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The author asserts her moral rights generally in respect to this Witness Statement.

**On behalf of: Respondent**
**Witness: G.Downs**
**6th statement**
**Date of statement: 5th June 2009**

**C1/2009/0073**

**IN THE COURT OF APPEAL**

**Secretary of State for Environment, Food and Rural Affairs v Georgina Downs**

**SIXTH WITNESS STATEMENT OF GEORGINA DOWNS**

1 I am Georgina Downs, of "Reflections", Runcton Lane, Runcton, Chichester, West Sussex, PO20 1PT and I am the Respondent in these proceedings. I make this, my sixth statement, to respond to points made in the third Witness Statement of Paul Hamey, dated 18th May 2009, and which was submitted to the court on 19th May 2009, on the second day of the Court of Appeal hearing. Document references are given as follows. For documents contained in the bundle accompanying this sixth statement: **[Tab */*]** (giving tab no./page no.); the pagination for this bundle appears at the bottom right hand corner of each page; the numbering re-starts with each tab. For documents contained in the four core bundles: [CB*/*/*] (giving core bundle no./tab no./page no.); for documents contained in the original trial bundles (numbered I-V): [TB*/*] (giving Trial Bundle no./page no). Except where otherwise stated, I depose to the truth of the facts contained in this statement from my own knowledge.

## Introduction

2 I would like to start this statement by reminding the court that, in relation to Mr. Hamey's third Witness Statement: 1) it was submitted at a very late stage (on the second day of the hearing), even though the Appellant had over 6 months to submit any comments in response to the medical report material (that had been submitted by the Respondent prior to handing-down of the judgment **[CB4/E/5-10]**), and the Appellant did not submit *anything* in its accompanying documentation to its

Appellant's Notice and Skeleton argument lodged in January 2009, nor at any other time *prior* to the COA hearing; 2) the letter from Dr. Myhill interpreting the results of my blood and body fat samples was the same letter referred to and quoted from in §48 of my first Witness Statement **[CB1/O/9]** dated 22nd October 2006 and which was not challenged *at any time* by the Appellant (despite having had over 2 and a half years to do so); 3) Mr. Hamey's third statement contains a considerable number of factual inaccuracies, which I would like to correct, in relation to my own personal health situation, and also in relation to the other parties mentioned in Mr. Hamey's third statement (eg. Dr. Sarah Myhill and Biolab); 4) Mr. Hamey is not a medical professional, and in fact it is important for the court to note that there are no medical professionals or doctors at all at the Chemicals Regulation Directorate (CRD), which was formerly the Pesticides Safety Directorate (PSD).[1]

3 The court only accepted Mr. Hamey's third statement on the basis that I would have the chance to respond and therefore I have done my best in the time available, to set out in this sixth statement my response to some of the points made by Mr. Hamey and to correct the various factual inaccuracies he has made.

4 I have included a number of exhibits to this sixth statement that are referred to at various places in the information below.[2] These exhibits consist of:-

(1) Letter from Dr. Sarah Myhill, dated 29th May 2009, responding to some of the points made by Mr. Hamey in his third Witness Statement. **[Tab 1/1-2]**.

(2) Comments from Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, PhD, BPharm, MRIC, CChem, dated 27th May 2009, responding to some of the points made by Mr. Hamey in his third Witness Statement. **[Tab 2/1-4]**.

[1] This was also previously noted by the Royal Commission on Environmental Pollution (RCEP) in its 2005 report, as §5.57 stated, *"The positioning of both delivery and policy responsibilities within the PSD appears to have made it more difficult to address health issues relating to pesticide exposure. Although health issues are important there are no medically qualified staff at the PSD."* **[CB2/I/87].**

[2] Other source material is contained in various weblinks or references detailed within this sixth statement.

(3) Study published in the Lancet in November 1999, entitled, *"Reduced bone formation after exposure to organophosphates,"* by JE Compston, S Vedi, AB Stephen, S Bord, AR Lyons, SJ Hodges, BE Scammell. **[Tab 3/1-2]**.

(4) Data sheet for Mevinphos prepared by the International Programme on Chemical Safety (IPCS). **[Tab 4/1-3]**.

(5) Bibliography entitled, *"Pesticide Lindane and Breast Cancer Risk,"* by Cornell University, prepared as part of its "*Program on Breast Cancer and Environmental Risk Factors*". **[Tab 5/1-22]**.

(6) Data sheet for Diazinon prepared by the International Programme on Chemical Safety (IPCS). **[Tab 6/1-4]**.

(7) Abstract of the study entitled *"Health Effects of Diazinon on a Family,"* by J.G Dahlgren, H.S Takhar, C.A. Ruffalo and M. Zwass, published in 2004 in *Clinical Toxicology*, Volume 42, No. 5, Pages 579-591. **[Tab 7/1]**.

(8) Factsheet regarding Carbaryl, by Caroline Cox, published in the Journal of Pesticide Reform, Summer 2005, Vol 25, No 2. **[Tab 8/1-6]**.

(9) One page sheet containing selected quotes from the study entitled *"Organophosphorus ester-induced chronic neurotoxicity"* by Professor Mohamed Abou-Donia, published in the Journal of Occupational Health Safety — Aust NZ 2005, 21(5): 408-432, in 2005. Also included in exhibit GD/9 is a one page biography of Professor Mohamed Abou-Donia, of the Department of Pharmacology and Cancer Biology, Duke University Medical Center in the United States. **[Tab 9/1-2]**.

(10) Table regarding the Immunotoxicity of Pesticides taken from *"Pesticides and Human Health: A resource for Health Care professionals,"* by Dr. Gina Solomon MD, MPH, Senior Scientist, Natural Resources Defense Council, Assistant Clinical Professor of Medicine, University of California, San Francisco, published in 2000. **[Tab 10/1]**.

5 Considering the seriousness and sensitivity of the content of this sixth Witness Statement and accompanying exhibits then I would request that the information provided herein is considered very carefully as it obviously involves information relating to my own personal health problems, pesticide exposure, and blood and fat test results etc. (Incidentally it is important for the court to note that the Appellant has not at any time challenged or questioned the actual confirmed chronic health problems that I have in themselves, (such as osteoporosis, long-term neurological problems etc.) Therefore that has not been in issue in this case, either in the court below or the Court of Appeal. The Appellant's argument/position in relation to my health problems is that exposure to pesticides sprayed in my locality could not be the *cause* of the chronic health problems that I have. (Considering the implications on the Appellant for it to accept otherwise, then its position in defending the pesticides it has previously approved as "*safe*" is not that surprising)).

**My own personal health problems**

6 At §65 of the Judgment, in relation to my own long term chronic health damage[3], Mr. Justice Collins concluded that there were "*very powerful reasons for concluding that there has been the necessary cause and effect*". (Judgment §65 **[CB1/K/26]**).

7 The 4 pesticides, Lindane, Mevinphos, Diazinon and Carbaryl, (amongst others, that were found in my system from the *limited* pesticide screen I had undertaken at Biolab[4]) were previously approved for use in the UK as crop-sprays. In relation to just these 4 pesticides *alone* (ie. before considering all the other innumerable mixtures of pesticides that I have been exposed to from over 25 years of crop-spraying in my locality), each one, just on its own, is neurotoxic and capable of damaging the central nervous system in humans, as well as other systems within the body, (as set out in more detail below).

[3] In relation to acute effects, Mr. Justice Collins found (at §40 of his Judgment) that there is *"solid evidence produced by the claimant that residents have suffered harm to their health (her own ill health is an example)...."* (See §40 to §47 of the Judgment at **[CB1/K/18-21]**).
[4] See §27 to §32 below for section relating to Biolab.

8 Dr. Myhill's letter, dated 13th April 2004, correctly described some of the long-term chronic effects of pesticides and pointed out that *"it is hardly surprising that the major effect of these chemicals is neurological and can extend into both central and peripheral and autonomic nervous system."* **[CB4/E/10]**. As I pointed out in §6 of my first Witness Statement at **[CB1/O/1-2]** my long-term chronic health problems are predominantly neurological and I suffer from many of the symptoms listed in Dr. Myhill's letter. I have previously been hospitalized with severe muscle wastage, muscle weakness and other chronic effects. The nature of the neurological symptoms previously led to tests and scans to rule out a number of neurological diseases, such as Multiple Sclerosis (MS), Motor Neurone Disease (MND) and Parkinson's disease.

9 Also, as was pointed out in the medical report material (that was submitted by the Respondent prior to handing-down of the judgment **[CB4/E/6]**), scans have confirmed that I have osteoporosis with a high risk of fracture and I am only 35 years old. There have been a number of studies that have found that pesticides, (in particular organophosphates), can cause impacts on bones leading to osteoporosis. I have attached to this sixth Witness Statement, as exhibit GD/3 **[Tab 3/1-2]** one such study published in the Lancet in November 1999, entitled, *"Reduced bone formation after exposure to organophosphates,"* by JE Compston, S Vedi, AB Stephen, S Bord, AR Lyons, SJ Hodges, BE Scammell. The study states, *"Bone histomorphometric analysis in 24 agricultural workers with chronic organophosphate exposure showed significantly lower bone formation at tissue and cellular level than in healthy controls."* **[Tab 3/1]**.

10 In the attached comments by Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, (at exhibit GD/2 **[Tab 2/1-4]**), in relation to the impacts of pesticides on bones, he states that, *"In addition bone growth and turnover is compromised leading to osteoporosis, (Compston, Hodges et al 1999), with similar effects being found in sick Gulf War veterans of the 1990-1 Gulf war, (Compston, Hodges et al, 2002)…It is ironic that the widely used standard for optimum bone density is that found in young women age 32. Miss Downs was found to be suffering from osteoporosis before this age. This is very unusual."* **[Tab 2/2]**.

11 Therefore Dr. Myhill's letter, dated 13th April 2004, again correctly pointed out that pesticides can be *"toxic to bone resulting in increased risk of osteoporosis…"* **[CB4/E/10]**.

12 As was pointed out in the medical report material (that was submitted by the Respondent prior to handing-down of the judgment **[CB4/E/6]**), the blood and fat tests I had carried out at Biolab were *limited* to only those pesticides that they were able to test for at that particular laboratory at that time, as part of their pesticides screen. Considering there are hundreds of different pesticides used in agriculture and that I have been exposed for over 25 years then going by these results there are likely to be many other pesticides present in my blood and body fat in addition to those identified in the aforementioned tests.

13 In addition to the information provided within this sixth Witness Statement, to add further support in relation to pesticides being implicated in my own personal chronic[5] health problems, I have enclosed a letter from Dr. Sarah Myhill, dated 29th May 2009, (at exhibit GD/1 **[Tab 1/1-2]**) that states, *"I have no doubt that Georgina's chronic long-term health problems are due to her repeated exposures to mixtures of agricultural pesticides sprayed near her home throughout the last 25 years. There is a considerable body of scientific evidence to support her case. As I said in my letter to former DEFRA Minister Alun Michael in May 2004, and which I understand is also before the court, the Government knows full well that there is a very serious public health problem here and are simply chosing to ignore it and all the solid evidence relating to it."* **[Tab 1/2]**.

14 Also, see the attached comments by Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, (at exhibit GD/2 **[Tab 2/1-4]**), as in relation to my chronic health problems Professor Hooper states, *"Miss Downs has*

[5] As detailed in §4 and §8 of my first statement at **[CB1/O/1-2]** the acute effects I have suffered following exposure to pesticides, in particular flu-type illnesses, headaches, sore throats covered in blisters, as well as blisters/ulcers in the mouth, (at times this could be as many as 20 at a time), were not minor or mild and left me seriously affected, (even before considering the chronic long-term health problems that I have). These acute effects are well recognised acute effects of pesticide exposure and the safety data sheets for pesticides can carry warnings of these types of acute effects. Therefore exposure to pesticides being able to cause the types of acute effects that I have suffered from is not in question as it is already accepted.

*been exposed to a variety of mixtures of well known toxic chemicals OPs, carbamates and pyrethroids, amongst others, with associated 'inerts' that may well enhance the toxicity of these compounds and/or exert toxic effects of their own. These compounds are well known to accumulate in body fat and fatty tissues like the brain where the neurotoxic effects are exerted. At the same time storage of these compounds in these tissues will provide a depot for the ongoing release of low doses of these compounds thereby maximising their toxic effects. The combination of repeated acute high dose exposures and repeated low dose exposures to these compounds will serve to maximise the toxic effects of these compounds. Miss Downs has been a credible and informed witness whose situation rings true. I have no doubt that her chronic ill health is due to her exposures to mixtures of agricultural pesticides of various classes, particularly OPs, carbamates and pyrethroids. There is a considerable body of scientific evidence to support her case."* **[Tab 2/3]**.

15 Also, it is important to reiterate the point I previously made in my first statement at §48 **[CB1/O/9]** that in addition to the health problems I have now, I have no idea what other chronic health effects may yet be to come that could be as a result of my ongoing exposure to pesticides in crop-spraying, particularly as I was exposed from a very young age.[6]

16 Also, as I have pointed out previously, as can be seen from Dr. Myhill's letter dated 13th April 2004, I have received medical advice to the effect that *"the most important aspect is to avoid ongoing exposure"* to pesticides, **[CB4/E/10]** which is obviously impossible in the kind of situation that myself and other rural residents are living in.

17 I shall now respond to the points made by Mr. Hamey in his third Witness Statement in the order they arise.

[6] Also as I pointed out in footnote 14 of my third Witness Statement, **[CB1/S/20]** it should also be noted that once someone is suffering from chronic long-term health problems (like myself and many other residents who contact me) then they fall into the bracket of a *vulnerable group* where any further exposure to pesticides (irrespective of whether pesticides was the cause of their pre-existing health problems or not) can be deleterious. Also see §5 of Route Map 4 at **[CB4/B/38]** that points out that *"irrespective of the quantifiable harm to health, the exposure has inevitably made the Claimant more vulnerable in the long-term to a number of diseases (**Fadayeva §88; 4/771; 2/746; 2/683 §3.24; Downs 2 §86**)."*

**The status of Dr. Myhill (§2 to §5 of Mr. Hamey's statement)**

18 In §3 of his third Witness Statement, in relation to Dr. Sarah Myhill, Mr. Hamey states, *"She is not on the Specialist Register, and not on the GP register."* The letter from Dr. Myhill, dated 29th May 2009, (at exhibit GD/1 **[Tab 1/1-2]**) shows that what Mr. Hamey has said is not factually correct. Dr. Myhill points out that *"I have a special interest in ecological medicine. This is a recently established speciality. The British Society for Ecological Medicine has its own specialist register, which is endorsed by the Institute of Biology and the General Medical Council. I am a Specialist on that register."* **[Tab 1/1]**.

19 In §4 and §5 of his third statement, Mr. Hamey has been somewhat selective in what he has cited from Dr. Myhill's website. One paragraph in particular that he has omitted and that the court should be aware of is where Dr. Myhill points out on her website[7] that, *"The information in this website is based on my experience of over twenty five years in general National Health Service practice and private practice. I describe what works for me…My special interest is in treating fatigue and in preventive medicine."*

20 Dr. Myhill goes on to state that, *"…many doctors are not familiar with the many tests now available to diagnose the root causes of illness."*

21 Dr. Myhill is a well respected doctor who has specialized in pesticide related health problems for many years.[8] The court should note that Dr. Myhill has recently had a paper published in the *International Journal of Clinical and Experimental Medicine (IJCEM)*,[9] where papers are subject to peer-review to meet the "*rigorous standards of academic excellence*."[10]

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[7] Source: www.drmyhill.co.uk

[8] Considering the lack of training for national health service GP's and specialists, in relation to the adverse health impacts of pesticides, (which was also recognised by the Royal Commission on Environmental Pollution in its 2005 report) then it is not surprising that Dr. Myhill assists many patients with pesticide related ill-health, as she is one of only a handful of medical doctors in the UK who has considerable direct experience and interest in this field.

[9] Source: www.ijcem.com

[10] Source: www.ijcem.com/guidelines.html

22 The website for the *International Journal of Clinical and Experimental Medicine (IJCEM)*[11] states, *"International Journal of Clinical and Experimental Medicine (IJCEM) (ISSN 1940-5901), is an open access online journal. It is founded by a group of academic physicians and scientists around the world, who are devoted to the advancement of clinical diagnosis, treatment and scientific exploration of human diseases."* [12]

23 Under the IJCEM *"Peer Review Policy"*[13] it states, *"All manuscripts are subject to peer review and are expected to meet the rigorous standards of academic excellence. The authors should provide up to 4 potential peer reviewers with detailed contact information including e-mail address. These should be experts in their field of study, who will be able to provide an objective assessment of the manuscript. If not provided, potential reviewers will be recommended by the Editorial Board members."*

24 The Editorial Board[14] for the *International Journal of Clinical and Experimental Medicine (IJCEM)* includes academic physicians and scientists from around the world including a number from the well-renowned Harvard Medical School.[15]

25 It should be pointed out that the two other co-authors of the paper[16] that Dr. Myhill has recently had published in the *International Journal of Clinical and Experimental*

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[11] Source: www.ijcem.com/index.html

[12] Under the IJCEM *"Author Guidelines"* at http://www.ijcem.com/guidelines.html it states, *"The International Journal of Clinical and Experimental Medicine (IJCEM, ISSN 1940 5901) is an open access online journal dedicated to publication of original work in all areas of Biomedical Sciences including, but not limited to biomedical research, internal medicine, surgery. OB-GYN, pediatrics and infectious disease. IJCEM also welcomes papers from other fields related to the study of human diseases, such as pathophysiology, pharmacology and epidemiology. IJCEM is primarily devoted to original clinical, translational and experimental research papers, but will also publish editorials, review articles, case reports, letters to the editors and meeting reports. The goal of IJCEM is to provide a free forum for rapid dissemination of clinical and basic observations that will enhance our understanding, diagnosis and management of human diseases."*

[13] Source: www.ijcem.com/guidelines.html

[14] Source: http://www.ijcem.com/editorial_board.html

[15] Under *"About IJCEM"* at http://www.ijcem.com/aboutus.html it states, *"The International Journal of Clinical and Experimental Medicine will be submitted to the National Library of Congress in Washington DC for indexing upon publication of the first issue. It will then be evaluated for indexing and abstracting in PubMed in the first year of publication. When the citation data becomes available, usually in the second or third year of publication, IJCEM will be submitted to Science Citation Index and other databases for indexing and abstracting."*

[16] Although this paper entitled *"Chronic fatigue syndrome and mitochondrial dysfunction"* is not directly about pesticides, for information the abstract can be seen at http://www.ijcem.com/812001A.html

*Medicine (IJCEM)*, are John McLaren-Howard (formerly of Biolab Medical Unit and who undertook and tested my blood and fat samples in 2004) and Norman E. Booth PhD, Department of Physics and Mansfield College, University of Oxford.

26 Therefore Mr. Hamey's attempts to call into question Dr. Myhill's reputability are unfounded and completely unacceptable, (especially as a non-medical professional himself).

**The status of Biolab Medical Unit (§8 to §10 of Mr. Hamey's statement)**

27 In §8 of his third Witness Statement, in relation to Biolab Medical Unit, Mr. Hamey states, *"No mention is specifically made of an analytical service for pesticides, however in a couple of newsletters (dated 2003) mention is made of a "pesticide screen"."*

28 The biochemist John McLaren Howard who specialised in pesticide screening has now moved to another laboratory, so Biolab no longer offers the pesticide screen (which was obviously available at the time I had the tests done in 2004).

29 In response to §9 of Mr. Hamey's third Witness Statement, in relation to Biolab's accreditation, having enquired with Biolab the situation is as follows. Biolab is not yet an Accredited pathology laboratory, although they are working towards a formal application for Accreditation. However, they do participate in external quality control schemes for all the tests for which a scheme is available. Also importantly, because Biolab provides tests that doctors have difficulty obtaining from "*routine*" pathology laboratories, then there are a number of the Biolab tests for which there is currently no external quality assurance scheme and therefore Biolab depend on stringent internal quality control using, amongst other controls, commercially available control samples.

30 Under "*Quality Assurance*" on Biolab's website[17] it states, *"Biolab assists doctors with the detection of nutritional imbalances in their patients by the use of the latest*

[17] Source: http://www.biolab.co.uk/doctor.html

*validated scientific methodologies and reporting systems to ensure optimal care. Quality assurance is essential in clinical laboratories for the provision of precise and accurate analytical results for reporting to the doctor. Quality assurance encompasses a range of measures to ensure the reliability of investigations. These include test selection, obtaining a satisfactory sample, analyzing it correctly and recording the result promptly, as well as the appropriate interpretation of the results. Biolab procedures are well documented and the results are carefully recorded so that they can be referred to for many years in the future. Biolab laboratory tests are internally quality controlled to keep a check on day-to-day analytical variations. Biolab also participates in a range of CPA (UK)-accredited quality assurance schemes for trace elements, vitamins, enzymes and antioxidants operating under the UK NEQAS code of practice. We also participate in a scheme run from the Centre for Disease Control in the USA. Biolab laboratory staff regularly attend a range of continuing professional development seminars to ensure satisfactory knowledge and competence of all analytical procedures."* [18]

31 In §10 of Mr. Hamey's third statement, he states, *"The pesticide residues stated to be found (lindane, mevinphos and diazinon) are relatively straightforward compounds to analyse by using commonly available lab equipments and standard GC techniques."* I can confirm that the pesticides found in my samples undertaken by Biolab were indeed detected using standard gas-liquid chromatography.

32 In relation to Mr. Hamey's comments in §10 of his third statement, regarding Carbaryl, I can confirm that Carbaryl was definitely detected in the fat cells taken from both my breast and my buttock. In fact, Alpha-Naphthol, which is a Carbaryl metabolite, was also detected in both the samples taken from my breast and buttock. (It should be pointed out that most analysts would regard the detection of Carbaryl

[18] Also Biolab's website (at http://www.biolab.co.uk/docs/infoleaf.pdf) states, *"The laboratory is staffed by a highly qualified team and participates in a number of external quality control schemes. Many of our tests require expensive high-tech instrumentation which is not ordinarily available in hospital pathology laboratories. Samples for standard pathology investigations are sent to the London Clinic Pathology Department (or other laboratories by prior arrangement). Biolab is actively involved in researching the effects of nutrition and the environment on health and disease. We have our own library of research material and have published many papers in medical and scientific journals."*

and one of its major metabolites as more than sufficient evidence for its presence in the sample).

**Fat solubility of pesticides (§11 and §12 of Mr. Hamey's statement)**

33 In §11 of Mr. Hamey's third statement, he states, *"The assertion is made that "Pesticides are very fat soluble compounds" (paragraph 2). This is not true of all pesticides"* and in §12 Mr. Hamey states, *"It is also stated that "Once absorbed they spend little time in the blood…and redistribute to the fat" (paragraph 2). In fact only lipophilic ones will potentially behave like this."*

34 This is not correct, as the majority of pesticides are significantly soluble in fat. For example, in the comments from Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, dated 27th May 2009, (at exhibit GD/2 **[Tab 2/1-4]**), in relation to the accumulation of pesticides in body fat, Professor Hooper states, *"A commonly measured physicochemical property is the partition coefficient that is usually expressed as a logarithm of the distribution, log P, of the pesticide between water and an immiscible organic phase. The most widely used immiscible organic phase is n-octanol which is thought to mimic the properties of body tissues. Log $P_{o/w}$ octanol/water is preferred by some on theoretical grounds. The values quoted in the scientific literature vary somewhat depending on the nature of the water immiscible, hydrophobic phase. For example, a compound with log $P_{o/w}$ = 2, is 100 times more soluble in the hydrophobic phase than water and would be expected to accumulate in body fat."* **[Tab 2/1]**.

35 In relation to some of the pesticides detected in my body fat samples, Professor Hooper states, *"Of the compounds of interest in this case that were detected in Georgina Downs' body fat samples undertaken by Biolab in 2004, mevinphos is, atypically, miscible with water and many organic solvents, alcohols, acetone but not hexane, log $P_{o/w}$ =1.2;*[19] *making it 15 times more soluble in fat than in water. Log P for diazinon = 3-4.*[20] *(This is estimated to be 10 times higher than that of diazoxon*

[19] Source: on page 3 of exhibit GD/4 **[Tab 4/3]**.
[20] Source: http://eprints.utm.my/1464/1/JT38C%5B5%5D.pdf (on page 4)

*(Log $P_{o/w}$ =2-3) the active metabolite of diazinon). For Carbaryl, a carbamate with a similar biological action to OPs, log P=2.36."* [21] **[Tab 2/1]**.

36 Professor Hooper then points out that, *"Pyrethroids*[22] *have even higher values with log P ~ 4, whilst the bioaccumulative and persistent organochlorine pesticides,*[23] *DDT, DDE etc. are even higher with Log P= ~5-6. Even many years after the organochlorine compounds were banned most people today have residues of these compounds and chemically related xenobiotics in their body fat. The biological distribution of pesticides in body tissues and organs has been studied in a number of cases and these studies show that the distribution of these substances is not uniform,* ***with body fat and the brain being tissues where the accumulation is greatest. This is consistent with the 'fatty' nature of these tissues."*** **[Tab 2/1-2]**.

37 Professor Hooper goes on to state, *"Some OPs are formulated as the thiophosphates in which usually one but sometimes two oxygen atoms have been replaced by sulphur atoms. This has the effect of reducing the neurotoxicity and pesticide activity whilst at the same time markedly increasing the relative solubility in fat. It was initially thought that these sulphur containing analogues of OPs would be less toxic in use and application since they had to be metabolised with the replacement of the sulphur atoms by oxygen atom(s). These active compounds are named after the parent*

[21] Source: http://www.epa.gov/ttn/atw/hlthef/carbaryl.html

[22] It is important to point out that the pesticide screen I had undertaken at Biolab, (which was limited to only those pesticides that they were able to test for at that particular laboratory at that time, as part of their pesticides screen), did not to my knowledge include testing for any pyrethroids. However, although I have not been able to obtain the information on what pesticides have been used (in relation to the majority of the years that crop-spraying has taken place in the adjoining fields) from the farmers involved, I was previously provided with some very *limited* information in relation to some of the pesticides used between 1998 and 2004. I then, out of my own enquiries with the various manufacturers for those particular pesticides, obtained the safety data sheets for them. These safety sheets were included in the original Administrative Bundle I Volume II at pages 742 to 776. As can be seen from those safety data sheets a number of the pesticides used included pyrethroids. For example, the product Hallmark, a pyrethroid insecticide **[TBI/vol II/768]**; the product Decis, **[TBI/vol II/772];** and the product Dovetail, is a pyrethroid and carbamate insecticide mixture **[TBI/vol II/764]**. From the limited information we were previously provided with by the farm manager, these particular pesticides have been sprayed repeatedly on the surrounding fields in our locality. Considering there are hundreds of different pesticides used in agriculture and that I have been exposed for over 25 years, then there are likely to be many other pesticides present in my blood and body fat, which could obviously include various pyrethroids.

[23] As can be seen from Dr. Myhill's letter of 13th April 2004, there were a number of organochlorines detected in my body fat (as well as in my blood), eg. Lindane, DDT, DDE, HCB, PCBs, amongst others. Some of these, in particular Lindane, and DDT (which includes its metabolite DDE), were previously approved for use in the UK as agricultural (and horticultural) pesticides.

*compound eg. diazinon a thionophosphate is metabolised to the phosphate, diazoxon. Some OPs are used in the 'oxon' form but these are more neurotoxic and dangerous to apply. Mevinphos is one such compound it "is a highly toxic compound in EPA (Environmental Protection Agency) toxicity class I". An unexpected property of the sulphur analogues of OPs is their ability to inhibit esterase enzymes in the liver that are responsible for the breakdown of pyrethroids. This allows pyrethroids to remain longer in the body increasing their toxicity. This is probably a major factor in the mutual synergy between pyrethroids and OPs (see Abou-Donia). Hence the attempt to synthesise less toxic OP analogues has led to increased toxicity of other pesticides that may be used at the same time, namely pyrethroids. It has been found that repeat low dose exposure to OPs gives rise to a much more toxic effect, 30 -100 times, than a single acute high dose of the same pesticide, (Abou-Donia). Residents can experience both repeated acute high dose exposures and repeated low dose exposures.* ***One aspect of this increased toxicity is the storage of OPs in body fat and the brain****. In effect this provides a depot effect whereby a low dose of the pesticide is slowly released from these stores over several days/months or longer giving rise to enhanced toxic effects."* **[Tab 2/2]**.

**Various points regarding blood and body fat testing to detect for the presence of pesticides (§13 to §16 of Mr. Hamey's statement)**

38 In relation to §13 to §16 of Mr. Hamey's third statement, it is important to point out that Biolab's pesticides screen was testing for the presence of pesticides *in general*, and therefore this covered both pesticides used for agricultural, as well as non-agricultural purposes. It was not stated that the Biolab pesticides screen was only testing for pesticides used for agricultural purposes in either §48 of my first Witness Statement **[CB1/O/9]** or in the medical report material that was submitted by the Respondent prior to handing-down of the judgment **[CB4/E/5-10]**). In fact, where chemicals were detected such as p-dichlorobenzene and polybrominated byphenyls (referred to in §13 and §14 of Mr. Hamey's third statement), Dr. Myhill had clearly pointed out in her letter dated 13th April 2004, that these are chemicals which *"are inevitable as a result of modern exposures."* **[CB4/E/9]**.

39 **However, a number of the pesticides found in my blood and/or body fat samples were pesticides used for agricultural purposes.**[24] As was pointed out in the medical report material (that was submitted by the Respondent prior to handing-down of the judgment **[CB4/E/6]**), the blood and fat tests I had carried out at Biolab were *limited* to only those pesticides that they were able to test for at that particular laboratory at that time, as part of their pesticides screen. Considering there are hundreds of different pesticides used in agriculture and that I have been exposed for over 25 years then going by these results there are likely to be many other pesticides present in my blood and body fat in addition to those identified in the aforementioned tests.

40 I was previously informed by Biolab, as well as other laboratories around the country that I made enquiries with, that to be able to test for many of the commonly used agricultural pesticides you have to know what pesticides you have been exposed to to know what to test for. **In the absence of being provided the information on what pesticides are being used by farmers (as there is no legal obligation under the Government's existing policy for farmers to provide this information) then how can any process of testing for detection of those particular pesticides even begin!**

41 Therefore as I pointed out in §20 and §21 of my first Witness Statement **[CB1/O/4]** to obtain definitive proof of causation in relation to chronic long term effects for residents is currently obstructed by the Defendant's very own policy.

42 As I have continued to argue from the outset of my campaign in 2001, mandatory requirements for access to information are absolutely imperative. Not only is it beneficial for residents and other members of the public who are exposed to pesticides sprayed in their locality so that they are able to know what they are being exposed to, it is also vital to be able to: 1) test for the presence of those particular pesticides in blood and/or body fat; 2) enable doctors to give the correct assessment and treatment of anyone who suffers adverse health effects (whether they be acute or chronic), as a doctor cannot possibly make a proper assessment of a patient's health

[24] Namely Lindane, DDT, (and its metabolite DDE), Mevinphos, Diazinon, Carbaryl (and its metabolite Alpha-Naphthol).

effects unless this information is kept and provided;[25] 3) feed back into the monitoring system, otherwise pesticide related ill-health statistics will never have a hope of being accurate or complete; and 4) provide crucial information for epidemiological purposes, as there is no way to trace exposure and correlate effects when there is no knowledge of what has been used and thus what people have been exposed to.

**Lindane (§17 of Mr. Hamey's statement)**

43 In §17 of his third Witness Statement Mr. Hamey states, *"She* [Dr. Myhill] *alleges that raised levels of lindane were found in fat, and this is "extremely toxic and a known carcinogen" and linked to breast cancer in many scientific studies" (paragraph 4). However: (1) Lindane was used as an agricultural/horticultural pesticide, also were some amenity and home garden products. No uses have been approved since spring 2002. Also it is/was available as lindane lotion/shampoo for animals and humans (control of scabies, head lice)."*

44 In response to this, first of all my family and I do not use any pesticides in our home and garden; we have not used any pesticides as lotion or shampoo for animals or as headlice treatment. We have lived next to pesticide sprayed fields since the early 1980's and have been repeatedly exposed to pesticides throughout every year for the last 25 years. Therefore irrespective as to whether Mr. Hamey says that no uses of Lindane have been approved since spring 2002,[26] Lindane was approved for use in agriculture from the time my family and I moved to the present address in 1983 until 2002, which is almost 20 years. **My father was previously informed by the farm**

[25] This has even been recognised by the Appellant itself, as documentation formulated for Ministers consideration by DEFRA's *Chemicals and Nanotechnology Division* in preparation of the Government's response to the RCEP report and recommendations, pointed out the benefits of access to the necessary chemical information in relation to gaining the appropriate medical assessment and treatment, as DEFRA officials stated, *"Benefits are in potentially improved health care from being able to diagnose or eliminate any pesticide related effects on bystander health."* **[TBIV/510/para 106]**. (NB. The Appellant, ACP and PSD (now CRD), often incorrectly refer to both residents and bystanders under just "bystanders" as per the statement referred to here. As set out in Ground 1 of my Judicial Review claim, residents and bystanders are two separate exposure groups and therefore should be referred to as such).

[26] Although there is no indication from Mr. Hamey as to what the use up period was for Lindane, as just because it has not been *approved* since Spring 2002, does not necessarily mean that that was the same as the use up date for existing stocks.

**manager that Lindane was definitely used over the years on crops in the adjoining fields.** [27]

45 Therefore the evidence clearly indicates that the raised levels of Lindane found in my body fat samples are as a result of exposure to pesticides used in agriculture.

46 It should be pointed out that the results of my body fat tests showed that the levels of Lindane in my breast were twice as high as those in my buttock. **[CB4/E/9]**.

47 Dr. Myhill correctly pointed out in her letter of 13th April 2004 that Lindane is *"extremely toxic and a known carcinogen"* and that *"Lindane has been linked to breast cancer in many scientific studies."* **[CB4/E/9]**.

48 To support this, I have attached to this sixth Witness Statement, as exhibit GD/5 **[Tab 5/1-22]**, a bibliography entitled, *"Pesticide Lindane and Breast Cancer Risk,"* by Cornell University, prepared as part of its "*Program on Breast Cancer and Environmental Risk Factors*". This bibliography contains a vast numbers of studies regarding the toxicity of Lindane and the risks of various chronic effects from exposure, such as breast cancer, as well as other cancers, mutagenic effects, reproductive effects, immunotoxic effects, hormone disrupting effects, and effects on cell proliferation, cell cycle, and cell communication. As can be seen from the attached bibliography, the order of topics include:

- Review Articles
- Review Articles and Book Chapters on the Toxicology of Lindane and its Metabolites
- Studies in Humans
  - Epidemiological Studies on Breast Cancer Risk
  - Occupational Exposure and Cancer Risk
  - Childhood and Cancer Risk

[27] Although the material has not been set forth before the court previously in this case, my mother also had the pesticide screen undertaken at Biolab at the same time as myself in 2004 (although in relation to the fat samples she had just the one taken from the buttock fat and not the breast, whereas I had fat taken from both places). She also had the same pesticides detected as I did, including Lindane, (although she had 2 additional organochlorines detected, Aldrin and Dieldrin) which definitely indicates that we have been exposed to the same pesticides from the same source, ie. that of crop-spraying in the adjoining fields. I should also point out that my mother has suffered for many years from some of the same long-term neurological effects as I have.

Levels in Human Breast Milk, Adipose Tissue and Blood
Case Reports on Toxicity
- Studies in Experimental Animals
Long Term Exposure and Cancer Bioassays
Mutagenicity
Toxicity
- Estrogenicity and Hormone Disruption
- Reproductive Effects
- Effects on Cell Proliferation, Cell Cycle, and Cell Communication
- Immunotoxic Effects
- Exposure and Pharmacokinetics in Humans and Animals
- Lindane Residues in Food and Environmental Fate

49 In response to Mr. Hamey's comments in his third statement regarding Lindane, Dr. Myhill states in her enclosed letter dated 29th May 2009, (at exhibit GD/1) that, *"I do not pretend to be a toxicologist, but I am capable of reading scientific references and there are a whole host of references in the literature indicating a link between lindane and breast cancer. I believe that the evidence is so compelling that I have little doubt that lindane is indeed causal with respect to breast cancer."* **[Tab 1/1]**.

50 As said at §47 above, this is absolutely factually correct as can be seen from the attached bibliography.

51 The Cornell University bibliography also completely counters the review undertaken by the Committee on Carcinogenicity (COC) that Mr. Hamey relies upon in §17(2) of his third statement that concluded that there was *"insufficient information to draw conclusions between human exposure and breast cancer."*[28]

52 It should also be pointed out to the court that the United Nations Environment Programme (UNEP) recently announced that Lindane was amongst a number of chemicals to be proposed for listing under the Stockholm Convention on Persistent Organic Pollutants (POPs). The press release issued from the United Nations

[28] In the context of this, I would remind the court that the COC was one of the Government advisory committees involved with providing advice to the Government regarding the resident and bystander issue following a completely inadequate consideration of the issue, in which the COC did not assess the relevant evidence, or were even aware of what the arguments were in the first place regarding the resident and bystander issue. See §162, §163, and footnotes 238, 239 and 240 of my second Witness Statement at **[CB1/Q/117-118]**.

Environment Programme on 30th April 2009[29] stated that, *"…all of these chemicals share four properties: they are highly toxic; they are stable and persistent, often lasting for decades before degrading into less dangerous forms; they evaporate and travel long distances through air and water;* ***and they accumulate in the fatty tissue of humans*** *and wildlife."*

53 The UNEP press release also states, *"Ministers and officials from 150 governments are meeting this week to advance global efforts to rid the world of some of the most hazardous chemicals produced by humankind."*

54 The UNEP press release goes on to state, *"The risks posed by such chemicals are profound and these toxic substances leave chemical footprints around the globe. Farmers, pregnant women, young people, the unborn and certain remote communities such as those in the Arctic are particularly vulnerable," said UN Under-Secretary General and UNEP Executive, Achim Steiner."*

**Mevinphos (§19 of Mr. Hamey's statement)**

55 In §19 of his third Witness Statement Mr. Hamey states, *"Dr. Myhill also says "Mevinphos" is found in fat (paragraph 5). However: (1) It has not been an agricultural pesticide in the UK since the early 1980's."*

56 I have been exposed to crop spraying since the early 1980's, therefore unless Mr. Hamey has got access to the full information of all the pesticides used over the last 25 years in my locality (which to my knowledge he has not), then he is not in a position to say, as he does in §19(4), that *"Exposure, if confirmed, unlikely to be from application adjacent to Ms. Downs' home."*

57 As can be seen from the comments above at §34 to §37, Dr. Myhill correctly stated in her letter dated 13th April 2004 that, *"Pesticides are very fat soluble compounds. Once absorbed they spend little time in the blood (measured in days and weeks) and redistribute into the fat."* **[CB4/E/9]**. Dr. Myhill went on to state, *"We are told by the*

[29] http://www.unep.org/Documents.Multilingual/Default.asp?DocumentID=579&ArticleID=6144&l=en

*authorities that these chemicals are supposed to be rapidly broken down in the body and excreted, but as you can see from this result, these pesticides are persistent, recognizable and will continue to poison the body in this form."* **[CB4/E/9]**.

58 In response to Mr. Hamey's comments in his third statement regarding Mevinphos, Dr. Myhill states in her enclosed letter dated 29th May 2009 (at exhibit GD/1) that, *"…the fact that mevinphos was present in Georgina's fat despite a low level of lipid solubility indicates exposure must have been high for it to have been found at all!"* **[Tab 1/1]**.

59 As pointed out earlier in §35 above, in relation to Mevinphos, Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, points out in his comments (at exhibit GD/2) that, "…*mevinphos is, atypically, miscible with water and many organic solvents, alcohols, acetone but not hexane, log* $P_{o/w}$ *=1.2; making it 15 times more soluble in fat than in water."* **[Tab 2/1]**.

60 This counters Mr. Hamey's assertion in §19(2) of his third Witness Statement that, *"The compound's Kow log P = 0.127 which indicates that it is not likely to accumulate in fat."*

61 In the data sheet for Mevinphos prepared by the International Programme on Chemical Safety (IPCS), included at exhibit GD/4 **[Tab 4/1-3]**, under acute symptoms from exposure via the inhalation route, it states, *"Pupillary constriction, muscle cramp, excessive salivation. Blurred vision. Sweating. Nausea. Vomiting. Diarrhoea. Abdominal cramps. Dizziness. Convulsions. Unconsciousness."* **[Tab 4/1]**. In the next column regarding prevention of exposure it states, *"Strict Hygiene! Avoid Exposure of Adolescents and Children!"* and then advocates *"breathing protection."*[30] **[Tab 4/1]**. Then in the next column under *"First Aid"* it states, *"In all*

[30] Although an operator will be required to wear appropriate protective equipment, residents and other members of the public, who may be only inches away, breathing in the very same airborne droplets, particles and vapours that workers are required to have protection from would not be expected to wear personal protective equipment (PPE) while going about their business in their own private homes and gardens. Therefore as Mr. Justice Collins rightly recognised (see eg Judgment §§15, 29, 35, 45) the use of pesticide labelling to alert users of the risks, and of recommendations for PPE (eg. protective clothing, gloves, respirators, etc.), is of course entirely useless as a way of protecting residents and bystanders.

*cases consult a doctor!"* and advocates *"Fresh Air"* and to *"Refer for medical attention immediately."*[31] **[Tab 4/1]**. Under acute symptoms, from exposure via the eyes, it states, *"Blurred vision"* and in the next column regarding prevention of exposure to the eyes it states, *"Face shield, or eye protection in combination with breathing protection."* [32] **[Tab 4/1]**.

62 The data sheet then goes on to detail the hazard symbols and risk and safety phrases in relation to the product packaging and labelling which contains a number of very serious warnings[33] including, *"Danger," "Fatal if swallowed," "Fatal in contact with skin," "Fatal if inhaled vapour,"* [34] *"Causes damage to the nervous system."* **[Tab 4/2]**.

63 Cornell University's *Pesticide Information Profile* sheet for Mevinphos[35] states that, *"Mevinphos is one of a class of insecticides referred to as organophosphates. These chemicals act by interfering with the activities of cholinesterase, an enzyme that is essential for the proper working of the nervous systems of both humans and insects."*

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[31] The data sheet goes on to state, *"Specific treatment is necessary in case of poisoning with this substance; the appropriate means with instructions **must** be available."* **[Tab 4/3]**. Therefore again this underlines the importance of knowing the pesticides involved in relation to gaining immediate medical attention. This has even been recognised by the Appellant itself, as documentation formulated for Ministers consideration by DEFRA's *Chemicals and Nanotechnology Division* in preparation of the Government's response to the RCEP report, stated, *"Benefits of direct access to spray records will mostly be for acute exposure where time is potentially critical in terms of determining correct treatment."* **[TBIV/510/para 106]**.

[32] The same point applies as that made in footnote 30.

[33] Which are listed in the data sheet under "*GHS Classification*." This is the *Globally Harmonised System of Classification and Labelling of Chemicals (GHS)*, which provides a harmonised basis for globally uniform physical, environmental and health and safety information on hazardous chemical substances and mixtures. See http://ec.europa.eu/enterprise/reach/ghs/index_en.htm

[34] Under "*Important Data*" the data sheet points out that the routes of exposure in which the substance can be absorbed into the body include "*by inhalation of its aerosol, through the skin and by ingestion*." **[Tab 4/3]**. It then points out, under "*Inhalation Risk*" that *"A harmful contamination of the air can be reached rather quickly on evaporation of this substance at 20°C."* **[Tab 4/3]**. Considering the number of times that spraying can take place between April and September then the temperatures would have, on many occasions, well exceeded that of 20°C, during the time this pesticide was approved for use in the UK. Obviously residents and other members of the public will not have been aware of the clear warnings including, *"Fatal if inhaled vapour"* in the absence of any mandatory prior notification or access to information requirements, and so will not have known the information necessary to make informed and knowledgeable decisions to protect their health and the health of their family from any harm. The same applies obviously to *any* pesticides used near resident's homes, children's schools and playgrounds etc. as residents and others exposed, who are not operators, are not legally entitled to know this information and therefore will not be aware of the risks and adverse effects involved in *any* exposure, let alone prolonged repeated exposures that residents will receive.

[35] Source: http://pmep.cce.cornell.edu/profiles/extoxnet/metiram-propoxur/mevinphos-ext.html

64 Under "*Toxicological Effects*" and then "*Acute Toxicity*" the Cornell University sheet states, *"Mevinphos is highly toxic through all routes of exposure, including ingestion, dermal absorption and inhalation. Poisoning affects the central nervous system, the cardiovascular system, the respiratory system and the eyes. The greatest occupational hazard is absorption of mevinphos through the skin, lungs, and mucous membranes. Its toxic action is direct and quick, regardless of the route of exposure. In humans, symptoms of poisoning have appeared within as little as 15 minutes or 2 hours after exposure to mevinphos, but onset of symptoms have been delayed for as long as 2 days. As with all organophosphates, mevinphos is readily absorbed through the skin. Skin which has come in contact with this material should be washed immediately with soap and water and all contaminated clothing should be removed.* [36] *The severity of mevinphos poisoning will determine the number and types of symptoms which will result. Poisoning is also influenced by the length and concentration of exposure. Persons with respiratory ailments, recent exposure to cholinesterase inhibitors, impaired cholinesterase production, or with liver malfunction may be at increased risk from exposure to mevinphos."*

65 It then states, *"An early and important symptom of mevinphos poisoning from dermal exposure is impairment of judgment or the ability to reason. Other symptoms of poisoning from exposure to the insecticide include giddiness, tightness in the chest, blurred vision, tearing, hearing irregularities, loss of muscle coordination, slurred speech, mental confusion, breathing difficulty, increased blood pressure, convulsions, coma. Several children were made ill by unknowingly wearing clothing which had been contaminated with mevinphos."*

66 The Cornell University sheet then states, *"The organophosphate insecticides are cholinesterase inhibitors. They are highly toxic by all routes of exposure. When inhaled, the first effects are usually respiratory and may include bloody or runny nose, coughing, chest discomfort, difficulty breathing or shortness of breath, and*

[36] Again, during the time this pesticide was approved in the UK, residents (and bystanders) will not have known that this was the correct action to take if they had not seen the safety data sheet information that advises to do this in the event of exposure (and in any event they may not have even been aware that they had been exposed to pesticides in the first place).

*wheezing due to constriction or excess fluid in the bronchial tubes. Skin contact with organophosphates may cause localized sweating and involuntary muscle contractions. Eye contact will cause pain, bleeding, tears, pupil constriction, and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours. These may include pallor, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, eye pain, blurred vision, constriction or dilation of the eye pupils, tears, salivation, sweating, and confusion. Severe poisoning will affect the central nervous system, producing incoordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids, and eventually paralysis of the body extremities and the respiratory muscles. In severe cases there may also be involuntary defecation or urination, psychosis, irregular heart beats, unconsciousness, convulsions and coma. Death may be caused by respiratory failure or cardiac arrest."*

67 Under "*Toxicological Effects*" and then "*Chronic Toxicity*" it states, *"Repeated or prolonged exposure to organophosphates may result in the same effects as acute exposure. Cholinesterase-inhibition resulting from mevinphos exposure can persist for two to six weeks. Repeated exposure to small amounts of this material may result in an unsuspected inhibition in cholinesterase levels. This can cause symptoms, such as weakness, lack of energy, and lack of appetite,* ***that are similar to other illnesses, such as the flu.***[37] *Other effects reported in workers repeatedly exposed include impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia. Severe symptoms of cholinesterase-inhibition may be produced in a previously exposed person, whereas symptoms of cholinesterase-inhibition may not be obvious in a person that has not been previously exposed to the same concentration of mevinphos. The monitoring of cholinesterase*

[37] As detailed in §4 and §8 of my first Witness Statement at **[CB1/O/1-2]** I regularly suffered from amongst other things, flu-type illnesses, (as well as headaches, dizziness, giddiness, tinnitus and memory and concentration problems, muscle wastage and weakness, which are all neurologically based symptoms).

*levels through regular blood testing is highly recommended for those persons with exposure to this material."*

68 Although this may happen for operators there is no medical surveillance for residents and other members of the public exposed to organophosphate pesticides, which for residents can be on a regular basis, (as well as exposure to mixtures of all other classes of pesticides used in crop-spraying). Obviously in the absence of any access to information and prior notification requirements, then many residents may not even be aware that they have been exposed, or what they have been exposed to.[38]

69 Under "*Organ Toxicity*" it states, *"Mevinphos primarily affects the nervous system through cholinesterase inhibition, by which there is a deactivation of cholinesterase, an enzyme required for proper nerve functioning. Acute pulmonary edema, or the filling up of lungs with fluid, and changes in the structure or function of salivary glands, were seen in rats that were exposed to an air concentration of 14 ppm for one hour. Rats given dietary doses of 10 or 20 mg/kg for 13 weeks exhibited degeneration of livers, kidneys and cells lining the salivary, tear and other glands, as well as clinical signs of poisoning."*

**Diazinon (§20 of Mr. Hamey's statement)**

70 In §20 of his third Witness Statement Mr. Hamey states, *"She* [Dr. Myhill] *also alleges that "Diazinon" is found in fat (paragraph 5). On the other hand: (1) It was approved for use on carrots until 1995, and approved for use on mushrooms (indoors) until 2000. Therefore it was unlikely to be applied adjacent to Ms. Downs' home."*

[38] The Cornell University sheet also points out that, *"Persons who work with organophosphate materials for long periods of time should have frequent blood tests of their cholinesterase levels. If the cholinesterase level falls below a critical point, no further exposure should be allowed until it returns to normal."* Again, residents and other members of the public exposed to organophosphate pesticides, which for residents can be on a regular basis, (as well as exposure to mixtures of all other classes of pesticides used in crop-spraying), will a) not be having any medical surveillance in relation to their exposures to even know whether their cholinesterase level have fallen below a critical point, and b) in any event, will not able to prevent further exposures from occurring anyway.

71 Again, as stated in §56 above, I have been exposed to crop spraying since the early 1980's, therefore unless Mr. Hamey has got access to the full information of all the pesticides used over the last 25 years in my locality (which to my knowledge he has not), then he is not in a position to say that Diazinon *"was unlikely to be applied adjacent to Ms. Downs' home."* (Also see earlier comments at §35 and §37 above, regarding the detection of Diazinon in my body fat samples).

72 Like Mevinphos, Diazinon is also an organophosphate pesticide.

73 Regarding the various classes of pesticides, the European Commission has stated[39], *"Under real life conditions, acute and chronic adverse effects associated with exposure to the common classes of pesticides can vary a lot for a given substance or substance class. Conversely, different substances or substance classes can cause similar symptoms. For example, the following have been reported for certain classes of insecticides:*

- *ORGANOPHOSPHATES can cause headaches, pain, weakness, numbness in extremities, dizziness, damage to memory, mood control, chest tightness, loss of coordination, uncontrolled urination, seizures, death due to respiratory failure;*
- *CARBAMATES can cause headaches, genetic mutations, vomiting, birth defects, dizziness, reduced fertility, seizures, kidney damage, shortness of breath, nervous system damage;*
- *PYRETHRINS and PYRETHROIDS can cause lack of coordination, deep lung allergy, convulsions, pneumonia, muscle paralysis, vomiting, asthma and death due to respiratory failure."*

74 Residents can be exposed (unknowingly) to all these classes of pesticides, along with other classes, (as well as to innumerable *mixtures* of these and other classes), repeatedly, throughout every year, and in many cases, like my own situation, for

[39] *"Impact Assessment of the Thematic Strategy on the Sustainable Use of Pesticides"* **[TBIV/771].**

decades, with no protection from the risks, and related acute and chronic adverse impacts.

75 In the data sheet for Diazinon prepared by the International Programme on Chemical Safety (IPCS), included at exhibit GD/6 **[Tab 6/1-4]**, under acute symptoms from exposure via the inhalation route, it states, *"Pupillary constriction, muscle cramp, excessive salivation. Laboured breathing. Nausea. Vomiting. Dizziness. Convulsions. Unconsciousness."* **[Tab 6/1]**. In the next column regarding prevention of exposure it again states, *"Strict Hygiene! Avoid Exposure of Adolescents and Children!"* and then advocates *"breathing protection"* (see earlier comments regarding the data sheet for Mevinphos at §61 and footnote 30). It then says *"Avoid inhalation of mist"* (which is obviously impossible in the kind of situation that myself and other rural residents are living in). Then in the next column under *"First Aid"* it again states, *"In all cases consult a doctor!"* and advocates *"Fresh Air"* and to *"Refer for medical attention immediately"* (see earlier comments at §61 and footnote 31). It also says *"Artificial respiration may be needed."* **[Tab 6/1]**. Under acute symptoms, from exposure via the eyes, it states, *"Redness. Pain,"* and in the next column regarding prevention of exposure to the eyes it again states, *"Face shield, or eye protection in combination with breathing protection"* (see earlier comments at §61 and footnote 30). Under acute symptoms, from exposure via ingestion, it states, *"Abdominal cramps. Diarrhoea."* **[Tab 6/1]**.

76 The data sheet goes on to state under, *"Effects of short-term exposure,"* that, *"The substance is mildly irritating to the eyes and the skin. The substance may cause effects on the nervous system, resulting in convulsions and respiratory depression. Cholinesterase inhibitor. The effects may be delayed. Medical observation is indicated."* **[Tab 6/3]**. Under *"Effects of long-term or repeated exposure,"* it states, *"Cholinesterase inhibitor; cumulative effect is possible."* **[Tab 6/3]**.

77 A study published in 2004 in *Clinical Toxicology*[40] entitled *"Health Effects of Diazinon on a Family,"*[41] found persistent neurological effects in all 7 members of a

[40] Volume 42, No. 5, Pages 579-591. The abstract is included at exhibit GD/7 **[Tab 7/1]**.

family that had been exposed (in June 1999) to Diazinon through cutaneous absorption and inhalation. The abstract of the study which can be seen at exhibit GD/7 **[Tab 7/1]** states, *"Acute symptoms in the family members included headaches, nausea, skin irritation, runny nose, and vomiting. The family was first evaluated at 3 months and then 3 years after the acute poisoning. There were persisting neurological symptoms of memory loss, decreased concentration, irritability, and personality changes of varying degrees in all family members. Objective neurological findings of impaired balance, reaction time, color vision, slotted pegboards and trials making were present in the three older children who could be tested. Neuropsychological evaluation revealed evidence of organic brain dysfunction in all seven family members."* **[Tab 7/1]**.

78 In addition to the long-term neurological impacts, the abstract of the study states, *"In addition, we observed skeletal and endocrine effects likely attributable to the diazinon poisoning."* **[Tab 7/1]**. The study found that bone growth difficulties were present in four of five children, and that one child had delayed menarche. **[Tab 7/1]**.

**Carbaryl (§21 of Mr. Hamey's statement)**

79 In §21 of his third Witness Statement Mr. Hamey states, *"She* [Dr. Myhill] *also says "Carbaryl" is found in fat (paragraph 5). However: (1) It was approved for use on turf (worm control) until 1998, and approved for use on apples until 2001. Therefore is was unlikely to have been applied adjacent to Ms. Downs' home."*

80 Again, as stated in §56 and §71 above, I have been exposed to crop spraying since the early 1980's, therefore unless Mr. Hamey has got access to the full information of all the pesticides used over the last 25 years in my locality (which to my knowledge he has not), then he is not in a position to say that Carbaryl *"was unlikely to have been applied adjacent to Ms. Downs' home."* (Also see earlier comments at §32 and §35 above, regarding the detection of Carbaryl in my body fat samples).

[41] By J.G Dahlgren, H.S Takhar, C.A. Ruffalo and M. Zwass.

81 Carbaryl is a carbamate pesticide. I have attached to this sixth Witness Statement, as exhibit GD/8 **[Tab 8/1-6]**, a 2005 factsheet produced by a scientist in the United States regarding Carbaryl.

82 As can be seen, the factsheet states, *"Carbamates affect human nerves in much the same way as they affect insect nerves. The nerves they affect are different, however. In insects carbamates affect the central nervous system. In humans they affect the interactions between nerves and muscles."* **[Tab 8/2]**.

83 Under *"Symptoms of Carbaryl Poisoning,"* the factsheet states, *"Symptoms of poisoning in people exposed to carbaryl include irritated, swollen, congested, stinging or burning eyes as well as sore or burning throat, chest tightness, wheezing, sweating, dry heaves, nausea, and vomiting, according to reports collected by the California Environmental Protection Agency."* **[Tab 8/2]**.

84 Under *"Effects on the Nervous System,"* the factsheet states, *"Given that carbaryl's pesticidal activity results from disrupting nerve function, it is not surprising that the nervous system is affected in laboratory tests. In a study sponsored by a carbaryl manufacturer, exposure to a single dose of carbaryl caused behavioral changes (decreases in activity) as well as decreases in the activity of acetylcholinesterase. These effects occurred at all dose levels tested in this experiment."* **[Tab 8/2]**.

85 Under *"Ability to Cause Genetic Damage (Mutagenicity),"* the factsheet states, *"The National Institute for Occupational Safety and Health labels carbaryl as a mutagen and has identified over 20 studies conducted in the 1970s and 1980s documenting carbaryl's ability to cause genetic damage. A recent study provided more details about this ability to cause genetic damage. A group of scientists led by a toxicologist from the Institut National de la Recherche Agronomique (France) studied carbaryl in 2001. They found, in human cells, that carbaryl stimulates the activity of an enzyme that transforms carbaryl into a compound that causes damage to DNA, the genetic material in living organisms."* **[Tab 8/2-3]**.

86 Under *"Ability to Cause Cancer (Carcinogenicity),"* the factsheet states, *"In 2001, EPA classified carbaryl as "likely to be carcinogenic in humans," based on a study sponsored by a carbaryl manufacturer which found that carbaryl caused malignant blood vessel tumors in laboratory mice. In this study, as well as in an additional study of laboratory rats, carbaryl also caused kidney and liver tumors. Consistent with these laboratory experiments, two recent studies have found an association between use of carbaryl by farmers and an increased risk of the cancer non-Hodgkin's lymphoma (NHL). One study, conducted by researchers from Yale University, the National Cancer Institute, and the University of Nebraska, found that farmers in four Midwestern states who used carbamate pesticides "had a 30% to 50% increased risk of NHL."…The second study, led by a researcher from the University of Saskatchewan, found that the risk of NHL in Canadian farmers was significantly increased by exposure to carbaryl."* **[Tab 8/3-4]**.

87 Under *"Effects on the Developing Brain,"* the factsheet states, *"A study sponsored by a carbaryl manufacturer showed that carbaryl exposure during pregnancy and nursing affects the developing brain. The study showed that two parts of the brain were smaller in offspring of exposed animals than in unexposed offspring."* **[Tab 8/4]**.

88 Under *"Carbaryl on Skin,"* the factsheet states, *"Carbaryl is absorbed through skin and* ***ends up in a variety of tissues and organs****. Scientists from the Institute of Agricultural Medicine (Poland) showed that carbaryl applied to skin of laboratory animals* ***ended up in the liver, blood, and brain****."* **[Tab 8/4]**.

89 Again, this counters Mr. Hamey's assertion in §21(3) of his third statement, that Carbaryl *"does not accumulate in tissues, but is rapidly metabolised and excreted."*

90 There are also sections in the attached factsheet (at exhibit GD/8 **[Tab 8/1-6]**), regarding Carbaryl's ability to disrupt hormones and the endocrine system; as well as effects on the immune system; amongst other known effects, such as on sperm and in relation to miscarriage.

**Combined/synergistic effects, accumulative toxicity (§22 of Mr. Hamey's statement)**

91 In §22 of his third Witness Statement Mr. Hamey states, *"Dr. Myhill alleges that there is "Good evidence…Combined effects…are far greater than…effects in isolation." (paragraph 6). However: (1) This is not supported by the COT WIGRAMP report, nor the more recent opinion from the EFSA PPR Panel. (2) Dose addition has been demonstrated where different compounds have similar effects, but where no response is seen to individual compounds their combined presence is not a concern."*

92 The full statement that Dr. Myhill actually made in her letter dated 13th April 2004 is that, *"There is now good evidence that with chemicals one sees a cocktail – that is to say that the combined effects of chemicals are far greater than their effects in isolation. Therefore, the accumulative toxicity of these chemicals is likely to be high."* **[CB4/E/9-10]**.

93 Dr. Myhill's statement is factually correct, and there are a number of studies to support this. First of all, a study published in "*Toxicology*," in January 2002 entitled, *"Interactions between pesticides and components of pesticide formulations in an in vitro neurotoxicity test,"* by J.C. Axelrad, C.V. Howard, W.G. McLean.[42] Some important statements from this study include:

a) *"Organophosphate (OP) pesticides are often used in combination with one another and with the components of formulations. Evidence already exists for interactions in the neurotoxic effects of OPs through interference with metabolism, but there is also potential for interactions related directly to cell damage. The purpose of this work was to investigate this possibility for OPs and the components of one of their common formulations in vitro. NB2a neuroblastoma cells were induced to differentiate in the presence of the OPs diazinon and chlorpyrifos, in combination with a commercial formulation (identified as Commercial Formulation 1) of the compounds and, independently, the components of that formulation."*

[42] The abstract of this study can be seen at:- http://www.ncbi.nlm.nih.gov/pubmed/11960678

b) *"Synergism was detected between combinations of:…chlorpyrifos and…pyrethrum; chlorpyrifos and one of the solvents (regular spirit) found in Commercial Formulation 1."*

c) *"The data suggest that exposure to multiple OP-containing pesticide formulations may lead to synergistic neurotoxicity by a direct mechanism at the cellular level."*

d) *"Where pesticides are used in combinations, there is a potential for interactions not only between the pesticides, but also between pesticides, solvents and potentiators."* (I referred to this in my second Witness Statement at §8(c) and related footnotes **[CB1/Q/7-8]** and §56(g) and related footnotes **[CB1/Q/53-55]**).

e) *"An additional factor that might contribute to synergism in vivo would be alterations in the blood–brain barrier known to occur as a consequence of exposure to OPs and pyrethroids (Gupta et al., 1999)."* (It is important to point out to the court that the inhalation route of exposure to pesticides is highly significant in relation to impacts on the blood-brain barrier. This important barrier can be breached by some chemicals at very low levels including OPs so that other toxins can enter directly, (Vogel JS, Garrettt A, Keating II, Buchholz BA. *Protein Binding of isofluorophate: in Vivo after Coexposure to Multiple Chemicals*. Environmental Health Perspectives 2002;110suppl 6:1-7; Abou-Donia MB, Goldstein LB, Dechovskaia A, Bullman S, Jones KH, Herrick EA, Abdel Rahman AA, Khan WA. *Effects of daily dermal application of DEET and epermethrin, alone and in combination, on sensorimotor performance, blood-brain barrier, and blood-testis barrier in rats*. J Toxicol Environ Health A. 2001 Apr 6;62(7):523-41). A further point is that the blood-brain barrier can be by-passed when compounds are carried by intraneuronal transport into the brain, (Ashford AN, Miller CS. *Chemical Exposures: Low Levels and High Stakes*, 2nd Edition, John Wiley, New York, 1998)).

f) *"Thus, the result is relevant to the situation of exposure to multiple agents; as…the hazard associated with exposure to chlorpyrifos in formulation may be*

*amplified several hundred-fold in the presence of a second formulation or another pesticide."*

g) *"In conclusion, these data present evidence that, in addition to possible interactions between pesticides and the products in their formulations at the level of metabolism, there is a potential for interactions that lead to enhanced toxicity at the level of the nerve cell…**The results demonstrate the potential for a substantial increase in neurotoxicity accompanying various combinations of pesticides and their formulations. Increasing the number of different products to which an individual is exposed might be expected to increase the potential for such interactions.**"*

94 As set out in detail in §56(g) of my second Witness Statement at **[CB1/Q/53-55]** residents and communities are exposed on a long-term basis to mixtures of pesticides, repeatedly sprayed, in their locality, throughout every year, and in many cases, like my own situation, for decades. This could realistically result in exposure to literally hundreds[43] of different pesticides and other chemicals, and residents could receive relatively high dose exposures (on a regular basis), along with lower dose exposures (on a regular basis) from the contamination of their surrounding environment.

95 A study by Mohamed Abou-Donia MB, entitled *"Organophosphorus ester-induced chronic neurotoxicity"* was published in the Journal of Occupational Health Safety — Aust NZ 2005, 21(5): 408-432, in 2005. Some important statements from this study include:

a) *"Repeated small exposures have cumulative effects. Early symptoms of chronic organophosphorus insecticide exposure **are influenza-like symptoms**.[44] As exposure continues, clinical manifestations appear until a full picture develops."*

b) *"Organophosphorus ester-induced delayed neurotoxicity is a neurodegenerative disorder characterised by a delayed onset of prolonged ataxia and upper motor*

[43] Or even more.
[44] As stated in footnote 37 above, §8 of my first Witness Statement at **[CB1/O/1-2]** pointed out that I regularly suffered from amongst other things, flu-type illnesses.

*neurone spasticity from a single or repeated exposure to organophosphorus esters."*

c) ***"Chronic or subchronic exposures to small daily doses of organophosphorus compounds are more toxic and efficient in producing OPIDN than large single doses."***

d) *"Organophosphorus compounds have greater access to the neurotoxicity target through inhalation and skin penetration than the gastrointestinal tract,* ***with inhalation being the most effective route of entry,*** *preceded only by intravenous injection."*

e) *"…severe cases of OPIDN that involve damage to the central nervous system would persist, as the central nervous system does not regenerate."*

f) *"Alterations to the cytoskeletal structure are prominent features in some neurological diseases and chemically induced neurological disorders."*

96 I have included a few other statements taken from this study at exhibit GD/9 **[Tab 9/1]**. I have also included at exhibit GD/9 **[Tab 9/2]**, a one page biography of Professor Mohamed Abou-Donia, of the Department of Pharmacology and Cancer Biology, Duke University Medical Center in the United States, as he is one of the leading experts in relation to chemically induced neurodegenerative disorders, as well as the synergistic effects of pesticide mixtures. (Although please note that when I spoke to Professor Abou-Donia recently he pointed out that the biography is one from a few years ago as it has not yet been updated, and therefore it does not list his most recent publications and research studies).

97 In addition, regarding increased toxicity due to potentiating or synergistic interaction from exposure to mixtures of different pesticides and other chemicals used in agriculture, (again in response to §22 of Mr. Hamey's third Witness Statement), I would also refer the court to the recent study (published in March 2009) in Tab K of Core Bundle 4, entitled, *"Parkinson's Disease and Residential Exposure to Maneb and Paraquat From Agricultural Applications in the Central Valley of California,"*

by Sadie Costello, Myles Cockburn, Jeff Bronstein, Xinbo Zhang, and Beate Ritz. Some important statements from this study include:

a) In the summary information at the beginning on page 1 it states, ***"Exposure to both pesticides within 500 m of the home increased PD risk by 75%...This study provides evidence that exposure to a combination of maneb and paraquat increases PD risk, particularly in younger subjects and/or when exposure occurs at younger ages."*** **[CB4/K/1].**

b) Page 1 of the study also states, *"…pesticides applied from the air* ***or ground*** *may drift from their intended treatment sites, with measurable concentrations* ***subsequently detected in the air,*** *in plants, and in animals* ***up to several hundred meters from application sites****…."* **[CB4/K/1].** (As can be seen in paragraphs 72 to 80 of my first Witness Statement **[CB1/O/13-14]** pesticides can travel in the air vast distances through long-range transportation and there are a number of studies before the court to show that pesticides have been found miles away from where they were originally applied (eg. Alarcon *et al*, 2005 at **[TBII/794-804]**; Lee *et a*l, 2002 at **[TBI/vol II/998-1007]**).

c) On page 3 of the study it states, *"We did not find increased risks of PD among subjects exposed to paraquat alone during the years 1974–1999. While the rarity of sole maneb exposure (4 subjects) precluded any meaningful interpretation of the maneb-only results,* ***combined exposure to both maneb and paraquat increased the risk of PD by 75%*** *(odds ratio (OR) ¼ 1.75, 95% confidence interval (CI): 1.13, 2.73), an effect estimate which was essentially unchanged after adjustment for occupational pesticide exposure (OR ¼ 1.74, 95% CI: 1.11, 2.72)."* **[CB4/K/3].**

d) On page 3 it also states, *"Furthermore, for younger (≤60 years) subjects, exposure to both maneb and paraquat in both windows* ***increased PD risk as much as 4- to 6-fold****…"* **[CB4/K/3].**

e) Page 3 of the study goes on to state, *"Exposure to either maneb or paraquat alone during 1974–1989* ***also increased risk of PD in younger subjects*** *(OR ¼ 2.27, 95% CI: 0.91, 5.70)."* **[CB4/K/3].**

f) On page 3 and 4 of the study it states, *"In this population-based case-control study, agricultural application of both maneb and paraquat* ***within 500 m of a residence*** *during the period 1974–1999* ***greatly increased the risk of developing PD****, especially when exposure occurred between 1974 and 1989 or when PD was diagnosed at a younger age (≤60 years). Exposure to both pesticides during the earlier time window (1974–1989) also doubled the risk for older cases.* ***Associations were particularly strong for younger-onset patients (≤60 years), who would have been children, teenagers, and young adults during the exposure period: Among those exposed in the earlier time window, risk was increased more than 4-fold with exposure to both pesticides and more than 2-fold with exposure to just 1 of the pesticides. Consistent with some theories regarding the progression of PD pathology (25), these data suggest that the critical window of exposure to toxicants may be years before the onset of motor symptoms which lead to diagnosis."***[45] **[CB4/K/3-4].**

g) On page 6 of the study it states, *"****Persons living near fields sprayed with maneb and paraquat may also be exposed to a host of other agricultural chemicals****. **When we controlled for the influence of other groups of pesticides suspected a priori to be risk factors for PD in our study, the odds ratios for combined maneb and paraquat exposure and PD in the younger subjects were still in the 3- to 6-fold range and statistically significant;** however, our precision decreased, probably because of correlated exposures. Correlation between pesticides is an*

[45] §56(i) of my second Witness Statement at **[CB1/Q/57-59]** was in relation to exposure for vulnerable groups, such as babies and children etc. where the health risks are increased. Also it is important to note that the pesticides and Parkinson's study at Tab K of CB4 appears to be only related to exposure through air (both in terms of drift as well as ambient air), but does not appear to include other exposure factors, such as children playing in gardens etc., (although it is noted on page 6 of the study that it acknowledges that, *"Such strong binding could result in contaminated soil getting blown or tracked into homes by wind, pets, and shoes, thereby increasing exposure for persons who live closer to agricultural application sites…"* **[CB4/K/6]**). Therefore the risk would be increased *even further* when the overall exposure for residents in totality is included in the exposure calculations, (ie. exposure to innumerable mixtures of pesticides from all exposure factors, via all exposure routes, for decades etc.)

*inherent problem when assessing the effects of human exposure.* ***However, since adjustment for other pesticides did not remove the association for maneb and paraquat, our data provide compelling evidence that these 2 pesticides may in fact affect PD risk in humans, as has been suggested by animal experiments."*** **[CB4/K/6].**

h) Page 6 goes on to state, ***"Our analysis has confirmed 2 previous observations from animal studies: 1) exposure to multiple chemicals may potentiate the effect of each chemical (of interest, since humans are often exposed to more than 1 pesticide in the environment) and 2) the timing of exposure is important. To our knowledge, this is the first epidemiologic study to provide strong evidence that 2 specific pesticides, suggested by animal research as potentially acting synergistically to become neurotoxic, strongly increase the risk of PD in humans, especially given combined exposure and when encountered earlier in life."*** **[CB4/K/6].**

98 This study again supports the argument that the Appellant's existing risk assessment for *bystanders* which is based on exposure to only *one* individual pesticide at a time, is wholly inadequate in relation to residents, who are exposed in a *realistic* residents exposure scenario, to *mixtures* and combinations of pesticides over the long-term.[46]

99 A recent major research report on Gulf War Illness entitled, "*Gulf War Illness and the Health of Gulf War Veterans: Scientific Findings and Recommendations*," by the US Research Advisory Committee on Gulf War Veterans' Illnesses, U.S. Department of Veterans Affairs, in Washington, D.C, and published by the U.S. Government

[46] As I have pointed out previously, when residents are exposed over the long-term to ongoing mixtures and then go on to suffer a chronic illness or disease it will be almost impossible to know which pesticide led to the illness or whether it is actually more likely to be as a result of synergistic effects of mixtures of pesticides and the long term cumulative build up in the individuals. Also it is important to note that there can also be a long latency period between exposure and any chronic effects and therefore exposure could lead to a chronic condition 5, 10, 15 years later, or longer. Both these points were recognised by the RCEP in §1.16 of the RCEP report that stated, *"If there is an effect, it could relate to a single acute exposure with a long latency or to repeated doses over time, that can be described as chronic exposure. Linking chronic health effects to a particular exposure or pesticide will not usually be possible. Different pesticides are used during the year to deal with different problems…or from year to year as different crops are grown. The difficulty is further compounded by changes in the pesticides available commercially. New pesticides are developed and older ones withdrawn."* **[CB2/I/10]**.

Printing Office, in November 2008,[47] makes a number of important statements regarding the human health adverse impacts of pesticide exposure, including combined exposures of mixtures of pesticides, and synergistic effects.[48]

100 A few of these important statements,[49] regarding the human health adverse impacts of pesticides, particularly in relation to the neurotoxicity of pesticides and related neurological damage/injury and adverse effects, include the following.

101 On page 151 the report states, *"Chemical pesticides and insect repellants have been in widespread use since the middle of the last century. Many pesticides are neurotoxic by design, that is, they are developed to kill insects by attacking their nervous systems. Thousands of compounds have been developed for use against different insects and other pests, for application in different settings…Many thousands of research studies have evaluated biological effects of pesticides in animal models and health effects in human populations. Detailed information from this large literature is compiled in comprehensive textbooks on pesticide toxicology. New findings related to toxicological effects of pesticides and insect repellants continue to emerge at a rapid pace.* ***In recent years, this research has provided important insights into health effects of lower-level and chronic pesticide exposures in human populations, including associations with persistent symptomatic illness."***

102 Page 151 of the report goes on to state, *"In the last several years, major reports have been released internationally by government and scientific panels concerning effects of pesticide exposures on the public health. These reports have raised awareness of recent findings on potential associations between pesticides and a broad spectrum of human diseases, including difficult-to-diagnose multisymptom conditions. This*

47 http://www1.va.gov/rac-gwvi/docs/GWIandHealthofGWVeterans_RAC-GWVIReport_2008.pdf

48 Considering Appendix I of the Royal Commission on Environmental Pollution (RCEP) report **[CB2/I/152-154]** was related to *"Lessons from the Gulf War Syndrome,"* then it is important for the court to be aware that this recent major research report identifies a causal link between pesticide exposure and Gulf War illness. For example, this can be seen on page 225 of the report under *"Pesticides"* where it states, *"Taken together, all available sources of evidence combine to support a consistent and compelling case that pesticide use during the Gulf War is* ***causally*** *associated with Gulf War illness."*

49 Which I only refer to as they are related to the human health adverse impacts of pesticide exposure, including combined exposures of mixtures of pesticides, and synergistic effects, and not to get engaged into issues involving Gulf War Syndrome itself.

*includes a review of the scientific literature on health effects of pesticides from the Ontario College of Family Physicians, which concluded that "there is a high level of consistency in results to indicate a wide range of pesticide-related clinical and subclinical health effects" and that "exposure to all the commonly used pesticides—phenoxyherbicides, organophosphates, carbamates, and pyrethrins—has shown positive associations with adverse health effects."* [50] *The report urges physicians to become more aware of health effects of pesticides in order to better educate the community and treat their patients. A 2005 report on human health affects of agricultural pesticides from the British Royal Commission on Environmental Pollution described the complex issue of discerning chronic health effects resulting from unmeasured and varying combinations of pesticide exposures. The Commission noted that "the clinical awareness of general practitioners and specialists needs to be improved in order to improve the investigation of people with chronic, ill-defined health effects."*

103 On page 152 the report states, *"Organophosphate compounds are a large and diverse family of chemicals that include hundreds of different pesticides, as well as several types of chemical warfare agents. They are among the most extensively studied of any chemicals in toxicology. Acute effects of excess exposure to OP pesticides are those previously described for AChE inhibiting chemicals generally, and include effects on the central nervous system, autonomic nervous system, and skeletal muscles. There are also a growing number of indicators that OPs can exert neurotoxic effects through mechanisms other than AChE inhibition. In addition, recent studies in animal models have demonstrated that repeated exposures to OP pesticides, even at relatively low doses, can produce persistent neurochemical and behavioral alterations that do not occur with single exposures at similar or higher doses."* [51]

[50] The Ontario College of Family Physicians 2004 *Pesticides Literature Review* was included in full in the original Administrative Bundle I Volume I at pages 445 to 632.

[51] The report gives the following references to various studies demonstrating this: (1) Al-Badrany YM, Mohammad FK. Effects of acute and repeated oral exposure to the organophosphate insecticide chlorpyrifos on open-field activity in chicks. *Toxicol Lett.* 2007;174:110-116; (2) Kaur P, Radotra B, Minz RW, Gill KD. Impaired mitochondrial energy metabolism and neuronal apoptotic cell death after chronic

104 On page 153 the report states, *"Neurotoxicologists from the U.S. and the U.K. have proposed the existence of a long-term OP-associated neurotoxic syndrome that develops with chronic low-level OP exposure or persists after substantial recovery from acute OP poisoning.*[52] *Persistent central nervous system effects are notable in the proposed condition, as opposed to the prominent peripheral effects associated with OPIDN. Based on his and other research teams' investigations in animal models, Dr. Mohamed Abou-Donia of Duke University has suggested these persistent symptoms are associated with neuronal oxidative injury and apoptotic cell death in multiple brain regions following OP exposure."*

105 On page 154 of the recent report relating to Gulf War Illness it states, *"Pyrethroids* [a class of pesticides] *are neurotoxicants and, at very high doses, can have adverse effects on both the central and peripheral nervous systems. Their best known mechanisms of action involves effects on voltage-gated sodium channels that result in alterations in nerve cell excitability. Multiple studies have also described additional pyrethroid targets and effects that include enhanced release of acetylcholine and alterations in other neurotransmitter systems."* [53]

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dichlorvos (OP) exposure in rat brain. *Neurotoxicology.* 2007;28:1208-1219; (3) Kobayashi H, Suzuki T, Sakamoto M, et al. Brain regional acetylcholinesterase activity and muscarinic acetylcholine receptors in rats after repeated administration of cholinesterase inhibitors and its withdrawal. *Toxicol Appl Pharmacol.* 2007;219:151-161; (4) Prendergast MA, Terry AV, Jr., Buccafusco JJ. Effects of chronic, low-level organophosphate exposure on delayed recall, discrimination, and spatial learning in monkeys and rats. *Neurotoxicol Teratol.* 1998;20:115-122; (5) Raheja G, Gill KD. Altered cholinergic metabolism and muscarinic receptor linked second messenger pathways after chronic exposure to dichlorvos in rat brain. *Toxicol Ind Health.* 2007;23:25-37; (6) Terry AV, Jr., Gearhart DA, Beck WD, Jr., et al. Chronic, intermittent exposure to chlorpyrifos in rats: protracted effects on axonal transport, neurotrophin receptors, cholinergic markers, and information processing. *J Pharmacol Exp Ther.* 2007;322:1117-1128; (7) Terry AV, Jr., Stone JD, Buccafusco JJ, Sickles DW, Sood A, Prendergast MA. Repeated exposures to subthreshold doses of chlorpyrifos in rats: hippocampal damage, impaired axonal transport, and deficits in spatial learning. *J Pharmacol Exp Ther.* 2003;305:375-384; (8) Trevisan R, Uliano-Silva M, Pandolfo P, et al. Antioxidant and acetylcholinesterase response to repeated malathion exposure in rat cerebral cortex and hippocampus. *Basic Clin Pharmacol Toxicol.* 2008;102:365-369.

[52] The report gives a number of references to various studies in relation to this including: (1) Abou-Donia MB. Organophosphorus ester-induced chronic neurotoxicity. *Arch Environ Health.* 2003;58:484-497 (selected quotes from the 2005 version of this study, published in the Journal of Occupational Health Safety — Aust NZ 2005, 21(5): 408-432 is included as exhibit GD/9 **[Tab 9/1]**); (2) Jamal GA, Hansen S, Julu PO. Low level exposures to organophosphorus esters may cause neurotoxicity. *Toxicology.* 2002;181-182:23-33.

[53] The report gives a number of references to various studies in relation to this including: Tonini M, Costa LG, Candura SM, et al. Interaction of the pyrethroid insecticides tetramethrin and cypermethrin with enteric cholinergic transmission in the guinea-pig. *Neurotoxicology.* 1989;10:707-715.

106 Page 154 of the report goes on to state, *"In humans who have been exposed occupationally or by accident to high doses of pyrethroids, symptoms have included nausea, facial tingling, dizziness, headache, fatigue, burning and itching of the skin, eye irritation, and respiratory symptoms.* [54] *At extremely high doses, convulsions and loss of consciousness can occur."*

107 On page 155 of the report it states, *"Lindane is a neurotoxicant that blocks the action of the neurotransmitter gamma aminobutyric acid (GABA) by altering the flow of ions through neuronal membranes.*[55] *This leads to a persistent hyperexcitation of post synaptic membranes, primarily in the central nervous system.*[56] *Release of other neurotransmitters can also be affected, including alterations in levels of dopamine, serotonin, and norepinephrine.*[57] *Animals given high doses of lindane develop behavioral changes, loss of balance, and seizures.*[58] *Effects vary both with the dosage and with the dosing schedule. Behavioral and neurochemical effects of a single exposure to lindane have been shown to differ from those of repeated, lower-dose exposures.*[59] *Lindane is also associated with chemical kindling, the phenomenon by which chemical exposures potentiate a persistent increase in the sensitivity of brain*

[54] The report gives a number of references to various studies in relation to this including: Zhang ZW, Sun JX, Chen SY, Wu YQ, He FS. Levels of exposure and biological monitoring of pyrethroids in spraymen. *Br J Ind Med.* 1991;48:82-86.

[55] The report gives the following references to various studies demonstrating this: (1) Joy RM, Walby WF, Stark LG, Albertson TE. Lindane blocks GABAA-mediated inhibition and modulates pyramidal cell excitability in the rat hippocampal slice. *Neurotoxicology.* 1995;16:217-228; (2) Narahashi T. Nerve membrane ion channels as the target site of insecticides. *Mini Rev Med Chem.* 2002;2:419-432.

[56] The report gives the following reference in relation to this: Ecobichon DJ. Toxic effects of pesticides. In: Klaasen CD, ed. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. Sixth ed. New York: McGraw-Hill. 2001:763-810.

[57] The report gives a number of references to various studies in relation to this including: (1) Joy RM, Vogel SM, Narahashi T. Effects of lindane upon transmitter release and end-plate responsiveness in the neuromuscular junction of the frog. *Neuropharmacology.* 1987;26:1223-1229; (2) Rivera S, Sanfeliu C, Sunol C, Rodriguez-Farre E. Regional effects on the cerebral concentration of noradrenaline, serotonin and dopamine in suckling rats after a single dose of lindane. *Toxicology.* 1991;69:43-54.

[58] The report gives the following reference in relation to this: Vucevic D, Hrncic D, Radosavljevic T, et al. Correlation between electrocorticographic and motor phenomena in lindane-induced experimental epilepsy in rats. *Can J Physiol Pharmacol.* 2008;86:173-179.

[59] The report gives the following references in relation to this: (1) Gilbert ME. Repeated exposure to lindane leads to behavioral sensitization and facilitates electrical kindling. *Neurotoxicol Teratol.* 1995;17:131-141; (2) Rivera S, Rosa R, Martinez E, et al. Behavioral and monoaminergic changes after lindane exposure in developing rats. *Neurotoxicol Teratol.* 1998;20:155-160.

*cells to electrical stimuli and seizures.*[60] *Human overexposure to lindane is also associated with tremors, ataxia, and seizures, and several deaths have been attributed to lindane poisoning. In addition, studies of brain tissues of Parkinson's disease patients, at autopsy, have demonstrated significantly elevated levels of lindane and dieldrin, compared to controls."* [61]

108 On page 156 of the report it states, *"In community and occupational studies, chronic low-level exposure to pesticides has frequently been associated with increased rates of symptoms and multisymptom illness…* [62] *The majority of studies have focused on effects of exposure to pesticides in general or effects of organophosphate pesticides. Compared to unexposed controls, populations chronically exposed to pesticides, either in relation to their occupation or where they live, have consistently been shown to report higher rates of symptoms that include memory problems, difficulty concentrating, headache, fatigue, difficulty sleeping, nausea, respiratory problems, and mood alterations."* [63]

[60] The report gives a number of references to various studies in relation to this including: (1) Albertson TE, Walby WF, Stark LG, Joy RM. The effects of lindane and long-term potentiation (LTP) on pyramidal cell excitability in the rat hippocampal slice. *Neurotoxicology.* 1997;18:469-477; (2) Joy RM. The effects of neurotoxicants on kindling and kindled seizures. *Fundam Appl Toxicol.* 1985;5:41-65.

[61] The report gives the following references in relation to this: (1) Corrigan FM, Wienburg CL, Shore RF, Daniel SE, Mann D. Organochlorine insecticides in substantia nigra in Parkinson's disease. *J Toxicol Environ Health A.* 2000;59:229-234; (2) Fleming L, Mann JB, Bean J, Briggle T, Sanchez-Ramos JR. Parkinson's disease and brain levels of organochlorine pesticides. *Ann Neurol.* 1994;36:100-103.

[62] The report gives the following reference in relation to this: Kamel F, Hoppin JA. Association of pesticide exposure with neurologic dysfunction and disease. *Environ Health Perspect.* 2004;112:950-958.

[63] The report gives a number of references to various studies in relation to this including: (1) Gomes J, Lloyd O, Revitt MD, Basha M. Morbidity among farm workers in a desert country in relation to long-term exposure to pesticides. *Scand J Work Environ Health.* 1998;24:213-219; (2) Kamel F, Engel LS, Gladen BC, Hoppin JA, Alavanja MC, Sandler DP. Neurologic symptoms in licensed pesticide applicators in the Agricultural Health Study. *Hum Exp Toxicol.* 2007;26:243-250; (3) Kamel F, Hoppin JA. Association of pesticide exposure with neurologic dysfunction and disease. *Environ Health Perspect.* 2004;112:950-958; (4) London L, Nell V, Thompson ML, Myers JE. Effects of long-term organophosphate exposures on neurological symptoms, vibration sense and tremor among South African farm workers. *Scand J Work Environ Health.* 1998;24:18-29; (5) Richter ED, Chuwers P, Levy Y, et al. Health effects from exposure to organophosphate pesticides in workers and residents in Israel. *Isr J Med Sci.*1992;28:584-598; (6) Steenland K, Dick RB, Howell RJ, et al. Neurologic function among termiticide applicators exposed to chlorpyrifos. *Environ Health Perspect.* 2000;108:293-300.

109 On page 166 the report states, *"Synergistic effects of mixtures of anticholinesterase pesticides have been described in the medical literature for over 50 years."* [64]

110 On page 167 the report states, *"Interactive effects of potentially hazardous chemicals can occur in different ways. Compounds can affect the degree to which other chemicals are taken into the body or the efficiency with which they are neutralized and eliminated from the body. Both processes can directly affect the dose of chemical delivered to target organs, and can also affect the duration of the delivered exposure. Once in the body, compounds can also interact with one another biologically, altering effects of one another on specific biochemical processes in the brain or the periphery."*

111 On page 168 the report states, *"a growing body of research has provided important insights into how concurrent exposure to multiple chemicals can alter metabolism of toxic substances."*

112 On page 173 of the report it states, *"Combinations of lindane, malthion, and, permethrin have been reported to act synergistically to increase markers of oxidative stress, stimulate increased levels of antioxidant enzymes, and produce necrotic and apoptotic cell death in thymocytes.*[65] ***And, using a novel in vitro screen for neurotoxicity, significant interactive effects have also been demonstrated when chlorpyriphos was combined with pyrethrum or with a solvent commonly used in pesticide formulations.***" [66]

113 On page 218 of the report it states, *"…research in animal models indicates that different classes of pesticides used during the Gulf War can have long-term effects on the brain, including effects on learning and behavior.* ***Repeat, low level exposures to***

[64] The report gives the following reference in relation to this: Frawley JP, Fuyat HN, Hagan EC, Blake JR, Fitzhugh OG. Marked potentiation in mammalian toxicity from simultaneous administration of two anticholinesterase compounds. *J Pharmacol Exp Ther.* 1957;121:96-106.

[65] The report gives the following references in relation to this: (1) Olgun S, Gogal RM, Jr., Adeshina F, Choudhury H, Misra HP. Pesticide mixtures potentiate the toxicity in murine thymocytes. *Toxicology.* 2004;196:181-195; (2) Olgun S, Misra HP. Pesticides induced oxidative stress in thymocytes. *Mol Cell Biochem.* 2006;290:137-144.

[66] The report gives the following reference in relation to this: Axelrad JC, Howard CV, McLean WG. Interactions between pesticides and components of pesticide formulations in an in vitro neurotoxicity test. *Toxicology.* 2002;173:259-268. (I referred to this study at §93 and footnote 42 above).

***organophosphate pesticides have been shown to have persistent effects that differ from effects of single exposures, even at higher dosage levels***."

114 On page 279 of the report it states, *"Inhaled exposure to the types of chemicals that trigger MCS*[67] *symptoms have also been reported to induce or exacerbate diagnosable medical conditions. These include, most prominently, asthma, rhinosinusitis, and inflammation of the upper airway.*[68] *Physicians who treat chemical injury have also reported that headache, depression, arthritis, and a persistent intestinal dysfunction syndrome can be precipitated by common chemical exposures."*

115 Regarding Mr. Hamey's reliance in §22 of his third Witness Statement on the recent opinion from the EFSA PPR Panel. As I detailed in §3 of my third Witness Statement **[CB1/S/1-3]** the EFSA PPR Panel opinion was regarding exposure to two or more pesticide residues in combination in food,[69] which, as I pointed out previously, is very different to the context of resident (and bystander) exposures to pesticides for the following reasons:-

a) The exposure scenario for residents is completely different to the pesticide residue levels that are found in food. Residents exposure is long-term, chronic and cumulative and involves exposure to innumerable mixtures of pesticides that can be at high doses and levels, whereas exposure to pesticide residues in food will (normally) be at a low level, far lower than exposure to residents exposed during

[67] The report on Gulf War Illness also has sections referring to studies linking exposure to pesticides and other chemicals with the onset of both ME/CFS and MCS. (In the context of this point, I would remind the court of the article that I wrote in June 2006 regarding the links between pesticides and ME/CFS and MCS at **[TBII/814-818]**. This article included the comment from Dr. Terry Mitchell, a haematologist and NHS regional clinical champion for ME/CFS, where he stated, *"The links between chronic illness and pesticide exposure seem significantly robust for action to be taken by those whose duty it is to guard the nation's health."* **[TBII/818]**).

[68] The report gives the following references in relation to this: (1) Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS). Persistent asthma syndrome after high level irritant exposures. *Chest.* 1985;88:376-384; (2) Meggs WJ, Elsheik T, Metzger WJ, Albernaz M, Bloch RM. Nasal pathology and ultrastructure in patients with chronic airway inflammation (RADS and RUDS) following an irritant exposure. *J Toxicol Clin Toxicol.* 1996;34:383-396.

[69] The PPR Panel opinion states, *"Therefore, at this stage the PPR Panel restricted its consideration of combined risk assessment to exposures from residues in food that could arise from plant protection products."* Source: http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1178712607885.htm

and after the actual application process, from all exposure factors, and via all exposure routes.

b) The requirement of the Directive is quite clear in relation to the need for Member States to carry out the appropriate assessments for all relevant *humans* that may be exposed to establish that there will be no harmful effect to the health of those humans. Therefore specific and relevant exposure assessments for whoever may be exposed must be undertaken and those assessments have to be realistic for the exposure scenario concerned.[70] As set out in my second Witness Statement the exposure scenario for residents inevitably involves long-term exposure to innumerable mixtures of pesticides as agricultural pesticides are rarely used individually, but are commonly sprayed in mixtures near to residents' homes, children's schools etc., quite often consisting of 4 or 5 different products mixed together. (In the context of this point, I would remind the court of the points made in my second Witness Statement at §8(c) and related footnotes **[CB1/Q/7-8]** and §56(g) and related footnotes **[CB1/Q/53-55]**. Also §17 of my first Witness Statement at **[CB1/O/3]**). As stated in §8(c) of my second Witness Statement, each product formulation in itself can contain a number of different active ingredients,[71] as well as other chemicals, such as solvents, surfactants and other co-formulants (some of which can have adverse effects in their own right,[72] even before considering any potential synergistic effects in a mixture(s)). Therefore the fact that the Appellant has not included the assessment of mixtures into its

[70] As highlighted in footnote 26 of my second Witness Statement**,** Annex 3 §7.3 of Directive 91/414/EEC under the heading of *"Data on exposure,"* states, *"The risks for those in contact with plant protection products….depend on…**the route, the degree and duration of exposure**…"* **[CB2/N/155].**

[71] This can include mixtures of pesticides from the same classes of pesticides or can be a combination of pesticides from different classes, eg. the safety data sheet for Dovetail, a product which has been used on the surrounding fields to our home, is a pyrethroid and carbamate insecticide mixture **[TBI/vol II/764]**.

[72] In the comments, dated 27th May 2009, by Malcolm Hooper, Professor Emeritus of Medicinal Chemistry at Sunderland University, included at exhibit GD/2, Professor Hooper states, *"A great deal of work has been done in connection with the formulation of pesticides. The active compound, eg. OP or pyrethroid, is always formulated with a number of other compounds- detergents, dispersing agents, wetting agents etc. These are often referred to as 'inerts' but in a number of cases these so-called 'inerts' have been found to possess biological activities of their own which can increase the toxicity of the active compounds. A well known example is the presence of various phenolic compounds in OP pesticides. Phenols were subsequently removed from the formulations."* **[Tab 2/1]**. (Also see the data sheet for Mevinphos prepared by the International Programme on Chemical Safety (IPCS), included at exhibit GD/4 that states, "*Carrier solvents used in commercial formulations may change physical and toxicological properties."*) **[Tab 4/3]**.

exposure assessment model is a significant omission, as to fulfil the requirement of establishing no harmful effect of a pesticide product *"having regard to all normal conditions under which it may be used,"* (Directive, Article 4(1)(b) **[CB2/N/9]**) would have to include not only when that product is used on its own, but also when used in combination with any other products considering, as said, that the normal conditions of use in relation to agricultural pesticides is that they are commonly sprayed in mixtures. However, as repeatedly stated throughout my second Witness Statement the specific pesticide exposure scenario for residents (as opposed to bystanders) would need to include in the exposure calculations all the exposure factors relevant for residents and via all exposure routes over the long-term, as no exposure assessment or resulting risk assessment for **residents** can be accurate or complete if some of the exposure factors are ignored in the exposure calculations, which they currently are. Therefore it is about all the various exposure factors that are currently missing from the Appellant's current approach (of which exposure to mixtures is just one) as highlighted at §56 of my second Witness Statement **[CB1/Q/42-60]**.[73]

116 §127 to §152 of my second Witness Statement **[CB1/Q/99-111]** set forth the evidence to show that the Government confirms acute adverse health effects from exposure to pesticides in its own monitoring system, (both local and systemic effects), but has not taken *any* action to prevent these acute adverse impacts occurring. This was recognised in §40 to §47 of the Judgment at **[CB1/K/18-21]**. In §40 Mr. Justice Collins concluded that there is *"solid evidence produced by the claimant that residents have suffered harm to their health (her own ill health is an example) or, at the very least, doubts have reasonably been raised as to the safety of pesticides under the regime which presently exists: see the Sweden case at paragraph 161. It is clear*

[73] Yet pesticides are not supposed to be approved for use until risk assessments have been undertaken to establish that there will be *"no harmful effect directly or indirectly"* on human health. Eg. see European Commission document at **[TBII/746]** that says *"A directive of 1991 on the placing on the market of PPPs seeks to prevent risks at source. It requires that a very comprehensive risk assessment is carried out for each active substance and for the products containing the substance, before they can be authorised for use."* In the absence of any risk assessment for residents then it cannot possibly satisfy the applicable legal duties under Directive 91/414 and the equivalent UK legislation of establishing no harmful effect. (Also see §57 to §59 of my second Witness Statement at **[CB1/Q/61-62]**).

*that the precautionary principle must apply."* In §47 Mr. Justice Collins concluded *"Had they* [the ACP] *appreciated that the evidence was solid and that the conditions come within the scope of the Directive inasmuch as they constituted harm to human health, a different approach ought in my view to have been adopted. There has in my judgment been both a failure to have regard to material considerations and a failure to apply the Directive properly."*

117 As I said in my address to the court on the 19th May 2009, residents suffering repeated acute health effects from exposure to pesticides from crop-spraying (which can occur on a regular basis for those living near sprayed fields) can result in an increased risk of developing cumulative chronic long term effects. As said previously, this has been recognised by the European Commission in important statements that clearly acknowledged that those who are regularly or repeatedly exposed to pesticides, including those living in the locality to sprayed fields, may have a higher risk of a number of chronic effects, illnesses or diseases: (see §1 and §86 of my second Witness Statement **[CB1/Q/1-2 and 78-79]**). For example, as recognised in §7 of the Judgment at **[CB1/K/4]** the European Commission has clearly stated that:

*"Long term exposure to pesticides can lead to serious disturbances to the immune system, sexual disorders, cancers, sterility, birth defects, damage to the nervous system and genetic damage."* **[TBII/746].**

*"There are various sources for continuous exposure, like the consumption of polluted water, pesticide residues in food, regular application of PPP over many years**, or residential proximity to it and consequently direct exposure via air. People regularly or repeatedly exposed to or working with pesticides, may have a higher risk of incidence of cancer or other chronic diseases, birth defects, cancer in offspring, stillbirths and reproductive problems, skin rashes and disorders, disturbed enzyme and nervous system."*** **[TBIV/771].**

118 Footnotes 180 and 183 of my second Witness Statement **[CB1/Q/97-98]** detail some of the risk and safety phrases and other toxicological information on pesticide safety data sheets that contain warnings in relation to systemic effects, as well as long-term chronic cumulative and irreversible effects. These are on the actual safety data sheets themselves and are therefore chronic and permanent effects clearly recognised by the

manufacturers of the products. (Examples of safety data sheets for a few of the pesticides that I was previously informed have been sprayed on fields in my locality can be seen at **[TBI/vol II/742-769]**).

119 In relation to chronic effects and irreversible permanent effects, Cornell University's teaching module *"Toxicity of Pesticides"* states, ***"Irreversible effects are permanent and cannot be changed once they have occurred. Injury to the nervous system is usually irreversible since its cells cannot divide and be replaced. Irreversible effects include birth defects, mutations, and cancer."*** **[TBIV/793].**

**Chemical levels in blood and body fat (§23 of Mr. Hamey's statement)**

120 In relation to Mr. Hamey's comments in §23 of his third statement, Dr. Myhill states in her enclosed letter dated 29th May 2009 (at exhibit GD/1) that, *"It is also important to note that in fat biopsies chemicals are measured in milligrams per kilogram. This refers to milligrams per kilogram of fat sample. Levels of chemicals in blood are measured in micrograms per kilogram, i.e. there is often a hundred to thousand fold difference. So organs of the body where they have a high proportion of fat such as the brain and bone marrow will have higher levels of pesticides than areas of the body which have lower levels of fat. This helps to explain why pesticides are particularly toxic to the brain and immune system."* **[Tab 1/1]**.

**Immunotoxicity and teratogenicity (§24 of Mr. Hamey's statement)**

121 In §24 of his third Witness Statement Mr. Hamey says that the statements made by Dr. Myhill in her letter dated 13th April 2004 that "*pesticides are immuno-toxic*" as well as being "*teratogenic*" **[CB4/E/10]** are incorrect. This is simply not correct as what Dr. Myhill said was absolutely right.

122 For example, under one of the study questions in Cornell University's teaching module *"Toxicity of Pesticides"* it states, "***Pesticides can: cause deformities in unborn offspring (teratogenic effects), cause cancer (carcinogenic effects), cause mutations (mutagenic effects), poison the nervous system (neurotoxicity), or block the natural defenses of the immune system (immunotoxicity).*** *Pesticides can also*

*have: local or systemic effects; immediate or delayed effects; reversible or irreversible effects; singular, additive, or synergistic effects."* **[TBIV/799].**

123 Also I have included a one page table at exhibit GD/10 **[Tab 10/1]** taken from *"Pesticides and Human Health: A resource for Health Care professionals,"* by Dr. Gina Solomon[74], MD, MPH, Senior Scientist, Natural Resources Defense Council, Assistant Clinical Professor of Medicine, University of California, San Francisco, published in 2000.

124 The table lists a number of different pesticide types (although not all) such as carbamates, including Carbaryl, organophosphates (OPs), and organochlorines, including Lindane. As can be seen from the table, they all have adverse effects on the immune system.

**Concluding comments**

125 In relation to the chronic adverse health effects reported by rural residents (eg. various cancers, leukaemia, non-Hodgkin's lymphoma, neurological conditions, including Parkinson's disease, ME, asthma, amongst others), Mr. Justice Collins concluded at §46 that *"there is evidence that some long term illnesses may be attributable to pesticide exposure"* and at §47 that *"there is sufficient material to raise a real doubt as to long term harm in some cases."* **[CB1/K/21]**). As pointed out in footnote 57 of the Respondent's Notice, in the absence of any assessment for a residents specific exposure scenario, (nor any investigation into the chronic adverse effects reported by residents), the Appellant *cannot* say that there is no association between the various chronic effects, illnesses and diseases reported by residents and pesticide exposure, as the Appellant has no evidence to support that assertion.

126 In my address to the court on the 19th May 2009 I pointed out that in §34 of his Judgment Mr. Justice Collins quoted from the Appellant's predecessor, (the Ministry

[74] Dr. Gina Solomon was the principal author, and Dr. O.A. Ogunseitan, PhD, MPH, Associate Professor, Department of Environmental Analysis and Design, School of Social Ecology, University of California, Irvine, and Dr. Jan Kirsch, MD, MPH, Assistant Clinical Professor of Medicine, University of California, San Francisco, were co-authors. There were also contributions by the Physicians for Social Responsibility and Californians for Pesticide Reform.

of Agriculture, Fisheries and Food (MAFF)) in a 1975 document, where MAFF stated that, *"The repeated use of pesticides, even in small quantities, can have cumulative effects which may not be noticed until a dangerous amount has been absorbed."* This clear statement from 34 years ago shows that the Government has always been well aware of the cumulative effects of pesticides, but *again* has not taken *any* action to prevent the exposure, risks and adverse impacts occurring for those exposed.

127 The Appellant's continued line that there is no evidence of harm from pesticides, or any evidence of combined exposures and/or synergistic effects, is just untenable and inexcusable. The evidence is there, and has been there for a considerable time, the Government is just determined not to act on it, which is not in line with the proactive approach of the European Directive regarding the protection of human health. (Also see §219 to §222 of my second Witness Statement at **[CB1/Q/147-148]**).

128 I would like to point out to the court that the only reason that this additional material in relation to my own personal health problems, and blood and fat test results etc. is being submitted now, is to respond and counter specific points made for the first time by Mr. Hamey, on behalf of the Appellant, in his third statement. As said earlier in §2, the letter from Dr. Myhill interpreting the results of my blood and body fat samples, was the same letter referred to and quoted from in §48 of my first Witness Statement, dated 22nd October 2006 **[CB1/O/9]** and which was not challenged *at any time* by the Appellant (despite having had over 2 and a half years to do so) until the CA hearing.

129 It is important for me to reiterate that this case is not dependent upon proving causation. So far as the Directive is concerned, my arguments would arise irrespective of whether I personally had suffered adverse health effects, because I would still have been exposed to the risk of harm, and continue to be.[75]

[75] As far as my claim under Article 8 ECHR is concerned, my position is that it is unnecessary for Article 8 purposes to have to be putting forward evidence of a direct causal link between crop-spraying and my own ill-health for the reasons explained in §5 of Route Map 4 at **[CB1/N/38].**

130 As I have continued to argue throughout my case, there has been (and continues to be) an inherent fundamental failure at all levels to protect rural residents and communities from exposure to pesticides. The principle aim of pesticide policy and legislation is supposed to be based on the risk of harm and not that harm has to have already occurred. Therefore the Government should not be exposing people to any risks. **Thus, my case has always been centred on the fact that under EU and UK law people are not supposed to be put at risk of suffering *any* harm, (whether it be acute or chronic effects), from exposure to pesticides.**

131 The Appellant itself is well aware of this fact as the Appellant has consistently paraded the virtue that if any risk to human health were identified, then there would be rapid action to prevent the authorization and use of pesticides. Some of the previous statements that have been made by, or on behalf of the Appellant, regarding the action that would be taken if there is a risk to human health are:-

a) *"If there were a documented risk to humans the use simply would not be approved"* (Downs second Witness Statement at footnote 288 **[CB1/Q/142]**);

b) "*If we believed based on the evidence that there was a risk to health then there would be very rapid action"* (Downs second Witness Statement at footnote 288 **[CB1/Q/142]**);

c) *"We already apply a very precautionary approach in the regulation of pesticides…We do not wait until there is evidence of an adverse effect before we react to restrict the use of a pesticide; the reverse is true. There has to be positive evidence that there will not be adverse effects before a pesticide is allowed on the market"* (Downs second Witness Statement at footnote 194 **[CB1/Q/103]**);

d) *"If a link between human disease and a pesticide were considered to be proven or even likely and if the product was still on the market its approval would either be modified to reduce exposures or the approval could be revoked entirely."* **[TBIV/564]."** (Downs second Witness Statement at footnote 194 **[CB1/Q/103]**).

e) *"If there is scientific evidence that use of a pesticide may harm human health, that is considered unacceptable"* and that, *"the system does not trade off the benefits and risks of pesticide use. If the risks are unacceptable, approval for use is refused, whatever the benefits,"* (DEFRA's previous statement as highlighted in §16 of the Claimant's grounds and at Downs second Witness Statement at §204 and footnote 284 **[CB1/Q/139]**);

f) *"If we thought that current margins of safety for a pesticide gave insufficient protection to neighbours, we would recommend that the use be banned rather than relying on a buffer zone to reduce exposures"* (Downs second Witness Statement at §62 **[CB1/Q/63]** and §65 of the Claimant's skeleton **[CB1/A/49]**).

132 **Therefore all these statements are on the Appellant's very own stated case.**

133 However, even though there is a clear health risk,[76] (and even further than there being a risk to health, there is, as Mr. Justice Collins found *"solid evidence"* (including in the Government's very *own* monitoring system) that residents have suffered harm to their health), no action has been taken by the Appellant to protect the health of residents and others in the countryside from exposure to pesticides. This is inconsistent with the Appellant's previous categorical statements regarding the *immediate* action that would be taken if there is a risk to human health.

134 I would like to point out in response to a question by Lord Justice Sullivan during the hearing, that the RCEP did not assess all the same evidence and arguments as has been set forth in the court, as the RCEP said it was outside their remit for their crop-spraying enquiry, the remit of which had been agreed with DEFRA.[77] This means

[76] Which has even been accepted by the ACP itself, (eg. at **[TBII/633]**). Also see §152 and §209 of my second Witness Statement at **[CB1/Q/111 and 142-143]**.

[77] Also it is important for the court to note that neither DEFRA, HSE nor PSD (now CRD) provided the RCEP with the HSE's Field Operations Directorate (FOD) reports which contain the raw data of the ill-health incidents reported to the HSE and assessed by PIAP. The RCEP secretariat has researched the files of papers and has not found evidence that these FOD reports, as opposed to the largely statistics based PI reports, were submitted for the RCEP enquiry. Therefore the FOD reports were not seen or considered by the RCEP. As I pointed out in §82 of my second Witness Statement, at **[CB1/Q/74-75]** the FOD reports are not published, as they are only produced by HSE for the sole purpose of submitting to the ACP for consideration, in relation to each year they are related to. In fact, it would appear that I am the only person

that the RCEP report was *still* predominantly related to spraydrift and not all the exposure factors in totality, via all exposure routes, as per a residents exposure scenario. Having said that, even based on the more limited basis the RCEP looked at the issue, compared to the evidence and arguments contained in my second Witness Statement, the RCEP still found *"serious shortcomings"* in the Government's approach and that the policy was inadequate to protect public health.

135 Also, it is important for the court to note that there have been significant recent developments since the time that the RCEP report was published in 2005, (which is now 4 years ago). For example, the study regarding pesticides and Parkinson's in rural residents (published in March 2009, as referred to at §97 above); as well as various other studies that have found associations between pesticides and various chronic adverse health effects, illnesses and diseases. Therefore the RCEP report has been superseded by subsequent studies and findings. Another example of this is the RCEP's finding that there was a "*plausible"* link between exposure of bystanders and residents to pesticides and chronic[78] ill health (RCEP report §2.65 **[CB2/I/34]**). As pointed out in footnotes 156 and 161 of the Claimant's Skeleton below the RCEP's finding was subsequently superseded by the important statements issued by the European Commission in July 2006 *confirming* the chronic long term health impacts of pesticides, including for those living in the locality to sprayed fields. See §1 and §86 of my second Witness Statement at **[CB1/Q/1-2]** and **[CB1/Q/78-79]**.

136 It is very important for me to clarify a fundamental aspect of this legal case for the avoidance of doubt. The Royal Commission's report was only a small part of my original Judicial Review challenge in relation to Ground 3. Therefore the Respondent's case itself has always been based on my arguments and evidence as set out in my 5 Witness Statements, in particular the second Witness Statement. This was

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outside of Government departments and officials, (as well as the ACP), to have requested and obtained these reports for the purpose of submitting them as evidence for my Judicial Review legal challenge.

[78] The RCEP fully accepted that acute effects can be, and are, caused by pesticides. Eg. §2.9 of its report stated, *"The evidence from the residents and bystanders visited identified a series of well-defined acute symptoms immediately following pesticide spraying. These include upper and lower respiratory tract irritation, eye irritation, skin rashes, headaches and, in susceptible subjects, asthma attacks."* **[CB2/I/22A]**.

recognised by Mr. Justice Collins in his Judgment, eg. at §39 Mr. Justice Collins stated, "*The alleged inadequacies of the model and the approach to authorisation and conditions of use have been scientifically justified. The claimant has produced cogent arguments and evidence to indicate that the approach does not adequately protect residents and so is in breach of the Directive."* **[CB1/K/18]**.

137 Therefore in relation to Grounds 1 and 2 of the original Judicial Review challenge, as well as the Human Rights Ground, I would reiterate the importance of my second Witness Statement, as it sets out the very important factual detail and arguments that provided the critical basis of my case and original challenge, (and subsequent Judgment from Mr. Justice Collins), concerning the *legality* (in EC and domestic public law terms) of the Government's policy and approach in view of the overriding public safety duty as required by the European Directive and the UK equivalent legislation regarding the protection of human health.

138 There are many hundreds, even thousands more scientific studies I could submit regarding the associations between pesticides and acute and chronic adverse effects. However, I tried to keep that to a minimum in the materials I submitted before the court below, as this case is about points of law. The arguments I set out in my 2nd statement were based on the Government's very own documents and findings, and that showed that the Government has fundamentally failed to: 1) protect residents; 2) act on the evidence of the risk of harm, and further than that, 3) act on the evidence of harm that is occurring; 4) act on its own findings of exceedances of the AOEL, in some cases an *order of magnitude* higher (any exceedance of which, under the Directive, is supposed to lead to authorizations being refused). All of which, as set out above, on the Appellant's own previously stated case, would lead to immediate action of authorizations being refused (or trigger prohibition if already approved).

I believe that the facts stated in this witness statement are true.

Signed:

Georgina Downs Date: