CONFERENCE REPORT

ORGANOPHOSPHATE SHEEP DIPS AND HUMAN HEALTH

A Seminar For

Farmers, Medical Practitioners

and Policy Makers

THE ROYAL SOCIETY
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NFU
Representing Farmers and Growers

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Friday 2 June 1995

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10.00 OPENING PLENARY
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■ **Chairman's welcome & introduction** – *Geoff Watts, Presenter 'Medicine Now'; BBC Radio 4*

■ **The farmer's perspective** – *Tony Pexton, Deputy President, NFU*

■ **The medical perspective and the commitment to patients** – *Dr Fleur Fisher, Head of Ethics, Science & Information, BMA*

■ **An overview of the delayed effects of organophosphates** – *Dr Timothy Marrs, Senior Medical Officer, Dept of Health*

■ **The crucial questions that need to be answered** – *Peter Beaumont, Director, The Pesticides Trust*

10.45 COFFEE BREAK
City of London Rooms

11.15 PLENARY SESSION
Wellcome Lecture Hall

■ **The impact of OPs on the nervous system** – *Dr Goran Jamal, consultant clinical neurophysiologist, Southern General Hospital, Glasgow*

■ **OPs and psychiatric disorder** – *Dr Robert Davies, consultant psychiatrist, Rydon House, Taunton*

■ **OPs and human health: an agricultural case study** – *Dr Bernhardt, general practitioner, Kent*

■ **Clinical toxicology - dealing with OP poisoning in practice** – *Dr Alex Proudfoot, Head of the Edinburgh Centre of the National Poisons Information Service*

12.45 LUNCH
City of London Rooms

14.00 OPEN PANEL DISCUSSION
Wellcome Lecture Hall

Panel

■ Dr Anne Spurgeon, *Institute of Occupational Health, Birmingham*

■ Roger Cook, *Director, National Office of Animal Health*

■ Dr Fleur Fisher, *BMA*

■ Ian Gardiner, *Policy Director, NFU*

Contributions from the audience

15.15 CLOSING PLENARY
Wellcome Lecture Hall

■ Closing remarks from the chairman

■ Closing remarks from Dr Fleur Fisher, BMA

■ Closing remarks from Tony Pexton, NFU

■ Chairman closes seminar

15.30 TEA *City of London Rooms*

A Seminar for Farmers, Medical Practitioners and Policy Makers
Friday 2 June 1995

FOREWORD

SirDavid Naish

President of the NFU

THERE IS increasingly convincing evidence that organophosphate sheep dips can harm the health of the farmers who use them. The recently published research by the Institute of Occupational Health points to "subtle" long-term effects on the nervous system. There is also a growing volume of anecdotal evidence of ill-effects, so this must be taken very seriously.

On the other hand there is also the evidence of our own eyes of the pain and suffering caused to sheep by the parasites and diseases which OP dips are designed to deal with. So we have a dilemma here. The need to protect human health is of course of paramount importance. But we cannot ignore the need to care for our animals in the best, most practical way possible.

It's an issue which the NFU has felt for some time must be given a higher place on the political and public agenda, in order that those who have suffered ill-effects from OP dips – and those who are potentially at risk – can receive the help they need.

It was for this reason that we decided to join with the British Medical Association in staging a seminar in London to enable sheep farmers and other interested parties to listen to and to question leading specialists in this field, and to bring about a productive exchange of views and information.

As this report shows, we did not reach any clear-cut conclusion. That may have to wait for the further research which was demanded from all sides.

Our seminar did however point the way to consensus with the BMA on a number of key points:-

- Farmers who go to their general practicioner complaining of health problems they believe could be related to OPs must tell him if they have been using OP dips
- The Department of Health must make GPs fully aware of the allegations linking OP dips with human health problems
- A national database should be set up to collate all the information about OPs, the symptoms of OP poisoning, and possible links with human health
- There is a need for a simple reporting system for sufferers and a single centre to which they can report
- The Department of Health and the safety authorities must give the issue a high priority.

The BMA has undertaken to discuss within its own appropriate committee and with the appropriate Royal Colleges the means by which more information can be provided to GPs to help them identify OP-related illness, and I am grateful for this.

We in the NFU will be pursing the same objectives through our own internal channels to raise farmer awareness of the risks in using OP dips – and the need for them to dip safely, using proper protective clothing, whatever the difficulties this entails. We will also continue to press manufacturers for the provision of alternative products at reasonable prices.

Our seminar has thrown up several areas where more research is urgently needed. But I believe its real significance is in pointing the way to the action which can and must be taken now – action in which the NFU will play its full part.

A Seminar for Farmers, Medical Practitioners and Policy Makers
Friday 2 June 1995

CHAIRMAN'S WELCOME AND INTRODUCTION

Geoff Watts

Presenter
'Medicine Now'
BBC Radio 4

ON BEHALF of the National Farmers' Union and the British Medical Association, welcome to this meeting on organophosphate sheep dips and human health.

Before we begin, let me say just a brief word or two about the sort of meeting that this is supposed to be – or, perhaps I should say, what it is not intended to be. The idea is not to come up with some kind of policy or course of action which either of its organising bodies will seek to follow. Even if that were the intention – and it is certainly not – I doubt that it would be possible.

I have no doubt there is going to be a lot of grinding of axes today, metaphorically – at least, I hope it will only be metaphorical – but these will not be specifically NFU or BMA axes. Neither body is using today in the hope of persuading you to a particular viewpoint or course of action.

So what is the intention? It is quite simply to present you with evidence and views from a handful of people with a particular knowledge of or interest in this topic and then offer you in the audience a chance to question what you have heard and also put forward your own views on the present and future use of organophosphates. So today will be a mixture – part educational and part opinion-seeking.

I think if there were any doubts in the mind of the general public that this issue is now firmly on the agricultural agenda, they were dispelled when sheep dipping finally made it on to *The Archers*. Perhaps, one day, when this issue has been resolved – whether it is 10 years or 20 years or whatever, and it is simply history – someone standing in the bar of The Bull in Ambridge will look back and say: "You remember what trouble they used to have over – what were they called – those organophosphates? Of course it was that meeting in that Royal Society place in London where things began to get sorted out."

Who knows what will come out of today?

THE FARMER'S PERSPECTIVE

Tony Pexton

Deputy President NFU

CAN I first of all echo the chairman's welcome and say how pleased we are in the NFU that so many delegates are joining us here today. We have got a very varied audience – a range of informed points of view. Open and informed discussion is the only way to advance the understanding of this highly topical but complex issue – an issue for which, on current evidence, I believe there is no easy or quick solution.

Let me say, also, how important it is for the NFU to have the British Medical Association as our partners for this seminar – not only for the knowledge and experience that the BMA brings to any serious consideration of issues of health but because GPs themselves must be able to identify and report cases of OP poisoning quickly and accurately. If, as a result of today, we can increase GP awareness to identify symptoms at a local level, and thereby contribute to the scientific evidence available, we will have taken important steps, I believe, to properly understand both the nature and the scale of the problem.

I have stressed the complex nature of the problem. Now let me outline the complexity from the point of view of the farmer.

Sometimes we have to use chemical substances which, if they are mishandled or used inappropriately, could be injurious to health. We use OP sheep dips for one simple but extremely important reason – animal welfare. We use them to prevent the development and the spread of diseases amongst our animals. Fortunately, not many of us have seen the effects of sheep scab – but it is an extremely nasty and a painful disease of the skin and the fleece, and preventive treatment can relieve a great deal of pain and suffering.

Here we have the farmers' dilemma. We care for our animals and we want to provide the best treatment. At the moment, there is no widely available single alternative treatment for scab, blowfly and other extremely unpleasant diseases facing our flocks. But, in recent years, more and more cases of human health problems seemingly linked to the use of OPs have come to light.

Two years ago, a review of OPs by the Veterinary Products Committee commissioned by the Government resulted in advice that there was no scientific justification for banning OPs but nevertheless asked that greater care should be used when handling them. Despite this we have members reporting breathing difficulties, nausea, dizziness and even paralysis. Indeed, a recent Health and Safety Executive report commissioned from the Institute of Occupational Health in Birmingham, identified what were called 'subtle' effects on the nervous system in users.

Thus the NFU finds mounting evidence suggesting a direct link between OP dips and human health problems and that is extremely worrying. One of the questions we need to debate is what safeguards are effective in handling and using OP dips while there is no effective alternative for control of sheep scab – and whether what is effective is also practical for the farmer and the farm worker.

There is a strong view among many sheep farmers that the safety equipment and clothing required to provide adequate protection as the Health and Safety Executive advises make it impossible physically for the farmer to do the job.

So this is the farmers' dilemma. Can we, while ensuring the welfare and protection of the operator, give our livestock the protection from diseases that we wish? What, amid the weight of claims and counterclaims, does the best available science say about OPs and human health? Is the safe use of hazardous substances a fact of farming life and a matter of common sense, or does adherence to the letter of health and safety guidelines render OP dips impractical?

If these dips are a danger, what are the practical and realistic alternatives? Where does the responsibility for research and development of alternatives lie? Finally, how complete is the scientific picture? What further work is necessary and whose job is it to make sure that that work is done?

In the meantime, what do farmers and medical practitioners do? What information do they need to get on with their respective jobs while all these and other questions are debated and remain unresolved?

The NFU's membership contains farmers with a range of views on these issues and we are all of us here to listen and to learn. Looking at the range of speakers and the participants, I hope and believe that we will learn a great deal from the discussions today.

In closing, can I again, on behalf of the NFU, thank all of you – speakers and participants – for coming here today. I am sure that as a result of today's deliberations we will make progress towards understanding better the nature of the problems that we are here to discuss.

THE MEDICAL PERSPECTIVE AND THE COMMITMENT TO PATIENTS

Dr Fleur Fisher

Head of Ethics, Science & Information, BMA

FOR MANY YEARS we in the BMA have been concerned with the relationship between the environment and human well-being. While a doctor's responsibilities are seen to relate primarily to their individual patients, they also have a duty to safeguard the wider public health and, through organisations such as the BMA, doctors work collectively towards the wider goals of reducing risks and promoting safe practices to protect humans and the environment from harm.

As a standing committee of the BMA, the Board of Science and Education has investigated many areas of public and medical concern, including for instance the risks from hazardous waste, transport systems and air pollution, road safety and agrochemical toxicity – and, most recently, reflecting on our own working background, we have looked at the environmental risks within the health care industry.

I can remember the impact in 1962 of Carson's *Silent Spring*. That was really the first popular expression of the growing concern about the indiscriminate use of agrochemicals, especially – then – DDT. Since then, public and professional anxiety has grown steadily, focusing on the entire range of the potential consequences of their use. These consequences include changes in the water quality, the health of livestock and wildlife, and the effects on people involved in the manufacture of agrochemicals as well as those using them, who are well represented here today – and, of course, the risks to consumers and the general public.

In 1988, the concerns were such that they prompted the BMA to undertake a study on pesticide toxicity in the UK, with particular reference to the effects on human health. Our resulting report was approved by the BMA Council in 1990 and we finally published it as an illustrated book for both professionals and general readers in 1992. This stimulated unprecedented media and industry attention. We even had patients who were concerned about their exposure to agrochemicals sending us their medical history and case notes and asking us for help.

As a result of this quite in-depth investigation, the BMA endorsed the principle that until a more complete understanding of the toxicity is achieved, the benefit of the doubt should be given to protecting the environment, the worker and the consumer, and we called for the continued reduction in the use of agrochemicals and for alternatives to be used when available. But the major issue that came from our report was that workers require instruction, training and protective clothing to minimise their exposure according to their working practices.

Chemicals can enter the body through three major routes – through the skin, inhaled in the lungs, or ingested through the gut. Contamination through the skin with subsequent absorption of a chemical is undoubtedly the major cause of accidental poisoning. Although the skin is not equally permeable to all chemicals, the importance of wearing full protective clothing is clear because we know that organophosphates may be absorbed by all three routes.

We will be hearing in a lot more detail today from Dr Marrs about the toxicology of OPs and from many other experts as well, but I just want to touch on a couple of the main points.

In our 1992 report we raised the possibility of health effects from exposure to organophosphate sheep dips, particularly the effects of acute toxicity. The human body contains something like 5-10 billion nerve cells which receive electrochemical impulses from the periphery and from everywhere in the body and send responsive signals back to various glands, muscles, etc. The brain functions as a sort of switchboard for the system and we know that this affects the seat of our emotions and mood, the seat of memory, personality and thought.

Substances such as organophosphates can cause an acute effect by inhibiting an enzyme called cholinesterase. This then leads to a build-up of a chemical called acetylcholine which is one of the major neurotransmitter chemicals involved in the transmission of nerve impulses. As a result, in acute toxicity cases, a range of symptoms can occur including tension, muscle weakness and exhaustion, anxiety, irritability and headaches.

We know that in addition to neurological signs and symptoms of acute poisoning, OPs may cause two delayed effects from the acute high dose. There is a delayed polyneuropathy – that is where many parts of the peripheral nervous system are affected at the same time – and also what we call neuro-behavioural effects, where the effects of OPs on the nervous system are reflected in behaviour.

There have been follow-up studies of survivors of acute agrochemical poisoning episodes which suggest that a few people – just a few – continue to experience persistent neuro-behavioural symptoms. It's not news really; we have known that since 1966 when Tabershaw and Cooper

followed a group of 117 workers who had experienced systemic poisoning by OPs. After three years they found that some of these had continued problems with vision, headaches and gastro-intestinal disturbances.

The possible effects of long-term low-level exposure to any toxic substance are more difficult to identify than those following acute exposure – for two reasons. Firstly , anybody experiencing an acute exposure is likely to be aware of the incident, particularly as many agrochemicals and their solvents have a distinctive noxious smell which makes people aware they have been exposed. Secondly, it is more likely in an acute case that the possible or probable cause will be identified – and that the effects on health will be observed very soon after that noteworthy exposure incident.

So we might expect evidence regarding the long-term neurological side-effects of OP sheep-dips to be much less clear. But the BMA's position to date has been to agree with the sentiment that "absence of evidence is not necessarily evidence of absence", and we called for further research. Since then there have been two significant developments.

Firstly, following the recommendation of the Medical and Scientific Panel of the Veterinary Products Committee, research proposals for epidemiological studies to detect illness in humans related to sheep-dipping are currently being invited by the Ministry of Agriculture, the HSE and the Department of Health, with a closing date of June 1995, and they have made additional money available for that.

Secondly, the next piece of news or extension of data is the recent survey by the University of Birmingham's Institute of Occupational Health, funded by the HSE, which looked at chronic OP exposure among sheep dippers. This was designed to investigate the possibility that long-term exposure to low levels of OP-containing sheep dips could perhaps cause lasting changes in the central nervous system. We have already seen the mechanism by which that may occur.

The IOH study compared neuropsychological performance of 146 sheep farmers who were exposed to OPs during sheep dipping with 140 non-exposed quarry workers, chosen for their controls because they have a similar heavy outdoor kind of work and had similar socio-economic and rural backgrounds. I am sure you will be hearing more about this later today.

The BMA welcomes such research, but we feel there is now a need for further studies to extend and confirm or otherwise those findings. What sang out from that report and was deeply concerning is that fewer than one-third of the dippers participating in the study used waterproof gloves and fewer than half used waterproof protection above the waist. Such low levels of protection continuing even today highlight the point that we made several years ago regarding the importance of training workers in the safe use of agrochemicals.

The COSHH regulations mean that all operators must be trained in the safe handling and application of chemicals. Thus all users of sheep dip and their employers who are legally liable need to ensure continuing training that covers all aspects of use, the hazards posed by OPs and the vigilant application of knowledge to safe working practices – and, of course, emergency action in case of accidental exposure. For doctors this is particularly important – health surveillance and record-keeping are absolutely vital.

The first point of contact for most farm workers who have experienced OP poisoning, whether acute or chronic, is likely to be their friendly local GP. How likely is it that the non-specific symptoms of OP poisoning will be recognised by the family doctor?

Doctors can only fulfil their commitment to patients if they receive adequate training in the basic science of toxicology. We don't need every doctor to be an expert toxicologist, but every doctor needs to be aware of the possibility of illness that can result from exposure to toxic substances. Depressingly, researchers reveal there is a consistent pattern of very limited toxicology training in the undergraduate medical programme. The best, we have found, is 12 hours. The worst – in five and a half years – is a single hour, and it is basically focused on the toxic effects of chemicals and medicines, with very little about any other aspects of toxicology.

So it is obvious, isn't it, that doctors need more training and practical experience in the diagnosis and treatment of patients who may have been exposed to agrochemicals. It is certainly arguable that there should be much more toxicology training for medical students and also that up-to-date knowledge should be made available to practising doctors – for instance, through the development of relevant on-line databases and of course seminars like today's.

I have raised some of what we believe are crucially important issues relating to the health of farmers and the need for both doctors and farmers to be aware of the potential damaging effects from all agrochemicals in general but OPs in particular.

I am looking forward to hearing the views and the expertise of the other people on the panel and what I think will be very important discussion sessions. I am looking forward to the final session – where we will, I imagine, discuss in debate whether there should perhaps be controls on OP sheep dips.

AN OVERVIEW OF THE DELAYED EFFECTS OF ORGANOPHOSPHATES

Dr Timothy Marrs

Senior Medical Officer, Department of Health

I AM going to occupy my 15 minutes by talking briefly about organophosphates and their toxicology and then about the delayed effects. I am going almost to ignore the acute effects of organophosphates because there quite simply isn't time to deal with the whole subject. I hope that Dr Proudfoot will talk about these to some extent later.

Those in the farming community will know that the anticholinesterase organophosphates are widely used on arable crops as insecticides and occasionally as fungicides, on farm animals as insecticides and anthelmintics and in particular as sheep dips, and on pet animals at home as insecticides – and some also are candidate chemical warfare agents.

The organophosphates have actually been around since the 1930s – so it's about 60 years or so. They were discovered by a German pharmacologist called Schrader who was interested in producing new and experimental insecticides. The acute cholinergic syndrome was investigated in the 40s. Delayed neurophathy, which was mentioned by the previous speaker, was discovered in the 1950s – by accident, actually – and treatment has been well worked out since.

It is worth remembering that the intermediate syndrome – a new facet of organophosphate poisoning – was only actually described in the 1980s – that is 50 years after these compounds were first described.

To explain what I will talk about later I need very briefly to go into the chemistry, I am afraid, of these compounds.

Organophosophates, at least the sort we are talking about, are derivatives of phosphoric acid, phosphates, phosphoroythiorates and S-substituted phosphoroythiorates. It is worth mentioning that there are some major differences between these types, and this must be remembered when you are looking at the epidemiological and other experimental literature. In particular, the phosphoroythiorates are much less toxic to mammals than the other types, because they need to be desulphurated before they are actually active as anticholinesterases.

The same is true of the phosphonic acid derivatives where the phosphoroythiorates of that type are also much less toxic. On the other hand, to make it even more complicated, particularly for the non-chemist, the S-substituted phosphoroythiorates of this type are one type of chemical warfare agent.

In addition, there are other types of organophosphates, such as phosporamidates and propetamphos, one of the ones that is used in sheep dips, is one of them. There are also phosphinates, which are not anticholinesterase and have very different toxic properties, and also the trialkylphosphorothiorates.

The actual toxic and insecticidal action of organophosphates is much the same at the molecular level. They react with the serine residue – an OH group on cholinesterase, which is an enzyme described by the previous speaker. They are organophosphorylated and thereby inactivate, and the result is that acetylcholine accumulates at the synapse.

What happens after that depends on the individual organophosphate. With some, for instance, like the nerve agent soman, you get very rapid ageing, and to all intents and purposes the enzyme does not reactivate.

With insecticides, generally the next thing that happens is that the di-alkylphosphoryl enzyme splits up back into the enzyme and the organophosphate, and the result is the enzyme becomes active again and the patient recovers. In some cases you get loss of an alkyl group to form this complex, which is much more stable – that is the ageing process, and in that case the patient only gets better when he makes some more enzyme, which obviously takes longer.

Moving on to the sheep dips in specific terms, there are three organophosphates at the present time on sale for use in sheep dips – propetamphos, which is a phosphoramidate, diazinon, which is a double-bond S-type phosphorothiorate, and chlorfenvintos, which is a di-ethyl phosphate.

These are sold as organic solutions containing other substances, so you immediately have two toxicological problems. Firstly, there is the man who is exposed to the formulation which is an organic solvent solution of organophosphate. Then there are the other people – most of the people – who are exposed to the actual sheep dip as it is made up for use, which is typically diluted x 500 in water. They are exposed to what in all intents and purposes is an aqueous solution of organophosphate. Any toxicologists here will know that the properties of toxic chemical are often very different, depending on what solvent they are in.

The one exception to the use of OPs in sheep-dips is flumethrin, which is an alphacyano type synthetic pyrethroid. The toxicological properties of the synthetic pyrethroids are quite different from the organophosphates, so I do not intend to mention flumenthrin dip except that it is an alter-

AN OVERVIEW OF THE DELAYED EFFECTS OF ORGANOPHOSPHATES

...Continued

native for those farmers who have problems with organophosphates.

Moving on, I want to talk about the delayed effects of organophosphates. As I said earlier, I am leaving out acute organophosphate toxicology, although that is the thing that kills people and is responsible for all these deaths in the Third World – I am going to leave it out because this seems not to be the problem we want to talk about today.

We have a received wisdom to contend with. The received wisdom is that the acute cholinergic syndrome is reversible, totally, providing the patient survives it. Organophosphorus-induced delayed neuropathy is irreversible and there are no other irreversible effects, and that is what you will find in elementary textbooks of toxicology.

A corollary to that is that organophosphorus-induced delayed neuropathy can be predicted by what is called the hen test which we shall talk about later. Therefore, OPs that are not positive in the hen test do not have any irreversible effects. That is what you will find in the text books.

However, this is where the problems arise – there are three areas where there is some evidence of delayed effects which do not appear to be related to delayed neuropathy or to delayed neuropathy as shown by the hen test. One is central effects, effects on the central nervous system – that is to say, the brain. Secondly, there are effects in the periphery – either myopathy, that is damage to muscles – or neuropathy, which may or may not be related to delayed neuropathy, but obviously not to delayed neuropathy as predicted by the test which is usually used.

I would like to talk first about the central effects of organophosophates. Again, we have a conventional wisdom to contend with. The conventional wisdom is that severe cholinergic poisoning, of the sort that gives you convulsions and anoxia, will produce structural changes in the central nervous system. That in fact has been demonstrated in experimental animals and shown to occur in humans. Those changes are irreversible. I am talking about doses close to the lethal dose – what we do not know is what happens at lower doses. The conventional wisdom has been in the past that at lower doses – those that do not cause convulsions – you will not get irreversible changes in the central nervous system.

There have been a lot of studies done on this problem – one or two were mentioned by the earlier speaker. There are studies in animals, which may be behavioural studies or electro-physiological studies – that is, things like EEGs. In humans, there are studies of the same sort on factory workers, on pesticide applicators – and, least satisfactory from the point of view of the questions we want answered, on ex-poisoning patients.

Reviewing all these studies, it is difficult to draw overall conclusions because the results are often discrepant. For example, that by Maizlish et al on diazinon was negative while two studies on sarin by Duffy et al on workers and by Birchfield et al on primates, which is another name for monkeys, both showed some changes in the EEG. You have to remember that sarin is an organophosphorus nerve agent, and therefore possibly different from insecticides.

The rest of the studies have all been those which have included acute poisonings. The problem with acute poisonings is that these are people who are admitted to Poisons Units with acute organophosphate poisoning, then discharged and looked at six months to a year later. One of the problems with using them is is that they may include people who have tried to commit suicide – they are obviously not completely normal, in the terms of their central nervous system. These studies may also include people who are very severely poisoned to the point that they may have had convulsions, and therefore you would expect long-term changes.

The older studies are generally rather small, but there are two that are worth mentioning in a little more detail. One is by Savage et al from a number of Poisons Units in the United States, which found abnormalities in people who had been admitted to Poisons Units and then discharged. However, they did not exclude severe poisoning, so it is possible that all the effects they observed were due to that small number of poisonings who convulsed. Secondly there is the Rosenstock study carried out on Nicaraguan agricultural workers – a smaller study than Savage but somewhat better matched in terms of matching of tests and controls. This study found subtle abnormality on psychometric testing.

Various criticisms have been made of those studies. Not all the criticisms are applied to all the studies and a lot of them are very easy to make because this sort of epidemiology study is extremely difficult to design. The earlier ones tend to be anectodal, as you would expect. Mostly, there is poor exposure data; that is to say, you do not know to how much organophosphate people were exposed, because of the type of studies they were. The symptoms tend to be rather vague. In some cases the exclusion criteria are not appropriate for answering the question we want answered; for instance, in the Savage study, where they included severe poisonings. In some cases there were not any controls and in the primate studies the numbers were clearly insufficient to draw any conclusions.

Unfortunately, the most robust studies were on nerve agents, which of course is not necessarily extrapolatable to insecticides and, secondly, there was non-exclusion of severe poisoning in the Savage study.

One has to conclude from those studies that the answer to the question –'is there a long-term

effect of organophosphates on the CNS at subconvulsive doses?'– has not really been answered.

I am going to speak very briefly about Dr Spurgeon's study – very briefly, because she is here in the audience and she will be talking later. For the same of completeness, I just wish to compare it with the previous ones.

This was a study carried out on 150 sheep farmers with approximately the same number of controls matched as far as possible. There was no difference in memory or learning capacity but dippers showed poorer sustained attention and mental processing. I would like to point out that, although some of these findings are similar to those of the Rosenstock and Savage studies, the two previous studies both found major differences in memory capacity between the test groups and control groups. So the observations are not exactly the same.

For the sake of completeness, before I move on to the peripheral nervous system, I would like to point out there are one or two cases of associations being drawn between more specific central nervous system pathology and organophosphorus exposure – for example, Parkinsonism and schizophreniform and depressive psychosis. The numbers are quite small and it is difficult to be clear whether there is a cause and effect relationship.

I want to move on to the peripheral nervous system – firstly, with regard to the muscle side of things. Intermediate syndrome was described in the 1980s – and, as I said earlier, this is something of a lesson, because organophosphates have been around since the 1930s, and 50 years seems rather a long time to find a new syndrome. It is a proximal limb paralysis which occurs one to four days after acute poisoning. It is not responsive to antidotes and its importance is that it may kill acutely poisoned people because you need respiratory support. Obviously in a western Poisons Unit it will not kill.

The interesting thing from the point of view of what we are talking about today is this. Is the myopathy that has been observed in experimental animals and in a few human fatal poisonings at post mortem the pathological correlate of the intermediate syndrome and of the post-junctional jitter – an electrophysiological disturbance which has been observed by the group from Newcastle with ecothiopate? The reason for the interest in that is that it was generally assumed from experimental studies that the pathology went better within 14 days or so, whereas the electrophysiology seems to go better much more slowly.

Peripheral neuropathy, mentioned very much earlier, is a very serious syndrome and starts seven to 14 days after exposure. It is a partial or complete paralysis which affects the legs greater than the arms and recovery does not occur or is partial. It affects large diameter, long axons, which is why it affects the feet worse than the hands and, as I said, it is very serious. There are pathological changes seen in the axons, spinal cord and medulla. Organophosphorus-induced delayed peripheral neurophathy seems to be due to inhibition of an esterase called neuropathy target esterase, followed by ageing; it is not anything to do with acetylcholinesterase inhibition. The other point about it is that different OPs cause it. Some OPs that are not powerful anti-cholinesterases, for instance tricesyl phosphate, cause delayed neuropathy and others do not. The insecticide that is known to cause it is leptothos, and there are queries over some other OPs.

The policy of regulatory bodies is to use the hen test, which is a very good detection method for delayed neuropathy in experimental systems, to keep off the market organophosphates that cause delayed neuropathy.

However, there have been two recent bits of work which have raised questions about whether this hen test does exclude all the ones that you think they do. One is by McConnell et al, who showed reduction in ability to detect vibration and touch with methamidophos in agricultural workers. The other is by Kelly et al from Newcastle, who found that with mipafox there appeared to be delayed neuropathy at much lower doses than was expected. So it is possible that the hen test does not detect quite as well as you think it does.

Even if you do have a prediction method for delayed neuropathy and it does work, that does not exclude some sort of other peripheral nervous problem. One only has to look at the literature to find that the reduction in conduction velocity has been found in greenhouse workers, for instance, and other sorts of agricultural workers in citrus groves.

I hope I have shown you that in the central nervous system, the peripheral nervous system and the muscles there are many unanswered questions which deserve further research. I hope I have also shown you how complicated is the interpretation of the vast literature on organophosphates and how difficult it is to draw hard and fast conclusions.

THE CRUCIAL QUESTIONS THAT NEED TO BE ANSWERED

Peter Beaumont

Director
The Pesticides Trust

WHAT ARE the crucial questions to be asked about OP sheep dips and human health? I should preface my talk with one or two general remarks. The Pesticides Trust is a charity concerned with the health and environmental implications of pesticides – treating sheep dips for the moment as pesticides. We look at pesticide and agricultural policy in developing countries and the European Union as well as the UK.

We do not say that pesticides should be banned. Nor are we saying that disease – particularly scab or strike – should go untreated. But we are for the more effective regulation of pesticides, and the promotion of alternative means of pest control.

If you look at other countries, the World Health Organisation's best estimate is that about 3 million people each year suffer severe, acute, non-intentional pesticide poisoning, and the best advice is that most of these are due to OPs. The conclusion from that is that many OP formulations are simply not safe to use in developing countries – not because they are not good pesticides but simply because the conditions of use make those formulations unsafe for users, and I think that may be the situation that we are approaching here.

Having said that by way of preface, let me now get on to some of the questions I think we will be debating during the course of today.

Firstly, what is the incidence of OP poisoning, particularly in the UK? If you look at some of the formal or informal surveys that have been carried out – and I am thinking, for example, of the surveys of the Pesticide Exposure Group of Sufferers, or South West EPA, both of whom do valuable professional and unpaid counselling work, or the NFU, or the Agmed consultancy or the work of the Poisons Unit – there seems to be a considerable number of sheep farmers who suffer adverse effects. My guesstimate is that possibly 1% of the 100,000 or so sheep farmers may suffer a range of adverse effects ranging from the mild to the acute, the chronic and the more serious adverse effects. I would like to see other people's guesstimates this afternoon, but I think that gives us the range of the problem.

Secondly, we do perhaps need a single unified system of adverse reaction reporting for pesticides. I think the general public and the users of pesticides in general find the present system extremely confusing. We know that a study has been done by the Birmingham National Poisons Unit on the feasibility of a single system. I understand it was completed nearly two years ago. We wait for its publication.

Thirdly, I would like to advocate that there should perhaps be a change in the risk assessment process for pesticides, including sheep dips and agricultural pesticides. I do not think there is any dispute that to assess the hazard – that is, the harm that can occur from a chemical – you need an independent expert group, and the British system is very good at providing that. I think the problem arises when you then have to move to assessing the risk – which is the likelihood that harm will result from the use of those chemicals. At present there is not sufficient involvement of the users in the assessment of risk from agrochemicals.

I think it goes without saying in the 1990s that those who use pesticides should now have a view on the acceptable level of risk. It is one thing to say that the chemical is safe to use but it is another thing if you do not actually have to use the chemical yourself. I think the views of users on the acceptable level of risk now have to be taken into account.

The fourth point I want to make is that it seems a lot of people can use OPs for years and have no adverse effects. If you read the correspondence columns of the *Farmers' Weekly*, you can see the argument raging week by week – people who have dipped for years and have had no problems, and people who have dipped and now have serious problems. I think there needs to be a recognition that perhaps for some people there may be a predisposition, there may be a sensitivity, or there may be an idiosyncratic reaction to particular chemicals.

I would like to see – and I hope it is not asking the impossible – some form of prediction or diagnosis of which people these may be, because at the moment we often only find out too late. We will also, of course, need treatment for those people, but I think there needs to be a recognition that for some people OPs are simply not safe to use. That may not be a function of the particular OP molecules – it may be that some people are just more sensitive than others.

The fifth point I would like to consider is this – how can risks and exposure be reduced? My view is that there has been too much emphasis on personal protective equipment. The legislation that governs the use of chemicals at work is the COSHH legislation, which I think is one of the better examples of European health and safety legislation. However, what COSHH says is that

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you must first try to prevent exposure. You can do this by elimination – but nobody is suggesting in these cases you do not use pesticides, except in very rare cases – or you substitute a particular chemical for a less hazardous chemical. I think a lot of people are going to say that the active ingredients on the market that are not OPs are going to be the preferred choice for many farmers in the future.

The next strategy that COSHH advocates is controlling exposure. That can be done by technical or engineering controls, or by operational controls – by which is meant safe dipping systems, washing facilities and so on.

The final line of defence – and it is the last line of defence – is personal protective equipment. I think that there is too much emphasis placed on personal protective equipment, because it has to be both adequate and suitable. I am not convinced, and I do not think many farmers are convinced that personal protective equipment, even if it is well-fitting and maintained, is a sufficient guarantee of protection against exposure, particularly for those people who are particularly sensitive. I think that for those people, use of non-OPs must be a prerequisite.

My sixth point is that to cope with the problems we are discussing we need a comprehensive Government policy on reducing pesticide use. We have a problem in that sheep dips are a small market and it is unlikely that the industry by itself is going to develop sufficient alternative products. It is not only sheep dippers who particularly run the risk of high exposure to OPs – so also do horticultural workers in greenhouses, and grain storage workers. There are also reports of adverse reactions in these working situations. A comprehensive pesticide reduction policy should aim at reducing use, reducing risk to health and the environment, and reducing the dependence of agriculture on chemical pest control.

Finally, the most important thing which should come out of today is an informed discussion amongst members of the BMA and the NFU and the great and the good who run those organisations. What is needed is a joint policy statement from doctors and farmers, if it is possible to agree on these very difficult issues.

If doctors and farmers can agree a case for reducing the use of OPs and promoting the use of alternative products and perhaps making people use either alternative products – or, if they are not safe to use, alternative methods of pest control – the farming community and the medical community would have a lot to be grateful for.

THE IMPACT OF OPS ON THE NERVOUS SYSTEM

Dr Goran Jamal

Consultant Clinical Neurophysiologist, Southern General Hospital, Glasgow

FARMERS are engaged in regular sheep dipping with compounds that contain a number of neurotoxic substances including organophosphates, phenols and various solvents. The possible health effects of long term exposure to these compounds have recently been taken up by the public and the media.

The organophosphates are recognised as toxic substances with acute, intermediate and delayed effects. The acute toxic effects of organophosphate compounds occur within 24 hours, mainly in the form of cholinergic effects and are well documented. The acute cholinergic effects are due to phosphorylation and subsequent inhibition of acetylcholinesterase. In addition, an intermediate neurotoxic syndrome has been described with proximal limb and neck flexor muscle weakness but probably with a different underlying pathophysiological mechanism.

It is also known that some organophosphate compounds may induce a "delayed" polyneuropathy unrelated to inhibition of acetylcholinesterase and often unrecognised as the clinical features may be easily overlooked. This delayed neuropathy is associated with phosphorylation of serum neuropathy target esterase followed by "ageing" of the enzyme complex. Organophosphate induced delayed neuropathy has been produced by many different organophosphate compounds with evidence of both peripheral and central neurotoxicity in experimental animals. It has been observed to occur in animals with accidental poisoning and in humans.

There is experimental evidence that organophosphate induced delayed neuropathy may be more frequent among the users of organophosphate compounds than formerly thought. There is also recent evidence emerging to suggest that the occurrence of the OPIDN is not limited to those compounds which inhibit NTE. In addition to the above there has been some recent evidence suggesting that long term exposure over a long period of time to small quantities of these compounds produces chronic damage of the peripheral and/or central nervous system.

This chronic toxic effect is believed to be the consequence of cumulative pathophysiological changes from frequent exposure to non-lethal doses. Furthermore, some authors now believe that organophosphate compounds which have been regarded as innocuous may produce an axonopathy after prolonged exposure under suitable conditions. It is likely that chronic intoxication as a result of repeated exposure to very small doses could result in cumulative poisoning which may produce sub-clinical effects initially but render the individual susceptible to further toxic insults, thus producing progressive effects on the nervous system.

The other potentially neurotoxic substances in sheep dip are organic solvents. Recently it has become evident that chronic low level exposure to a number of solvents may produce slowly developing peripheral and/or central nervous system disorders.

We have been performing studies to investigate and determine the presence of any peripheral and/or central nervous system dysfunction in farmers using sheep dips. A battery of sophisticated neurological and neurophysiological techniques has been developed in our department to test both peripheral and central nervous system function in a group of farmers and in a group of sex and age matched control subjects. These neurophysiological techniques are sufficiently sensitive to detect early or sub-clinical signs of neurotoxicity in humans.

The results of our preliminary but continuing studies provide some evidence of peripheral nerve damage in the farmer group who have been exposed to sheep dip in the form of distal axonopathy involving both the motor and sensory fibres. All fibre populations including large myelinated, thinly myelinated and unmyelinated fibres are affected. These findings are comparable with those in neuropathies associated with toxic chemicals such acrylamide, arsenic and thallium. Distal axonal degeneration is the principal and earliest feature of organophosphate induced delayed neuropathy in both experimental animals and humans. It has been suggested that motor involvement is more prominent in organophosphate induced delayed neuropathy but sensory loss is invariably present upon careful clinical examination. Distal axonal degeneration involving both sensory and motor fibres is also a feature of chronic exposure to solvents.

In toxic neuropathies, including those related to organophosphate compounds and solvents, it is thought that the vulnerability of nerve fibres is related to axonal length and diameter; large diameter long axons, both motor and sensory, are thought to be more susceptible than small diameter shorter axons. This assumption has been primarily based on morphological data but more recent electrophysiological, clinical and morphological data suggest that this is not the case and

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that all fibre populations are equally vulnerable. With the practice of combining several agents in a given product, complicated biological interactions and synergistic toxic effects may be encountered.

Our nerve conduction study findings are generally similar to those observed previously in toxic neuropathies including solvent neuropathy and organophosphate induced delayed neuropathy in experimental animals and humans. It has also been claimed that changes in sensory rather than motor potential amplitudes are more sensitive physiological markers for screening patients exposed to neurotoxic organophosphate compounds. Our results strongly support this hypothesis.

The precise biochemical abnormality responsible for the development of the organophosphate induced delayed neuropathy has not been clarified but a selective metabolic lesion of the neurone, possibly involving phosphorylation of cellular components, has been postulated. In other toxic neuropathies, distal axonal degeneration has been correlated with abnormalities of axonal transport, physiochemical changes of proteins or alteration of the properties of the axonal membrane. In experimental organophosphate induced delayed neuropathy it has been suggested that neuropathy target esterase, when phosphorylated, may be a marker of the development of a delayed neuropathy if this is followed by "ageing" of the phosphorylated enzyme complex. The cellular role of this enzyme and whether or not it is directly involved in the production of the neural damage is unknown.

None of the farmers in this study, when tested, had any biochemical evidence of very recent acute exposure to organophosphate compounds. Therefore, neuropathy target esterase activity would probably not have been affected since its activity returns to normal well in advance of the development of long-term electrophysiological effects of organophosphate compounds in animals.

Further studies of the association of these electrophysiological effects with exposure to sheep dip should include measurement of lymphocyte neuropathy target esterase, red cell acethylcholinesterase and urine organophosphate metabolite concentrations in those farmers recently exposed to sheep dips.

More extensive investigation of low level occupational exposure to potential neurotoxic preparations containing organophosphate compounds and other substances is required. Little is known about the long term neurological consequences of mild and repeated exposures which may have important health risks for those using these compounds.

terns. The trouble is that the numbers are small, but I would again leave it with you that there is strong evidence to suggest a major effect of OPs on serotonin systems and that this may, at least in part, explain the very high risk of suicide in farming folk. It is also perhaps interesting that another group of people with a high risk of suicide are vets, and they too are severely exposed to OPs.

Let me now turn to another group of psychiatric disorders. Many OP-exposed farmers have been labelled as hysterics. What we see are a variety of neurological disorders, often quite bizarre – difficult to explain. The so-called non-specific symptoms form part of these, and because the GPs and the neurologists cannot find any clear rationale to explain them, in due course the poor farmers are labelled hysterics or – perhaps more commonly these days – depressed.

So what is the fate of such a farmer? He is referred on to a psychotherapist – probably not medically qualified. If he accepts the explanation offered by the psychotherapist for all his symptoms, then he is a good patient and, presumably, in due course, after many many years of psychotherapy, gets a little bit better. If, on the other hand, he tells the psychotherapist, 'no, you have got this absolutely wrong; this is down to OPs', he is labelled a bad patient; he refuses to accept the interpretation and is discharged with that label to his GP and there the case is closed.

Fortunately, some of us – Dr Jamal and myself and others – take these cases a little more seriously, and I am now beginning to see patients with these bizarre neurological disorders. I have two patients at the moment – OP-exposed – who developed partial seizures when performing a certain action. Actually demonstrating that this is a true organic effect is very difficult, but we are about to acquire some rather sophisticated EEG equipment with which I hope we may be able to clarify the situation.

The message is, then – do not label people hysterics. Hysteria does occur, but I think it is absolutely essential that medical practitioners are prepared to say they do not know. Speaking now to the GPs and the psychiatrists and neurologists here, even if a neurological syndrome appears to defy current views of anatomy or physiology, do not discard it as hysteria. If you want to make a psychiatric diagnosis, you need positive grounds to do it. Psychiatric disorder, including hysteria, is not an exclusion diagnosis.

Finally, then, may I come to the need for further research.

I am going to make a plug here for the Proceedings of a conference which took place last September which is published by the South West Environmental Protection Agency, in which some of the complexities and confounding factors in doing research on causation of psychiatric disorder by this sort of mechanism are gone into in much more detail.

In view of these difficulties, there is another way of tackling things, rather than relying on epidemiology – because in order to produce epidemiologically significant results for a common disorder such as depression, you would actually need numbers in excess of the total numbers farming in the UK. I would suggest we develop the so-called bottom-up approach of looking at the neurochemical and neurobiological effects of OPs.

I have mentioned serotonin effects, and much more work needs to be done to elucidate these. But OPs affect other systems as well – they phosphorylate glutamate receptors. Apart from being involved in epilepsy, glutamate receptors also control proliferation of dendrites – the tree-like growths of the nerve cell where contacts are made. This may possibly explain the cognitive deficits noted.

The analogy between a telephone system and the brain has been drawn. It is not an exact one but in this case it is good enough. If you have uncontrolled, random development of synaptic connections, it is rather like a telephone call from London to Bristol being routed through Stockholm. That is going to slow processing. The effects on epilepsy from glutamate receptors also would stand out.

Finally, OPs induce a process known as kindling – central to the genesis of epilepsy and possibly related to impulsive aggression and other behaviours which are frequently reported in farming communities.

More work needs to be done. There is no doubt that organophosphates not only influence acetylcholine but have wide and varied effects on central nervous system function. We need now a lot more work to specify what those effects are, but I think we are not very far off drawing an almost exact cause and effect relationship with regard to affective disorder.

I am not suggesting that if you are exposed to OPs you will develop depression. What I am saying, as the previous speaker said, is that we are talking about vulnerabilities. Everybody carries a certain vulnerability to depression and vulnerability is conferred by genetics, by early life-experiences, by stress – both acute and chronic – and other factors. What I would propose is that being exposed to organophosphates shifts your vulnerability along, and therefore you are much more likely to develop this psychiatric disorder than had you not been exposed.

Some people have expressed a difficulty in accepting that environmental toxins and similar substances can in fact induce psychiatric disorder. This perhaps is most common in those who take a non-biological view of mental illness as a whole. It is however very well established that whole groups of compounds can produce profound mental disorder, and I would draw the example of the old anti-hypertensive drug reserpine.

This was a drug known to Hindu medicine as a

tranquilliser and used in the 50s and to some extent the 60s to reduce blood pressure. However, it became clear that patient after patient was becoming profoundly depressed and very, very suicidal on this drug. This finding actually opened up the whole area; it was a major contributor to an understanding of the chemistry of depression because this drug depletes, among other things, serotonin in the nerve terminals, and there was a clear cause and effect relationship between a drug with a central neurochemical action and the development of a major depressive illness. Again, it was a vulnerability question; not all patients given reserpine became depressed, but a proportion did, and many who did probably would not have become depressed had they not been given the drug. The analogy with organophosphate exposure, I would suggest, is almost exact.

The purpose of this conference is to try to formulate at least the beginnings of policy or strategy as to where we go. Research is essential, and not simply epidemiological research. We need basic science done and this is expensive and must be funded.

Quite what the toxic effects of the OP alternatives are I really do not know and this causes great problems; you may actually be jumping out of the frying pan into the fire. But, if we are to develop these agents, then there must be routine neurotoxicology done. It is a regrettable fact that the only country which has required routine neurotoxicology on pesticides was the former Soviet Union. Unfortunately the quality of the work was not brilliant, but at least they made an attempt.

It is time we made an attempt and ensured that every agent which is used in this way is run through a series of animal studies to look at the effects on key neurotransmitters and major nervous system function.Whilst this work is being done I would strongly advocate a moratorium on the use of OPs.

OPs AND HUMAN HEALTH: AN AGRICULTURAL CASE STUDY

Dr Bernhardt

General Practitioner
Kent

I WILL be presenting the case of Mr Gary Coomber, who I believe has heart disease as a result of organophosphate exposure. He is now 36. This gentleman suffered recurrent myocarditis. He has had a cardiac arrest on one occasion and been resuscitated. He now has evidence of heart damage and a degree of cardiomyopathy. This gentleman was fit and well before June 1991.

At age 1.5 years he had whooping cough and at age 16 years a fractured femur. There is really not much remarkable about that. In June 1991 he developed a flu-like illness – vomiting, chest pain, and was really quite unwell. He was admitted to hospital, where the diagnosis given was myocarditis.

That episode recurred within two weeks of using propetamphos sheep dip. He recovered and was well one month later. Fairly extensively investigated, the cause of the myocarditis then was thought to be viral. I think quite a lot of people with myocarditis get the cause diagnosed as viral in the first place.

He remained well up until June 1992. Again, we have the same thing happening – flu-like symptoms, chest pain, went to hospital, had a VF cardiac arrest, resuscitated, transferred to London. Heart muscle biopsy investigations again showed the diagnosis to be myocarditis. Yet again, we have got the episode occurring within two weeks of using the same sheep dip. It took slightly longer to recover this time – about three months gradual recovery to apparent full health. The cause was now thought to be either viral or unknown.

Coming to 1993, fortunately he remained well during that year. I do not know whether I can take any credit for that, but I told him to stay away from organophosphates after the episode in June 1992, which he managed to do.

So we have had June 1991, June 1992, and now we have May 1994 – a similar time of year. Again, we go down with chest pain, flu-like symptoms, sent to hospital, myocarditis diagnosed. It did not occur after sheep dipping this time – it recurred after unintentional contact with dipped sheep and walking through a field sprayed with organophosphate three days earlier. Again, from contact to symptoms developing was less than two weeks. He was unwell for quite a bit longer this time – it took about five months to recover. He is not in full health now. He does have evidence of heart damage.

To summarise that, I therefore have a previously fit farmer, three separate episodes of myocarditis, one cardiac arrest, each occurring within two weeks of exposure to organophosphates. He has been to see quite a lot of specialists. He has been comprehensively investigated so far by both them and myself trying to look for a cause. The evidence so far does not support an infective (viral) cause or a connective tissue disease cause for the illness.

If you look at the timing of the events – it has been June 1991, June 1992, May 1994 – that would suggest to me that he is exposed to something in his work environment or his home environment that triggers the problem If it was viral, or if it was due to some other cause like a connective tissue disease, I would have thought it could have happened at any other time of the year. The myocarditis develops within two weeks of organophosphate contact. That to me – I know this sounds pretty basic – would suggest that organophosphates actually are the cause.

Since 1991, he has not used organophosphate products at other times. If he had done and he had not had a recurrence, I would be less inclined to think they were the cause. If you look through the literature, organophosphates are known to cause myocarditis. They are also known to cause fatal arrythmias. He has had both of those.

If you want to go into this in more detail – I hope this doesn't become boring:-

- In 1980 we have a paper describing congestive cardiomyopathy from long-term organophosphate exposure.
- In 1992 a letter in the *BMJ* – a 28-year old farmer with ventricular tachycardia using OP dips in the preceding two months, got better, told to stay away from it; recurrence two months later after handling dipped sheep.
- *American Journal of Cardiology* in 1982 – six patients described with ventricular arrythmias after OP poisoning.
- Paper from 1966 – that is quite an old paper – Journal of the *Indian Medical Association* – toxic myocarditis found in OP poisoning necropsies.
- *Indian Journal of Medical Research* in 1984 – cardiac toxicity in pesticide formulators exposed to organophosphates.
- 1989 paper describing ECG changes from OP intoxication
- *Indian Journal of Medical Sciences* , 1970 – ECG changes, ventricular arrythmias, myocardial damage on necropsy in OP poisonings.
- *Journal of the Royal Society of Medicine* in 1981 – OP poisoning and complete heart

block.

- 1979 – another paper – OP poisoning causing arrythmias, transient picture looking like myocardial infarction, histology showing myocardial damage, arrythmias occurring on days 3-15 after exposure.This paper suggests that organophosphates have a direct toxic cardiac effect.

To reiterate, in Mr Coomber we have myocarditis, cardiomyopathy – at some stage in his various appointments with specialists he was thought perhaps to have a myocardial infarction, but that is not now substantiated, and each exacerbation occurred shortly after organophosphates exposure.

I contacted the Department of Health in 1994 and asked them the question – were organophosphates the cause of the problem? – and explained the situation. The reply was that it was unlikely that organophosphates were the cause of the myopathy and some other cause was likely. Guy's Poisons Unit were contacted in 1992 – this was after the cardiac arrest in hospital – and did not have a contribution to make. He has been seen by Guy's Poisons Unit in 1994 – they thought then that possibly an environmental exposure, possibly organophosphate, was the cause.

My views are at obvious variance with the Department of Health. With that evidence, I feel it is the cause. I think one of us is right and one of us is wrong and it will be interesting to see how things develop.

Garry Coomber is under the care of Professor McKenna at St George's Hospital in London. He feels now that in view of Mr Coomber's illness and its timing it would be consistent that OPs were the cause. He does intend further research. He has another patient with a very similar background including a VF arrest and exposure to organophosphate.

I have been in my amateur attempts trying to find more people similarly affected. A visit to a 42-year old sheep farmer in Surrey who had quite severe dilated cardiomyopathy, who is now very much better since he has avoided OPs, reveals two to three more local, similarly affected sheep farmers that use OPs.

I do not know how many people there are out there who perhaps, if I am right, have the diagnosis of myocarditis that could be due to organophosphates. I think – I do not know – that the most likely diagnosis they are given is that the cause is viral. If there is one thing that I hope this talk from me produces it is that patients going to their GPs or their specialists with myocarditis or cardiomyopathy should specifically be asked if they have organophosphate exposure. Only by the awareness of a possible link with organophosphates and myocarditis and dilated cardiomyopathy can we get some progress made and perhaps an epidemiological study.

Dr Fleur Fisher said earlier that it would be useful if GPs had increased toxicological knowledge. I do not know how useful this would be because, until we know what are the full effects of OPs, we will not know what we are trying to look for or diagnose. My recognition of the possible problem in 1992 met with little if any interest.

I was asked to base my talk on how other GPs could be helped if they get presented with a possible organophosphate poisoning case. It has taken me three and a half years to get to this stage and I believe it is the cause, my patient believes it is the cause and now Professor McKenna believes it is the cause. I would not know how to advise.

CLINICAL TOXICOLOGY: DEALING WITH OP POISONING IN PRACTICE

Dr Alex Proudfoot

Head of the Edinburgh Centre of the National Poisons Information Service

IT IS quite clear already that there are more questions than answers to this particular problem. What I propose to do is to speak a little about acute organophosphate poisoning, raise an issue which will suggest that we are all perhaps being a little narrow-minded, and lastly, suggest how we should go about approaching the patient who has chronic symptoms that might be the result of organophosphate exposure.

There is a spectrum of organophosphate intoxication. There is the very acute form with its intermediate syndrome and delayed neuropathy of which we have heard something this morning. There is also sub-clinical poisoning, where the amount of cholinesterase in the plasma or red blood cells is significantly diminished, yet the patient does not have much in the way of symptoms, and there is chronic poisoning.

Acute poisoning with organophosphates is a rarity in the UK. It is a problem 90% or more of which occurs in developing countries. In the latest large series which I saw published about 1993, two-thirds of acute poisonings admitted to hospital in India were the result of suicide attempts, one sixth were the result of accidents and the final sixth occurred during the normal use of these compounds. So, while there is a real problem, I am not sure that it can be resolved very easily in this country.

We do however have means of dealing with acute organophosphate poisoning, depending on how severe it is. For the severely poisoned patient, we need to clear the airways - they are almost certainly full of frothy secretions which have to be removed, breathing may be impaired because of problems at neuromuscular junctions, and a period on a ventilator may be necessary. Convulsions have to be controlled. These are obviously emergency measures. Once they are implemented and the patient is in a stable state, we have to decontaminate him and consider whether antidotes should be used. I do not wish to go into this because it is really not relevant to this morning's discussion.

Let me return to chronic poisoning. We have heard that there may be chronic symptoms after acute poisoning episodes, but we are perhaps more interested in those which occur without a preceding acute poisoning. Many groups in society are blaming toxins in the environment for their symptoms. It is not just a matter of organophosphates - a whole lot of other pesticides are being blamed. If we look at the chronic features we might get from organophosphate poisoning, they are vague and do not lend themselves readily to measurement. I do not have to tell general practitioners that fatigue, depression and irritability, for example are extremely common. The same holds in my clinic, but most of the patients have never been exposed to pesticides in any major way.

It is serious that so many people in present day society have such symptoms but I would strongly suggest that they are not due to organophosphates. There are a whole lot of other illnesses which are relatively new and being attributed to a number of things in the environment - the chronic fatigue syndrome, myalgic encephalomyelitis, whether it is due to viruses and so forth, multiple chemical sensitivity and "environmental" illnesses of various sorts. If you look at the symptoms of the patients in these groups, there is a great amount of overlap. I am not suggesting that they are all the same, but there is a significant overlap which I find difficult to explain.

Most doctors, having had a scientific training, would like to know what lesion they are trying to put right when dealing with chronic symptoms due to organophosphates. Is it damage to the structure of some organ or other? Is it an abnormality of function or the harmony between one body system and another? Or is it more basic, involving biochemistry at the level of cells? I also need to think of how I am going to identify the various problems that might be corrected. Exposure is relatively easy to confirm if it has been recent since we can measure the cholinesterase activity of plasma or red cells and see if it is reduced. Measuring the effects of exposure is much more difficult but we have already heard of sophisticated nerve conduction studies and a variety of other tests which can be used.

Then there is the question of susceptibility. In respect of acute poisoning there are good reasons for some people being more susceptible than others. The amount of cholinesterase in the blood is determined genetically and, even if it is present in quantity, it may not be of the usual quality and, as a result, not work in the correct way. I do not know the factors which might predispose to chronic symptoms.

I am in no doubt that the first thing doctors must do is to take the patient seriously. If they do not, there will be all manner of problems. Unfortunately when patients allege that their symptoms are the result of poisoning, they may not be taken

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quite so seriously as when they blame something more mundane. The important starting point is to get the trust of the patient. What I find very sad is the tragic group of patients - fortunately small at the moment - who are totally convinced that there is an external toxic cause for their symptoms. Nothing you tell them will budge that belief, which may or may not be correct. Doctors can usually neither prove nor disprove it.

If examination of the patient and all the tests fail to reveal an abnormality the doctor's competence may be questioned or the patient may refute comparison with commonly accepted normality on the grounds of increased susceptibility which, again, doctors can neither prove nor disprove. We end up with far more questions than we have answers and as a consequence of the understandable dissatisfaction which may result, patients may consult a variety of doctors up and down the country. We would all like to prevent this happening and may be able to do so by taking the patient seriously enough from the outset.

If I were a general practitioner, I would start with the patient's history. We are interested in symptoms and their relationship to exposure but we must remember that farmers who have used organophosphates have probably used all manner of other pesticides as well. Not only the active ingredient needs to be considered but also the solvent in which it is diluted. Nor is it simply a matter of exposure at work, it is a question of what happens at home as well, particularly hobbies and gardening. It may be useful to get the patient to compile a list of all the chemicals he might have used in these various areas and they can then be considered in the context of his symptoms.

The patient must then be examined, but even a detailed neurological examination is a crude tool even if it is an important starting point. Assuming nothing turns up, other investigations are indicated to exclude more orthodox diseases. These are aimed at ensuring that the kidneys and liver are working satisfactorily: that the thyroid gland is not underactive, since this could be the explanation for some of the symptoms attributed to organophosphate exposure. Diabetes must be excluded, as must vitamin B12 deficiency. A chest x-ray is also a good idea. Then there is the question of whether or not cholinesterase activity should be measured. This will depend on the services available locally. If they cannot perform this analysis, the National Poisons Information Service will be able to direct you to an appropriate laboratory. But I would not hold out much hope of getting an abnormal result if there has not been recent exposure.

That, I think is the starting pack of investigation which a general practitioner might carry out and which I do myself. The history taking, examination and investigations take time. They cannot be done in five minutes and I allocate at least an hour for every new patient who has a problem that might be related to an environmental toxin. After that, there is homework to be done - not only on the results of the investigations but on the list of chemicals and pesticides that the patient produces. It is a very time consuming process.

In addition, you may have to do other investigations depending on the nature of individual symptoms. It might be important in some cases to carry out a brain scan to see if there is an abnormality that might more reasonably explain the patient's condition than exposure to a pesticide. We have already heard that nerve conduction velocities may have to be measured and neurobehavioural tests carried out. These are important and highlight the fact that no single discipline within medicine is going to come up with the right answer. There must be a multidisciplinary approach if we are going to progress. It is not just a matter of referring the patient to a general physician or clinical toxicologist - they need help from x-ray specialists, neurophysiologists and others who are able to perform the sophisticated tests which will help doctors to reach a decision about the patient's mental state and ability to react to stimuli.

The general practitioner has a problem. He has to find a focus through which he can obtain these services and none is readily available. However the clinicians within the various centres of the National Poisons Information Service would be prepared to see some of these patients and sort them out as best possible.

In conclusion, doctors need to establish the trust and confidence of the patient and that means taking time with them, discussing things, and being sympathetic because, whether or not the doctor believes that the symptoms are related to a poison, they can neither prove nor disprove it. If doctors take an antagonistic position at the outset they are lost and so is the patient.

Investigations to exclude other disease are important as is the need to seek expert help. At the end of the day, conventional medicine at the moment is unlikely to be able to offer any specific treatment. We have heard about the link between serotonin and organophosphates, and perhaps some of the newer antidepressants will become the treatment of choice in due course, but that has yet to be decided. At the present, unfortunately, symptomatic treatment is all that can be offered and may include sessions with clinical psychologists or psychiatrists, depending on the patient and the complaints - it has to be tailor-made.

Finally, prevention is far better than cure in this situation. We should either abandon organophosphate sheep dips – but not before ensuring that what might replace them does not carry greater risks – or reduce exposure by improving practices during sheep dipping.

MORNING DISCUSSION

Chairman — Now it is time for some questions to our four speakers on the panel. What we want at this stage is questions to the people whose presentations you have heard.

Dr Julian Kenyon, *Centre for the Study of Complementary Medicine, Southampton* — Dr Bernhardt – your case study. Professor McKenna at St George's – is he doing anything effectively therapeutically for this patient or is he just simply researching the case? If so, if he is doing something therapeutically effective, what is that?

Dr Bernhardt — Garry Coomber at the moment is on no treatment at all. He is fairly well at the moment. I do not think his disease is currently progressing – therefore he does not need any treatment. When you talk about therapy for his condition, unless his heart is becoming worse, then he does not need any therapy, as far as conventional medicine would have it.

Dr Jean Monro, *Brakespear Hospital, Hemel Hempstead* — I have been dealing with the neurotoxicological effects of poisons on patients – not necessarily only OP poisonings. I want to put a question concerning the way in which research might be conducted and to ask the speakers' opinions about it. They have all identified different aspects of illness in these patients, some neurological, some cardiological, some general in that fatigue has been mentioned, and some psychiatric – so that if an epidemiological study were to be considered, it would be difficult to home in on any of those and get a conclusive response. I have a suggestion, which is that the patients who are badly affected should be the ones who are studied, not those who are excluded because of something. They should be the ones who are studied and they are the ones who can provide their own longitudinal effects of therapeutic intervention – so that, if they happen to have neurological disease, it is the neurological disease that is followed in them and if they have a cardiological disease, then it is the cardiological. There is no point in trying to lump these patients together into one large group when you will miss what is going to be an effective therapy for them because they may not all have the neurological disease, they may not have the psychiatric disease.

Chairman — This is a plea for the better understanding of the natural history of whatever disease might be caused.

Dr Monro — I am asking that they consider the functional ability of the individual as a therapeutic outcome so that it should not be done as a sort of double-blind gross study for a whole group. It should be done as an individual functional assessment for a particular patient.

Dr Davies — I wholeheartedly endorse what you are saying. It is essential for us to look at the most severely affected patients. Can I, though, make the point that the HSE study needed to be done and it needed to be done in a highly controlled exclusive way. At least now we have data which nobody can assail. There is nobody who can rubbish the HSE report or at least rubbish the conclusion that OPs produce cognitive impairment – that is now set in stone. Now we need to go on and find out what really happens to people exposed to OPs and other neurotoxins. I would, however, make the plea that we *must* look at the basic science; we have got to look at what these agents do to the various systems within the brain and, indeed, elsewhere – so it is basic science, not just epidemiology.

Dr Jamal — What you refer to is what we call case control studies. That is important – it is important to understand more about the effects – but I do not really think it is a substitute for a well conducted, well-planned epidemiological study.

Dr Routledge, *Newcastle College* — A question to Dr Proudfoot. I am coming from a different direction. I lecture in pesticide contamination in food, and a second-stage judgement was offered in my favour by the European Commission about the problem of the contamination of drinking water with sheep dip. There is widespread exposure to sheep dip pesticides in the population, and the symptoms in the general population that you see similar to the sheep farmers may in fact have that cause.

Dr Proudfoot — I should apologise to the meeting for my comment that some of these patients who have got symptoms similar to those that are being linked with chronic organophosphate toxicity have never seen an organophosphate in their life. Quite clearly, they have. I accept all that Dr Routledge has said. OPs are in the water, they are in other foods and so forth. What I was trying to

do – and obviously went over the top in trying to make the point – was that the exposure which one gets from drinking water and through food is relatively small compared with the levels of exposure that one would expect to get in farmers who have been sheep dipping.

Dr Douglas, *GP from Fort William, Scotland* — I have a couple of patients who I am convinced have long-term effects from OP poisoning. The chairman has repeatedly asked the question – what do general practitioners want? What I want and what my colleagues want is succinct information and guidelines. We need specific guidance on the best timing for post-use samples. I would suggest within 36 hours – but something simple like that.

Chairman — When is the right time to do this testing?

Dr Bernhardt — I think there are papers out that show ECG changes in patients exposed to organophosphate – yet their cholinesterase levels are normal. You do not know what these substances do. In my opinion I do not think the cholinesterase level is useful.

Dr Davies — On that, can I offer one piece of advice to general practitioners everywhere when they are dealing with depressed and anxious OP-exposed patients. Avoid drugs with anti-cholinergic actions like the plague because OP-exposed patients are exquisitely sensitive to these drugs – they go into urinary retention, they develop awful dry mouths, often to the point of ulcers, and maybe there is your marker – extreme sensitivity to anti-cholinergic drugs. The pharmacology of this immediately seems to be a bit perverse – there you are, increasing acetylcholine levels, but what happens to receptor sensitivities? My strong suspicion is that after exposure patients develop sub-sensitive acetylcholine receptors and, when you reduce the input to those still further by using a tricyclic antidepressant or similar drug, they develop hellish problems – and that may be your way in.

Dr Jamal — You must understand the limitation and the sensitivity of any test that you use. If the acetylcholine esterase level does not change, do not exclude exposure to organophosphate – that is a very important message. It is exactly the same thing with any biological test – there are false negative, false positive. You must also understand from what is being said that depression of acetylcholine esterase level is not related to this effect that we are talking about.

Chairman — I think that further illustrates the problems that the GPs are up against. If you do the test and you do not get a positive result, you still cannot conclude from that everything is well.

Dr Keith Eaton, *British Society for Allergy and Environmental Medicine* — A question to Dr Davies to slightly broaden the field of discussion. Would he not agree, when he discusses stress, that there is a very large number of papers mainly in the psychiatric literature on the adverse effects of stress on the immune system – and could this not also be relevant in this area?

Dr Davies — Absolutely. Stress has effects on the immune system, as of course does depression. The prognosis of cancer patients who are depressed is much worse than those who are not depressed – so, yes, I wholeheartedly agree.

Elizabeth Sigmund, *South West Environmental Protection Agency* — Dr Martin Johnson, who was with the Medical Research Council, wrote to me just before he retired saying if he had the time and money he would study at least six other enzymes that might be involved with the use and exposure to OPs. Cholinesterase levels are an absolute blunt instrument. There are other enzymes which should be looked at.

Chairman — Essentially, is there a case for studying in more sophisticated detail the kind of enzyme abnormalities or effects that you might find? Would anyone care to say whether that is practicable or feasible?

Dr Jamal — Any neurologist with general knowledge will say to you that there are hundreds of enzymes in the axon of plasm, in the membrane of the nerve cell and in fact you are absolutely right. I spoke with Martin Johnson myself, and the direct answer to my question was that it was by pure chance that they tripped their feet over NTE. There are very many other enzymes which are related to the nerve function which are absolutely vital and they have not been looked at. We do not know, even now, 30 years later, how the delayed neuropathy is produced.

Dr J Kenyon, *Southampton* — I wonder if there is a European dimension on this – whether other countries in the world have the same perception of OP chronic toxicity as we are having today – and, if I may ask another question of Dr Bernhardt, what were the cholinesterase levels in his patient, please? Had he actually absorbed any organophosphate?

Dr Bernhardt — Garry Coomber's cholinesterase level, when it was done, which was some time after his illness, was normal – as far as I can remember.

Comment from floor — Serum or red cell?

Dr Bernhardt — Both were done. I think there was a slight decrease in one of them and the other one was normal. I really do not think that this dis-

proves any of the fact that it is the cause of his problem. I think the cholinesterase test is, as Liz Sigmund says, a terribly blunt tool.

Chairman — The other part of that question was a wider view – what goes on elsewhere in the world?

Dr Jamal — I can only say that the authorities in Australia have been in touch with me – they have indicated that they are worried about this problem. They brought to my attention that they do have cases reported to them of a similar nature. I have got two medico-legal cases from Australia about this issue.

AFTERNOON DISCUSSION

Chairman — Now it is the session in which we are going to be calling upon you, in the body of the hall, to make most of the contributions this afternoon. Let me introduce the panel who are on the platform here with me:

- Dr Anne Spurgeon of the Institute of Occupational Health in Birmingham, whose name is of course well known to you on account of the recent report published in *The Lancet*
- Roger Cook, Director of the National Office of Animal Health
- Dr Fleur Fisher of the BMA, who we have already heard from this morning
- Ian Gardiner, Policy Director of the NFU.

Before we come to you in the body of the hall, I am going to ask Anne Spurgeon if she will briefly summarise the IOH report in *The Lancet* – so that we have some idea of the conclusions it came to, and then I will be asking Roger Cook if he would care to comment on that report. First of all, Anne Spurgeon.

Dr Anne Spurgeon — I will just summarise the main design and results of our study that has been alluded to several times in different ways this morning. We were basically trying to answer the question – are there chronic effects resulting from exposure to OP sheep-dips? – and we were looking at chronic effects on the nervous system by using neuro-behavioural tests. We were trying to look at behavioural outcomes that would indicate some effects on the central nervous system. We are having some controversy about our choice of quarry workers as a control group – but it was difficult to find a suitable control group, because most farmers use neurotoxicants of some kind, so in the end we settled on quarry workers.

We compared the two groups on a range of psychological or neuro-behavioural tests as they are sometimes called. These are basically tests of cognitive function, so we are looking at things like reaction time, information processing, memory, learning – those sort of processes – and we adjusted for all the other things that might affect psychological performance, like age, educational level, and long-term alcohol consumption. We either matched for those or we adjusted for them statistically in the analysis. We excluded people who were suffering from pre-existing diagnosed disease of the nervous system and, as has already been mentioned this morning, we also excluded people who had a prior head injury.

We were trying to look at what appeared to be normal farmers doing their normal work, using OP dips on a regular basis, and trying to find out if any effects they had were actually due to OPs. We gave them seven psychological tests. We found effects on three of those tests – tests that were concerned specifically with the ability to sustain attention and also ability to process information. The effects were a slight slowing of information processing and a slight slowing of reaction time – fairly subtle effects. We did not find any effects on any of the other tests, which were largely concerned with different types of memory and learning processes. So the results did follow a certain pattern from the psychological point of view, and we concluded from this that there were subtle effects on cognitive functioning in this group of farmers. This was not a group of farmers who appeared to be ill in any way. The effects were not the kind of effects that would have made them likely to go to their doctors complaining that they were suffering from certain symptoms. But even in this group of apparently healthy farmers we did find some subtle effects on cognitive functioning which, as far as we can tell – because we did a very carefully controlled study – were likely to be due to OP exposure.

Chairman — So the general view you take is that, in your study at least, you were fairly confident that there was an effect, that you did detect something?

Dr Spurgeon — Yes. We are fairly confident there was an effect, albeit a fairly subtle one, but we are confident there was an effect.

Chairman — Let us come to Roger Cook of the National Office of Animal Health, speaking on behalf of the manufacturers. How do you respond to this report?

Roger Cook — I have the benefit over this audience of having been to a meeting the HSE organised last week where Dr Spurgeon gave a full presentation of her report. A number of comments came out of the audience, really as to whether the control groups were quite as good a match as they might have been. This morning we heard reference from Dr Davies, among others, about the stress that is particularly affecting farmers. There were some doubts about whether that factor was mirrored in the quarry workers and whether this was having some effect on the psychological tests. What was also interesting was that in four out of

the seven tests there was no difference found. What I think we all have to learn from this, in particular, is that Dr Spurgeon's enquiries confirmed what we have all been concerned about, which is the lack of protective clothing used by many of the sheep farmers (shouts of "rubbish").

Elizabeth Sigmund, *South West Environmental Protection Agency* — Completely irrelevant – your usual excuse is, the farmer is to blame.

Chairman — Mrs Sigmund, we will come to you in a minute.

Roger Cook — I was summarising on Dr Spurgeon's report and she does state that as a fact in her report. One of the things we have to recognise is what Tony Pexton said this morning, that there is a very important job to be done in protecting the sheep of this country. We have the biggest sheep population in Europe – 40 million sheep. The parasites which attack them are very damaging. They do need controlling, so that work has to continue in some form. If I can just make a couple of points on this morning's presentations – I found it very interesting in Dr Marr's presentation that he put emphasis on the very diverse properties of organophosporus compounds. They are not a single group – they are a very wide group. In trying to analyse all the information dispassionately we have to be very careful not to argue from the general to the particular. We need to look at the effects of the three organophosphorus compounds used in sheep treatment in this country and not get too side-tracked by the properties of other organophosphorus products which are not used.

The other thing which is important to all of us, whatever side of the debate we are on, is that we need to recognise the vagueness of the symptoms. It is important for doctors to be aware of the possibility of OP poisoning – but equally, it is important for them, when they embark on this process of detection, to find out what is wrong with their patient, to recognise the vagueness of the symptoms, and to make sure that we have got to the right diagnosis so that at the end we can give the right treatment.

Chairman — But as far as the continued use of OP dips is concerned, you think farmers should be carrying on?

Roger Cook — I think they should continue to have the choice. An important point to make is that there is a range of products available – dips and alternatives, pour-ons, and now an injection. What the farmer has got to do is look at all the circumstances that he is faced with – the parasites he needs to control, the particular circumstances on his farm – and choose the right product. Once he has made that choice, he has to read the label and he has to follow whatever advice is given on that label.

Chairman — Let me come to Mrs Sigmund now. You do not entirely agree with Mr Cook on this. You think that OPs should be banned.

Elizabeth Sigmund — I think that any chemical that has been demonstrated to have been causing the sort of symptoms that we are seeing among hundreds of our farmers must be withdrawn until the proper scientific tests have been done. We are still unclear as to the type of enzymes that are being affected. We are still very unclear about many of the effects which different OPs are causing. The most telling thing that Roger Cook said is that we have got to differentiate between the chemicals. It is absolutely true, and if he can give us clinical evidence that one of those three OP chemicals is not capable of causing neurological and neuropsychological damage, then he should stop trying to blame the farmers for not using recommended protective clothing. The recommendations for these have been changed repeatedly and, even up till last year, were never recommending any form of breathing protection whatsoever.

It is not necessarily the OPs being inhaled into the lungs that is the root of exposure. It can be OP particles that are absorbed in the nasal tissues. Only last year did you start to tell these farmers – who have done as you have told them – that there is a possibility that under certain conditions, under which many farmers are still working, OPs can actually cause damage, and that they should be using recommended respirators.

Roger Cook — I think the important thing to recognise is that this advice that has been given over the years is collective advice; it is not the advice of one individual or one company. This is advice which the various safety authorities, the safety experts, have passed to the farming community in the form of advice on the labels. The question of whether you change advice is a 'no win' situation. If you do not change it, you get accused of not listening; if you do change it, you are accused of being at fault in the past. All we can do is update advice as information becomes available, and that is what has been happening over many years.

Chairman — Can I seek clarification from someone in the audience who might know this? We have got the question of whether the right protective clothing is available and the right advice is being given. Then there is a separate question of whether people are following the advice they are already being given. There seem to be contradictory views here. Can anyone offer guidance on this particular point?

Brenda Sutcliffe, *Lancashire* — In my family there are six of us – the complete family were very badly affected in 1992. We had blood tests done at Guy's which showed cholinesterase levels ranging between 8.24 and 8.75. The second cholinesterase test done later showed

cholinesterase levels of 25. There were something drastically, dramatically wrong with my family. I *am sorry – I am emotional – most women are* emotional about their families. Mr Cook knows my feelings very well; he has been told often enough. Nobody told us about using face masks.

Chairman — This is obviously a very emotional issue, understandably, and no-one is going to condemn you for that. What I was trying to find out was a fairly basic factual question – the extent to which anyone knows whether farmers by and large do follow the instructions they are given.

Brenda Sutcliffe — We did.

Chairman — Anyone else like to comment on that?

David Henderson — I worked for 19 years in the animal health industry. I am no longer employed by any of the pharmaceutical companies. The recommendations on protective clothing have changed very little over the last 20 years. It is quite typical of Liz Sigmund to try and shout down people and to tell half-truths. That is no way to conduct this debate. She has had her say – perhaps some of the rest of us can. The guidelines on protective clothing have changed very little over the last 20 years. The labels have been provided to people like Liz Sigmund – she has seen them. Anyone else can see them.

It came out in this latest study, which is seriously flawed, that many farmers do not wear protective clothing. It has come out in previous reports done by the Institute of Occupational Medicine for the Health and Safety Executive that a large number of farmers do not use the recommended protective clothing.

Dr Anne Spurgeon — I think you are referring to our report. Bearing in mind that it is approximately two years ago now that we asked these questions, we did find that that in our particular survey of 146 sheep farmers the vast majority wore waterproof footwear, and I think it was about a third wore waterproof trousers – but apart from that, they did not wear protective clothing. That is not to say that I am on the side of blaming the farmers. I am just stating what we found, and this was based on asking farmers what they usually wore when they were dipping. So it was their own report of what they wore.

Chairman — This would not be unusual in occupational health – to find a group of people who are given instructions which they then do not follow.

Vera Chaney, *Safe Alliance* — I know a number of farmers who find it is impossible to work in hot weather wearing the protective clothing that is advised. What sort of community are we in putting on the market the most dangerous chemical and then telling people that if they use it they *have got to dress up in a capacity that would make* them ill. You might as well say to them it is safe if you use it standing on your head. Lots of farmers will not report the health problems they have because of this issue. Even small farmers that work as a co-operative tell me that, if they are dipping 800–1,000 sheep a day in hot weather, it is absolutely impossible to wear the protective clothing and work at the speed that they have to work. Surely, this is one of the basic problems that we are all facing. The chemical companies are hiding behind their instructions that protective clothing has to be used and the poor farmers find that they cannot use that protective clothing and are suffering ill-health in the process. Surely this is the issue we should all be looking at. Take it off the market if it cannot be used without protective clothing (*applause*).

Chairman — Can I follow through that issue of whether protective clothing would be practicable or not? We have someone in the audience who has been with the RAF – Dr Laurence Leeming-Latham – who has some experience of the kind of protection against chemicals that the military use. Would you care to comment on that – this whole question of whether it is actually feasible to protect yourself against chemicals like this and do a job as physical as sheep dipping?

Dr Laurence Leeming, *Latham* — Yes of course it is feasible – but often it is simply not practical for reasons which have been clearly stated by others here this afternoon.

Chairman — Let us put it back to Mr Cook, because we obviously have a problem here. If farmers are not wearing protective clothing, it may be because it is just not practicable to do so. How do you respond to that on behalf of the industry – because you can see their dilemma?

Roger Cook — I can indeed, but I have also talked to agricultural contractors who dip far larger numbers of sheep than an individual farmer does, and they tell me that they do find it possible to wear the protective clothing on a regular basis. I also know from company staff, who themselves wear the protective clothing when they dip large numbers of sheep in trials, that they are able to do it. But I think the point one has to make, and this is fundamental to the whole business of health and safety law, is that if you find you are not able to wear the necessary protective equipment for any industrial operation, then you should not be carrying out that operation – you should be looking for an alternative way of doing it. Today there are many different products on the market.

Unnamed speaker from floor — There weren't, though, were there? Until 1992 there was nothing

but OPs on the market. We certainly didn't have a choice when we were forced to use them to dip.

Roger Cook — I am sorry, that is not correct. It is a fact that there have been pyrethroid dips available for a much longer period than that.

Alan Dalton, *TGWU* — My trade union, which represents agricultural workers, is very much concerned that we get a ban on these products immediately. Protective clothing is a last resort. We should not be talking about protective clothing. Dr Bernhardt, who I think has made a very important contribution, said he would not know how to advise people. That was a very frightening statement from a GP – but an honest statement. We support a ban totally. I was very unimpressed with the HSE support for the research they published recently. They tried to downgrade that support at a press conference two weeks ago. We will be supporting full compensation claims for our members. This is a battle in the war against pesticides.

Chairman — Before I take the next comment from the audience, let's just get a quick response from the representatives of both the BMA and NFU on the platform about whether one should simply ban these things and then cope with the problems as they come up. Ian Gardiner, what about that from the point of view of the NFU?

Ian Gardiner — We are talking about a set of compounds which are very useful in sheep farmers' lives – they are already licensed .There have been concerns raised by people around this room – concerns which we share – and we will want to come back at the end of this conference to see where we go from there. But I do have to remind people that the NFU has a lot of sheep farming members who have used OPs for many years and do not have health problems. It is a silent majority who are not represented noisily in this room.

Chairman — Would that silent majority also resist any attempt to ban them, do you think?

Ian Gardiner — I think they would be concerned about the effects on their businesses. Can I just say that it is not my position to determine whether things should be banned or not; that should be done by the Government's advisory committees of independent experts working on the best science. I really think it is improper to ask an association of farmers whether they want a chemical banned or not. We do not have the expertise – we are not medical or scientific experts. Our job is to ensure, if a product is licensed subject to conditions, that our members know and understand those conditions, and we use our best endeavours to make certain that they protect themselves and of course protect their workers.

Chairman — Well, let's see what Fleur Fisher has to say, because the BMA does campaign on all sorts of things.

Dr Fleur Fisher — Yes, we do, particularly on subjects which affect individual patients' health and the health of the public. I think there is a very interesting parallel here. In medical terms, we talk about informed consent – that is, patients consent to treatment about which they know the risks and the benefits in their particular case. It is our job as doctors, supported by research workers, to explain what are the risks and what are the benefits of a particular course of treatment. The patients then have to make up their minds. We do not always do that as well as we should, but that is certainly what we should be doing in every case. If we take the parallel here, that would mean to say that farmers would be faced with what the benefits are of using OP sheep dips, because they are very effective for a troublesome parasite, and that has big economic effects – but farmers also need to know the risks as far as we know them.

Chairman — Clearly – and it is not surprising – there is a strong swell of opinion in the hall towards banning these things. Are there any sheep farmers here who actually would not wish to see these things banned? Would they dare speak?

John Thorley, *National Sheep Association* — I am not a sheep farmer, but I do represent the National Sheep Association, which, like the NFU, has been extremely concerned about the OP debate for a great number of years. We had our own closed conference on this subject in 1989. One of the worries we have is that if OPs are removed the armoury that we have left is extremely limited. It is a serious dilemma. The other side of it is that the majority of sheep farmers do not recognise the problem.

Chairman — In the audience we have Lady Mar, who is a sheep farmer. You have suffered as a consequence of these things and you now want them banned, don't you – so how do you respond to sheep farmers who say, no – we need these things.

Lady Mar — I have never actually asked for a ban. I have asked that they be proved to be absolutely safe before we use them again – so there is a subtle difference. Can we go back to this business of protective clothing and just a little bit of the history of sheep dip?

Organophosphate sheep dips were brought in because they were supposed to be safer than organochlorines, so the farmer was misled into thinking these products are safer than the ones he had been using previously. Initially we were told to wear rubber boots, rubber apron or leggings, and rubber gloves only when using the concentrate – there was nothing about face protection,

nothing about wearing a waterproof jacket, nothing about wearing waterproof headgear.They then discovered that phenol, which is one of the constituents of sheep dip, rotted rubber. The gloves were rotting on their hands. Their wellington boots were rotting. When I looked at the rubber apron that I had been wearing, you could see straight through it – it had rotted the fibres. It was not until about 1992 that the recommendations were changed – that we should wear these vinyl things.

We have had a discussion about the practicality of wearing the clothing. I know when I am gardening for half an hour in rubber gloves that my hands get sopping wet. To expect someone to handle sheep, particularly when they have been recently shorn, so they have not got a lot of wool to grab hold of, is impossible. You get frustrated and you throw the gloves off. In fact, we have not dipped sheep since 1992 – we now use the pour-on products.

In the period between 1985 and 1992, when dipping was compulsory, there were something like 17 OP dips which were MAFF recommended, and two pyrethroid dips. The pyrethroid dips were about twice the price of the OPs. Sheep farmers are extremely conscious of costs. There is not a lot of profit to be made on a lamb, and they were not going to spend twice the amount on their dip if they did not feel that it was absolutely necessary. At that stage, they were not aware of the dangers of OPs. There was nothing on the container which said that this is a dangerous product. The disinfectant came with a hazard warning symbol on it, but there was nothing on the sheep dip container itself until last year, when MAFF – or the manufacturers – were finally persuaded to put an orange skull and crossbones on it.

So we have a pattern of the farmer being deluded into thinking these products are safe, going back to the 1960s, and today we discover that these things are not safe.

Chairman — Before we get Mr Cook to comment on that, can I ask you one further question. The effect of what you are saying is that these things are jolly good, they are doing what they are supposed to do, but they are pretty bad for the humans who use them and it is not practicable to use them safely. What are you suggesting, then? You say you do not want to ban them – but you would require further evidence which presumably means withdrawing them in the meantime?

Lady Mar — If someone can develop a means of dipping sheep and handling sheep afterwards. Remember that sheep dip remains in the fleece for several weeks afterwards; this is another of the advantages of sheep dip. I have been affected by sheep that have been dipped that were 30 yards away from me.

Chairman — What do you think should be done right now?

Lady Mar — Right now we have got to withdraw them for the moment. We have got to have a moratorium on it.

Chairman — So dipping would stop using these materials?

Lady Mar — Yes, there are alternatives.

Chairman — An awful lot of sheep farmers are not going to accept that, are they?

Lady Mar — It is up to them – it depends upon how much they value their health. The gentleman sitting on the end here says - "I've been dipping sheep for years". Perhaps he would be one of Anne Spurgeon's test cases – he has no symptoms at the moment. Maybe he would get them later on – you simply do not know.

Chairman — Roger Cook – too unsafe to be used under any circumstances?

Roger Cook — I think one of the things that has come out of Lady Mar's and other people's comments is the importance of information, the importance of choice. It ought to be clear now to anybody who has picked up a farming magazine in the past two or three years that there is a debate about this. I think there is a considerable awareness now among the farming community of concerns about OPs and the need to take them seriously. With that in mind, it is interesting that a very large proportion of the sheep farming population continue to buy OPs, even though there are an increasing range of alternatives available. So certainly there is a large proportion of the sheep farming population that wishes to continue to use them and is making that choice in the light of all the information .

Robin Maynard, *Soil Association* — I work for the Soil Association. Our farmers do not use organophosphate sheep dips. They manage perfectly well by good husbandry, operating closed flocks and acting for curative rather than prophylactic or preventive reasons. They will use the alternatives - they will use the flumethrines – but, as they say to me, these are jolly expensive and how can you expect the majority of farmers to pay twice the price to make that shift? If they are using Vetracin it is costing them about 50p a sheep. So if Roger Cook is actually wanting to win back the hearts and minds of his market and reassure the public, why doesn't his industry voluntarily at least give farmers a real choice by saying all these alternatives will be no more expensive than the OP dips. Then they will make a choice – and I bet you'll have a lot of OP dips left on the shelf.

Roger Cook — There are two practical problems there. First of all, the modern products are a lot more expensive to make. But the other point is that when you are comparing costs, you have to look at the total cost of using some of the alternatives which do not involve the dipping process. You have to look at the total cost of the operation, not just the cost of the can you buy.

Chairman — What about a Government subsidy to correct the price difference?

Roger Cook — I think you had better ask the Government about that.

Chairman — We would if they were here, but they are not.

Jane Rees, *Glamorgan* — My husband suffers from organophosphate poisoning and so does my daughter, who is only 20 years of age. I would like to ask Mr Cook one question: Could you tell us why, then, compulsory dipping was stopped? Why do we no longer have to dip and why will it not be reintroduced? If everything is so safe, then why is there any need? Why will the Government not tell us – you have to dip? Or is it that they are frightened that, by making us do it, we will sue them?

Roger Cook — I think, again, you had better ask the Government there, but I believe their decision was based on a lot of aspects rather than the simple question of dips themselves.

Chairman — Yes, it is unfair to hold Mr Cook responsible for the motives of the Government which have been open to many different interpretations.

Jane Rees — I watched you on a programme on Sunday where you indicated that it was the Government that put the instructions on the sheep dipping cans. That is what you said, Mr Cook. You said that all the information was given by the Government to tell us how to use these dips. May I further go on to say that I remember when we bought sheep dip there were gloves included for the job and they were no more than washing-up gloves. Everyone seems to have forgotten all these points, but we were there; we worked, we did the job. We are now very, very ill. To Dr Spurgeon I would like to say that from her report it would appear that farmers who had said they were not affected *were* affected – so how on earth does anybody in this country know how many sheep farmers are affected, because they all think they have not been affected?

Chairman — Let us get a comment from Anne Spurgeon, because it is one interpretation, isn't it, of your findings, that the problem is far more widespread, simply because no-one has actually looked for the subtle effects of it.

Dr Anne Spurgeon — Certainly we are not denying that we found effects – we found effects in asymptomatic farmers, yes. They were not farmers who were to all intents and purposes ill. I am not denying that we found effects.

Dr Erick Millstone, *Sussex University* — I would like to ask a question of Anne Spurgeon as to precisely how many farmers were excluded who had reported that they were feeling the kinds of effects that you were looking. I am interested also in asking also a question of Ian Gardiner, because something rather puzzles me. When he was saying the NFU will not take responsibility, he characterises the Government's expert advisory committees as being independent – but he did not make it clear of whom they are supposedly independent. Members of the Advisory Committee on Pesticides and other similar expert advisory committees are, surely, Mr Gardiner, allowed to act as paid consultants to the pesticides industry, and many of them do so. Therefore the question is how can any reliance be placed upon the judgements of such committees unless and until they are staffed purely with people who either are not allowed to or who refuse to act as paid consultants to the pesticides industry, until all the safety evidence is in the public domain, and until the public are also able to judge the criteria by which that safety data is evaluated?

Chairman — We will get Anne Spurgeon's comments first of all in response to the question of the people who are excluded in her study.

Anne Spurgeon — In a sense, the answer to your question is none. We only excluded people who had pre-existing nervous system disease. People might like to argue that those pre-existing diseases might have been initially caused by OPs but that is what we decided to do. (*question from floor* - how many were there?). I do not know the exact figures but something like half a dozen, suffering from different diseases.

Chairman — Let's hear from Ian Gardiner now about this question of independence.

Ian Gardiner — First of all, the one place where the experts who make these decisions are independent from is the Government, which is an important point. Eventually, society has to decide who it is going to trust to take decisions. You can leave it to Ministers, who will know very little about the subject but can make the decision on democratic grounds, or you can have it deputed to people who understand the subject – and it is a fact of life that, in practice, some of the people who understand the subject will be involved in working for commercial interests in that field. Others

OPEN PANEL DISCUSSION
...Continued

will not be. It is a question whether, at the end of the day, you trust those people to give proper advice or you do not. All I can say is that the NFU as an organisation does trust those people to give proper judgements.

Dr Fleur Fisher — I think one of the issues here is that the research and the evidence on which these decisions are made does need to be in the public domain. That seems to me to be very important.

Vivian Howard, *Liverpool University* — We do quite a lot of testing on minimal damage in the nervous system, which is starting to be used in the pharmaceutical industry now. Lady Mar asked whether these things could be withdrawn until there was absolute proof of their damage. I am afraid I do not think that absolute proof is going to be available. Nobody denies that these organophosphorus substances are toxic – in fact they are designed to be toxic to do their job. Now, health risk assessment, on which licensing is given, assumes some of the following:-

- That we know the most sensitive end-points for toxicity and that we have the tools to be able to detect them
- That we know what the current levels of exposure are in the places where those most sensitive end-points will be
- That we know about synogistic effects with other pollutants or chemicals that are around at the time
- That we know the pharmacogenetics of it – ie, that we know that some people are going to be idiosyncratically hypersensitive to it
- That we can control globally and locally the levels of these chemicals when we use them.

Having listened to this morning's discussion, it seems to me that we do not really know the answers to any of those questions. As scientists and medics, we like to be able to address scientific problems where there will be a definitive answer at the end with some level of scientific proof. I do not think that that is an available option with this set of problems. Dr Proudfoot pointed out this morning that we are not just necessarily looking at this one chemical – we are looking at a whole cocktail of chemicals in which we live. So, therefore, we maybe have to take a weight of evidence approach. This is something we should maybe discuss. At what level of evidence do we say we take action? We cannot have absolute scientific proof – perhaps we should look for something called reverse onus. Perhaps the producer should be made to prove to some agreed level of confidence that his product is not toxic before he is allowed to use it. Once you have a licence to use one of these now, it seems that the complainant has to prove to a pretty high level of proof that it is causing harm before it can be withdrawn.

Chairman — This question of putting the boot on the other foot, how do you fancy the prospects of reverse onus?

Roger Cook — This of course is what the licensing system is about and always has been. When you apply for any animal medicine licence – or, I think, any pesticide licence – it is up to the applicant to provide the data to satisfy the Advisory Committee that the product can be used safely in accordance with the recommendations, and it is on that basis that you get your licence. Although dips have been on the market for a long time, they have been subject to many reviews, particularly in recent years, and a vast amount of new data has been put forward, not just from the industry but from other sources as well. So the burden of proof is already on the licence holder, on the applicant for the licence, to satisfy the committee of their product's ability to be used safely.

Chairman — You are not accepting that, are you?

Vivian Howard — If you talk to people in the pharmaceutical industry, they will agree that they do not have the tests available, although people are trying to develop them, to detect some of the most sensitive end-points of minimal change. I think we are talking about what is represented by minimal changes here; we are looking at measurements which are quite subtle. The acute toxicity we seem to understand quite well, but it is the level at which we can actually detect the effects that is in question. Are we applying the right tests? I suspect we are not, for the standard licensing of these chemicals.

Joanna Wheatley, *Berkshire* — I would like to take up a point on this burden of proof. When we talk about testing, we do not test chemicals on humans; it is against the Geneva Convention. Therefore, these things are licensed without any tests on humans. We know that these organophosphates are specific to humans in their effects. What we need is proper surveillance after licensing. Ironically, in this country the surveillance is carried out by the same people who license the chemicals and it would appear that the surveillance just is not being done properly. This whole system has to be changed. I personally worked, before farming, for a chemical company. We had blood tests every couple of weeks and we were monitored – and when the blood tests were not at the right level for cholinesterase, we were withdrawn from using them. They would wait until the blood levels had returned before we went back to using. If that sort of system had been in place and made mandatory on licensing, we would never be in the situation we are in now. When those first few OPs were first licensed and put out to be used in the general community, the farmers who were using them should have been monitored, and their GPs would then have been aware of these chronic

effects that could come up.

Furthermore, I would like to say to Ian Gardiner, when he says not all farmers are affected, that we did a little survey in our own NFU branch. All the sheep dippers had been affected. None of them had ever been to their GP, but they had all had headaches and what have you. We look around in our little area and there are masses of farmers and farmers' wives who have gone with ME symptoms, who have had all the testing for MS, and then gone back out. They have recovered, yes, but it should have been picked up because they should not go back and use these chemicals again.

One last point – we do not have a department at NFU headquarters for farmers' health. We have got an animal welfare department, but we have nothing for human welfare or farmer welfare. We are front-line troops – we should be properly surveyed, we should be properly monitored.

Chairman — Sounds like one for you, Ian Gardiner. Should you have a farmers' health department?

Ian Gardiner — We do have a staff member who deals with health and safety on the farms and relates to the Health and Safety Executive on all fronts, so I do not accept the premise on that. But of course I hope that everybody who works for the NFU is conscious of the need to protect the welfare of farmers, in all senses – not merely the economic one, with which we are normally associated, but all functions of farm life.

John Armitage, *Somerset* — John Thorley mentioned the National Sheep Association meeting held in London in 1989. There are about half a dozen of us in the room who were at that meeting. There were five farmers there, and we all said there was a problem. There were five dipping contractors, and they all said there wasn't a problem, because they made their livelihoods out of dipping sheep. There were five manufacturers and, of course, they all said there wasn't a problem. There were 12 experts there from various departments, and it is a matter of debate which way they split. But certainly the five of us who were farmers felt afterwards that the experts had split straight down the middle – half of them thinking there was a problem and half of them thinking there was not a problem.

This is the sad thing to my mind, that this meeting could well have taken place six years ago – because most of the people who are here today who are sufferers from OP poisoning were sufferers then. If some people five or six years ago had taken the bull by the horns and said there was a problem with OPs, and perhaps if this sort of conference had been called then, just maybe we would not be in the mess we are in today.

There are many people to blame. Why did the NFU not get off the fence and tell us that there was a problem with these OPs? – it was not for lack of trying from the South West. The South West sent up numerous resolutions to my certain knowledge about OPs and dipping. They got to headquarters, nothing happened. Our Regional Director, Anthony Gibson, well-known for his anti-OP views, all right, but he did take the trouble to conduct a survey and that showed that there were a lot of people who were ill, not seriously ill, but ill. How many anecdotes does it take to make a story, I ask myself. We have been told for years, it is all anecdotal, there is no scientific proof. We have all heard it and, quite honestly, it just does not add up.

Jane Monro, *Brakespear Hospital* — I want to make a comment about the obligation society has to the people who have become ill. They were being forced to use organophosphates compulsorily for dipping sheep right up till 1992 and I think that the Government, the National Health Service and the producers of the pesticides have an obligation to these people. Whether it is proven or not exactly how they were made ill, they are saying that there is an association between usage and their ill-health. These people have got to be funded for treatment. There are treatments available, there is in fact a book by Nicholas Ashford and Claudia Miller called *Chemical exposures, low levels, high stakes*. Nicholas Ashford is Professor of Toxicology at the Massachusetts Institute of Technology. There is a lot of information in that book about how people can be treated and I do feel that, if this forum can take back a message to the powers that be, it should be that. Society should take care of those whom it has obliged to become ill on its behalf.

Roger Cook — Can I correct just one point. I regret to differ but, in fact, it is not correct to say that farmers were forced to use OPs up until 1992. It is true that dipping was compulsory but the availability of other products was there throughout the compulsory dipping era. (shouts of "No!" and "at twice the price").

Chairman — Twice the price – yes, I think we rehearsed that argument earlier on.

Roger Cook — The fact of the thing is that they were not forced to use OPs.

Ray Bryant, *Somerset* — I am one of the affected farmers and I am a member of the NFU. For six years I have been trying to get to the bottom of this. We had used these dips for several years. In the autumn of 1989 my wife became ill whilst ducking the sheep. I spoke to the manufacturers for advice because we were dipping on another farm in a few weeks' time. They assured me that it must have been a virus – there was nothing wrong. I took their advice – we carried on and then, two weeks after that, I handled sheep through the market after 36 hours of wet weather and became

very, very ill. I thought it was the end of the road. I now know I was not given the proper advice.

As an NFU member, my main aim has been to try and highlight this problem to at least alleviate any more people going through what we have gone through. I am glad that the NFU have actually got off their backsides and got this meeting today but I wish they had listened before.

I have nothing against dipping sheep. The problem of sheep scab has got to be looked after. We had always dipped our sheep every year, every summer, irrespective of whether there was a compulsory dip or no. We had not got a scab problem. We used the products we were told to use, we have become ill, it has ruined our health, it has ruined our business, then they stopped the compulsory dipping. Nobody cares less about the scab and it is actually spreading like wildfire, so where have we gone? We have actually ruined our health doing a job we were forced to do for something we hadn't got and now we have got it.*(laughter and applause).*

Chairman — Ian Gardiner, do you want to comment on the NFU's tardiness – or lack of it – in taking this problem on board?

Ian Gardiner — No, I think it has already been said that whenever you change something you can always be accused of not having done it before. The important thing is that we are looking at this and we intend to take the results of this seriously.

Chairman — Anything you want to add to that, Roger Cook?

Roger Cook — The manufacturers and the sheep farming industry as a whole were very concerned when compulsory dipping came to an end – not because the compulsory dipping itself disappeared, but because of the removal of the notifiable status of scab. This is something that we all regret and the sooner it can be changed the better. It is interesting that in Northern Ireland notifiable status has been maintained. That does mean that the authorities working with the sheep farming community there can very easily get to grips on the problem and do have the legal resources to do something about it.

Dr Keith Eaton, *British Society of Allergy and Environmental Medicine* — I think we need to broaden the discussion a little bit. One point that seems to be so basic that perhaps we are in danger of overlooking it – for pesticides to work, they must be toxic. There is the question of the wider effects of pesticides. We have a considerable biodiversity in the human population, which means that some are more subject than others to adverse effects .We should not, I think, base our presumptions on the best case scenario. Pesticides have been measured in animals and in humans – they actually do get into the tissues. Lassiter in 1983 showed positive levels, including levels for pesticides that had not been on the market for over a decade, and this is in human tissues. We can also measure enzyme systems that are affected by OP exposure. The point is that measurements can be done.

Enfys Chapman, *Pesticide Exposure Group of Sufferers* — We have been interested in the problems that sheep dip sufferers have been having since 1989, when it broke in *Farmers' Weekly*. We ourselves were set up in 1988. The NFU has been referring people to me for counselling for quite some time now and, in the sample of people that I now speak to, there seems to be a high proportion of young farmers who have been sensitised possibly by the use of warble fly dressings and sheep dip. They are now suffering from other problems like cancer, aplastic anaemia and other things which are much more difficult to deal with. They are young, they have been sensitised in the past, and now they are considerably at risk.

Chairman — I'll take one more question and then we had better move into the final session of today.

Martin Burtt, *Chairman of the NFU Livestock Committee* — I am a sheep keeper and a user of OP dips and have done so for as long as OP dips have been available and, touch wood, have suffered no ill-effects. What I am concerned about is, of course, the sheep. They have had very little say in this matter today. The welfare of sheep must be nearly as important as what happens to the people who are operating and dipping the sheep themselves. I hear the people that have got a problem with OPs and I am tremendously sympathetic to them. But what I have to say to you is that, unless and until an alternative to OP dips is available, I shall continue to use and ask for OP dips. I feel perfectly within my rights. We wish an alternative would come along and I would urge Mr Cook and his organisation to come along hurriedly with an alternative dip to OPs. But in the absence of that farmers will, as he has said, continue to buy OP dips, because we have the sheep's best interests at heart as well. Anyone who has had scab in his sheep will know that it is a horrendous disease. They suffer tremendously from this. We must not forget that that is a very important aspect.

Chairman — With that plea to Mr Cook hanging in the air, we must wind up this session. Let me on your behalf thank the four members of the panel for taking part and doing their best to answer your questions.

CLOSING PLENARY

Geoff Watts

Presenter
'Medicine Now'
BBC Radio 4

WE ARE NOW into the final session of today, in which it is my happy duty to offer some sort of overview of what has being going on, and then we will get the closing remarks from two of the speakers whom you have heard today.

I think to summarise properly what has gone on today would be impossible in the sense of producing a neat, straightforward account – the feeling of the meeting was this, that or the other. It is not that kind of meeting and I did not imagine that it would produce that kind of result.

Last night I jotted down what seemed to me to be the kind of issues and the questions that would raise themselves today and I have been curious to see how far answers would be provided to those questions. Needless to say, I suppose, that in many cases the answers simply have not come up and, where they have, they are contentious and disputed.

The first thing I asked myself was – do organophosphates cause long-term damage to human health? The answer would appear to be – yes, if the dose is large enough to cause acute symptoms of poisoning. If the dose is going to cause acute symptoms, the chances are that there will be something lingering on. The problem begins when you start talking about doses below those required to cause acute poisoning. Do organophosphates cause damage under those circumstances? We have the study from the Institute of Occupational Health – Anne Spurgeon's study – which is coming out with something which is actually quite difficult to dismiss. The effects are fairly subtle – nonetheless, subtle effects can be important, if they involve behaviour and neurological function, as indeed they seem to do.

We have heard a great deal on the medicine of the issue today, but the difficulty is the lack of agreement about the problem itself. It is not as if we have a situation where we can say–'Look, this is a problem; we can agree about the existence of the problem; now, what do we do about it?' The problem is that there is disagreement even about the problem itself. The most extreme is whether the problem even exists at all. Under those circumstances, it is actually very difficult to get beyond first base and I think we have seen that today. Constantly we have found that we are coming back to this question. Is there a problem?– not, are people imagining it, but is the problem the one they perceive it to be? I thought this was captured rather neatly in the two presentations we had from two of the speakers this morning – Dr Davies and Dr Proudfoot. Dr Davies was arguing that sometimes problems can only be seen by those whose eyes are prepared to look for them. He argued that unless you actually go out and make the effort to look for the possible damage, the possible side-effects of these things in the long term, you simply will not see them – and this does not mean they are trivial. Subtle problems are not necessarily trivial problems.

The other side of that, of course, is that patient and doctor together can then work themselves into a spiral of disagreement, whereby the patient becomes ever more convinced that he or she understands the source of whatever problem they have had and the doctor becomes ever more convinced that the patient is wrong – and you get a cycle of mutual antagonism. Now, this is not unique to this field at all. I come across this all the time, as a medical journalist.

I was heartened by – I think it was Dr Proudfoot – who, even while expressing a certain scepticism, was emphasising that doctors must take the patient seriously. If farmers are coming to doctors and saying that they have a problem, the last thing that the doctors should be doing is taking a cavalier attitude towards them. I am not saying that does happen; I dare say it happens in some cases.

Going back to that list of questions that I was talking about – can the effects of organophosphates be avoided by the use of protective clothing? We have heard a lot of disagreement about that. The consensus in this room would appear to be No. Likewise, the view expressed by a number of people would appear to be that the kind of protective clothing which might offer you total protection against the effects of these things is virtually impracticable – and it is not much use having an agent which can only be used with protective clothing which renders the job itself almost impossible.

Would animal welfare suffer if these agents were abandoned? From what I hear today, yes – the answer is it would. By what amount and to what degree I really have no idea and, from what I gather, there is disagreement among farmers themselves about the extent to which this would be a problem.

Would sheep farmers suffer economically if these agents were abandoned? Yes, I presume that they would. If you assume that there is a serious need for these things, then they are going to suffer economically and, as I understand it, some sheep farmers are very much at the margins anyway

economically and something like this might just serve to push them over the edge.

We always hope, don't we, for a magic bullet which is suddenly going to solve all our problems. Today we have the drug or agent which does what we want but at unacceptable price – tomorrow, perhaps, the new drug or agent which does what we want without our having to pay the price. It could happen, but the chances of its happening seem fairly remote in the near future. And when alternatives to these things do come along, they they may be as effective – they may be more effective – but it is almost certain that they will have side-effects and problems of some kind themselves. So to rely wholly on the industry to develop something which is going to solve all our problems is, I think, pretty slim.

We seem to have a series of options of things that we can actually do here and now. None of them are very satisfactory. Obviously, one of these options is more use of protective clothing. We have heard the problems about that. More effective protective clothing – likewise, the same problems. You can dress yourself up in a Noddy suit of the kind that you use for nuclear and biological warfare, but it is not practicable to go dipping sheep under those circumstances.

An outright ban on organophosphates themselves? I suspect if one took a poll in this room there would probably be a healthy majority in favour. But there is no point in doing that, because the sort of people who are most likely to attend a meeting like this are probably not representative of the entire community. It is natural enough that people with strong feelings about these things attend a meeting like this. They are the people who are most likely to say – 'I don't care about the practical problems; these things have damaged my health, they must be banned.' So, although the mood in this room might favour an outright ban on organophosphates I rather doubt that the farming community as a whole would go for that option.

Another possibility, of course, would be to try to take account of the practical consequences of a ban. You could say, right, we'll have a ban on these agents but with some form of compensation for the consequent effects on the income of sheep farmers. I know nothing of the economics of this, I have no idea how such a thing would be viewed by the Government – dimly, I should think – or whether it would be practicable. But anything can be done if there is a will to do it. There is another possibility – we just carry on as we are; we muddle through. I do not think that is terribly satisfactory, and I am sure no-one in this room does.

Another possibility – carry on with organophosphates, but with some kind of no-fault compensation for those who do suffer. There are plenty of precedents for this kind of thing. Essentially, you are agreeing to pay people who have taken risks and suffered. That would be one basis for the continuing use of organophosphates, provided that the farmers concerned had the freedom of choice. They would be free to go on using them, knowing that there was a risk and knowing that they would be compensated at the end of it.

The final one is always, of course, more research. So often this phrase comes up and in the end there is an awful banal truth about it. Yes, of course you need more research and there is just the possibility that increased effort to develop alternatives will come up with something – but, even if it does, it will not be a magic bullet and it is probably going to be a medium-term solution.

So policy-makers face unenviable choices, because policy-makers very seldom are able to make black or white decisions – they are always choosing in shades of grey. They are always making compromises. One thing we can be certain of is that, short of these things suddenly disappearing overnight because something has come in to replace them, whatever is done is going to be a compromise. I would hope that you would be able, all of you – both in the industry and in the farming community – to see some way by which you can forge some kind of compromise between you in whatever policy is finally decided. The more openness we can have about making that policy, the better. Openness in Governmental circles is not something we are known for in this country, which is regrettable. The more that organisations like the NFU and the BMA can themselves bring these things out in the public, discuss them, encourage openness about them, the more likely we are – I suspect – to be able to reach compromises which policy-makers can then put into effect without causing undue offence or undue pain or distress to more people than is absolutely necessary.

One other hobbyhorse of mine – the fourth R. Reading, "Riting", "Rithmetic" and Risk-Assessment. There is an abysmal lack of knowledge among the vast majority of people in this country – even including, I suppose, some scientists – about the nature of risk. We live in a world in which there is an increasing number of things which cause us risk, and if we are going to make sensible decisions we need to have some notion of what it is to say that this is riskier than that.

Finally, if you ask me what today has achieved, I have not the foggiest idea. We probably will not know for months, we may not know for a couple of years – we may not know at all. But I think that a meeting like this has to be put on in a kind of spirit of faith that there is only one thing worse than arguing about these kind of problems and that is not even to mention them. At least this meeting today has made it clear that these things are on the agenda, and they must be discussed.

CLOSING PLENARY

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Dr Fleur Fisher

Head of Ethics, Science & Information, BMA

IT HAS BEEN a pleasure to be here today. I have learned an enormous amount. First of all, talking about risk, the BMA has recognised that understanding of risk is absolutely crucial to people in making decisions about their everyday health and about their everyday behaviour. We produced a book called *Living with Risk* which won the Copus Prize – a prize for the public understanding of science. We have turned it now into a game which is available at schools, so kids in secondary schools can use it and begin to understand.

There have been some positive developments since we published our report on *Pesticide chemicals and health*, but there really are a lot of problems remaining. It seems to us in the BMA that a comprehensive approach to the control of all agrochemicals is needed.

I think one of the other things that has come out today is that our understanding of the functioning of the brain and the nervous system is a bit like those old-fashioned mediaeval maps where bits they did not know about were called *Terra Incognita*. We know something about it, but it seems to me that there is an enormous amount we still do not know. Even that which is known by specialists like Dr Jamal is not generally understood and put into practice by those of us who are generalists – in general practice or in other parts of the profession.

I think today's conference has revealed lots of reasons to be concerned about OPs and health. It is not absolutely clear what we need to do to prevent or minimise harm to the farm worker or the local community or the environment in general. First of all, post-marketing surveillance is extremely important. We need to be clearer on what we should be doing about that.

What I have also learned is that the generality of people who are using OP sheep dips do not understand the potential for risk and are unable to make genuine risk assessment.

We need to make sure that, in the current world, with lots of toxins in the environment, understanding and appreciation of that as an aspect of medicine needs to be fed in in some way into the already heavily over-loaded undergraduate medical curriculum. We certainly need to make general practitioners and all doctors much more aware of this aspect of medicine.

There is a lot that we do not know about the functioning of the nervous system, but a lot that we do we do not share, and I think the job of the medical profession is to press for the information that we do have to be made more easily available to the working doctor

CLOSING PLENARY

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Tony Pexton

Deputy President NFU

WE HAVE HAD what I can only describe as a hugely successful day today, and that shows the value of our working on this subject with the BMA. Speaking for the NFU, I can assure you that we are determined to maintain the momentum created by this meeting. I anticipate that this will be the first in a series of many meetings between the NFU and the BMA to consider this whole issue.

If anything has come out of today, it is that there is a lot of work to be done.

We see that there is a great deal of research that is needed – both into the effects of OPs and, of course, into effective alternatives. I believe that a national database to collate all the information that there is about OPs and their symptoms, and any possible links with human health, would be hugely helpful. I believe there is a need to consider a simple reporting system and a single centre to report to. Again, it would help to collate the information that is coming forward.

From my point of view as a farmer, I have realised that if I am going to my GP and complaining of something that might be related to OPs, I have got to tell my doctor that I have been using or have been exposed to OPs. It does not help him if he is flying blind.

At the same time, GPs must be made fully aware of all the allegations linking OP dips with human health problems.

This, again, is something that has come out of today – there is perhaps not the level of awareness that there might be among GPs about the effects that some of these substances can have on the individual. The Department of Health and the safety authorities have got to give this issue a very high priority. I know that the BMA is going to be discussing with its appropriate committees and the appropriate Royal Colleges how best to ensure that more information does get to GPs to help identify the problems.

From our side we in the NFU will be pursuing the same objectives through our network of communication, our internal channels, to make sure that farmers are aware of the situation as it is perceived to be and as things develop.

My sincere thanks to you, Chairman, to the speakers and all of you here for what I believe has been a hugely valuable day

BIOGRAPHIES

CHAIRMAN

GEOFF WATTS *Presenter, 'Medicine Now', BBC Radio 4*

Geoff Watts read zoology at King's College, London, and spent a year doing cancer research at St Mary's Hospital Medical School before moving to the Institute of Ophthalmology to work on lasers. Having completed a doctoral thesis he left research for scientific and medical journalism. For many years he was the deputy editor of *World Medicine* magazine. He now spends much of his time broadcasting, and has been the regular presenter of BBC Radio 4's prize-winning *Medicine Now* programme since it began more than 10 years ago.

SPEAKERS

TONY PEXTON *Deputy President, NFU*

Tony Pexton runs two adjacent farms at Watton, near Driffield, East Yorkshire. After study at Harper Adams Agricultural College he spent two years travelling through Australia, New Zealand, the Far East, USA and Canada - working on farms as he went - before returning to the family farming business in 1967. He was awarded a Nuffield Scholarship in 1967 to study sheep farming in New Zealand and in 1987 visited the USA to study cereal production and state support for arable farming. He became a council member of the NFU in 1981 and served as chairman of the Cereals Committee for five years. He was elected Vice President in 1992 and became a Deputy President earlier this year.

DR FLEUR FISHER *Head of Ethics, Science & Information, BMA*

As head of her division at the BMA Dr Fisher has responsibility for scientific and ethical affairs, clinical audit, career progress of doctors, information services and the BMA Library. She is also Director of the BMA Foundation for AIDS. Before joining the BMA as a member of staff, she was a long-standing member of the BMA Council, the Committee for Public Health and Community Health Doctors, and was the public health representative on the General Medical Services Committee. She joined the BMA secretariat in 1991 after six years in NHS general management - running the Community/Mental Health Unit in Macclesfield, with responsibility for psychiatric, mental handicap, rehabilitation, paediatric and community services.

DR TIMOTHY MARRS *Snr Medical Officer, Dept of Health*

Dr Marrs is in charge of the Pesticides and Veterinary Drugs team, Dept of Health Advisor on Advisory Committee on Pesticides, Pesticides Incidents Appraisal Panel, and Working Party on Pesticide Residues, 1990. Past posts held are Senior Registrar, Dept of Experimental Chemical Pathology, Westminster Children's Hospital/Kingston Hospital, Surrey; Senior Lecturer/Consultant, Westminster Medical School, in charge of clinical chemistry and toxicology laboratories at St Stephen's Hospital Chelsea; Medical officer (Research) Ministry of Defence, Second in charge of toxicology and pathology MOD, and Senior Medical officer (Research) MOD. Dr Marrs has also written for several publications on the subject of poisons.

PETER BEAUMONT *Director, The Pesticides Trust*

Peter Beaumont has been Director of The Pesticides Trust, a charitable public interest group concerned with the health and environmental implications of pesticides, since its inception seven years ago. As a qualified solicitor he formerly had his own practice in London for 10 years. He is a member of the MAFF Working Party on Pesticide Residues, and the author of *Pesticides, Policies and People: A Guide to the Issues* as well as numerous papers on pesticides.

DR GORAN JAMAL *Consultant Clinical Neurophysiologist, Southern General Hospital, Glasgow*

Dr Jamal has been NHS Consultant at the Institute of Neurological Sciences (INS) and honorary senior clinical lecturer in neurology at the University of Glasgow since June 1988. He is Director of the Peripheral Nerve and Autonomic Unit, Director of the Video-Telemetry Epilepsy Monitoring Unit at the INS and Director of EEG services to the West of Scotland. He is also a consultant to the Glasgow Royal Infirmary and Western Infirmary. Dr Jamal has a particular interest in neurotoxicology based on the experience of five years as neurologist at the Institute of Neurology in Baghdad and is continuing to work on the effects of organophosphates on the nervous system.

DR D R DAVIES *Consultant Psychiatrist, Rydon House, Taunton*

Dr Davies is Consultant Psychiatrist in Somerset, covering an area from the Quantocks to the Devon border of Exmoor. He was formerly Consultant and Honorary Senior Lecturer in Rehabilitation Psychiatry, Grampian Health Board. He is a member of the British Medical Association and a countryside member of the National Farmers Union. He is temporary adviser to the World Health Organisation on Information Technology applied to Psychiatry 1992.

DR RICHARD BERNHARDT *GP, Kent*

Dr Bernhardt MB ChB is a General Practitioner who has practised in the Weald of Kent for the past three years.

DR ALEX PROUDFOOT *Head of the Edinburgh Centre of the National Poisons Information Service*

Dr Proudfoot is Consultant Physician with the Royal Infirmary of Edinburgh NHS Trust and Director of the Edinburgh Centre of the National Poisons Information Service and has had particular experience of the management of poisoned patients throughout his professional lifetime. He has contributed to the literature on pesticide poisoning, is a contributor on toxicology to several major textbooks and is the author of *Acute Poisoning, Diagnosis and Management*. He is an independent member of the Advisory Committee on Pesticides and current chairman of the sub committee on Pesticides.

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A Seminar for Farmers, Medical Practitioners and Policy Makers
Friday 2 June 1995

NOTES