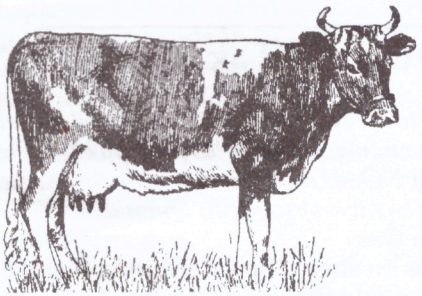
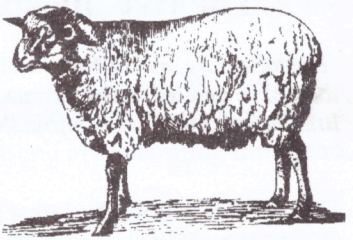
CAUSE & EFFECT





The Search for Truth

A collection of papers relative to the link between organophosphate poisoning and ill health in farmers, soldiers and the general public.

This booklet contains a small selection of papers collected over many years as part of detailed unofficial study by Brenda Sutcliffe and her contacts around the world.

Brenda, a retired Civil Servant and sheep farmer for over fifty years in the hills of Lancashire, and her family were terribly effected by the use of organophosphate as a sheep dip. Ever since, and during a painfully slow recovery from the effects, Brenda has been one of the most vociferous voices against the use of OPs in the UK.

During this time, Brenda has forged links with many useful allies around the world but has not yet been able to persuade anyone in authority to admit the part that OPs have played in decimating a large (and not fully quantified) number of farmers, soldiers and the British Beef industry; as well as causing untold damage to the public at large.

The aim of this collection is to bring into the public domain some of the papers and information which is on public record as *not existing.* The heads of certain international chemical companies, and indeed heads of government, may not like you to know some of the undeniable facts contained in this booklet. Also included are some of the background facts which have led to some of the disasters of the last twenty years and relevant contacts and links for further study and support.

Brenda's message is one which should be heeded by everyone.
If just one parent is persuaded not to use OP headlice shampoo through reading this then her efforts have not been in vain.

The five questions YOU should ak NOAH:

The following are the five questions to put to the National Office of Animal Health (NOAH), printed in the Farmer's Guardian on July 11th 2003 as part of a letter from Brenda Sutcliffe. You can find the address for NOAH on the links page in this booklet.

Question One

How do we follow the latest instructions on sheep dip by ensuring that the sheep do not drink or inhale any of the dip wash?

Question Two

When parents use Malathion as head lice treatment, are they told to avoid skin contact and that death is usually due to respiratory failure?

Question Three

The manufacturers of Crufomate are resisting publishing the hazard fact sheet. Isn't it the duty of NOAH to do it for them?

Question Four

In view of the fact that the shepherds, the soldiers and the children with head lice are now being subjected to the use of pyrethroids, shouldn't they all be made aware of the symptoms of this form of poisoning?

Question Five

When Ministry vets were advised not to approach an OP dip by closer than 14 feet, wouldn't it have reduced the suicide figures if shepherds had been given the same advice?

CRUFOMATE: ***An Organophosphate used as treatment for Warble Fly.***

There has long been a belief in the farming community that the treatment of animals with chemicals has been the root cause of BSE but to believe something and to prove it are two quite different things. I am now confident that we have proved the theory.

My interest in the organophosphate group of chemicals and the danger they represent, goes back ten years to 1992, when myself and my family were devastated by the compulsory use of organophosphate sheep dips. To learn more I had to make enquiries overseas and I owe a great deal to the E.P.A. Washington who were very generous with their help and information.

I have never seen any difference between what happened to the cattle with BSE and what happened to the poisoned shepherds. When you can't walk between two gates in a field without falling over, you know you have a problem and when you begin to lose the power of speech and reasoning then you become absolutely sure. The sight of afflicted cattle on T.V. filled many of us with dread.

My scientific background is none existent but it seemed a good idea to start with the Health and Safety Data sheets of all the products relating to sheep dip. The information was astounding and explained why the farmers and the shepherds had never had sight of them until 1993. I then began to collect data on the compounds used for treatment of warble fly infestations in cattle; Crufomate was the first . This was followed by Coumaphos, Triclorfon and last but not least, Phosmet.

Researching any subject is difficult and costs time and money but when the subject is a closed book it becomes nigh on impossible. The obstacles become even worse when the people who should be helping are becoming more and more obstructive.

I cite The Veterinary Medicines Directorate, The Health and Safety Executive, Local Environmental Health Offices, The Department of Health, and of course the mouthpiece for the chemical companies, The National Office of Animal Health (NOAH).

The shepherds got together and pooled information and what a wonderful group of people they have turned out to be. Black's Veterinary Dictionary 1973, 10th edition became the focus of much attention. There we discovered the symptoms of Crufomate poisoning if the cattle were overdosed. At the same time we were looking at the Government document APS/1 which stated that if one OP made one ill, then one should never ever use another OP. Pity the cattle couldn't talk at the time. Over a period of time, some cattle must have been subjected to treatment with four different organophosphates. Finding scientific data on all of the compounds used turned out to be a test of patience and frustration for me. Coumaphos and Triclorfon presented no problem. Crufomate was something else again.

The people I tried to get information from turned out to be either unwilling or unable to help. I suspect the former. After seven years of searching we eventually ran around the house shouting "Eureka". The Health Authority New Jersey, U.S.A. placed it on their web site. For the benefit of all I will repeat it here.

SUMMARY

Crufomate can affect you when breathed in and by passing through your skin. Crufomate can damage the testes. Breathing Crufomate can cause rapid fatal organophosphate poisoning with headache, sweating, nausea and vomiting, diarrhoea, loss of coordination and death. Repeated exposure may cause personality changes. High or repeated exposure may damage the nerves.

IS THERE ANYONE OUT THERE WHO IS STILL IN ANY DOUBT ABOUT WHERE BSE ORIGINATED FROM?

The BSE inquiry was a complete waste of tax-payers' money. The manufacturers of Crufomate were Dow Agrosciences Ltd. Their submission to the enquiry stated that they had withdrawn from the animal health business about 1980 and had no records of that part of their business prior to their withdrawal. Was this because they saw everything going pear shaped I ask. They now have a copy of the hazard fact sheet, I sent them a copy, and they should waste no time in putting it in the public domain.

There are a number of interesting factors about organophosphates which are not widely known. They were originally designed as nerve gases and then supposedly modified to be used as insecticide. They are extremely efficient at destroying certain enzymes in the blood of all warm blooded creatures, man being no exception. The first enzyme we investigated was cholinesterase. This facilitates messages getting to and from the brain. The expected norm in man varies between 40 and 80 units per 100 mls of blood. When I was blood tested I had 8.25. The cholinesterase enzyme will try to put itself right over a period of time but never reaches its starting point. It is essential that blood tests and done within 24 hours of exposure. Discovering that the enzyme Paraoxinase is important to the functioning of the heart muscles was interesting to say the least. The destruction of this enzyme leads to Cardiac Arrest which is usually fatal. The most amazing factor of all the scraps of information is that there are any cattle, sheep, or farmers, left alive in the UK after so many years of enforced abuse.

We need to look carefully at all the Government bodies and agencies which have been involved in his gigantic cover-up.

1. **Local Environmental Health Offices** supervised the sheep dipping at all farms. Their officers were no better equipped the most farmers. When we collapsed in 1992, there was a blank refusal to help. We needed to remove spent dip from our yard and were told to do it ourselves. When our local councillor called a meeting of local farmers to discover if there were any other victims in the locality, the authority refused to send a representative. They stated;

IF THESE PEOPLE WERE MADE ILL, IT IS THEIR OWN FAULT, THEY WERE GIVEN GUIDELINES.

2. **The Health and Safety Executive.** They have a great deal to answer for. The Medical Services Document MS 17 is their responsibility. When they came to rewrite this paper the draft paper actually mentions the Suicide Impulse of many victims. The fact that hundreds of shepherds have committed suicide after using OP sheep dips hasn't yet occurred to them. The MS 17 never reached the farmers and the rewrite was completed without the suicide clause. They later went on to threaten campaigners with libel action and at the same time closed down a wool grading factory at Carnforth because they couldn't guarantee the safety of the workers from organophosphates poisoning. In 1991 they will in the process of finalising their own research into the health of farmers to the use of organophosphates. The research was devastating. They were aware then that organophosphates degrade through heat and age to become tetraethyl pyrophosphate. They should have been aware that it took only 45 minutes to destroy 20 head of cattle in Texas. Their reaction to this set of devastating research defies belief. They called a meeting. The HSE the VMD and the National Office of Animal Health all attended on 25.2.1991 and now would you believe **THEY HAVE ALL LOST THE MINUTES OF THAT MEETING. IT HASN'T PASSED MY NOTICE THAT IF THEY HAD TOLD THE TRUTH AT THAT TIME MY FAMILY WOULD HAVE BEEN SAVED A GREAT DEAL OF TRAUMA AND MANY HUNDREDS OF FARMERS WOULD STILL BE ALIVE.**

3. The **Veterinary Medicines Directorate** is the agency which is supposed to investigate all adverse reactions to veterinary products. They have yet to find a confirmed case of organophosphate poisoning among the farmers who have managed to find their way through the maze of forms. Every report which have looked at very blandly states that there is insufficient evidence to support the diagnosis. They even managed to ignore the reports from HSE doctors which support or confirm the diagnosis. Papers appear to be lost or destroyed as do medical records.
Marks out of ten for the VMD = Zero.
Whilst the minutes of the 1991 meeting may have been destroyed, the people who were in attendance are still around. They should be called before the select committee on health at the House of Commons and asked for explanation of the research and their own involvement in suppressing this information.
The Veterinary Products Committee are responsible for the licensing of all these products. They certainly have a laid-back attitude to the user's welfare. There is no communication between themselves and the victims. Letters to any of these quangos are part of a game called Pass the Parcel. The letter Is passed to another Ivory Tower for reply and when you finally get an answer you will have forgotten what the question was in the first place.

The HSE will never implement the 1968 Medicines Act against the chemical companies on the shepherds' behalf. Section 6 relating to substances at work is quite explicit. **The shepherds are entitled to a Police Investigation** - always supposing that Murder by Remote-control is still not legal in Britain today.

Brenda Sutcliffe, April 2003

Submission to BSE Enquiry – April 1998

Name: Brenda Sutcliffe
Status: Sheep Farming background for last fifty years / Retired Civil Servant

IN 1992 myself and my family were diagnosed as suffering from Organophosphate sheep dip poisoning by blood tests carried out at Guys Hospital, London.

The fact that we all nearly died was seen as an irrelevance by Government departments. There was no treatment offered, or indeed and acknowledgment of our predicament. In simple terms, we were to be ignored to enable Government to cover its tracks – after all, they were responsible for licensing all veterinary products. In the face of such stonewalling, I decided to make my own enquiries, and contacted the following establishments for information – I am extremely grateful for their help:

The World Health Organisation
The Environment Protection Agency, Washington, USA
The University of New Jersey, USA The Pesticide Trust
The Royal Society of Chemistry, London
The Hazards Society, London

Plus many private individuals. I am also extremely grateful to Mr Rod Knight (lawyer) of Keith Park & Co. Solicitors (39 Barrow Street, St. Helens, Lancashire) for his support and advice. Mr Knight as copies of all the papers in my Possession.

With the help of the Royal Society of Chemistry, I obtained scientific information on all the OP compounds used as Sheep dips, i.e. Diazinon, Propetamphos, Chlorfenvinphos, Carbophenothion; and via a third party, went on to Crufomate, which he had used as a warble fly dressing. I later explored all the Ops which had been used in the Gulf War.

What I began to find extremely worrying from this collection of, information was that all OPs were cholinesterase inhibitors – the damage caused was cumulative, and research using laboratory animals showed that most were carcinogenic and also responsible for malformations in up to and including the third generation of offspring.

The destruction of the central nervous system, immune system and peripheral nervous system was also apparent with most OP compounds.

My next move was to collect as many published scientific papers as I thought were relevant. Copies of the following are enclosed:

1) *Poisoning on the Farm* by Dr Redhead, published in the Lancet in 1968. This paper explains the importance of the cholinesterase levels in man. It should be borne in mind the Ops were designed to attack the cholinesterase in insects, and will of course have the same effect on cattle.

2) *Effects of Chronic Organophosphate Pesticide Exposure on the Central Nervous System* by Richard J Korsak and Miles M Sato, published in 1977. This paper is important because it exposes the frontal lobe brain damage suffered by exposed victims.

3) *Environmental Trichlorfon and Cluster of Congenital Abnormalities* by Andrew E Czeizel *et al*, published in the Lancet in 1993. This paper is devastating in its content, and the fact that it has been ignored in the UK is unbelievable. The effect on the foetus is beyond words.

There are many other papers in my possession, which are of course available to the Inquiry, but I hope to have painted an adequate picture of then effects of OP poisoning on the insect, human and animal species for the present. I move on...

As mentioned above, I received an enquiry about Crufomate. The information on this compound, plus the sight of BSE-afflicted cattle on television, made me aware of the alarming similarities in both our situation and that of the cattle/. To put it bluntly, the human OP victims were going off their heads and off their legs! The cattle were becoming more obvious than the shepherds (more than 900 shepherds have committed suicide since 1984).

Black's Veterinary Dictionary, 10th Edition (1973) provided quite a lot of answers where it describes the effect on cattle treated with Crufomate for warble fly. The VMD deny knowledge of Crufomate but the British Veterinary Association should still have the results of all the testing done on cattle before they gave their blessing to its usage. I have persistently called for the testing of cholinesterase levels in cattle before and after treatment for warble fly. This is unnecessary – it has been done, and the results should be made available to the Inquiry by the BVA as a matter of urgency. The maternal transmission is explained in the following paper:

Reproduction and Growth of Progeny of Female Mice Mated After Treatment With Crufomate – author M A Khan – Research Station Lethbridge, Alberta, Canada; published in the Journal of Scientific Health in 1981. It is interesting to observe that when the first damning scientific papers on a product are published, the chemical companies react in a somewhat vigorous manner – by withdrawing the offending compound and substituting it with something considerably worse.

I consider that I have proved the link between OPs and BSE. What I could never see was a link between BSE and nvCJD, unless nvCJD was some other form of chemical poisoning. Had these two conditions been the same, there would not have been any OP victims left alive to tell the tale. In any case, the Department of Social Security have been paying out Industrial Injury Benefit to OP victims in industry for decades.

There a very few, if any, recorded deaths from chemical poisoning in the UK, but there are numerous reported deaths from "glue sniffing", "inhalation of lighter fuel", etc. Autopsies on all these deaths would presumably show frontal lobe brain damage, plus failure of the respiratory tract – but I digress. The nearest I have come to identifying nvCJD as a form of chemical poisoning is a version of Pyrethroid poisoning, the symptoms of which are identical to those exhibited by nvCJD victims.

I have requested Mr Rutter of the VMD to forward my findings to SEAC. He has intimated that he has done this, although I have had no response from SEAC. I also passed my findings to Mr Frank Dobson, the Minister of Health, on 23rd February 1998, without receiving any reply to date. I have also sent to the Inquiry, prior to this paper, such scant information on Pyrethroids as is available, in the hope that it may be of assistance to any further victims diagnosed with nvCJD.

Finally, the chemical companies should be invited to submit all research papers to the Inquiry – I am sure they have much more than I do, and some effort should be made immediately to help the thousands suffering shepherds. It is also of extreme importance that every scrap of information relating to Pyrethroid poisoning be made available to the general public as a matter of urgency. The fact that these compounds are being used as a head lice treatment for children is appalling.

Dow AgroSciences

Statement of David Farrant to the BSE Inquiry

[This witness has not been asked to give oral evidence in Phase 1 of the Inquiry]

Dow Agriculture had an Animal Health division between approximately 1960 and 1981 when the group was disbanded. In that period the division did sell a product for warble fly based on the active ingredient Crufomate with a registered name of Ruelene. However we have no records available from the period and our statement in answer to your questions has been drawn up based on one or two people's memory of the period. We cannot therefore guarantee the accuracy of the statement.

Agricultural Chemicals

1. Dow AgroSciences has a range of insecticides, fungicides and herbicides approved by the Pesticides Safety Directorate for use in crop protection on farms.
2. The products are approved for use on various agricultural crops. We have no products used on livestock.
3. Full approved product labels, technical training for distribution and Safety Data Sheets are supplied.
4. The organochlorine molecules have been replaced in many crop situations byorganophosphate products because of the environmental persistence of organochlorine molecules. The change has been driven by environmental and regulatory authorities worldwide.

Warble Fly Treatment

5. Dow Agriculture, as the company was at the time, did have an animal health division and sold a treatment for warble fly. This was approximately 1960 to 1981 when the animal health group was disbanded.
6. The active ingredient was crufomate.
7. The brand name for the product was Ruelene.
8. Ruelene 25E – the rate used was a 1:3 dilution with water to give a 6% pour on using 1 fluid oz/cwt bodyweight up to a maximum of 8 fl.oz. Later (late 1970's) Ruelene 7R was produced which was a ready to use pour on formulation used at the same rate as the 25E.

9. The dosage in the UK was the same as in other countries.
10. A regulatory package that met the requirements of all countries at the time of sale was produced.
11. As far as we are aware no specific studies were undertaken on the effects of combination treatments.

Contact with Government Departments

12. At the time Ruelene was sold the then Animal Health Group in Dow were in discussions with the government on an eradication programme but we understand Ruelene came off the market before the programme came into force in the UK.

David Farrant
Registration Specialist
UK and Eire

Issued on behalf of the witness by:
The BSE Inquiry Press Office
www.bse.org.uk

"The cause of death in poisoning by organic phosphorous compounds is usually respiratory failure and consequent anoxia but may be cardiovascular in origin. Four factors (excessive secretion of the respiratory tract, bronchoconstriction, weakness of the muscles of respiration, and failure of the respiratory centre) may contribute to respiratory failure. … In a few instances, death has followed profound brain damage that occurred, usually early in the course of poisoning, as a result severe anoxia. …

Some organic phosphorus compounds produce an immediate "CNS depressant" effect, ranging from incoordination to deep anaesthesia following IV injection. At the same time respiration may be affected. A large dosage is required for all compounds for which the effect has been demonstrate and, by necessity, all of them are of low toxicity."

Description from the Royal Society of Chemistry

From the BSE Inquiry Website: www.bse.org.uk

Annex 1: Warble fly outbreaks and the use of organophosphates

Introduction

1 This Annex describes how the Government responded to the incidence of warble fly infestations in the 1970s and 1980s. Warble fly was a significant cattle pest in the 1970s and 1980s throughout England and Wales in particular, the greater incidence being in the southern half of the country. Surveys had shown that the average incidence of cattle affected by warble fly passing through livestock markets was over 40 per cent in England and Wales and about 20 per cent in Scotland.

2 The larvae of the warble fly live within the body of the cow. Their presence may cause distress to the animal and can have severe economic consequences. The meat may be damaged, and heavily infected hides can be useless for leather. Furthermore, milk yield can be reduced by as much as 25 per cent.

Eradication campaigns

3 In 1978 it was estimated that 38 per cent of cattle in Great Britain were infected with warble fly. The eradication campaign began with the Warble Fly (England and Wales) Order 1978 (SI 1197), which relied on compulsory treatment of affected animals in spring with recommendations for voluntary treatment of all susceptible cattle in the autumn.

4 On 15 March 1982 the Warble Fly (England and Wales) Order (SI 234) ('the 1982 Order') came into force. This made warble fly infestation in cattle a notifiable disease, gave MAFF veterinary inspectors power to serve compulsory treatment notices on farmers whose herds were found to be affected by warble fly, and allowed inspectors to restrict the movement of cattle until the treatment was carried out.

The 1982 Order required infected cattle to be treated in spring, with a follow-up treatment in autumn. The eradication schemes also provided for veterinary inspection of the treatment on farm, and the exemption of cattle from treatment under the authority of a veterinary inspector, if the inspector was satisfied that it was impracticable or inexpedient to treat the animal in question.

Livestock Farming

5 By 1983 only localised areas of low incidence remained, and the Warble Fly (England and Wales) (Infected Areas) Order 1983 (SI No 1382) *('the 1983 Order')* was introduced. 251 This allowed the Minister of Agriculture, Fisheries and Food to declare an area to be an 'infected area' if he or she believed or suspected that warble fly existed in that area, and to require treatment of cattle there.

6 Infected areas were declared at various times, notably after the hot dry summers of 1983 and 1984. After the 1983 Order came into force, Anglesey was declared an 'infected area'. In 1984 infected areas were declared in six localities; Anglesey, the Lleyn Peninsula in Gwynedd, east Cornwall, parts of Dorset and Wiltshire, parts of north Somerset and south Avon, and parts of the Dumfries and Galloway region. Further orders were made in 1985 covering west Cornwall, east Cornwall, much of south Devon, Dorset and parts of neighbouring counties including most of south Avon, and the Isle of Anglesey and the Lleyn Peninsula. In 1986 the areas subject to orders were west Dorset, neighbouring parts of Somerset, and south-west Devon including Dartmoor.

7 By January 1985, the incidence of affected cattle had declined from the 1978 figure of 38 per cent to less than 0.01 per cent.
8 In 1993 warble fly re-entered the UK in imported cattle, which resulted in the Agriculture Departments increasing serological checks on imported cattle. In England and Wales, 39 areas were subject to 'infected area treatment' in the autumn. From 1994, animals found to be infested were sent back to the country of origin. The Animals (Post-Import Control) Order 1995 required all imported bovines (other than those from Northern Ireland or those for immediate slaughter) to be treated with an approved warble fly treatment within 24 hours of arrival at premises of destination.
9 MAFF continues to run a comprehensive control programme under the legislation in force. Whenever infestation is found or suspected, either the individual animals or the herd, and in some cases all animals within three kilometres of the affected herd, are required to be treated, and movement restrictions apply. Blood testing is available where practicable, allowing treatment to be restricted to infested animals only. Regular publicity campaigns are run, and importers are reminded of their obligation to treat imported cattle.

Treatment and dosage for warble fly

10 Under the 1978 Order, farmers were required to treat their cattle with either Phosmet, Famphur or Fenthion – pour-on types of organophosphate (OP) insecticide – or Derris, a contact insecticide. This Order and the subsequent Orders required these products to be applied according to manufacturers' instructions.

Warble Fly Outbreaks And The Use Of Organophosphates

11 Pour-on OPs are effective because of the systemic nature of their action, which ensures the death of a high percentage of larvae before they complete their migration. Non-systemic or contact insecticides remain on the surface of the treated animal. Pour-on OPs were applied to cattle along the back of the head and the entire length of the spinal column.
12 The 1982 Order discontinued the use of Derris as an alternative to OPs, because the activity of Derris meant that it was only effective in destroying larvae as they emerged from the animal. The Government therefore considered it to be ineffective in helping to eliminate warble fly on a national basis.
13 Ivomec (active ingredient ivermectin), an injectable parasiticide for cattle, was introduced to the market in 1981 and provision for its use to treat warble fly was made in the 1982 Order. If Ivomec was used on dairy cows, milk could not be used from those cows for a period of 28 days, in contrast to a six-hour withdrawal period if OPs were used. Accordingly, Ivomec was more commonly used on beef cows and other cattle, as the financial penalties for dairy farmers were severe.
14 It has been estimated that the peak level of OP usage was around 1979–80, with a reduction in the following years as the infection became less common and the use of Ivomec increased.
15 However, Mr Mark Purdey, an organic farmer, suggests that OPs were used more extensively in the 1980s, and had a significant impact on the BSE story.

Hazard Sheet: ***Crufomate***

Trade or other Names:

Amidophos,
Montrel,
Ruelene,
Dowco 132

Identification:

Pure Crufomate is a colourless powder or a yellow oil. It is an Organophosphate pesticide applied on plants as a spray to kill insects and worms.

Handling & Storage:

Prior to working with crufomate you should be trained in its proper handling and storage.

Crufomate is not compatible with Strong Bases *(such as Sodium Hydroxide, Potassium Hydroxide);* Strong Oxidizing Agents *(such as Chlorine, Bromine and Fluorine).*

Store in tightly closed containers in a cool well-ventilated area away from heat.

Sources of ignition, such as smoking and open flames are prohibited where Crufomate is used, handled, or stored in a a manner that could create a potential fire or explosion hazard.

HAZARD SUMMARY

Crufomate can affect you when breathed in and by passing through the skin.

Acute (short-term) effects:
Exposure to crufomate can cause rapid, fatal organophosphate poisoning with headache, sweating, nausea and vomiting, diarrhoea, muscle twitching, loss of coordination, and death.
Chronic (long-term) effects):
Repeated exposure may cause personality changes, including depression, anxiety or irritability.
High or repeated exposure may damage the nerves with weakness "pins and needles", and poor coordination in arms and legs.
Crufomate may damage the testes (male reproductive glands).
Breathing Crufomate can irritate the eyes, nose and throat.

MEDICAL TESTING:
Before employment and at regular times after that, the following are recommended:
Plasma and red blood cell cholinesterase levels (tests for the enzyme poisoned by this chemical) If exposure stops, plasma levels return to normal in 1 to 2 weeks while red blood cell levels may be reduced for 1 to 3 months.

When cholinesterase enzyme levels are reduced by 25% or more below pre-employment levels, the risk of poisoning is increased, even if results are in the lower ranges of "normal". Reassignment to work not involving organophosphate or carbamate pesticides is recommended until enzyme levels recover.

If symptoms develop or overexposure occurs, repeat the above tests as soon as possible and get an exam of the nervous system.

Any evaluation should include a careful history of past and present symptoms with an exam. Medical tests that look for damage already done are not a substitute for controlling exposure.

Request copies of your medical testing. You have a legal right to this information.

Mixed Exposures:
Persons exposed to other chemicals which affect body cholinesterase (OPs, carbamates) may be at increased risk.

Source: New Jersey Department of Health & Senior Services

Hazard Sheet: ***Coumaphos***

Trade or other Names:

Agridip, Asunthol, Meldane,Muscatox, Umbethion, Co-Ral, Asuntol, Bay 21, Baymix, Dilice, Resistox, Suntol, Negashunt (Extoxnet)

Physical Properties:

CAS Number:
56-72-4
Description:
Tan, crystalline solid with a slight sulphur odour
Molecular formula:
$C_{14}H_{16}ClO_5PS$
Molecular weight:
362.78
Melting point:
90-92˚C
Solubility: insoluble in water: 1.5 mg/L at 20˚C, soluble in organic solvents
Specific gravity: 1.474

Regulatory Status:

AUSTRALIAN GUIDELINES
No guidelines.
Classified as harmful and toxic.
Schedule 7 poison (PESKEM, 1995).

OVERSEAS GUIDELINES
No occupational exposure limits have been established for coumaphos by OSHA, NIOSH or ACGIH (Extoxnet)

CATEGORY: Organophosphate USE: Agricultural

Coumaphos is an insecticide used for control of a wide variety of livestock insects, including cattle grubs, screw-worms, lice, scabies, flies, and ticks. It is used against ectoparasites, which are insects that live on the outside of host animals such as sheep, goats, horses, pigs, and poultry. It is added to cattle and poultry feeds to control the development of fly larvae that breed in manure. It is also used as a dust, dip, or spray to control mange, horn flies, and face flies of cattle (Extoxnet). In Australia registered for use on cattle, horses and sheep (PESKEM, 1995)

ACUTE TOXICITY: Coumaphos is highly toxic by inhalation and ingestion, and moderately toxic by dermal absorption. As with all organophosphates, coumaphos is readily absorbed through the skin. Skin and eye contact with this insecticide may cause mild irritation, as well as cholinesterase-inhibition. Some of the symptoms of acute inhalation of coumaphos begin immediately, or within four to 12 hours, of exposure. These include headaches, dizziness and incoordination. Moderate poisoning is characterised by muscle twitching and vomiting. Severe poisoning is indicated by diarrhoea, fever, toxic psychosis, fluid retention (edema) of the lungs, and high blood pressure. Symptoms of sublethal poisoning may continue for 2 to 6 weeks (Extoxnet).

CHRONIC TOXICITY: Repeated or prolonged exposure to OPs may result in the same effects as acute exposure including the delayed symptoms. Other effects reported in workers repeatedly exposed include impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise has also been reported (Extoxnet).

Carcinogenic: Coumaphos was not found to be cancer-causing, or carcinogenic, in tests done on mice and rats (Extoxnet)
Mutagenic: Gene mutation and DNA damage studies performed on bacterial cultures showed no evidence of mutagenicity (Extoxnet).
Teratogenic: Based on studies with rats and rabbits, EPA has determined that coumaphos is not teratogenic (Extoxnet).
Reproductive effects: Mice fed coumaphos at a dietary level of 100 ppm exhibited a decrease in the number of pregnancies, litter size, and surviving offspring (Extoxnet)

FATE IN THE ENVIRONMENT: Coumaphos was relatively immobile in a sandy loam soil and is unlikely to contaminate groundwater. It is relatively resistant to breakdown in water (Extoxnet).

ACTION ON ANIMALS: Coumaphos is highly toxic to birds, moderately toxic to fish and highly toxic to aquatic invertebrates. It also poses a moderate hazard to honey bees and a slight hazard to other beneficial insects (Extoxnet).

ACTION ON PLANTS: No information was found on the breakdown of coumaphos in vegetation (Extoxnet).

REFERENCES: Extoxnet. PESKEM. (1995) Australian Directory of Registered Pesticides and their Uses. Compiled by K. Hamilton, Centre for Pesticide Application and Safety, University of Queensland. Worksafe.

Definitions from Black's Veterinary Dictionary *(13th edition)*

TEPP

Tetra-ethyl pyrophosphate, used in agriculture as a pesticide, is a potential danger to livestock. A Texas rancher diluted one gallon of TEPP with water to make 120 gallons, and sprayed 20 head of cattle. All were dead within three-quarters of an hour. Symptoms of poisoning in a puppy comprised drowsiness, muscular incoordintion and vomiting. The antidote is atropine sulphate. *Please note that ALL OPs degrade to become TEPPs through age and heat.*

TERATOGENIC

Agents, called Teratogens, that are known to cause congenital defects. Such agents include drugs (e.g. thalidomide, griseofulvin) and viruses.

TERATOMA

A developmental irregularity in which the embryo, instead of growing normally in the uterus, develops structural defects or, in extreme cases, develops into a monster. The latter are comparatively common in cattle, and give rise to difficulty at parturition. 'teratology' is the study of monstrosities.

From The Columbia Encyclopedia, Sixth Edition, 2001.

Botfly (Warble Fly)

Common name for several families of hairy flies whose larvae live as parasites within the bodies of mammals. The horse botfly secretes an irritating substance that is used to attach its eggs to the body hairs of a horse, mule, or donkey. When the animal licks off the irritant, the larvae are carried into the host's mouth and later migrate to the stomach. They attach themselves to the lining, where they feed until ready to pupate, and then drop to the ground with the faeces. The larvae, which may cause serious damage to the digestive tract and weaken the animal, can be eliminated by a veterinarian. Sheep botflies lay their eggs in the nostrils of the host without alighting. The larvae work their way up into the head cavities causing fits of vertigo known as blind staggers; failure to eat because of irritability may result in death. Old World species of this family attack camels, elephants, horses, mules, donkeys, and deer. The warble flies, also called heel flies, or bomb flies, parasitise cattle and other animals. The larvae, called cattle grubs or cattle maggots, penetrate the skin of the host immediately after hatching; they migrate through the flesh, causing irritability, loss of weight, and decreased milk production, and then settle under the skin of the back, producing cysts, or warbles. Breathing holes made in the warbles by the larvae damage the hide. A species of human botfly found in Central and South America attaches its eggs to a bloodsucking mosquito that it captures and then releases. When the mosquito comes in contact with humans or other warm-blooded animals, the fly eggs hatch and the larvae fasten to the mammal's skin. The larvae bore into muscle tissue; infestation is called myiasis. For control methods, see bulletins of the U.S. Dept. of Agriculture. The botflies are classified in the phylum Arthropoda, class Insecta, order Diptera. Horse botflies are classified in the family Gasterophilidae; sheep botflies and warble flies are classified in the family Oestridae; the human botfly is classified in the family Cuterebridae.

From the letters page of the Farmers Guardian, 20/6/2003:

Key Report Unseen By Farmers

The Health and Safety executive can whinge all it likes, the fact remains that they stand accused of negligence with regard to organophosphates and the sheep dips in particular.

The M.S. 17 has still not been seen by the majority of farmers, not even the watered down version, which has obliterated the mention of suicide impulse. This version was made available to the transport and general Workers' Union. Did the NFU have a copy? Are these people more important than the folk who were compelled by law to use this garbage? How many farmers have succumbed to the suicide impulse may we ask, and how many more will become mere statistics in the future?

I have spent a good number of years in the civil Service and as far as I can remember we had no choice as to whether we replied to certain individuals who were asking awkward questions. Neither do I recall the threat to sue individuals who were making a fuss about something. We were paid to treat everyone with politeness and in accordance to the law.

The law! Would someone mind telling the HSE about the Medicines Act 1968 relating to substances at work? They owe us all the same duty of care and no-one in this country should be above the law.

When I started to forward submissions to Brussels many other people followed suit and some very kindly sent me copies of their submissions. It was enlightening that one person, who is not a victim, stated quite bluntly that there was evidence here of a conspiracy. I will go along with that. It is easily within their powers for the HSE to prove us wrong. They can begin by telling the farmers everything they know about pyrethroid poisoning. You know, the stuff that is now being used as sheep dip and sprayed on almost everything we eat. When I wanted information on this, I was told by the HSE that I had more information than anyone else. Hardly a comfort when the symptoms of this obnoxious stuff are the same as those of nvCJD victims.

The biggest crime of all committed by nearly all Government departments is that they are continuing to ignore the massive amount of scientific evidence which I have continually put forward proving that BSE was caused by the use of a chemical for warble fly. We now have the autopsy papers in part - not the full papers - but one glaring fact stands out, some died through anoxia, lack of oxygen to the brain. Is this what has happened to a lot of farmers and shepherds apart from the suicides and cardiac arrests?

Brenda Sutcliffe
Sheep Bank Farm. Littleborough, Lancashire

Letter printed in the *Rochdale Observer*, September 2002

Put This To The Test

I was interested to read of Dr Mason's talk to the Soroptomist International Group *(Observer 7th August)*. Stringent testing done on chemicals used in farming? Don't make me laugh.

There has been no long-term research done on the effects of people having to use this garbage – never mind eat it or use it on children for head lice.

The organophosphate group of chemicals is lethal and the Pyrethroids even worse, though they do have a quicker knock-down ability.

I wonder if Dr Mason has ever seen the Hazard Fact sheet on Crufomate, the first organophosphate used on cattle for warble fly. The summary reads as follows: Crufomate can effect you when breathed in and by passing through your skin. Crufomate may damage the testes. Breathing Crufomate can cause fatal organophosphate poisoning with headache, sweating, nausea and vomiting, diarrhoea, loss of co-ordination and death. Repeated exposure may cause personality changes. High or repeated exposure may damage the nerves.

Anyone wanting a copy of the full paper can contact me with a large SAE and a small donation to cover the cost of printing.

Is there anyone out there who is still in any doubt about where BSE originated? The manufacturers told the BSE enquiry that there were no papers in existence on Crufomate. They'll be delighted to know I've found some – I've sent them a copy but they haven't made it public – well who would admit to selling this garbage? What the chemical companies don't tell you is that there is no magic pill to cure OP poisoning and that the damage is cumulative. They don't tell you that the enzymes cholinesterase and panaoxonase are destroyed along with genes 192 and 55.

There are 27 known symptoms of OP poisoning ranging downwards from brain haemorrhage to pneumonia with a severe dose of gastro-enteritis in the middle. The second the government ban this garbage, the sooner the NHS will recover and we will stop seeing the carnage that has been brought on the farming community.

BRENDA SUTCLIFFE
Sheep Bank Farm, Littleborough

Copy of widely circulated letter from Brenda Sutcliffe, dated 13.6.2003

TO WHOM IT MAY CONCERN AND FOR FURTHER ACTION PLEASE
Re: ORGANOPHOSPHATE AND PYRETHROID POISONING

You will see from the attached information that I have asked some very pertinent questions of the Director General of the National Office of Animal Health. There is no obligation for Mr Sketchley to reply to my letter so I feel that we might get some response if many other people started asking the same questions. The silence of the chemical industry on this subject tells its own story but it is in the interests of everyone in this country today that we elicit the truth, particularly about the origins of BSE and nvCJD.

I trust that all recipients of this letter will realise the importance of this matter and take all the action necessary for us to reach a satisfactory conclusion.

Copy of letter from Mrs Brenda Sutcliffe to Mr Sketchley,
Director of National Office for Animal Health (NOAH), dated 16.5.2003

Dear Mr Sketchley,

I have no doubt that you have seen this week's Farmer's Guardian, particularly the OPINION COLUMN. I was intrigued with your comment that OP products are highly regulated and would not be allowed on the market unless they were absolutely safe. This opinion is very much outdated and in direct contrast to the information now being put out with Copper's own brand of garbage. Their Health and Safety Data sheet now advises that care must be taken to ensure that sheep do not inhale or drink any of the dip wash. Can you explain exactly how this can be done. We have the same sort of contradiction with the use of Malathion used as a headlice shampoo for children where the scientific data states, AVOID SKIN CONTACT AND DEATH USUALLY OCCURS THROUGH RESPIRATORY FAILURE. The manufacturers of this garbage don't tell the parents that either. Another interesting anomaly has arisen with the knowledge that Ministry vets were advised not to approach an OP dip by closer than 14 feet. This was said to me in my house while she was trying to convince me that our sheep needed dipping. We are living in very strange times, Mr Sketchley.

My problem is that I have never seen much difference between what happened to us and what happened to the poisoned cattle. We all have similar enzymes in the blood doing similar functions, e.g. cholinesterase and paraoxonase. The destruction of these enzymes causes tremendous problems as I am sure you know.

The biggest cause for concern at the moment is the fact that now, not only are the shepherds moving from organophosphates to PYRETHROIDS but that the latter is now the favoured method of insecticides being used by our soldiers in Iraq. I reached the conclusion long ago that it was some form of PYRETHROIDS poisoning which was responsible for nvCJD although I do not think it more than likely that a chemical trigger is needed to make them so virulent. We had better all start praying that I am wrong. Perhaps you can tell me if the regular testing which you claim has been done on these products has tested for this cross contamination time bomb.

I have requested that Dow Agrosciences make public the hazard fact sheet on Crufomate, the first OP used on cattle warble fly treatment. They have declined to do this. I am a but miffed about this as it took seven bloody years to find this information and as they had lost theirs I went to the trouble of sending them a copy. I enclose a letter from the Rochdale Observer which I think is quite specific. Perhaps as your department has spoken so eloquently for the chemical companies for a good number of years you can now find the right words to begin telling the world the truth. It will save me a lot of time and money.

It is one thing to dispense with a few hundred shepherds but quite another to put the whole of the British Army at risk.

Definition of Organophosphates (OPs) and Their Toxicology

1. The term organophosphate (OP) is usually used to describe derivatives of phosphoric and similar acids. However, it may be more accurate to describe such compounds as 'organophosphorous compounds'. This term covers a wide range of chemicals not all of which (for example, glyphosate and fosetyl aluminium both derivatives of phosphonic acid) display anticholinesterase activity.

2. There are many different OPs and they differ to some extent in their properties. Many OPs inhibit an enzyme known as acetylcholinesterase (the effect of which is described in paragraph 2). Some OPs react with other proteins such as neuropathy target esterase, and this reaction is associated with the effects detailed in para. 6.

3. Inhibitors of acetylcholinesterase affect certain nerve junctions in animals, as well as parasympathetic effector sites (the heart, lungs, stomach, intestines, urinary bladder, prostate, eyes and salivary glands). The transmission of impulses across nerve junctions involves the release of a transmitter chemical, which, in the case of many nerves, is acetylcholine. To stop the nerve continuing to transmit the message, the transmitter, acetylcholine, must be broken down immediately after it has had its effect. This breakdown is brought about by an enzyme, acetylcholinesterase. By inhibiting the enzyme acetylcholinesterase, OPs prevent the nerve junction from functioning properly. In the case of most OPs and all medicinal and pesticidal anticholinesterase OP products this effect is either reversible, the rate of re-activation of the enzyme being dependent on the chemical structure of the OP, or recoverable by synthesis of new enzyme.

4. OPs can be carefully selected, on the basis of their chemical structure, so that they are very effective agents against their target pest or insect and the risk to humans can be controlled by following the recommended precautions. The efficacy of OP products as pesticides and as human and veterinary medicines relates to the inhibition of acetylcholinesterase in the target pest species.

5. In humans, anticholinesterase OPs have broadly similar actions to those seen in other species. Acetylcholinesterase inhibition causes acute effects in humans and other mammals. The symptoms in humans, which generally occur when acetylcholinesterase activity has been reduced by about 50%, may include: headache, exhaustion and mental confusion together with blurred vision, sweating, salivation, chest tightness, muscle twitching and abdominal cramps. The severity of the effects depends on the degree of acetylcholinesterase inhibition. The more severe effects can include muscle paralysis leading to severe difficulty in breathing, so requiring respiratory support. Convulsions and unconsciousness can occur.

/continued...

Recovery depends on elimination of the OP product from the body and return of acetylcholinesterase activity. However, as noted in paragraph 1, not all OPs are anticholinesterases, and compounds such as glyphosate exhibit quite different toxic effects. Furthermore some non-OPs are anticholinesterases and these compounds have similar toxicity to anticholinesterase OPs, an example of this being the carbamate insecticides.

6. Some OPs may also work by another mechanism, that is, causing an OP-induced delayed effect on the peripheral nerves. This is known as OP induced delayed polyneuropathy (OPIDP). OPIDP is a delayed effect caused by die-back in the long nerves, thus affecting the limb extremities. OPIDP is associated with, but not necessarily caused by, inhibition of another enzyme known as neuropathy target esterase (NTE). The capacity of OPs to inhibit NTE and cause OPIDP does not correlate with their capacity to inhibit acetylcholinesterase. Any OP product which is shown by laboratory tests to be likely to produce OPIDP in humans, will not be authorised in the UK. A number of studies of OP products currently or previously used in UK sheep dip, have shown them to have no potential to produce delayed polyneuropathy in animal tests.

7. Another known toxicological effect of OPs in humans has been termed the intermediate syndrome. This can follow severe acute poisoning, sometimes as a result of a suicide attempt, and causes temporary paralysis of the proximal muscles (muscles nearest to the central line of the body e.g. respiratory, neck and upper part of limb muscles; the distal muscles of the limb are not affected so grip strength may be preserved). Since this includes the respiratory muscles, respiratory support is necessary to keep the patient alive. The precise reasons for the development of intermediate syndrome are not clear but explanations which have been advanced include myopathy (muscular damage), depolarisation blockade (blocking impulses at the neuromuscular junction and paralysing the muscles) and Guillain-Barre syndrome-like effects (muscle weakness, numbness and pins and needles in limbs).

8. There are postulated long-term effects of OPs following long-term low-level exposure. Some studies on low-level exposure have shown subtle effects (e.g. slower reaction times) in specialised tests for neurological function, whereas others have shown no change in different neuropsychological and neurophysiological tests. The alleged theories and mechanisms are sometimes not related to acetylcholinesterase activity.

Lord Walsingham on the Cause of nvCJD

It is worth remembering that the infected feed theory which says that scrapie-infected sheep brains in bonemeal, which was consumed by cattle, led to BSE which led to nvCJD in humans when this meat entered the food chain, is a purely speculative theory based on no hard evidence whatsoever. There is no evidence that there is a link between scrapie in sheep and BSE in cattle, or BSE in cattle and nvCJD in humans.

This letter from ***Lord Walsingham*** *titled* ***Vested interest in CJD 'cause'*** *was published in the Farmers Weekly of 31st May 2002.*

Brenda Sutcliffe (Letters, May 3) drew attention to the fact that organophosphorous (Dichlorfos) is now banned as an insecticide in the home, but organophosphorous insecticides are nevertheless still used on children's heads.

Some people (like me) think this could possibly amount for the youngsters killed by CJD. The infective feed theory, with the misformed prions, resulting from the disorder, being treated as the vectors, was given credence in the media in 1996 by the President of the Royal Society at that time, neurobiologist Sir Aaron Klug, who underwrote restrictions on feed and beef - but solely "on the precautionary principle".

The only evidence he had to go on was a half dozen trials of inter-species transmissibility, to which research had been diverted, of which he was good enough to send me the transcripts.

They were all inconclusive so far as identifying the vector for the disorder was concerned since they all used raw whole brain homogenate from cows with BSE, so they all contained all possible vectors (whatever caused BSE) as well as the misformed prions caused by the disorder; no tests were done for anything other than the prions as evidence of BSE.
No proper official trials had been carried out into OP as vectors then, so the had nothing else to go on, and none have been to date, seven years later! Given the negligent handling of the BSE outbreak by MAFF we would all have CJD by now if it were a simple infection rather than a genetic disorder due to manganese taking the place of copper in the formation of routine replacement prions in the brain cells of the few who, by bad luck, are genetically vulnerable to this malformation.

The rest of us will not get the disorder, unless flooded with manganese from industrial pollution like a population in Slovakia downwind of an Eastern Bloc chrome steel works, with more CJD in five miles than in the whole of the UK. OP captures all the copper atoms allowing manganese to take its place.

Mark Purdey has demonstrated scientifically the role of OP in triggering the BSE epidemic, but the establishment maintains its pretences to save itself embarrassment, as well as liability for damages.

The biological establishment is in the pay of the giant pharmaceutical companies who carry out most of the research. Scientific establishments are wooden ships, once in full sail before the prevailing wind there is no stopping them without shooting them out of the water.

Lord Walsingham
The Hassocks, Merton, Thetford, Norfolk

From www. bbc.co.uk Article posted Friday, 26 November, 1999, 17:34 GMT

Jury out on sheep dip

A government committee has concluded there is not enough evidence to prove organophosphate (OP) sheep dip has caused serious medical problems to people exposed to it at low levels.

However, the experts acknowledge an urgent need for more research into the impact of the chemicals - particularly on those who may have some genetic predisposition to suffer from OP poisoning. They also accept that OPs can cause brain damage at high levels, and that controls on their use may be advisable. Farmers exposed to the chemicals claim OPs cause chronic fatigue, memory loss and aching limbs. They have been campaigning for years to have the illness recognised, and for compensation for those whose health they say has been damaged.

A report by two psychiatrists published this summer found that farmers were 10,000 times more likely to suffer from mental disorders if exposed to OPs. The claims have been studied by the Committee of Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). Its report was published on Friday.

The committee concludes that the "balance of evidence" does not support the theory that prolonged or repeated low-level exposure to OPs can cause nerve damage or significant neuropsychological effects. If such effects do occur, the report concludes, they must be relatively uncommon. However, committee chairman Professor Frank Woods said: "There are sufficient indications in the science we looked at to change the way in which the compounds are used."

The Veterinary Products Committee, the Advisory Committee on Pesticides and the Committee on Safety of Medicines are now preparing advice on the implications of the COT report. Junior agriculture minister Baroness Hayman said the government took concerns over OPs "very seriously" and would respond to the expert advice as soon as possible.

Liberal Democrat MP Paul Tyler, chairman of the All Party Organophosphates Group, said the report contained nothing to suggest OPs were safe, and called for a total ban. He told the BBC: "The people who have been studied carefully are those who are still working and who are very fit. The people who are not fit, who have had to give up work because of exposure to OPs, have not been properly studied. In that sense, this report is still inconclusive. The government should do everything it can to try and find equally effective pesticides for use on sheep."

The National Farmers' Union issued a statement welcoming the call for new research as a "step forward". They said: "OP based dips have a vital animal welfare role to play in the control of diseases like sheep scab for which there are currently no alternatives available which are equally effective and do not have an environmental impact. This is a serious problem for the farmers who are affected and the NFU is committed to raising farmer awareness about the potential dangers."

Letter from Brenda Sutcliffe printed in the Farmer's Guardian on July 11th 2003

The five questions YOU should ask NOAH:

The Health and Safety Executive are not alone in their refusal to answer questions on the subject of organophosphates. They are in dubious company. On May 16, 2003, I wrote to the director of the National Office of Animal Health asking simple questions which every farmer in the country has a right to answers to.

1 How do we follow the latest instructions on sheep dip by ensuring that the sheep do not drink or inhale any of the dip wash?

2 When parents use Malathion as head lice treatment, are they told to avoid skin contact and that death is usually due to respiratory failure?

3 The manufacturers of Crufomate are resisting publishing the hazard fact sheet. Isn't it the duty of NOAH to do it for them?

4 In view of the fact that the shepherds, the soldiers and the children with head lice are now being subjected to the use of pyrethroids, shouldn't they all be made aware of the symptoms of this form of poisoning?

5 When Ministry vets were advised not to approach an OP dip by closer than 14 feet, wouldn't it have reduced the suicide figures if shepherds had been given the same advice?

I won't ask the idiotic question of where can we buy a rubber suit for the cows being treated for warble fly.

I now learn that to implement the Medicines Act 1968 on behalf of the shepherds the HSE would have to liaise with the Department of Trade and Industry. Why won't they then? Is it because it would destroy the export market in chemicals? It can't be because they have any concern for the peasant farmer overseas. Are these people told they need rubber coveralls before use, and a certificate of competence before purchase? Are all the labels on the cans printed clearly in a language applicable to the user and isn't it a bit presumptive to assume that they can read in the first place?

The Official Secrets Act is not a tool to protect civil servants, it is a tool of Government to hide the truth. Senior civil servants need reminding that there is a very serious issue of misprision which can carry a long custodial sentence. Six weeks after my letter to NOAH, I am still waiting for a reply. Perhaps other people will have a better response than myself.

The address to write to is:

The Director of National Office of Animal Health
3 Crossland Chambers
Gladback Way
Enfield
Middlesex
EN2 7HF

References, Web links & Further Reading...

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HM Government Department for Environment, Food & Rural Affairs (DEFRA):
DEFRA
Lower Ground Floor
Ergon House
c/o Nobel House
17 Smith Square
London SW1P 3JR
www.defra.gov.uk

National Office of Animal Health (NOAH)
3 Crossland Chambers
Gladbeck Way
Enfield
Middlesex EN2 7HF

Health & Safety Executive
HSE Infoline
Caerphilly Business Park
Caerphilly CF83 3GG
hse.gov.uk

Royal Society of Chemistry
www.rsc.org

Veterinary Medicines Directorate (VMD)
Woodham Lane
New Haw
Addlestone
Surrey KT15 3LS
www.vmd.gov.uk

BSE Inquiry Website:
www.bse.org.uk

US Environmental Protection Agency:
www.epa.gov

Farmers Guardian
Oliver's Place
Eastway
Fulwood
Preston
Lancashire PR3 OTP

Farmers Weekly
Quadrant House
The Quadrant
Sutton
Surrey SM2 5AS

New Jersey Department of Health and Senior Services
Right to Know Program
PO Box 368
Trenton, New Jersey
NJ 08625-0368
www.state.nj.us/health/

Nigel & Mark Purdey's Research Site:
www.purdeyenvironment.com

'Provet' Pet Health Care:
www.provet.co.uk

Independent Farming Website (UK):
www.farm.org.uk

Pesticide Information (UK):
www.pesticides.gov.uk

Pesticide Action Network:
www.pan-uk.org

UK Ministry of Defence:
www.mod.uk/issues/gulfwar/info/pesticides.htm

Australian Safe Working:
www.worksafe.gov.au

Internet 'Search Engine':
www.google.com
www.google.co.uk

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RECOMMENDATIONS

* Drastic Action - a complete ban on the production and sale of all OPs until the manufacturers have proved their safe use in the public domain. Of particular concern is the use of MALATHION (OP) and PERMETHRIN (SP) on children for head lice.
* DEFRA to contact all sheep farmers with a simple questionnaire to find out the exact number of affected shepherds.
* To investigate how many shepherds are in prison or psychiatric hospitals, to enable sympathetic treatment to be given. Large doses of Prozac are not to be given.
* The Industrial Injuries Benefit available to workers in the industry should be made available to the self-employed. Shepherds are finding it nearly impossible to obtain the basic State Sickness Benefit.
*The autopsy papers on BSE to be made public. Frontal lobe brain damage applies to humans as wells as cattle, and this damage, along with the symptom of "anxiety" =, is what is causing farming suicides.
* A Police Enquiry is necessary - too many people have died and will continue to die until a halt is called.
* Any future Police Enquiries into "Shaken Baby Syndrome" cases should include an environmental evaluation into the possibility that these deaths could have been caused by (a) head lice shampoo, (b) garden products, or (c) the most likely culprit - fly sprays. These babies have suffered massive brain haemorrhages without any of them suffering broken necks or whiplash injury. This would seem to be an impossibility given the fragility of a baby's neck.
* CJD victims should be investigated thoroughly as to their use of or exposure to OPs and especially Pyrethroids, and should be made aware of the possibility of help through the treatment for Pyrethroid poisoning - they do not appear to have many options left.

CONCLUSIONS

1 Crufomate was the "root cause of BSE".
2 The second mistake was moving on to use other OPs as Warble Fly treatment. This double dosing exacerbated to situation.
3 There has never been a case of horizontal transmission of BSE - so how has it crossed the species barrier?
4 Transmission has occurred via the sire -see Crufomate.
5 Transmission has occurred via the dam - see Trichlorfon
6 Animals born after the ban on suspect feed have become affected via the feeding of grain stored in silos, most of which is sprayed with ACTELIC D - another OP.
7 Organophosphates, as well as destroying the enzyme cholinesterase, also destroy the enzyme paraoxonase.
8 This will have a dramatic detrimental effect on the heart muscles, leading to cardiac arrest. Mild effects on humans will include strokes.
There is widespread use of artificial insemination in the dairy industry - can we be sure that the semen is not partially to blame for transmission> Many cattle have succumbed to BSE without having been treated for Warble Fly.
10 CJD is different to BSE - similar symptoms but not exactly the same. Top of my list of suspects is PYRETHROID poisoning, where the symptoms in small laboratory animals tested are exactly the same as those exhibited by CJD victims (for treatment, see paper by Box & Lee, Edinburgh).