dr Garside, for coming here today. The reasoning for our wanting to have you return to the Committee is some of the issues that relate to the evidence that you gave and also to the DAR report, which was the reason for the regulatory regime in the first place.

Before we get to that, I think everybody is aware that there have been various developments over the last few weeks, one of which is the European EFSA report. While we have you back in front of the Committee, we wish to try to tease out some of the issues there and to understand Bayer's position in relation to that. What weight should we accord to EFSA's recent assessment that neonicotinoids should not be used on crops that are attractive to honey bees? Have you had a chance to give us a response to that?

Dr Little: First things first: thank you very much for allowing us to give further evidence. I was going to start with an apology to the Committee for not being able to fully answer the questions around the environmental fate of neonicotinoids—I should be able to say that—hereafter known as "the neonics". Clearly, you will be aware that we followed that up by submitting further written evidence, and we are very happy today to come in with a little bit more experience in terms of people, like my colleague here, who knows a lot more about this subject.

On the EFSA report, what I would like to do is take issue with the particular question, because the EFSA reviews do not in any way recommend that neonicotinoids should not be used in flowering crops. That is not what it says——

Q373 Chair: Can I cut you short there?

Dr Little: Okay.

Chair: I did not suggest that. I was just asking for your response to the assessment that you have made and to EFSA's recent assessment.

Dr Little: All right, so what does the assessment say?

Chair: The assessment that Bayer has made.

Dr Little: Sure, of course, and we will give that very clearly. Essentially, our assessment is—and that of anybody, such as EFSA if you were to ask them—that what they were asked to look at was the difference between what we know at this precise moment about neonicotinoids and the regulatory system around it; what we may have to know if EFSA's suggestions on new regulations come up in the future; and what knowledge gaps there might be between those two areas of legislation.

Not surprisingly, you find knowledge gaps, as you always will if you decide in the future to increase the regulatory control. Our view is that those knowledge gaps are not insurmountable. We would argue that knowledge gaps are continually being plugged and always are, and that when you get a regulatory approval of a product, such as imidacloprid or whatever particular pesticide, that is only the start of the knowledge process—in other words, that is the point where you have the minimum information that you need for the regulatory approval of a product. We will continue and always have continued to increase our knowledge about these products, and we probably know more about neonicotinoids than most pesticides, let alone insecticides.

Q374 Chair: Can I take it from that that you support the assessment that EFSA carried out on neonicotinoids?

Dr Little: The assessments are what they are. Unfortunately, they decided to omit large quantities of information for various reasons. For example, despite being asked to look at field trials, they either did not have time or for whatever other reason did not do so. Therefore, from our perspective, they have not been able for whatever reason to include large quantities of data in their assessments, but you cannot complain about the assessments themselves.

Chair: No, but what I would like to know is that if you look at the graph in the EFSA report, there are many products where they have not been able to complete the risk assessment at this stage, but there are some that relate to honey bees where they have. So I am just trying to understand whether or not you support the assessment that they have made and where they have suggested that there are risks associated with it. It is a yes or no.

Dr Little: It depends on what you mean when you say "risks associated". If you like, we are talking about a knowledge gap between what we have and what we might have to have in the future. If those particular rules were implemented tomorrow, then, yes, there would be a risk, but—

Chair: Could I cut you short there? Does that mean that you would accept in those circumstances, where they have completed that risk assessment with the information available, that you would agree with their assessment that neonicotinoids should not then be used on crops that are attractive to honey bees?

Dr Little: No, because EFSA do not make a recommendation that these things are not used.

Chair: No—their assessment; I am not talking about their recommendation. If they have made an assessment that there is a risk, would you at least concur with their assessment?

Dr Little: We would agree that there is a knowledge gap. It does not mean to say that these things cannot be used. It means that there is a knowledge gap between where we are now and where we might be in the future.

Q375 Martin Caton: Just to clarify, yes, not a recommendation, but EFSA's own press release says, "Exposure to pollen and nectar. Only uses on crops not attractive to honey

bees were considered acceptable". I think that is fairly clear. That is not saying there is a knowledge gap; it is saying this use is not acceptable.

Dr Little: What it is talking about if you look into the report is that the knowledge gap in non-flowering crops is not as big as it is in flowering crops. So if those rules were put into place tomorrow—and they have not been addressed or validated by member states or the Commission—the knowledge gap for non-flowering crops would be smaller than it would be for flowering crops. That is what that statement is saying.

Q376 Martin Caton: So you accept the risk assessment that I have just read out?

Dr Little: I accept the fact that—

Martin Caton: These are not my words. These are EFSA's words.

Dr Little: I accept that the knowledge gaps in flowering crops are larger than they are in non-flowering crops.

Q377 Chair: But surely the point is that there are some knowledge gaps in the assessment that they did but there are some categories where they are quite clear in their assessment—where they say that there is a risk to honey bees. We just need to know first of all whether or not you would accept their assessment that there is a risk.

Dr Little: Okay; I would accept that if you only looked at the evidence that EFSA looked at, you would conclude that there was a risk.

Q378 Chair: All right, so the evidence that EFSA looked at, in Bayer's view, is not the whole picture? They were looking at the wrong evidence?

Dr Little: No, they were looking at an incomplete set of evidence, so there were large amounts of evidence that were out there that they did not use in their review. Because of the very strict method by which they looked at the evidence, they excluded wide areas of research, including things that we had in our own submission—for example, the German and French studies that looked into what happens in real situations. Hence, what you are left with, if you exclude those areas—as EFSA did, because they did not see that all of that information was complete—are studies that essentially show that insecticides have an effect on insects. So, yes, as a result, you would conclude there would be a risk issue.

Q379 Chair: Are you saying then—I have probably read you wrongly—that the European Commission should wait for actual harm to occur before managing that risk because you are saying that that information is incomplete?

Dr Little: No, of course not, because if EFSA had taken the full data set that was open to them, you would have seen all the work that has been done in real in-field situations, using real bees from real beehives in real fields. They decided not to include that data set in their assessment so, not surprisingly, what they ended up with is assessment of a risk. Also, in those reports, because that is not what they were asked to do, they have not looked at any stewardship, any mitigation or any other reasons why that risk assessment is not valid in a real agricultural situation.

Q380 Chair: All right. I am still not absolutely clear about what concerns you have about the regulatory regime that applies. You are somehow suggesting that because you do not accept the assessment that EFSA has made, because somehow it is incomplete, because it has not looked at all the different options, that somehow from your point of view that seems to relate back to the failure of the European regulatory system altogether.

Dr Little: No—on the contrary, the regulatory system looks at all things. So, for example, you will do your initial tests. For example, if you do a laboratory test where you

take a neonicotinoid and apply it to a bee, there is an effect on that bee. Whenever you see that effect, and you will tend to see it with whatever insecticide you use, you will have to do higher-tier studies, and then you will start to look at real-field studies where you can look at all the impacts on bee health, and those higher-tier studies, which were not looked at in these EFSA reviews, demonstrate that you can have safe use of these products in the field, in real agri-environmental situations.

Q381 Martin Caton: Sorry, I am going back to my first question. Are you saying that because they have not done these higher-tier studies you basically disagree with their statement that only uses on crops not attractive to honey bees were considered acceptable?

Dr Little: But I go back to my answer last time.

Q382 Martin Caton: Why can you not just answer that question?

Dr Little: Because the question is about what is it that EFSA were showing and what they were looking at was knowledge gaps. So what they do is they look at those knowledge gaps and say, "Are those significant enough in a particular crop for there to be a concern?" In a non-flowering crop, they are considering that those knowledge gaps are relatively small but in flowering crops, they are saying those knowledge gaps are relatively large.

Q383 Chair: Can I just try to look at it from a different perspective? In a way it could be argued that what the recent EFSA report did was to apply enhanced standards of environmental protection for bees in a way that was perhaps different or had moved on from the original regime that was first introduced—

Dr Little: I absolutely agree with that, yes.

Chair: —leaving aside whether or not the actual regime in the first instance was or was not fit for purpose. I wonder what views Bayer has of those enhanced standards that would have been underpinning the research or the assessment that EFSA has most recently made?

Dr Little: EFSA put together some ideas around what a regime might look like in the future, and that was published, I think, in the middle of last year. It is yet to be looked at in detail by the member states, who are involved in the regulatory process, to the point where they have yet to be validated, so there are some ideas about what you might want to do in the future. I think it is worth noting that if you talk about these knowledge gaps that occur between these theoretical enhanced regulatory—

Q384 Chair: Sorry, you keep on talking about knowledge gaps. I do not know what you mean by knowledge gaps. Can you just basically set that out for us?

Dr Little: All right, let us find an analogy. Essentially, you are in a situation where you are asked as a company to demonstrate that your products are safe, so they are safe to whatever level is being required from you. The European regulatory system is the toughest one in Europe and essentially if you can pass all of the tests in Europe, you can pass the test just about anywhere because they are seen as the gold standard.

The regulatory system moves on. Usually, it moves on incrementally, so there will be a recommendation that we would like to have a little bit more information here or a little bit more information there. What invariably happens is that you either already have that information and you submit it or you go away and get that information and submit it. Very occasionally—we saw it with the last major review of 91/414: there was a massive loss in products because there was a step change in what was being required from Europe. We lost something like two-thirds of all pesticide products and that was because either the data gaps

were so large that it was going to be difficult to plug them in the time allocated or it was not worth the effort of doing so. That is, the cost of doing so would be prohibitive.

Therefore, when you have those very big step changes, you see large losses in products, and that is always because of knowledge gaps. It is because you need to be able to plug where you are now to where you need to be next. In the case of EFSA's proposed guidance for insecticides, which was proposed last year, again the gaps are very big. This is where I come up with explaining about the knowledge gaps.

We have estimated that 96% of all pesticides, whether it is an insecticide or otherwise, would fail on that knowledge gap. There is a big knowledge gap between what we know now and what we would need to know in the future. So if your assumption that knowledge gaps equals a need to ban, the logic would therefore be that you would have to ban pretty much all insecticides and an awful lot of non-insecticides as well.

Clearly, that is not a sustainable situation to be in, and that is why we are very keen to make it clear that neonics are one class of compounds. They are very much in scrutiny at the moment, but they should not be treated any differently from any other product when looking at knowledge gaps and risk assessments.

Q385 Chair: Does that not beg the question that if that is happening, a company such as yours would need to have time to adjust what it is producing? It also fails to respond to the question that I asked previously, which was whether or not you agree with the tighter standards that were at the core of EFSA's most recent assessment.

Dr Little: The guidelines themselves are extremely onerous. They clearly have taken the gold standard further and would take a significant effort to bridge. Whether or not you agree whether it would be nice to have all of the information that EFSA has come up with, yes—and we are always working to get that information on all of our products. But to use that guidance as an excuse to take out particular products, for us does not seem appropriate or proportionate.

Q386 Chair: No, I was referring to enhanced standards.

Dr Little: Sure, and as I said, enhanced standards are part and parcel of our industry. What we are concerned about is any use of enhanced standards in a punitive way targeted at a particular type of chemistry.

Q387 Chair: So would you see this as being a punitive proposal?

Dr Little: As I said, if you were to—

Chair: If it were a proposal; if it were translated into a recommendation.

Dr Little: If it were to be translated untouched, it would be very onerous, but we would continue to move towards those standards, of course.

Q388 Martin Caton: EFSA in their report acknowledge that there are what you call "knowledge gaps" and that there are shortcomings because of data. They accept that, and they draw attention to where they find those shortcomings. Again, they did not hesitate to draw the conclusion that only uses on crops not attractive to honey bees were considered acceptable. They knew the knowledge gaps they had come across, but they still felt able to come to that conclusion and put that forward.

Dr Little: If those rules were put in place; I think in that particular case they make it clear in lines 35 to 45 or something like that.

Q389 Martin Caton: They are about the process of drawing up new rules, so obviously that is what they are going to be talking about, is it not?

Dr Little: Yes, that is fine, but we do not know what those rules will be once the member states and the Commission have looked at those proposals.

Q390 Martin Caton: Can you understand scepticism among observers of this? You have an independent body that reaches certain conclusions and does not make recommendations but makes an assessment, and then a company that has huge financial benefits from producing these products finds opportunities to criticise. Even if you just delay a ban or a moratorium, you are going to make a lot more money, are you not?

Dr Little: It is a difficult one for me to argue with, because by definition the fact that you have said that because we are a company we would say that—anything I say from that point onwards—

Chair: But presumably you would have done those—

Dr Little: —falls foul of that, but let me—

Q391 Martin Caton: I am just asking if you understand that there might be some scepticism when you criticise an independent body that comes to a conclusion.

Dr Little: All right: what is the consequence of a loss of neonicotinoids? Farmers will have to go back to the old way of doing things.

Q392 Martin Caton: There are questions on that. You have made those points before. I have taken up too much time, but you will have an opportunity to make those points.

Dr Little: What I was going to say was we have a very large portfolio of spray chemicals as well. What we would lose in one area, I have no reasons to believe we would not gain in another.

Q393 Zac Goldsmith: Just very quickly picking up on that point you made, before we go back to the specifics, are you saying that 96% of the chemicals on the market today are chemicals about which we do not know enough in order to be able to regulate them properly? That seems to be what you are saying. If that is the case, does that not suggest that the market is rushing way ahead of the science?

Dr Little: No—on the contrary, what we have is very good information on all products that are on the market. As I said, Europe has the gold standard on pesticide legislation. What I am saying, though, is if you make a massive step change in the regulations, and EFSA has come up with some ideas about what you might do, then an awful lot of products would have exactly the same knowledge gaps as neonicotinoids—in some cases would have bigger ones, because we know so much about neonics.

Q394 Mr Spencer: Dr Little, are you aware of any chemical product on the market where there is zero risk to insects?

Dr Little: If you are talking about an insecticide, it is purely about dose. It is purely about how much insecticide does that insect come up against. You will be aware that for a company to be able to sell an insecticide, you have to control your target species to a very large degree at the dosage that you will see in the field. So no, there are no products that have zero risk. The neonicotinoids are beneficial in many ways, especially on things like human risk assessment.

Q395 Mr Spencer: By conclusion, if you went to a system where there has to be zero risk, there would be no chemicals available to you?

Dr Little: Absolutely none at all.

Q396 Dr Whitehead: Just a brief thought on the question of what we do know and what we do not know: if we have gaps in knowledge, to what extent is it your understanding, particularly in terms of what EFSA has said, that the gaps are contingent or the gaps are primary? In other words, if you have a piece of knowledge that you do know about but is contingent on something you do not know about, clearly the validity of that piece of knowledge is undermined. If that is the other way around, then it is not. To what extent would you put the information that EFSA have put forward under either of those two categories?

Dr Little: That is a very good question. I will try to answer it. I think when you look at your normal data package and what you have to demonstrate, you have to demonstrate that you can control, as I have explained earlier, the things that you are supposed to control. You are not supposed to control the things that you are not supposed to control, so non-target organisms are out.

If it is an insecticide, it should not be having a big effect on either fungi or other sorts of biodiversity out there, so the specificity has to be there. On top of that, you are talking about safety—and that can be to mammals and non-mammals—and when you are talking about mammals, of course, you are quite focused in on humans. You would have to look at what is going on in the environment. All of these things are a huge data package that you have to submit, and then that is reviewed by a large number of people.

With these new proposals, it is about building on what we know, but it takes us a long way ahead of what we know in many cases today. It is not absolutely new information, although in this particular case what I will mention, and I mentioned it in the last evidence, I believe, is in this area of non-bee pollinators—because the focus in the risk assessment has always been about the impact on the honey bee. The honey bee is seen as the prime example of a pollinator, and they ask for information specifically on that in a large amount of detail.

The biggest areas from my perspective between where we are now and what EFSA are proposing lie in the area of understanding all non-honey bee pollinators. That is a huge area, and I think we discussed it at the last Committee meeting. You have one honey bee but 20 bumble bees or 200 solitary bees or maybe 2,000 other pollinators. Which ones do you select? That is where the knowledge gaps start to build up very quickly.

Q397 Chair: The Advisory Committee on Pesticides met yesterday, and we certainly have no way of knowing what the outcome of their discussions were or indeed what their recommendations will be to Defra Ministers. In terms of what you have just said to the Committee about EFSA, I wonder what your reaction would be in a hypothetical situation whereby they would recommend that there should be concerns about the risk to bees?

Dr Little: Sorry, risk to bees?

Chair: The whole risk assessment in terms of use of imidacloprid.

Dr Little: Yes, all right. Like you, I am not party to—

Chair: It is a hypothetical question.

Dr Little: —what is going on, but if ACP look at new evidence and feel that it changes their view on a particular type of product, then they have the ability to advise the Government accordingly.

Q398 Chair: But they would be looking at the recommendations of the EFSA Committee, would they not?

Dr Little: Yes, I guess they would, but the Advisory Committee on Pesticides is a group that advises the Government as to whether a product should be or should not be put on the market.

Q399 Chair: If they were to put forward a recommendation that, in this case, it should not be on the market—I am talking now particularly about imidacloprid—what would your response to that be, hypothetically?

Dr Little: To be honest, it is not my response. It is the Government's response.

Q400 Chair: But you are here representing Bayer.

Dr Little: Sure. We would be very disappointed, but we would look at the evidence that they took to come to that conclusion and if it—

Q401 Chair: But would you accept that evidence?

Dr Little: It is difficult for us to say. How do you mean "accept", because essentially the Advisory Committee is there to give advice? Do we accept that advice? It is not advice to us; it is advice to the Government. So if the Government looked at that advice and said, "As a result we will make a decision on whether this product is available in the UK", again, it is not for us to say that we disagree. Of course we disagree but if the Government makes that decision, then we have no choice but to comply. That is the way that these things work.

Chair: All right. We must move on. Martin Caton.

Q402 Martin Caton: All right. We do not know what ACP has done, but we do know what the Dutch Parliament did last week. It noted EFSA's risk assessments and proposed a European moratorium on all applications of neonicotinoids unless it is conclusively proven that they have no harmful effect on the health of bees. How do you feel about that political response to the growing evidence?

Dr Little: I think you used a very clear word—a "political" response. There is plenty of evidence out there that suggests that if they take all the evidence, there is no need for a ban.

Q403 Martin Caton: In the end it is going to be a political response. You have just pointed out to us that the ACP will make a recommendation to Government and Government will make a decision. That Government is by definition a political body. It is a decision made by politicians, so it is perfectly appropriate for a Parliament to come to the conclusions that the Dutch Parliament has.

Dr Little: Which is that unless you can demonstrate that these things can be used safely, they should not be used. We would argue that you can use these things safely.

Q404 Martin Caton: So you believe that you can, and presumably therefore you will provide to the Dutch Parliament something at least approximate to conclusive evidence that it is safe to use neonicotinoids.

Dr Little: What we have is evidence from real-life situations and we have discussed this both previously and this afternoon. When you look at what affects bee health in real situations, what it is not is pesticides; it is varroa, various viruses, habitat issues and nesting opportunities.

Martin Caton: That is a very selective choice of scientific evidence. Clearly, there is evidence pointing in the other direction—evidence that has led EFSA to reach the conclusions it has just very recently. But I think we will move on, Chair.

Q405 Chair: Is there any likelihood that whatever evidence you do have you might be prepared to put into the public domain?

Dr Little: The evidence around the safe use of these products is very well demonstrated.

Q406 Chair: The evidence that is demanded by the Dutch Parliament.

Dr Little: We absolutely will be supplying the Dutch Parliament with our views on the safe use of these products. Yes, of course.

Q407 Mr Spencer: I am asking you to speculate to a certain extent, but given that different member states have different rules in place for different products, I wonder why the Dutch Government called for a European ban rather than just one within the Dutch borders.

Dr Little: It is an interesting observation. You are right that there are a few countries that have taken steps to do something within their own borders. Up to now we have not seen any improvement in bee health as a result of those. But nevertheless, you are right; the Dutch could have made that decision. Why they chose to do otherwise, I think, is because they were aware that this was an area up for discussion.

There was evidence put on the table around Europe in terms of an impact assessment, and that impact assessment essentially shows that if these products were to be taken off the market, farming of certain crops becomes less competitive. Now, if you take it from a purely political perspective, there is a disadvantage in terms of competitiveness in taking off a product in one country if it is available to farmers in another. You ask me to speculate. It may well be that they took the view that it is better to lose these products across Europe rather than just in an individual country.

Q408 Chair: In fact, this same debate is going to be the subject matter of an inquiry, or at least a debate, by the European Parliament, is it not—the European Environmental Committee?

Dr Little: Sorry. Could you repeat the question?

Chair: Yes. In relation to the issue about the Dutch decision and the question from Mr Spencer as to whether or not that related to a wider European perspective, what I am saying is that it is the case, is it not, that the European Parliament will be having further debates on this? So, it is likely that it will be looked at in a pan-European fashion.

Dr Little: Absolutely. Yes, of course.

Q409 Mr Spencer: I wonder what commercial conclusions you draw from the EFSA revised assessments. Are you changing your business patterns or looking to change the direction the company is going in at all?

Dr Little: When we saw the initial proposed guidance last year, we of course made our own assessments of what knowledge gaps there are. Broadly speaking, if you look at the areas that EFSA looked at, we concluded that there were knowledge gaps. We then looked at the whole raft of information and said, "Okay, if those rules were to come in tomorrow, what extra would we have to supply?" In many cases, we believe that the higher-tier studies that have been done, and other areas to mitigate around that, mean that those knowledge gaps are nothing like as big as were suggested in those EFSA reports.

I go back to saying that whenever you get a regulatory approval of a product, it does not stop your process of understanding your molecules; we continue to work on them right up to the point where they are withdrawn from the market. That is a very normal process.

What I would say also is that where those knowledge gaps are quite big we are looking at mitigation, stewardship and making sure that these products are used in the safest way possible to minimise any risks as a way of mitigating against those knowledge gaps. Again I think that is important. If you say, "But we don't know what's happening specifically on this particular pollinator", then we will say, "Okay, what can we do in terms of how these products are used to minimise those impacts on that pollinator specifically?"

Q410 Mr Spencer: Obviously, you are suggesting you are engaged in R&D then, to fine-tune these products. But am I hearing you suggest that fine-tuning takes the format of how those chemicals are applied, rather than fine-tuning the chemical make-up of those products?

Dr Little: In some cases, it is about the formulation of the product. We talked in the last session about dust and things like that. So, how do you reduce dust levels to below 10% of what used to be out there? We believe we can do that. There is a lot of technical stuff in terms of making those products physically as safe as possible. But then we are looking at how these things are applied. Who are using what piece of machinery? How do we train farmers and professional contractors to use the products in a way that maximises their benefit and minimises their negative impact?

Q411 Mr Spencer: So in the light of the EFSA report, will you be investing less time in that sort of R&D, the same amount of time, or more?

Dr Little: Absolutely we will continue. But I would say that we have been doing that since the new proposed guidance notes came out. It is not as a result of these reviews, because we already had done our own review of what they meant. So that work has been ongoing for a long time but of course has been redoubled since we knew that new guidance was coming our way.

Q412 Mr Spencer: Right. Okay. Again, I know you do not want to break any commercial issues that you may have, but I just wonder if there is son-of-neonics on the horizon. How much investment is your company putting in to the next generation of products? Will we get to a point where this debate does not matter because there will be another generation of product that is safer and more effective?

Dr Little: Okay. The timescale for new-product development is somewhere in the region of 10 years. It is somewhere in the region of £300 to £400 million to bring something new. That is a very big investment and one that fewer and fewer companies are prepared to make, especially for products destined for Europe, simply because we have little confidence in what the regulatory system will be and we have no idea whether or not, by the time we get it to the market, we will be able to use it.

Neonicotinoids are ageing. For example, imidacloprid is already off-patent and is used by companies other than Bayer. However, they represented a massive step-change in human safety; traditionally, insecticides were quite a big problem in terms of their impact on operators and everything else. The development of a class of chemicals that had very, very low mammalian toxicity and were very good at controlling things at low dosage meant that, for us, the industry, farmers and the whole of the supply chain, neonicotinoids were seen as a major step forward. What is interesting is that we narrow down and narrow down what we consider to be acceptable for our products without necessarily recognising the huge steps forward that we have made over the last 30 to 40 years.

Q413 Mr Spencer: So, if we take a step back and speculate again and they are removed from the marketplace, what happens to European agriculture?

Dr Little: There is nothing major new coming through. Talking to all of the companies in this area, there are not these sorts of blockbuster insecticides coming through. What they tend to be are more narrow niche products that might work pretty well in certain circumstances but not in others. I think there is a real issue here—that we are losing technologies that are considered to be quite big steps forward elsewhere in the world and are making farming more and more competitive, being able to produce more and more food from

less land, from less water and from fewer inputs. We are in danger in Europe of almost enshrining some sort of museum agriculture.

Q414 Mr Spencer: So does Europe continue to grow those crops with old chemicals, or does it cease to grow them and import those products from other parts of the world? Which?

Dr Little: There are very few blanket bans or restrictions for countries anywhere in Europe, but what we are seeing is they become less competitive. You see a drop in yields. You see a return to using more insecticide in terms of sprays. That is the norm of what we are seeing. You do not necessarily see a massive reduction in yield because if you carry on spraying more and more, you can approximate the same level of control of the insect pests. We would always argue, though, that the trouble is that you are controlling insects that are living in a crop rather than just those insects that are eating the crop. That is essentially where seed treatments come into their own.

Q415 Chair: Just before I bring in Zac Goldsmith, you said—I think I got your words correct—that you have little confidence in what the regulatory system will be. I wonder if you could just slightly expand on what you really meant by that.

Dr Little: Of course. So, for example, I explained that previous major revisions of pesticide legislation led to the demise of a whole swathe of pesticides. The issue always is that if you take a gold standard and keep improving upon it, that is fine. But if you make a gold standard and say, "Next time we are going to make a platinum standard"—that is, these huge step-changes that are not happening elsewhere in the world—you end up having a regulatory system that is out of kilter with other countries.

We already see that with most places in the world having access to a lot more products than we see in Europe. Now, I personally absolutely support very, very strict regulation, but not to the point where, as we believe, you are taking out major advances in chemistry and agriculture with no discernible improvement in bee health—and that is our assertion—whereas other countries will continue to use these products.

Q416 Chair: Where does that lead you in respect of the value that you attach to the precautionary principle?

Dr Little: "The precautionary principle" is one of those expressions used essentially to damn anything that people do not particularly like. I think that the precautionary principle should have a proportionate addition to it that says, "Let us look at the reality of things"—if you like, "What do we know?" not always, "What don't we know?" What we know about these sorts of products is huge. We know an enormous amount about these products. So, it is not a question of looking at this and saying, "We don't know anything about these products. The precautionary principle says they should not be used". We have a long history of safe use of these products.

Q417 Caroline Lucas: I am concerned about where scientific evidence featured in your analysis. It seems to be much more driven by your assessment of what would happen to your commercial advantage, and indeed to pesticides on the market more generally, rather than saying that if we gather more evidence from the science that suggests that old regulations were not sufficiently stringent because we have new information and therefore we should build better regulations based on that new information. How does that fit into your very dismissive response about the precautionary principle?

Dr Little: I would argue that it is not dismissive at all. I think the precautionary principle has its place where you are essentially looking something that is very, very new. But this technology is not particularly new.

Q418 Caroline Lucas: More and more recent evidence is showing us that there is a bigger danger than was previously thought.

Dr Little: Yes, a bigger theoretical danger, but what we see in the field does not back up that those particular concerns are realistic in real agricultural situations.

Q419 Caroline Lucas: Does that mean that the whole EU regulatory system is based on flawed analysis?

Dr Little: No, it is based on risk. Again, whenever you do your initial experiments to show that there is a risk to bees of an insecticide, for example—and I should say that most insecticides fail the initial tests on bees because bees are insects and it is about dose—you have to do those higher-tier studies. It is very disappointing that EFSA either did not have time, or for whatever reason, did not look at the whole data set and preferred to narrow-in on to a very narrow data set that excluded all of the real agronomic situations.

Q420 Martin Caton: My colleague, Caroline Lucas, has largely dealt with this but you have just painted a caricature of the precautionary principle, have you not? As has been indicated, the precautionary principle is enshrined in European legislation and is not that, "Oh, we can see a bit of a risk, ban the stuff"; it is, "Look at the science". If the science reaches a certain level, then the precautionary principle kicks in. That seems to me a very sensible scientific approach, and it should not be dismissed in the sort of caricature that you just made.

Dr Little: I am sorry if you feel that is a caricature, but the fact is that in this particular case we have a huge raft of evidence that suggests you can have safe use of these products.

Q421 Martin Caton: That still does not excuse your dismissing the precautionary principle, and it certainly does not excuse your suggesting that the precautionary principle should only apply to new products.

Dr Little: What I am suggesting is that with new products there is less information. There are bigger information gaps. With products that have a history of safe use, there is a lot more information that suggests that these things can be used appropriately, in which case the precautionary principle does not really seem to apply unless there is convincing evidence to the contrary.

Martin Caton: Exactly.

Q422 Zac Goldsmith: I want to refer back to your previous evidence where you discussed the extent to which neonicotinoids accumulate in the environment. I shall read you what you said in answer to Caroline Lucas, who asked you about how long the chemicals persist in the soil. I quote: "But if you are looking at something like imidacloprid or clothianidin, you can be talking about a half-life of anywhere between 16 and, say, 200 days." Just before I go on with that, are you familiar with the bio-termite treatment called Premise in which the active substance is imidacloprid?

Dr Little: I am not aware of that particular treatment, but it does not surprise me that these treatments exist because termites are very sensitive to neonicotinoids.

Q423 Zac Goldsmith: Just before I go on, as far as I know, you commented on that very product on 30 March 2011 in *The Independent*. So it must have crossed your radar at some point. I am very happy to have you jump in at any point.

Premise was marketed in the US initially with a guarantee that it would kill termites for seven years, and I have a quote here from the promotional material. Bayer marketed Premise with this guarantee. I quote: "If Premise insecticide fails to stop termites at any time within seven years of initial treatment, we will gladly reimburse you for your product". So, I am interested in knowing how Bayer was able to provide such a guarantee.

Dr Little: I can certainly go back and find out, but what we are talking about here is obviously a termite bait with a formulation that keeps imidacloprid stable in that situation for a long time.

Q424 Zac Goldsmith: But with the same active ingredient that we are talking about now in the neonicotinoids.

What conclusion can this Committee draw from that guarantee about its propensity to accumulate in the environment, and how can we relate that—how can we reconcile that—with the evidence you provided us with when you last appeared in front of this Committee?

Dr Little: Okay. As I said, what you are talking about here is probably a block bait, or something along those lines, of a very stable form of imidacloprid—entirely different to what you would see in an agricultural environment. In an agricultural environment, you have active breakdown of these products in the soil. So it is an entirely different system.

Q425 Zac Goldsmith: How can you know that, given that a few minutes ago you said you were unaware of the product that two years ago you had written about in *The Independent*?

Dr Little: What I am saying is that I did not know the specific product that you mentioned. What I am also saying is that for something like termite control, you are not talking about termite control in a field. You are talking about termite control in a house, in which case you use an entirely different formulation that would keep that thing stable.

Q426 Zac Goldsmith: But there is a relevance also to bees. I have chunks of the promotional material here. In short, your literature describes Premise as "working because imidacloprid disorients termites and prevents them from grooming each other, which allows diseases to take hold". Now, obviously, there are similarities between the behaviour of termites and bees—they clean each other and exist in large colonies. So, what research, if any, has Bayer conducted that would shed light on the impact of this chemical on bees and honeybees in particular?

Dr Little: When you do your initial studies, you are looking at a large number of different factors in terms of the colony's susceptibility to disease: things like how long the bee brood will survive; the extent to which you see a change in behaviour; and whether a colony survives over winter—all those higher-tier studies that would be normally expected.

Q427 Zac Goldsmith: Can I just interrupt for a second?

Dr Little: Of course.

Zac Goldsmith: Can you point us to any research at all, either Bayer research or other research, that shows that this effect that the company brags about in relation to termites does not also apply to honeybees? Is there any research that you can point to?

Dr Little: I believe there is, and I will certainly come back to you on further evidence that we can show.

Q428 Zac Goldsmith: Just for the record, without casting doubts on your integrity, I want to cast doubts on your belief, given that you were not aware of the product's existence until a few moments ago.

Can I just make just one point? The imidacloprid's approval for use in the EU has been described, I think it was by you in our last session, as being environmentally sound because it is not bioaccumulative. I think that is what you said at the time. Given what your promotional material tells us about that very same chemical in relation to termites, surely at best it is a disingenuous claim.

Dr Little: No. Absolutely not. What we are saying is that in an agro-environment this particular product does not bioaccumulate. That is an assertion. What we can also do, and it is true for a number of other insecticides, is formulate them in a way that means that they are extremely stable and will work for a lot longer.

Q429 Zac Goldsmith: For seven years—seven-year durability.

Dr Little: Yes.

Q430 Zac Goldsmith: That is not in any way linked to bioaccumulation?

Dr Little: Not at all, because what you are talking about there is not repeat use. It is a single use. So you are talking about a formulation of a product that is not exposed to what a field application of a product would be exposed to and will have an effect over that time. Yes. I have no reason to believe that is not the case. It is entirely normal. For example, the stability of sugar in a sugar bowl is much different to that in a cup of tea. It is that different.

Q431 Zac Goldsmith: I am going to end this particular area by asking you if you could send us a detailed response to the points that I have just raised, when you have gone back and found the research that disproves that there might be a link.

Dr Little: Of course. Yes.

Chair: That would be very helpful. Thank you very much.

Q432 Caroline Lucas: I want to pursue the point of the accumulation in soil. Back in November, you told us that the imidacloprid had a half-life in soil of between 16 and 200 days. In December, you submitted some written evidence stating that in worst-case scenarios the half-life in normal soils would be variable, but could be around 288 days and would be expected to plateau upon repeated doses after three years. I wondered if you could tell us what caused you to change your calculation between November and December.

Dr Little: The difference between 200 and 288 days? It was with the help of my colleague here, Dr Garside, who went through it with me. I had been given information, which I believed to be correct, that we were looking at a half-life of up to around 200 days. Actually it is 288 days. If the question is how do we calculate a half-life of between 40 and 288 days, I am very happy to go through that.

Q433 Caroline Lucas: It would be interesting to know—288 days is very specific. It would be interesting to know a little more about that.

Dr Garside: These are measured days in the fields. So we performed 16 studies in the field across Europe, in central northern Europe and southern Europe, and in these you apply the compound once and then over a period of two years you measure its decline by taking samples of the soil—between 10 and 12 samples across the period of the study. Then, by measuring the difference in the concentration of imidacloprid in the soil, you can calculate how long it takes, with the half-life how long it takes to derive.

Just one point: these are done across Europe, and the half-life, because it is under field conditions, does vary depending on the particular year you are doing it and the climatic conditions. Just by coincidence, both the shortest and the longest half-life were both studies performed in Italy, but at different times.

Q434 Caroline Lucas: What is your response to UK trials? I know in the original submission there was an example from Germany and an example from the UK. Defra has told us that the UK trials in the '90s were based on a worst-case scenario, but they also confirm that those trials showed a half-life in the soil of around 1,300 days and that a plateau had not occurred after six years. So has Defra misinterpreted the UK evidence?

Dr Garside: This is a very different study from the one that we use to determine half-lives. Basically in this particular study it was a treated seed that was sown for six years.

Chair: When you say "in this particular study", can I just double-check—

Dr Garside: Yes. Sorry. I am talking about the UK accumulation study that is being discussed here. It is a very specific study that is not designed to derive half-lives. That is important, because when I just said about studies we do to derive half-lives—we take a lot of measurements of soil concentration. In this particular study in the UK, where it was a seed treatment, there was only one sample taken each year, and that was taken at the end of the year, just before the next sowing. So we do not have a measure of the concentration initially. We only have this measurement of one time a year.

This study was also different in its design. Normally when we do these studies they are designed to reflect common agricultural practice. In this particular study, the barley was sown and we took the harvest of the grain, but then the straw remained on the soil and the straw was chopped and shallow-incorporated back into the soil bed.

Q435 Caroline Lucas: It was a trial that you chose, or that was chosen, to be part of a demonstration within the assessment to demonstrate the long-term field dissipation?

Dr Garside: No, the study does not answer the question. I cannot speculate as to why the study was designed the way it was. But we generated the data. We have to submit it. So whether we believe now, looking back at a study performed from 1991, that it was a reasonable practice—I happen to believe it is not, because I know in the UK straw is quite a valuable commodity and therefore it is normally harvested. We still have the data, so we have to submit it and it has to be assessed by other member states.

Q436 Caroline Lucas: So why do you suppose it was done? If it was looking at something that was so utterly extraordinary that it would not normally happen, then why would someone pay for it to be done?

Dr Garside: I cannot speculate as to why the particular study was designed the way it was in 1991. All I can say is it was very early for this type of study and perhaps the design was not thought through properly. I can't speculate as to why it was done that way.

Dr Little: But other studies have been done.

Dr Garside: Yes. We have a study that was performed in Germany, not with seed treatment, where we do not have incorporation of a lot of organic material, and that study was done.

Q437 Caroline Lucas: Going back to the UK trial for a moment, though, do you think it demonstrates anything other than that imidacloprid has an unacceptable effect on the environment in a worst-case scenario?

Dr Garside: I do not think it demonstrates it has an unacceptable effect on the environment.

Q438 Caroline Lucas: Why? It is showing that it is not plateauing and it is increasing significantly.

Dr Garside: No. Because when we take into account the plateau, when we do risk assessments, if it passes the risk assessment demonstrating safety, then it is not an unacceptable effect on the environment.

Q439 Caroline Lucas: But we have new EU regulations that suggest that there should not be a half-life of more than 120 days. We are talking about something that has 1,300 days.

Dr Garside: No. There is no EU regulation suggesting there should not be a half-life of 120 days.

Q440 Chair: But is it not the case that the guidance subsequent to the regulation's coming into force, the supplementary guidance, laid that down as a guideline?

Dr Garside: It is not a cut-off. It is not a definitive figure that is greater than 120 days. That is not—

Q441 Caroline Lucas: We have here essentially a new regulation covering active substances that was introduced in 2009 that specifies that any plant protection substance approved for use in the EU must have a half-life in soil of less than 120 days.

Dr Little: Is that not a cut-off only if you have the other two?

Dr Garside: That is in combination. There are three criteria that apply. It is not a cut-off just on one criterion.

Caroline Lucas: Sorry?

Dr Garside: It is not a single cut-off criterion. It is also in combination with bioaccumulation and toxicity. So, it is not a figure that is itself a cut-off figure.

Q442 Caroline Lucas: Let me come back to the point that, however unusual the situation might have been around the UK trial, if those trials demonstrated a half-life of around 1,300 days and a plateau had not occurred after six years, if that were to be normal usage, would you agree that that would probably represent harm to the environment?

Dr Garside: I would say that if we did a risk assessment, so we look at it not in isolation, we look at the effect it has on the environmental organisms and if it has no effect and it passes the safety criteria, then in itself it is not having a detrimental environmental effect. The figure itself does not say that.

Dr Little: Also, as we have already made very clear, the experiment that was done was not to determine half-lives. Those experiments that were done to determine half-lives have demonstrated very clearly that you get a half-life of somewhere between 40 and 288 days depending on the circumstances.

Q443 Caroline Lucas: But it was about accumulation in the soil. It was about how soon the chemical is no longer present in the soil. If we have evidence that seems to suggest that after six years it is not plateauing, I do not quite understand why that would not represent harm to the environment.

Dr Garside: It related to a very specific set of circumstances, where this straw was ploughed back into it.

Q444 Caroline Lucas: I appreciate that. I am not arguing that this is an unusual scenario. What I am trying to get from you is: in that unusual scenario, if that were happening,

would you agree, from everything we know, that that would be damaging to the environment? Because you will know that the same regulation that talks generally about the number of days of half-life also says that substances should have no unacceptable influence on the environment.

Dr Garside: Yes.

Q445 Caroline Lucas: So, something that is not plateauing after six years and we would argue had this half-life of around 1,300 days—would you not say that would have an unacceptable influence on the environment?

Dr Garside: I would still stay you have criteria by which you define an unacceptable or an acceptable risk. If it passes those criteria in the risk assessment, then it cannot by definition be an unacceptable risk.

Q446 Caroline Lucas: But it did not. The interesting thing, of course, is if you go back to the original EFSA review, what EFSA said was—and I am reading here that EFSA picked up the issue of soil accumulation in the risk assessment—that at the two UK study sites, accumulation occurred over the full six-year duration of the studies and experts considered that a plateau was not reached. Now, that was in spite of the fact that the German authorities were saying that a plateau had been reached.

Dr Garside: Yes, because the German authorities commented that you cannot calculate the half-life from this study, and they commented on the design. EFSA also commented on the very fact I am mentioning about the design of the study, with the reincorporation of very high amounts of the straw. And in Germany the RMS looked at all the data that we had, not just one single trial.

Q447 Caroline Lucas: Let me ask you one last question. Are you confident that imidacloprid was subject to sufficiently rigorous and relevant environmental testing before it was approved for use as an active substance in the EU? Do you have absolutely no doubt about that whatsoever?

Dr Garside: Yes.

Caroline Lucas: Dr Little?

Dr Little: That is the good thing about having a whole weight of data rather than just a single time-point in a, as you have just heard, flawed experiment. I would much prefer to go with experiments that were designed to come up with answers that you need rather than focusing on a flawed experiment that essentially will definitely throw up a wrong sort of number if you do it in non-agronomically appropriate way. So, there are data on half-lives from across Europe that are in agreement that the half-life is indeed acceptable. Likewise, the accumulation data that has been done elsewhere according to appropriate criteria, appropriate methodologies, has come up with an accumulation that peaks at four years and you do not see any further accumulation.

Q448 Caroline Lucas: Let me just clarify one last thing. The reason why it was included in the original documents that were forwarded to EFSA for assessment was that there was a mandatory requirement to do that. Because the tests had been undertaken, it had to be put in there. Is that correct?

Dr Garside: Yes. Yes. Whether we comment on the design of the study or not, we generated a set of data. We have to submit it in our dossier. We cannot pick and choose the data that we use.

Q449 Chair: But that data that you submitted did not comply with the standards that were required for authorisation for the product.

Dr Little: That does not matter. We are obliged.

Chair: That does not matter?

Dr Little: Yes. We are obliged to submit all data. We do not have a choice. That is the rule. If you put data together, you have to submit it.

Q450 Chair: Surely if the data that you are submitting does not comply with what was being required in terms of the accumulation of the half-life in the soil—

Dr Garside: Sorry, in what sense do you mean that it does not comply?

Chair: My understanding is that the guidance that was subsequently issued when the initial regulation was reviewed required that there would be an assessment that would give assurances that there certainly would not be over 1,000 units for the half-life. So, the measurements that were done did not comply with what the regulatory regime was asking for.

Dr Garside: That particular study does not comply with the aims—

Q451 Chair: That study was what the initial authorisation was based on, was it not?

Dr Garside: No. It was not. The initial authorisation is based on all the data. So it is based on the 16 half-lives that we have derived from the different trials, also considering the accumulation study. But it is recognised that these studies have a lot of weaknesses because of the accumulation-type studies themselves. Now the requirement is to calculate a plateau based on the longest half-life that you measure in a field study. So, we no longer are required to do this type of study, because it is recognised that you cannot derive the information that you want from them. It is very difficult to determine whether you have a plateau or not when the climatic conditions change year to year.

Q452 Chair: True. But is it not the point that in the two UK studies, a plateau was not reached?

Dr Garside: Yes. From when you look at the data you do not appear to have a plateau, but this does not represent a relevant agronomic scenario. So we are not looking at the actual use, because you are ploughing this very large amount of material back into the field and that does influence the behaviour.

Chair: Okay. Mr Spencer, you wanted to come in.

Q453 Mr Spencer: Just to clarify for my own knowledge whether there is a difference in the impact on the environment between the chemical present in the soil or the chemical that is present in plant residue within the soil—

Dr Garside: I am not sure. What you tend to find is that when a compound is present in the soil for a length of time, the plants can no longer take it and it no longer has harmful effects on organisms. There is a difference between what we call a residue that has been there for six or nine months and one you apply freshly to the soil.

Q454 Dr Whitehead: I think we would lastly like to have a brief look at whether we ought to be considering neonicotinoids collectively or individually. Does imidacloprid have a different impact on pests and the environment from other neonicotinoids?

Dr Little: There are essentially two classes of neonicotinoids. The ones that are essentially imidacloprid, thiacloprid and thiamethoxam—these are used in the UK essentially as seed treatments. Then there are a number of other ones, possibly the ones that you will have come across are thiacloprid and acetamiprid, which have an extremely good profile in terms of non-target organisms, especially bees, and can be used to spray over a crop. So

basically there are two types. They are very different in their absolute toxicity in terms of target and non-target organisms.

Q455 Dr Whitehead: Bayer manufactures products containing both?

Dr Little: Bayer produces imidacloprid and clothianidin and also thiacloprid as a spray. Syngenta have thiamethoxam. Sumitomo also use clothianidin. Then there are a host of generic companies who use imidacloprid.

Q456 Dr Whitehead: As far as Bayer CropScience's sales are concerned, what proportion does indeed come from plant protection products containing imidacloprid?

Dr Little: Are we talking UK, or globally?

Dr Whitehead: Both.

Dr Little: In the UK, it would be a tiny fraction for imidacloprid. I think almost all of it now is generic in the UK. We use mainly clothianidin and again from the spray perspective, thiacloprid. Imidacloprid is irrelevant in terms of our turnover in the UK. The turnover on things like clothianidin is significant, but it is by no means the biggest product that we sell.

Q457 Dr Whitehead: When you say it is insignificant, is that because it is out of patent and, therefore, is only generically made?

Dr Little: Actually, the use of imidacloprid in the UK has largely been supplanted by clothianidin; so, very similar products, but from our perspective clothianidin is more suitable.

Q458 Dr Whitehead: The ACP said that they thought that imidacloprid use in the UK was declining very rapidly indeed.

Dr Little: Yes, absolutely. It is down to a minimal level; as I said, essentially a generic level.

Q459 Dr Whitehead: Is that the same in EU, worldwide, or is it just a local thing?

Dr Little: Imidacloprid remains a product that is used extensively globally. It depends a little bit on country by country, and essentially if you combine the use of imidacloprid, clothianidin and thiamethoxam, one or more of those will be used extensively in most places in the world. Not everywhere, but in most places.

Chair: Unless any of my colleagues have any further questions, at this stage I would like to thank you very much indeed for returning, Dr Little, and for making time available as well, Dr Garside. Thank you very much indeed.

Examination of Witness

Witness: **Professor Vyvyan Howard**, University of Ulster, gave evidence.

Q460 Chair: Welcome, Professor Howard. I realise that you have sat through the previous hearing, and the Committee members are aware that you do have travel plans to return home, so we are very conscious about—

Professor Howard: I think I am okay for the time being. I have a flight at 7 pm from Gatwick.

Q461 Chair: Okay; well, we will make sure you do not miss your flight.

I just wondered, first of all—you have had an opportunity to hear from our previous witnesses—whether or not by way of introduction you would like to give us your perspective

and make any comments on what you have just heard. We will also wish to refer to the role of the Pesticide Advisory Committee, which we understand met yesterday and will be making recommendations to Ministers. The floor is yours.

Professor Howard: Thank you, first of all, for the invitation to come and give evidence. There were some very interesting things said in the previous session, some of which I disagree with, but I think there is an emphasis on the economics of this, clearly. But I think that these neonicotinoids are a very good case study for what is deficient in the current risk assessments that we use. There was a lot of talk about data gaps, and a very interesting statement that Dr Little made was that, prior to neonics, pesticides represented a big problem for human health. I have never heard that before; we are always being told that they are perfectly safe, but this was a step change.

I think the things that were not discussed were the behavioural problems that these compounds seem to induce. In a way, because nicotine is known to affect the brain, there is a specific receptor that these compounds interfere with. It is also that particular part of the system, with acetylcholine as the transmitter, that is very important in the development of the nervous system.

When you do a risk assessment, the first step is hazard identification, and when you have identified a hazard the next step is characterising that hazard. That is expensive—lots of experiments and time. Then, finally, you do an exposure assessment, and then you do a risk assessment. It is all predicated on those first three steps. In this first step, hazard identification, one of the things you could have said was, "Well, this is a neuroactive substance so, therefore, we really ought to look carefully at the effect it has on the nervous system". Yet, as I recollect, most of the toxicity testing that was done was based on mortality. So these are standard pallets of tests over specific periods of time, from days to 14 days, to a medium-term study up to 90 days, and then multigenerational studies.

If we knew what we do now about the low-dose effects that affect the behaviour of these insects, I do not think they would ever get licensed; I do not think they would have been licensed. But at the time when they were licensed that was a data gap. Now they are licensed, they are continuing to be used, but clearly EFSA have identified that as one of the data gaps and have applied the precautionary principle.

The only other thing I would like to comment on about the previous presentation was that they clearly do not seem to understand precautionary principle. I published a letter in *Nature* about this a few years back, which I can furnish to the Committee. The precautionary principle is a tool that decision-makers can use at any step along the process—not just when it is new—of whether it is better to go on or not, taking the risks and benefits into account. That is what EFSA has done now. At this stage in the development of these compounds, they have weighed up the scientific evidence as it is, and they are basically applying the precautionary principle.

Q462 Chair: You would say that in the case of something already licensed, if further evidence comes up it is all right to go back to the beginning, as it were, and to undo that authorisation if the evidence is there.

Professor Howard: Yes. That exists, of course, in the pharmaceutical industry anyway, and pharmacovigilance is a kind of concept that people have been talking about applying to pesticides.

Q463 Caroline Lucas: Can I explore that a little further? It does seem quite extraordinary, in a way, that it does not already apply. I was really struck by the way in which Dr Little was basically saying that these chemicals have been around a long time, they do not

seem to have done any harm, they got through the tests that were provided at the time and, therefore, it is an odd thing to do to reapply the precautionary principle to them.

I guess what we are saying, just to try to make sure we are clear, is that if new evidence shows that those early tests were flawed because, for example, they did not take into account sublethal effects, then it is entirely proper and appropriate—indeed, essential—to apply that new bit of understanding that we have about sublethal effects to those older chemicals and to revise the standards by which they are judged. If what I have said so far is correct, then it seems to me to be very odd that there is not an automatic way in which that happens. Am I right in that—

Professor Howard: I think it is very difficult. It is not like pharmaceuticals, where they can just be whipped off by the relevant committee and there is no debate. I hope I was not saying that the tests that were submitted were incorrectly performed—I think they were probably okay—but—

Q464 Caroline Lucas: No, but the questions they were trying to answer were not the same questions we are trying to answer now, with the benefit of more—

Professor Howard: There has been a movement over the last decade or two, really, in the States and here in Europe, to try to get the risk assessments for pesticides moved away from these pallets of protocolised standard tests, to ask the developer to do relevant science.

A colony of bees is like an organism in itself. This bit of the population depends on that bit, and they are all in different states of development. So instead of looking at individual worker bees, which was originally what was done and toxicity tests were applied to those, one should look at the whole colony as the standard unit that you are trying to assess the outcome of. I do not think that was really done.

Q465 Mark Lazarowicz: First of all, sorry that I missed the beginning of your evidence; I had to go out for one second. Can you tell me a bit about your experience of the Advisory Committee on Pesticides and how far it really represents part of a coherent EU-wide system of regulation?

Professor Howard: I was on the ACP for six years, and I am a pathologist and toxicologist, so I was contributing in that way. I think the ACP does a pretty thorough job on applying the risk assessments that we have classically had over the years. Looking at these pallets of standardised tests, they look at pesticides in isolation and the toxicology has largely been predicated upon adult toxicology, although more recently developmental toxicology has come in. They try to come up with a regulatory level, and then they can recommend that they are licensed. To get on to annex 1 they have to be approved at EU level, and what the EU does is to farm out different pesticides to different equivalents of ACP in the different European countries and it feeds back into that.

The UK has been a rapporteur on a number of these. I remember Gaucho being discussed when I was on ACP. But it only goes as far as it does, and there are some areas where I think I would like to see what they do extended. I submitted a statement from the Endocrine Society yesterday by e-mail, so I think that is a very relevant document—and here I come back to one of Dr Little's statements; he said it is purely about dose. That is classical toxicology. It is Paracelsus: the dose makes the poison. I disagree with that, and most developmental biologists would disagree with it as well.

It is also to do with timing. What we are learning is that exposure to certain chemicals, which will have little effect on adults, will affect foetal development at a 1,000 times lower dose. So, in this development stage of life the toxicology is completely different, and the people who are making the running in the science here are not classically trained toxicologists—they are embryologists and developmental biologists. There is low-dose fatal

toxicology, and I think with these bee colonies it may be a factor. They need to have that expertise.

Q466 Mark Lazarowicz: Is that expertise not sufficiently in the ACP at the moment, then, in your view?

Professor Howard: Well, they do have developmental biologists, but this is a really new area. For example, in Ana Soto's lab in Boston they have shown that you can affect the development of the breast in way which looks suggestive of possible breast cancer later in life in an animal at 1:250,000 of the No Effect Level. That is just one example, but the document I have provided you with looks at a whole range of these things.

Q467 Mark Lazarowicz: Do you think the Government should be reviewing the membership of the ACP in terms of strengthening certain areas, or are they doing that anyway?

Professor Howard: I think they should have that expertise on board. There are other areas as well; I think the mixtures problem is another one. Again, I think this neonic is a good example because there is literature now showing that they may synergise with certain other things—fungicides and things like that. So there are some people around—Professor Kortenkamp, for example—who have done a lot of work on mixtures, and that sort of expertise, again, would probably be rather important. Another area is the nanotechnology that is coming into agro-delivery systems now. If you nanonise things, you actually affect the transport systems around the body, and that is another area. When I left ACP I said, "This is one that you have to watch because it is coming".

Q468 Mark Lazarowicz: If there is a disagreement within ACP, how would that resolve itself? Does it work almost entirely by consensus, or are there actual decision-making procedures that have to be applied?

Professor Howard: Most decisions are arrived at by consensus. Occasionally, there is a vote. I think the response that the ACP made to the Royal Commission on Environmental Pollution eight years ago—I was one of the four who dissented from that. There is room for dissent. Dr Chris Stopes wrote a dissenting opinion once on another aspect, so there is the chance to do that.

Q469 Mark Lazarowicz: Was that dissenting opinion then sent to Government along with the majority opinion as well?

Professor Howard: Yes. It is minuted and available, yes.

Q470 Zac Goldsmith: Just very briefly on the point you made—and it is something you have written about in the past—about the synergistic effects of chemical mixtures. Taken to its logical conclusion, if you were to assess all the different combinations of chemicals that are likely to react with other chemicals, you would be presumably setting yourself a task that is administratively impossible. Is it, therefore, possible to have a genuine precautionary principle? Is it possible to have a regulatory system that takes into account the impact of all these new chemicals coming into the market?

Professor Howard: You are right. We published papers where we compare two compounds together, and it is three years of a PhD to do that. Then, if you asked me to do three, I would be starting to struggle. If you permutated any three combinations from the number 1,000, you come up with a number like 15 million different combinations. That would all be at one concentration. You can see that the experiments very rapidly get out of hand.

You can obviously try to pick out the ones you think are going to be the most likely interactions and look at those, but this soup effect is a really problematic one. Professor Kortenkamp has been looking at mixtures of up to, say, 15 pesticides that are commonly there and at very low dose and finding synergistic effects, so it is clearly something we have to consider. But I agree that if you really want to go to town on that, then precaution is something that comes to the fore.

Q471 Mr Spencer: I am quite interested in your comments about chemicals and how they react together if they are applied, if you like, at the same time. But, clearly, if we move from a system of neonicotinoid seed treatment, we then move to an insecticide programme at the same time as a fungicide programme. You would be applying those chemicals at the same time and could exacerbate the problem.

Professor Howard: That is the status quo ante, isn't it? That is what has been going on for a long time in integrated pest management, the spraying of several things at once. There are various aspects to this; the chance of human exposure from spray drift is discussed and well aired. We are assured if sprayers take the right precautions and spray in the right conditions, that is minimal.

One thing that I am not sure has been fully aired yet with neonics is their ability—they are water soluble—to get into surface water. There have been studies in Holland and America that have shown that they are getting there. One question that I would certainly like to see covered would be what the significance is, say, of children drinking water that has these compounds in, because we know they are neuroactive. Again, I don't think that has been fully addressed or addressed at all in a risk assessment. But if there is evidence that they are getting into surface waters and maybe, therefore, drinking water, I would certainly flag that up as an area that needed to be looked at.

Q472 Mr Spencer: Just so we can get a feel of the scale of the issue, I wonder if you could make a layman's comparison to human exposure to nicotine. Could you compare the drinking of water, as you described, to the effect on a child in the back seat of a car where a mother smokes in the front? How far apart are those in terms of exposure to nicotine?

Professor Howard: I would have to go away and get a calculator out and have a look. I don't know. I would think the water would be rather lower, because we know that passive smoke has an effect on cot death. That is well documented. It is one of the big factors. Again, this is a neuroactive thing, that was Professor Fleming's big epidemiological study. If one parent smokes, there is a high risk, and if both parents smoke, there is a yet higher risk. This is certainly associated with tobacco smoke. Whether it is specifically the nicotine, nobody knows.

Q473 Peter Aldous: I will just say at the outset—it is on the Register of Members' Interests, but I am a partner in a family farm in Suffolk, arable and livestock. Just taking it further forward, Professor Howard, the possible impact of pesticides on human health: what pesticides do you regard as being particularly hazardous to human health?

Professor Howard: That is an interesting one. The one that I am most perturbed about, I think, is chlorpyrifos. There is a burgeoning scientific literature on the fact that low-dose chlorpyrifos during the foetal period, delivered by the mother, has neuro-behavioural effects on the offspring. Indeed, when I was on the ACP I went with Michael Meacher to Defra. We had a meeting with them about chlorpyrifos. They did reduce the ADI at about that time. I was putting all these papers on the table and saying that maybe they should consider going further than that, but they didn't. That is certainly one that I would like to see come under the spotlight.

Q474 Peter Aldous: Leaving aside fungicides and herbicides, which particular insecticides raise the greatest concerns in respect of their impact?

Professor Howard: Chlorpyrifos is an OP, isn't it?

Q475 Peter Aldous: Yes. Have you identified any cases of human health effects specifically from the use of neonicotinoids?

Professor Howard: No.

Q476 Peter Aldous: Thank you. I want to take further something that Mr Spencer commented on. One consequence of a hypothetical moratorium on the use of neonicotinoid seed treatments in the UK might be an increase in the use of foliar pesticide sprays. Would such change in agricultural practices lead to an increase in risk to human health?

Professor Howard: If they are improperly applied, yes, I think that could be argued, although systemic pesticides are a very new development and I think we are only beginning to understand what it means. But I think this threat to pollinating species, from a financial point of view as well as from an ecological point of view, is severe, as I read it. I think that EFSA is right to be proposing a precautionary stance on this.

Q477 Dr Offord: I wanted to ask you a couple of questions on the Royal Commission on Environmental Pollution. They produced their report in 2005 on crop spraying, and that was right in the middle of your tenure on the Advisory Committee on Pesticides. They concluded in their report—we do have sight of it here—that they could not draw firm conclusions on causality between recorded ill health and pesticide exposure. I wanted to gain your opinion on that and ask if you felt that they came to the correct conclusion.

Professor Howard: I think the conclusion is correct. The thing is that human exposure patterns are very complex. If you think about the two examples where medics have been able to say, "There is something going on here", one was thalidomide and the other was diethylstilbestrol, which was given to women in the 1940s to stop miscarriage, and 20 years later young women started turning up in clinics with a very rare cancer of the vagina. There were six or seven. They said, "What's going on?" With thalidomide it was such an obvious thing you couldn't miss it. "What's going on?" When someone put two and two together and went back to the mother's case notes there was the history of exposure, so you had an exposure history.

What we are dealing with, with pesticides, is diffuse low-dose mixture and nobody is monitoring who is exposed to what very much, and particularly they are not monitoring what the foetus is getting. It is almost impossible without that history to be able to say there is a tie-up. You can do these very large cohort studies and look at pesticide usage in certain areas—and they have done that with Maneb and things like that, with respect to Parkinson's—and you can get an inkling. But what they are saying is that there is a large degree of uncertainty and that was what a lot of the argument within the ACP was about. The RCEP were saying, "You're putting this forward in a much too confident way to say there is no risk. You are not in a position to give it that level of confidence". The majority of ACP argued back that they thought they could, and that was the nub of the argument then.

Q478 Dr Offord: But your answer to that question fits in well with your response through the Committee to the Royal Commission's report, and I understand that you cautioned about being too ready to acknowledge potential human health risk. But in response

to that position, would you say that those people who were reporting ill-health due to exposure to pesticides in some ways had a slightly psychosomatic effect?

Professor Howard: Most illnesses have one or both. The evidence that was put forward there was not a properly constructed epidemiological study, so I think it is very difficult to draw hard-and-fast conclusions. It is what is known as anecdotal evidence. But they put themselves forward, and there were quite a number of them with conditions. When you find clusters of things like that, then it often is worth looking further into, but it doesn't prove anything.

Dr Offord: Right; okay, that is great. Thank you.

Q479 Chair: Just finally in conclusion, I think you appreciate that this is a timely report that we are undertaking. In a way, there are fast-moving developments. We have had a retail ban, I think, either today or yesterday, and obviously we have had the EFSA report. Is there anything that you would wish to cover and raise with us that you have not had an opportunity to raise that is particularly pertinent to the stage we are at with our inquiry now?

Professor Howard: Yes. I think the main thing that I want to see introduced into regulatory process is a much closer look at subtle functional deficits. Hitherto, developmental toxicity has largely been measured by looking at gross malformations, spina bifida, skeletal malformations—things you can see with the naked eye. It is changing slowly but not fast enough, in my opinion.

I will give you examples of these subtleties. One would be, say, a reduced ability to produce sperm. You don't see any deficit by looking at the anatomy; you have to measure the physiology, and neuro-behavioural deficits obviously fall into that as well. The subtle deficits are the things that we are finding increasingly following exposure during the foetal period. I think if we get to a stage where we can manage to protect the foetus, then we protect everybody—that is the most vulnerable state.

Q480 Chair: When you say "we"—

Professor Howard: I mean society through its regulatory processes, yes.

Chair: Okay. Thank you very much indeed for going to such lengths to be with us this afternoon. We appreciate your evidence, and we shall see where our inquiry takes us. Thank you.