This is a scanned version of the full HSE report on its 1990 study of sheep dippers. Some sections of the report have not scanned properly but the main portions of the text are correct. Comments on the contents of the report are italicised and enclosed in text boxes.
The HSE produced a misleading sanitised "summary" of this report that did not contain the key information.
The HSE's findings were presented to the dip manufacturers at meeting early in 1991 and this initiated the process whereby the products were withdrawn from the market and replaced with different versions in 1993. The phenolic dips came onto the market around 1981 and were withdrawn in 1993. The problems with OP sheep dips seem to have arisen almost exclusively during the period 1981-1993.

Health and Safety Executive
FIELD OPERATIONS DIVISION
SHEEP DIP SURVEY 1990

xxxxxxxxxxxxxx - Agricultural Inspector
xxxxxxxxxxxxxx - Employment Medical Adviser

Internal Report May 1991

Not to be communicated outside the HSE without the approval of the authorising officer.

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These are all HSE staff. This report is very relevant to the licencing body - MAFF. Was a copy was sent to MAFF and, especially, the VMD/VPC?
Note the secrecy surrounding the contents of this report, which dealt with the mass-poisoning that was taking place. The HSE had responsibility for the safety of people using OP sheep dips.

INTRODUCTION

Over the last few years, there has been a growing interest within the farming community regarding possible health effects following the use of sheep dip. As they become aware that others are experiencing problems many farmers are voicing their complaints of ill health for the first time.

This report was written late in 1990. Farmers had been voicing concern for about ten years and the HSE and VMD had held meetings with concerned farmers since the mid-1980s and had diagnosed OP poisoning from OP sheep dip by 1987, if not earlier

This, plus the knowledge that sheep dips are due for relicencing and review, led us to undertake a survey with the following aims:

The review and re-licencing was the responsibility of MAFF, not the HSE. So why is the HSE carrying out this study? Was it asked by MAFF to become involved?

1. To look for evidence of personal exposure and possible absorption of major constituents of sheep dip, including solvents and phenols. The increasing availability of passive monitors made this possible, whereas in the past, cumbersome pumped equipment was impractical for such an operation.

If there was no practical way of testing, how were these products licenced as being "safe"?

2. To validate the measurement of urinary metabolites of specific organophosphates (OPs) with a view to its future use in the routine detection and monitoring of OP absorption. The advantages of this technique would be that it is more direct and more sensitive than the traditional cholinesterase estimation. It is also non-invasive.

This is fraudulent. The information that is needed concerns enzyme inhibition. The HSE is proposing to avoid that. The urinary metabolites are not specific to particular OPs. Metabolites such as DEP are produced by all diethyl OPs(that is a huge number of different parent OPs). This is part of the fraud whereby it is assumed that the metabolites such as DEP come from diazinon, when they might be formed from sulfotep, TEPP etc, or any diethyl OP. Metabolite testing enables the HSE to assume that the metabolites are generated from innocuous OPs when in fact they may come from more dangerous compounds such as TEPP which appear as impurities in commercial diazinon. The HSE's has chosen a method that enables it to define a potentially dangerous exposure as being "safe". Cholinesterase testing identifies potential damage to workers. Metabolite testing does not.

3. To take advantage of the availability of cholinesterase inhibition detection badges. These provide a quick field technique for detecting personal OP contamination, especially during post dip handling of flocks.

The badges do not measure workers' cholinesterase inhibition.

4. To address the need for correct and consistent guidance on protective clothing suitable for sheep dipping. Investigation of recent incidents had indicated that even the manufacturers were issuing free equipment of the incorrect standard.

Where are the reports on these "recent incidents"?

5. To use some of the blood samples collected for other purposes for phenotype research regarding individual differences in rates of esterase activity.

That would be interesting but the results are not reported.

Organophosphates, being the active ingredient, have traditionally been blamed for the symptoms reported following dipping. OPs, however are rarely formulated as pure compounds, but as a mixture with carriers, emulsifiers, etc. It is possible that non-specific symptoms such as headaches, reported after using these products may indeed be due to factors other than the OP.

The symptoms described tend to fall into three main categories - those which could be consistent with exposure to solvents, phenols or OPs. They are usually acute, experienced on the day or evening of dipping and lasting, at most, only a couple of days. There also have been anecdotal reports of delayed and long-term symptoms.

Non-specific symptoms such as fatigue and headache are similar to those which could be experienced following a long, hard day's work, or due to a viral or zoonotic infection.

Identifying the source of these symptoms is further bedevilled by the fact that the various chemicals present in the dips can often produce similar symptoms. Examples of these are shown in the following table:

headache	fatigue	blurred vision
pyrethroids	pyrethroids	phenols
solvents	solvents	OPs
phenols	epichlorohydrin	
glycol ethers	thiram	
OPs	OPs	

dizziness	nausea	salivation	sore throat/cough
pyrethroids	pyrethroids	pyrethroids	solvents
solvents	solvents	phenols	phenols
phenols	glycol ethers	OPs	epichlorhydrin
glycol ethers	OPs		

This indicates that farmers were being exposed to a battery of compounds that could give rise to the symptoms of which farmers complained. However, this list is incomplete. There is evidence that there were other toxins present.
Not all dips contained all of the toxins. The different toxins might explain why there was not a standard response to exposure. The above list also omits surfactants, which are considered to be mild toxins but which would contribute to penetration of eyes and to the formation of aerosols.
OPs can be dissolved in phenols, so why are the phenols differentiated from solvents? The answer is probably that the phenols were there for a different purpose - to increase the toxicity and persistence of the OPS. Why is the term "phenol" used to describe a wide range of compounds with different toxicities?
The HSE believes that dippers were exposed to epichlorohydrin, that contradicts a MAFF Parliamentary answer, which stated that the epichlorhydrin was broken down by water when the dip was mixed in the tub. If MAFF is right then epichlorohydrin's stabilising effect ceased as soon as it was mixed in the tub. Epichlorohydrin is a carcinogen. It has apparently been removed from the dips but has been replaced by propylene oxide, which has similar qualities. The presence of thiram is particularly interesting. Its commercial use is as a fungicide, so why was it included in the dips? Thiram appears to be a carbamate, so may have anticholinesterase effects
There is no reference to the fact that some of these compounds affect behaviour. The HSE appears to be unconcerned that the farmers suffering from vision defects etc were driving on the public roads and using dangerous machinery.

One of the most intriguing aspects of the problem is the fact that some farmers are affected and some are not. The answer to this phenomenon is not simple and often the person with ostensibly the greatest exposure is the least affected. Although working practices, protection worn and dipping sites do vary, these differences do not fully explain the mystery.

Individual variation in response to exposure may be due to genetic factors which determine enzyme systems available to detoxify, or even enhance effects of OPs and solvents.

Because of the cumulative effect of OPs, contamination from post dip handling and use of other cholinesterase inhibiting substances on the farm and in the home, may be contributory factors.

Cumulative poisoning is probably the key to OP sheep dip poisoning, but it would not be detected by a study of dipping because the cumulative effects would only be measurable during the post-dipping handling. The later CVL study found that handling sheep caused significant OP exposure.

A dose-effect relationship, especially at lower levels of exposure to OPs, has always been notoriously difficult to quantify. This is because clinical cholinergic effects in OP poisoning are due to the inhibiting action of the OP on nervous tissue cholinesterase.

The dose-effect relationship can be seen to be a chimera once cumulative poisoning is recognised.

As we are unable to measure nervous tissue cholinesterase we must measure blood cholinesterase which appears to reflect those present in the nervous system. This indirect method is the only one recognized to estimate the biological effects of exposure to OPs. Even in cases where classical cholinergic symptoms have been exhibited, it has been reported that blood cholinesterase has shown no significant change.1 This method is therefore of questionable value in confirming and quantifying OP absorption after the use of sheep dip, especially as the toxicity of the OPs used and the extent of exposure are of a lower magnitude than in other areas of usage.

Measuring blood cholinesterase will not help to assess the effects of inhaled OP that enters the CNS without spending time in the blood.
What is the basis for the statement that sheep dip OPs are of lower toxicity and the extent of is of lower magnitude than those in other areas of usage?

BACKGROUND

Sheep Scab

In the UK, sheep have traditionally been dipped to control a number of ectoparasites, principally lice, keds, scab mites and blowflies.

Sheep scab infection is a Notifiable Disease under the Sheep Scab Order 1986, and causes severe distress to infected animals. The scab mite, Psoroptes, can infect a variety of animals. Psoroptes ovis is specific to sheep. Adult mites lay eggs on the skin of the animal which hatch and mature to adults within 14 days. The mite feeds by scraping the skin, causing an intense reaction. The exuded serum dries to form scabs. The mites feed on the fresh skin at the edge of the scab and spread outward from the initial site of infection. If left untreated the entire body will be covered in scabs within 3 months.

Chemical Control

The scab mite was eradicated in the UK by 1952 through a programme of controlled dipping, primarily using organochlorine based products such as dieldrin and HCH (BHC). The mite reappeared in 1973 with the result that a variety of compulsory sheep dipping programmes have been instituted.

Scab had been previously eradicated without the use of OPs or organochlorines.

Organochlorine products were withdrawn from use in 1984 due to concern over residue levels in the meat and the development of alternative compounds capable of controlling the scab mite; namely, the organophosphorus compounds diazinon and propetamphos.

More recently a synthetic pyrethroid, flumethrin has become available for use.

Sheep are also treated to control other ectoparasites, such as blowfly, which is active during the summer: Such treatment can expose operators to the OP compounds already mentioned or to other more potent OPs such as chlorfenvinphos and chlorpyrifos. Flumethrin does not control blowflies.

All dip formulations used to control sheep scab must be approved by the Veterinary Medicines Directorate (VMD) of the Ministry of Agriculture Fisheries and Food (MAFF). They are classified as medicines under the Medicine Act 1968, not as pesticides, and are not subject to statutory control under the Food and Environment Protection Act.

So, there was no thought of environmental protection despite the quantities of toxins were entering the environment.

Control Application Methods

For the purposes of sheep scab control the only approved method is to plunge dip all sheep in a dip bath. Other delivery systems may be used to control the other ectoparasites. These systems include spraying, jetting and showering, which may result in greater operator contamination.

The dip bath can either be a short swim, tub or swim-around type. The sheep may be introduced to the dip either manually or by slip way entry.

For scab control the sheep must be fully immersed in the dip bath for a minimum period of one minute, with the head and ears being submerged at least once. This requires at least two operators - one presenting and one dunking the sheep. It is necessary to exercise restraint over the sheep to retain them in the dip. This is often achieved with a pivoting gate operated by a rope which eventually gets saturated by the dip solution.

The method of introduction into the dip can vary from that of gentle persuasion to actually throwing the sheep from some distance into the dip.

"Throwing the sheep from some distance"? Where did they see this?

A metal or wooden "dipping stick" is used to submerge the sheep's head; however, some operators prefer to simply push the head under with their booted foot. This latter method quite often results in total submersion of the operator's foot and leg.

Most of the sheep are able to climb up the ramp out of the dip into the draining pen but some of them, such as lambs with heavy fleece, near-term pregnant ewes or animals in poor condition, require manual assistance. The wet sheep stand in the draining pen and shake off the surplus dip which often showers the operator.

Near-term pregnant ewes being dipped in mutagens and fetotoxins! Animals in poor condition should not be dipped anyway.

Dip Preparation and Renewal
The recommended procedure for preparing the dip is to fill the bath with a known quantity of water and add the proportion of dip concentrate detailed by the manufacturer.

OP is removed and ("stripped") from the bath at a rate which is not proportional to the amount of water taken from the bath in the sheep's fleece. The dip therefore is replenished by one of four systems, depending upon the manufacturer's instructions.

1. Water and concentrate are added at a constant rate by an automatic metering system (eg, Powerpack).

2. Water is added constantly to maintain the dip volume with periodic addition of concentrate after a specified number of sheep have passed through (eg, Top Clip).

3. The dip volume is allowed to drop by 10% then replenished by a mixture made up at 1.5 times the original dilution rate eg, Ectomort).

4. The pyrethroid dip is replenished at the original dilution. Some dips require the addition of either phenolic disinfectant Or other bacteriostats, such as thiram, to prevent post-dipping lameness or to allow the dip to be used on the following day.

Other documents indicate that preventing post-dipping lameness was not the reason for adding phenols to the formulations. It's odd that thiram, a fungicide, is used as a bacteriostat.

Disposal

Once the dipping operation has been completed, the dip may be emptied immediately either by sucking it out with a vacuum tanker or by bailing it out with a bucket. The dip may then be cleaned with a disinfectant.

On other occasions, the dip is left in the bath until the next dipping period, in the belief that the long period will aid the natural breakdown of the product and decrease the environmental risk.

Farmers leaving dirty dipwash in the dip for six months then adding the concentrate to it! The dip would be full of urine, excrement, barbed wire and briars.
One possible explanation that has been put forward is that empty fibre-glass tubs can be pushed out by ground water, so the farmers may have left the tubs full until the next dip, when they would be emptied and refilled.

Protective Clothing

According to the manufacturers' recommendations, the only protection required when handling the concentrate is that of protective gloves and a faceshield. Further protection advocated by some manufacturers when dealing with dilute dip or freshly dipped animals varies, but generally involves the use of wellington boots and a waterproof bib apron.

In reality, the clothing worn varies from the minimum of jeans, T-shirt and wellington boots to full protection, including gloves, impermeable suits and airstream RPE. The boots, coats and leggings worn are usually those used for normal work-wear.

SHEEP DIP FORMULATIONS

Proprietary dip formulations contain a variety of chemicals in addition to the organophosphorus or pyrethroid active ingredient. Properties of some of these ingredients are as follows:

ORGANOPHOSPHORUS COMPOUNDS

The property of organophosphorus compounds which makes them effective as insecticides is their ability to inhibit the enzyme, cholinesterase. Although the action of OP is identical in both man and insects, selective toxicity exists due to differences in the rate of detoxification in man compared to insects. Insects break down OPs relatively slowly compared to mammals. OPs inhibit cholinesterase by forming a stable bond at an active site of the enzyme, therefore blocking its ability to function.

The enzyme, cholinesterase, is an essential component in the control of normal nerve impulse transmission, in that it breaks down the acetylcholine produced at nerve synapses. If the acetylcholine is not broken down, nerve impulses continue unchecked. It is this continuous neurotransmission which produces cholinergic effects which give rise to the signs and symptoms of OP poisoning - abdominal cramps, vomiting, excessive salivation, cold sweats, blurred vision, muscle twitching and tremors.
Clinical effects do not generally appear until plasma cholinesterase activity has fallen to 30% of normal pre-exposure values.2

OPs are occupationally absorbed through the skin, eyes and respiratory tract. Formulation as well as concentration must be considered when evaluating the rate of absorption into the body. Repeated absorption of small doses have a cumulative effect and can result in progressive inhibition of nervous system cholinesterase.

A direct contradiction of the CoT report, which did not consider the cumulative effect of small doses, although that characteristic has been known for half a century.
This reference to absorption through the respiratory tract is a contradiction of MAFF statements about inhalation being inconsequential because of the vapour pressure of diazinon. Vapour pressure is irrelevant to the inhalation of aerosols.
We see here the usual nonsense - OPs affect cholinesterase, which is taken as meaning that cholinesterase is the only enzyme affected. MAO and other enzymes are affected. There are other compounds in the human body that are more sensitive than cholinesterase to diazinon - Taurine, Glutamate and GABA are affected at doses too low to affect cholinesterase.

This occurs when repeated exposures occur within the cholinesterase recovery period and may be the result of handling contaminated clothing, dipped sheep, etc.

The recovery period can be as long as 120 days, and if there is a further exposure in the interim, the recovery period starts again. So, people exposed on a weekly basis through handling sheep could have their cholinesterase level cumulatively depleted by "safe" doses, ie doses that on their own would not be expected to cause damage. The exposure from post-dip handing six sheep was estimated as being over sixteen times the Allowable Daily Intake! Cumulative depletion of enzymes by OPs has been known since the early 1950s but was somehow missed by the CoT Report.

In addition to the acute effects, Organophosphorus Induced Delayed Polyneuropathy (OPIDP) has been recognized since the 1930s.
It is only recently that interest has been rekindled in this phenomenon. The signs and symptoms associated with this syndrome, which appears 1-5 weeks after acute exposure to an OP compound, are those of a distal peripheral neuropathy. Reported symptoms include leg cramps, numbness and paraesthesiae, followed by progressive leg weakness. The upper limbs may be similarly affected.
For OPIDP to occur, an enzyme, NTE (Neuropathy Target Esterase or Neurotoxic Esterase) present in nervous tissue, lymphocytes and other tissues must be inhibited by the OP. This inhibition can result in the formation of a stable non-reactive form of the enzyme. All reported cases have been due to ingestion of large quantities of concentrated chemical.

The OP dips could cause paraesthesia by a process other than OPIDP. The phenols and other ingredients also cause paraesthesia. A recent accidental exposure to the mixture of phenolics added to sheep dip caused the usual OP symptoms, including loss of feeling to the hands, with no OP present.
OPIDP by NTE-inhibition might be caused by the OP impurities even if the stated OP ingredient is not an NTE inhibitor.
A series of small exposures is more effective at inhibiting NTE than a single large dose.

Diazinon
Chemical Name:0,0-diethyl 0-2-isopropyl-6-methylpyrimidin-4-yl phosphorothioate

In its pure form, this compound is a colourless liquid with a low vapour pressure and relatively low toxicity. An Occupational Exposure
Standard (OES) of 0.1 mg/m3 (8 hr TWA) applies.
Diazinon is a relatively unstable product. It is broken down in the presence of UV light to form dioxodiazinon, hydroxydiazinon and other biologically active products. In the presence of water it is hydrolysed and will produce traces of monothio-TEPP, a more active cholinesterase inhibitor. Storage of diazinon over a long period also allows it to oxidize to dioxodiazinon.

Is there synergism between the various diazinon impurities? The published answer is "yes". When MAFF were asked in a Parliamentary question for details of the impurities section 118 of the Medicines Act was used to avoid giving an answer. The implication is that the impurities were regarded as part of the formulation. If they were not, then section 118 should not apply. The references to UV light and water describe the condition of the diazinon when it is on the sheep's back after dipping.
What are the vapour pressures of the impurities? What is the OES for impure diazinon?

In addition to its use in sheep dip, diazinon is present in various pesticides and veterinary medicines.

Metabolism in man

Diazinon is probably metabolised by the cytochrome P450 system. Metabolism occurs initially to diethylthiophosphate (DETP) then to diethylphosphate (DEP). Both these products can be detected in urine and provide a tool for biological monitoring.

"Probably"! They don't know how diazinon is metabolised?
They say that DETP and DEP are metabolites of diazinon and can therefore be used for biological monitoring. But DETP is also a metabolite of the highly toxic impurities of diazinon - sulfotep, MTEPP, diazoxon etc? DEP is metabolite of those, plus TEPP. So these metabolites give no guidance as to what OP has been metabolised and so this method is worthless, unless the OP involved in the exposure is analysed correctly. The presence of DEP merely indicates that a diethyl OP was involved. The presence of DETP merely indicates that a diethyl OP and sulfur were involved. A given level of DEP from diazinon might be derived from a harmless exposure. The same level of DEP derived from TEPP might be derived from a lethal exposure.
The cytochromwe P450 system is involved in the metabolisation of most of the dip ingredients , so potentiation would be expected.

Propetamphos
Chemical Name: Isopropyl 3-[ethylamino(methoxy)phosphinothioyloxy] isocrotonate

This compound is stable to UV light, during storage and relatively stable in solution.

Metabolism

Development studies have shown that propetamphos is metabolized by oxidation and that the major excretion product is probably exhaled Co2. It is unlikely, therefore, that a stable metabolite is available in the urine for the purposes of biological monitoring.

No metabolites for urine testing? So what safety tests are to be done for propetamphos?

PYRETHROIDS

Pyrethroids are synthetic pyrethrins which are less toxic than the natural pyrethrins. They have a very low vapour pressure.

Flumethrin, the only non-OP approved active ingredient for scab control, is a pyrethroid.

Although it is stated that there is no significant absorption through the skin, one study has shown that dermal absorption due to skin contamination whilst spraying can lead to acute intoxication.3 Pyrethroids are absorbed and distributed throughout the body and are rapidly metabolized and detoxified by esterase action. OPs can inhibit their degradation and as a result increase their toxicity producing CNS stimulation.

This potentiation was known in 1990 but no warning was issued.

Pyrethroids produce both local and systemic effects. The most common are those of facial sensations such as numbness, itching, burning, tingling and warmth, due to an excitation of sensory nerve endings. Systemic effects are headache, fatigue, dizziness, nausea and salivation.

The same symptoms again.

INDUSTRIAL SOLVENTS

All the dips used in the survey contained either Shellsol R, Solvesso 150, Solvesso 200, or medium oil. These primarily comprise a mixture of aliphatic hydrocarbons in the C11-C13 range. The disinfectant of both Deosan and Diazadip contains 8.5% of kerosene in addition to the other solvents. Kerosene contains C9-C16 hydrocarbons plus small fractions of aromatic compounds (xylenes) and of saturated rings (naphthalenes).

This is odd. Deosan was the same as Topclip and had the same product licence number. But apparently had different solvents.
There are references to xylenes potentiating the toxic effects of OPs.

These solvents are sold as performance products based upon their physical properties so have no precise chemical analysis; for instance, Shellsol R comprises C11-C13 in the boiling range of 205o-270oC with a flash point of SOoC.

Small amounts of contaminants may be present. Because of their high volatility compounds C9 and below will form no more than 0.2 - 1% of the mixture.

The lipophilic character of these chemicals allows them to be readily absorbed through the skin. Metabolism appears to be carried out by cytochrome P450.

The cytochrome P450 system appears to be the major metabolizer of not only aliphatic hydrocarbons, but also other constituents of the dips, such as phenols and OPs and is responsible for the metabolism of certain drugs such as paracetamol. It would seem, therefore, that this system in particular is being bombarded by the chemicals present in these mixtures, together with common drugs which may be used for headaches (following exposure to the dips!)

So there were several sources of potentiation and these involve the P450 system, which is subject to significant genetic variation.
This list of solvents appears to be incomplete. No reference to isopropanol. Some evidence of benzenes(carcinogenic), vinylidene and pentadecane being present, perhaps as contaminants.

GLYCOL ETHERS

At least 3 of the dips used in the survey (Coopers Powerpack Winter Dip, Ciba Geigy Top Clip, Bayer Diazadip) contain a glycol ether.

These are not highly volatile compounds, but are very readily absorbed through the skin and classified as harmful or irritant. Exposure produces non-specific effects such as headache, giddiness, loss of coordination and nausea.

EPICHLORHYDRIN

Two of the dips used in this survey (Diazadip and Deosan) contain this solvent. It is highly volatile with a vapour pressure of 13 mm Hg at 20 C, and has an OES of 2 ppm (8 hr TWA) which is under review.

This compound is readily absorbed through intact skin and by inhalation; it also penetrates rubber and leather.

It is highly toxic, causing possible long term health effects and is an animal carcinogen. In addition, it is corrosive, strongly irritant and a skin sensitizer. Inhalation causes coughing, shortness of breath and pulmonary oedema. A vapour level of 100 ppm produces eye irritation. It is a CNS depressant.

How many solvents did they need? Why is epichlorhydrin described as being a solvent? Its role is generally regarded as being a water scavenger - ie used to "round up" water molecules and keep them away from the OP in the can. Why was the UK sheep flock compulsorily dipped in a carcinogen? Note the human effects of epichlorhydrin. Why did some diazinon dips need epichlorhydrin and not others?

PHENOLS/CRESOLS

All the dips used during the survey (including the non-OP dip) contained some form of these related chemicals. Although they can be derived either from petroleum or coal tar, those appearing as constituents in sheep dip are coal tar in origin.

Phenol has a vapour pressure of 0.35 mm Hg at 25oC and cresols a vapour pressure of 0.1-0.24 mm Hg at 25oC. The odour threshold for phenol is 5 ppm.

Phenols and cresols are readily absorbed through intact skin and by inhalation. Phenols are extremely toxic; both phenols and cresols have an assigned OES of 5 ppm (8 hr TWA).

Phenols are noted mainly for acute systemic effects through skin absorption following significant exposure, which can be severe. They are corrosive and highly irritant producing burns and other irritant symptoms such as gastric burning, sore throat and shortness of breath. Other reported effects are watery eyes and blurred vision, increased salivation and non-specific symptoms such as headache, drowsiness and dizziness. Cresols produce similar effects but to a lesser degree.

The HSE report does not explain why phenols were introduced into the dips around 1980. Nor does it satisfactorily explain their role.
Phenols/cresols attack PPE, indeed virtually all of the ingredients have that effect. Why no reference to an acceptable exposure limit?
See the attached HSE Alert on phenols.

THIRAM (tetramethylthiuram disulphide)
Thiram is used as a bacteriostat for two dips, to which it may be added as a powder in a soluble sachet.

The ethyl form of this chemical is used as a pharmaceutical (disulfiram) to discourage the use of alcohol. Much of the available information on the effects of thiram is based upon the effects of disulfiram, although thiram is 10 times more toxic.

Disulfiram's use as an anti-alcoholic treatment is based on the fact that it blocks the metabolism of alcohol, resulting in the accumulation of acetaldehyde, which, in turn, produces unpleasant effects such as violent flushing, dyspnoea, headache, palpitations, tachycardia, nausea and vomiting.
Non-specific symptoms such as drowsiness, unpleasant taste, mild gastrointestinal disturbances and orthostatic hypotension are recognized side effects occurring at the beginning of treatment with this drug; ie, effects of the disulfiram without alcohol.

Thiram is a severe irritant to mucous membranes, a mild skin irritant and a potent skin sensitizer.
Its toxicity is increased in the presence of fat solvents, which promote absorption.
A warning is given with Thiram, that if alcohol is ingested within 72 hours of absorption, severe nausea and vomiting may occur.
Disulfiram, however, is eliminated from the body at a very slow rate and it may be detected in body fluids up to 7 days.

See the Extoxnet data on thiram. Thiram could account for all of the effects attributed to OP sheep dips. It appears to be a form of carbamate and may have anticholinesterase effects.
Thiram was considered as bacteriostat and an alternative to phenols in that role, which is odd in view of the fact that its commercial use is as a fungicide. It may be that thiram was included in sheep dips as a fungicide to prevent infections that might be falsely diagnosed as scab.

METHOD

Recruitment

A request for volunteers for this survey was published in a newsletter distributed by a local Agricultural Cooperative. This brought only two initial replies, a surprisingly small response compared to the large number of people expressing concern over the issue. A policy of pyramid recruitment was undertaken, as a result 43 "volunteers" were obtained, representing 25 dipping sites.

The first concern was that the survey population would be biased because the majority of people coming forward would be those having had symptoms in the past. This, in fact, was not the case. Many of the volunteers cooperated because they wanted to help colleagues who had experienced problems.

A second concern was that everyone would view the HSE team in its normal enforcement role and modify work practices accordingly. There was no evidence that this occurred.

We visited all participants prior to the dip to explain the aims and practical aspects of the survey. We completed guestionnaires covering normal dipping practice, sheep handling in the months prior to the survey and OP products used on the farm and in the home. We also enquired about past symptoms associated with exposure to sheep dip, relevant past and present medical histories, medication and other factors such as special diets which might influence the proposed biological monitoring.

Biological Monitorinq
Blood
In 3 cases, blood samples were taken before the dipping operation for the estimation of:
(1) baseline cholinesterase
(2) other enzyme systems (esterases)
using EDTA and plain tubes respectively.
In all the other volunteers, OP exposure during the preceding 60 day period could not definitely be ruled out. This was due to the use of OPs (either pesticides or veterinary medicines) and regular handling of sheep dipped during the summer months, mainly to protect against blowfly strike. In these cases retrospective baseline samples were taken in January/February 1991.

Post exposure blood samples were taken at the end of dipping for the estimation of (1) cholinesterase, (2) other esterases and (3) solvents.

So, they did not have a way of collecting a reliable baseline cholinesterase figure. That's a blot on the entire study.

Urine

Urine samples were collected for the estimation of OP metabolites (dialkylphosphates) and phenols.

1. pre and post dip - each day of dipping

2. post dip handling of sheep

3. When retrospective baseline blood samples were collected

Enviromental Monitoring

Atmospheric

Tenax passive samplers were worn by each operator, clipped to the lapel next to the breathing zone. A fresh monitor was worn each day of the dip. These were analysed for volatile products - solvents and phenols.

The atmospheric presence of OPs was not monitored as previous studies had reported none present.

Surface Contamination

Cholinesterase inhibition detection badges were used as a simple field method to detect the presence of OPs on skin, inside protective clothing and on fleece. They were also used during handling of sheep in the months following dipping when the following procedures were carried out - drenching (oral administration of veterinary medicine), vaccinating, docking shearing around the tail area), scanning, shearing and lambing.

In some cases, the badge tests were paralleled by quantitative laboratory analysis of either gauze patches or the garment itself.

Enzytec Pesticide Detector Kits provide a simple field method for the detection of cholinesterase inhibitors at low levels (typically 1-5 ppm). The kit includes detector badges which have two discs attached, one sealed under a foil cover (substrate disc) and one exposed (enzyme disc).

The enzyme disc is rubbed on the surface to be tested, then developed in reagent solution for three minutes. The disc was then removed from the reagent, the substrate disc uncovered and the badge folded to bring the enzyme and substrate discs into contact with each other for five minutes. At the end of this period the enzyme disc was examined for colour.

(i) white - positive (cholinesterase inhibitor present)

(ii) blue - negative

Dip Samples

Bulk samples were taken at some sites before and after the dip. This was to assess whether or not the replenishment system resulted in a higher concentration of dip chemical at the end of the period, thus creating a greater hazard to the operator.

Laboratory techniques for analysis of biological and environmental samples are listed in Appendix 1.

Observation

We initially intended to utilize a large team to carry out this survey. This proved impractical because of factors such as geographical distribution of sites and the need for close liaison and flexibility to adapt to last minute changes of dipping times and dates. Ultimately a team of two people visited all but two of the sites (either together or independently) for the purposes of personal observation of the site, weather conditions, dipping practices, and significant incidents.

Follow Up

In January/February 1991 we collected retrospective baseline blood samples and asked participants about any symptoms occurring either during the dipping or later.

The subjects will have been exposed to OPs throughout the period leading up to the taking of baseline blood samples, so the baseline figure will in fact be an inhibited figure and the post-dip results will falsely suggest that exposure at that time had not caused inhibition.

RESULTS

Cholinesterase

OP exposure is measured by the inhibition of plasma and red blood cell (rbc) cholinesterase. Diazinon and propetamphos mainly affect plasma cholinesterase, propetamphos being the more active. Inhibition is determined in the individual by comparing cholinesterase levels before and after OP exposure. Pre-exposure levels, however, can only be determined if there has been no contact with OP products in the preceding 60 days.

Blood cholinesterase estimations were carried out in 39 of the 43 volunteers. All the results fell within the predicted range for a normal population; ie, plasma cholinesterase was greater than 162 daU/L and red blood cell cholinesterase greater than 97 hU/L for all subjects.

Determining individual baselines for all but a few of the subjects presented difficulties. Residual OP persists in the fleece for some time after dipping and acts as a potential source of re-exposure during the normal handling of sheep; also, the farmer's intermittent use of other compounds containing OPs calls into question how often one actually achieves a true baseline measurement.

Cholinesterase levels vary in individuals. This intra-individual variation, together with the degree of precision achieved in our laboratory, allow an individual's plasma cholinesterase to vary up to 15%, and 10% in the case of red blood cell cholinesterase, before the change can be considered significant.4 In a population of 30-40 it is to be expected that one or two subjects will appear at the extremes of these ranges.

But since the HSE had no way of collecting correct baseline figures, no they valid conclusions could be drawn on this matter, nor could it be determined whether the sample conformed to a normal range of baseline cholinesterase levels. The same problem would affect all of their enzyme results. In order to be able to measure correct baseline enzyme levels it would be necessary to ensure no exposure in the previous 120 days, impossible among people who worked with dipped sheep and who may have been exposed to other products.

Plasma Cholinesterase

Pre and post dip exposure estimations were carried out on 37 subjects 30 of whom demonstrated decreased plasma levels.

These were insignificant except for one subject who showed a depression of 10%. He experienced no symptoms of ill health following this dip.

Red blood cell cholinesterase

In 30 subjects, red blood cell cholinesterase levels were higher In the post exposure samples than in the "pre-dip" or retrospective baseline samples. Two of these were at significant levels, ie, 12% and 13%, but in the reverse direction. There is no obvious explanation for this apparent trend; however, it is recognized that when red blood cell cholinesterase levels are depressed, a rebound phenomenon is triggered during recovery, whereby the red blood cell cholinesterase often overshoots its normal level. It is also true that red blood cell cholinesterase takes longer to recover than plasma cholinesterase.

These higher post dip samples therefore, may reflect a rebound recovery from an exposure during the summer. This would not be apparent in the baseline measurement as 34 of the 37 determinations were made retrospectively in Jan/Feb.

There were no reports from any of the participants in the survey of OP linked symptoms. This would be expected from these cholinesterase results. Although low plasma and red blood cell cholinesterase activity are consistent with OP exposure, normal levels do not prove a lack of exposure.

Cholinesterase estimations and percentage changes for the survey population are shown in Table 1.

None of these conclusions are valid, for the reasons explained above.

TABLE 1

	CHOLINESTERASE PLASMA			CHOLINESTERASE RBC				CHOLINESTERASE PLASMA			CHOLINESTERASE RBC		
CODE	PRE daU/L	POST	%	PRE hU/L	POST	%	CODE	PRE daU/L	POST	%	PRE hU/L	POST	%
B1	250	247	97	155	164	106	B2	290	288	99	154	166	107
C	447	427	95	148	156	105	D	314	328	104	150	159	106
E	272	236	86	154	162	105	F	349	364	104	175	176	100
G1	271	283	103	176	169	96	G2	295	283	96	157	162	103
G3	354	302	85	177	161	90	H1	316	313	99	183	184	100
H2	174	161	92	180	208	113	H3	286	266	93	162	169	104
X1	301	297	98	159	174	109	X2	234	228	97	178	180	101
J1	263	269	102	160	157	98	J2	296	274	92	158	159	100
K	292	301	103	170	179	105	L1	273	251	92	172	190	110
L3	312	295	95	163	179	109	L4	251	230	92	171	189	110
M1	270	262	97	165	172	104	M2	272	262	96	170	166	97
N1	284	236	83	144	161	110	N2	308	299	97	154	157	101
O1	281	249	88	196	201	102	02	255	238	93	151	160	106
P2	251	231	92	166	157	105	P3	338	369	91	188	194	105
Q1	273	273	100	156	163	104	Q2	290	284	97	165	167	101
R1	357	350	98	167	171	107	R2	287	252	87	177	186	105
S2	301	284	94	164	183	112	T	315	294	93	176	184	104
U	227	213	94	152	159	104	V	321	317	99	155	159	102
W1	268	279	104	158	168	106							

% - POST DIP READING AS A PERCENTAGE OF BASELINE

Changes in cholinesterase

	Plasma Decrease	Plasma Increase	Rbc Decrease	Rbc Increase
Total Number	30	6	4	30
Significant	1	-	-	2

Significant change Plasma > 15%
Significant change Rbc > 10%

The reference to higher enzyme levels because of "rebound" assumes that levels had been significantly reduced before dipping. How did that happen? The higher enzyme levels around the time of dipping might be caused by the fact that the baseline is an inhibited level and the low baseline figure's may have been caused cumulative inhibition after dipping and/or the exposure to more toxic OP transformation products through handling sheep. It is that type of exposure, not exposure at dipping, that may have caused the damage measured in victims by various studies.

Urinary Dialkylphosphates

Part of the survey was designed to validate the use of urinary dialkyl phosphates for monitoring OP absorption. The survey was well advanced before it became apparent that there was no known measurable metabolite of propetamphos. As 40% of the dips used in the survey contained propetamphos as the active ingredient, this resulted in a signi□icant reduction in the number of dialkyl-phosphate analyses.

As an alternative, the laboratory attempted to measure free propetamphos in the urine. This analysis poses several difficulties:

1. Propetamphos is broken down readily by factors such as a high urinary pH.
2. A relatively high absorption of propetamphos is probably necessary before it can be detected in the urine.
3. The potential for specimen contamination cannot be ignored.

The report contained a figure showing the metabolisation of diazinon but it did not scan well. The figure showed diazinon breaking down into DETP and "dioxodiazinon"(normally termed diazoxon or oxo-diazinon, ie diazinon where the sulfur has been replaced by an oxygen atom, which increases its toxicity dramatically. This conversion is the first part of the breakdown of diazinon in the P450 system). The figure also shows DETP and dioxodiazinon breaking down to form DEP.

The levels of DETP and DEP detected in the survey were 10 times lower than those reported in American studies, when no symptoms occurred.

The exact excretion times of dialkylphosphates are unknown. Where ipping occurred over several days, sometimes with short gaps in between, it was possible to collect serial pre and post urines for analysis. We were limited by having to rely on random samples taken at the end of the dipping day. In cases where serial samples were collected, some of them represented 24 hour specimens (first morning voids on day following exposure) and some represented 48 hour specimens. Although impractical under field conditions, the ideal situation would have been to analyse 24 hour, and even better, 48 hour total collections.

In a study carried out on orchard workers exposed to azinphos-methyl, it was shown that 13 out of 17 workers excreted·40% or less of their total metabolites in the morning void (totals varying from 5-80%).5 This indicated the unreliability of partial or random sampling and demonstrated only a weak correlation between the exposure and the 24 hour urinary output. A much stronger correlation was found for the 48 hour output, possibly due to a reduced effect of individual variation. It has been suggested, therefore, that a 48 hour total urine collection is a minimum requirement for estimating total dermal absorption of organophosphates.

The dialkylphosphate results in some cases did not accord with the observation of dipping practice. Some people who appeared to be exposed to a greater extent recorded no DETP in their urine samples whilst others, exposed to a much lower degree demonstrated detectable DETP. These results may be due to the limitations of random sampling or an insufficient interval between the end of exposure and collection of the sample.

In other words, they don't know what is going on and they don't trust their own methods - very wise.

Diazinon was used by 24 participants. No DETP was detected in the urine of 8 subjects whose samples were collected not more than 8 hours after the first exposure. This may be due either to the sampling method or may represent slow metabolisers of OPs. In one subject in this group the plasma cholinesterase level fell 15% following the dip, but.resulted in no symptoms. Eight of the remaining 16 diazinon users either dipped over several days, had cleaned the dip on the day before dipping, or were contract dippers with serial exposure.. Precise information regarding the time intervals between exposure and sample collection is not available on the remaining 8 subjects in this group.

In 4 cases DETP levels were relatively high compared to the remainder of the group, although still at a level considered insignificant.
Two of these were related (father and son). The father dipped, but was well protected, whilst the son gathered and assisted in introducing the sheep into the dip.
A third was a contractor who had close contact with wet sheep to move them down the ramp from the elevated draining pen. Although he wore gloves for dipping the palm of one was torn.

DEP was detected in samples from only three subjects.
Two were a husband and wife who used a dip formulation not used by any other person in the survey (Diazadip). In both these subjects the level of DEP detected was higher than the DETP; the husband had however discarded summer dip from the bath some days before the dipping.
The third subject had also cleaned the dip several days before dipping, but used old dip concentrate to make up the dip. It is possible therefore that during storage some diazinon had oxidized to another derivative, dioxodiazinon, which is capable of being metabolized directly to DEP.
Information on the age of the dip concentrates used by other participants in this survey is not available at present but warrants further investigation.
The factors determining the rate of metabolism of DETP to DET are unknown and could be time or dose dependent, or indeed dependent upon specific esterase activity.

The HSE could have simply analysed the products to determine what was present, in particular to identify OPs impurities and their proportion.

Urinary Propetamphos

The urines of 19 users of propetamphos were analysed for free propetamphos. It was detected in 2 of these.

One of the cases where it was not detected, a borderline fall of plasma cholinesterase (17%) occurred although he reported no symptoms.

Blood Solvents/Urinary Phenols

No blood solvents were detected in any of the subjects.
All urinary phenols were within normal limits taking into account endogenous sources and dietary influences.

Tenax

In practical terms the use of Tenax passive sampling tubes is ideal as they require no technical expertise and do not interfere with normal working practices. Their use in this survey to determine the presence of atmospheric solvents and phenols does however lead to some reservations. The results contained the following unexplained discrepancies.

1. Subject B1 was a contractor operating a mobile dip based on an articulated lorry. It had solid sides reaching a height of 1.5 m, resulting in a very enclosed work site. In this situation we would have expected to detect significant levels of solvent; in fact, only very low levels were found (0.3 ppm aromatic and 0.7 ppm aliphatic).

High solvent readings were anticipated on the basis that the operator complained of classical symptoms of solvent exposure, both during this dipping period and on previous occasions. These included headaches, alcohol intolerance and complaints of feeling constantly "high" throughout the dipping season. Skin absorption was also considered as a possible significant route, especially as biological monitoring showed that this man had absorbed propetamphos from the dip, probably via the skin.

A further factor which makes this particular case puzzling is the fact that in spite of the clinical history, no solvents were found in the blood. This estimation however, also required the use of tenax tubes.

The possibility that this person is demonstrating an idiosyncratic reaction to the substances to which he is exposed on a daily basis cannot be ruled out, nor is it possible to ignore the role of esterases in breaking down solvents and their potential to be inhibited by OPs.

The method is again found to be inadequate.

2. Subjects Hl;H3; These operators were working in an atmosphere in which the odour of phenols was strong and extremelv irritant, also experienced by the survey team. Analysis of the sampler revealed no phenolic compounds to be present.

3. Subjects Xl;X2; The samplers detected no phenols at this site which was very sheltered and smelt strongly of phenol.
The atmosphere was so irritant that both the dip operator and the observer were coughing during the latter part of the day.

Clearly the method is inadequate. The threshold for smelling phenols is 5 ppm, which ***was*** *also the acceptable exposure level. The study should therefore have found phenols and there should have been a conclusion that the products caused unacceptable exposures.*

The results for Hl;H2;Xl and X2 contrast sharply with another dipping operation conducted at a much more open, windy site, using an identical chemical where 4 ppm cresylic acid were detected.

4. Subjects Nl;N2; This dipping operation was conducted inside a shed. No contamination was detected by the samplers. Although there was no noticeable odour around the dip the observers independently reported feelings of unnatural tiredness and throat irritation. The farmers themselves did not complain of symptoms on this occasion, although they had been affected during

Again, evidence of unacceptable exposures and consequent symptoms.

4. Subjects Pl;P2; These 2 operators spent the majority of their time gathering sheep, and spent minimal time near the well exposed dip site. The tenax analysis, nevertheless, showed the presence of:

(i) aromatics which were known dip constituents

(ii) ketones, which, according to the manufacturers, are not dip constituents. Ketones are present in agricultural pesticide formulations. On this particular farm the dip was stored with pesticides: It is possible that the ketones collected by the sampler were present in the atmosphere of the store.

5. Subject P3; This man performed the actual dipping on the site where Pl and P2 were working. His tenax analysis showed the presence of glycol ethers. Although a glycol ether is present in this dip formulation, the 3 named in the analysis are not.

The results suggest that the co-formulants were very impure, which is confirmed by other evidence. If the co-formulants were so impure then the acceptable exposure recommendations would have been inappropriate. These results should have led to an analysis of the products to determine what people were being exposed to.

We have discussed the analytical and sampling techniques with both the laboratory scientists carrying out the analyses and the manufacturers of the equipment used (Perkin Elmer). There are, to date, unresolved differences of opinion with regard to the adsorbent material and gas chromatography columns used. If agreement is not reached before, a collection of duplicate samples at the proposed summer dip could allow comparison of the different techniques advocated. This may also resolve some of the apparent inconsistencies between clinical history, observations and laboratory results.

In retrospect, due to the dilutional factors created by monitoring in the open air, it may have been advantageous to use pumped equipment, particularly as we were looking for high molecular weight materials over a relatively short period of time.

Low levels of atmospheric contamination can only be analysed semi-quantitatively using thermal desorption techniques. This is because the errors may be as much as 50%.

TENAX RESULTS

CODE NO	SOLVENT PHENOL CODE	SOLVENT PHENOL PPM	SUBSTANCES DETECTED
Bl	Dl	0.3	Aromatic hydrocarbons
		0.7	Aliphatic hydrocarbons
X2	D2	0.1	Toluene
L4	D3	0.1	Xylene
Ll	D4	0.1	Xylene
Rl	D5	0.4	Isopropanol
P3	D6	0.1	Hydrocarbon
P3	D7	1.0	Glycol ethers
		0.04	Toluene
		4.0	Cresylic Acid
P2	D8	1.0	Toluene
		0.1	Methylethylketone
		0.1	Methylisobutylketone
Pl	D9	0.3	Toluene
S2	D10	0.05	)
		0.2	) Low C hydrocarbon
T	D11	0.1	Xylene & toluene
		0.1	Aliphatic hydrocarbons
U	D12	4.0	Cresylic acid
Q2	D13	0.2	Toluene & xylene
Q2	D14	1.5	Toluene
Ql	D15	0.2	Toluene & xylene
Ql	D16	0.2	Toluene

These results should have caused concern. Toluene, which is phenolic, is turning up repeatedly. Toluene is cited as causing the sort of neuropsychiatric damage that has been measured in OP sheep dip victims.

Blood Esterases

Research assays carried out subsidiary to the survey investigated whether the biological monitoring results, or symptoms reported, were related to the activity of other enzymes in the blood. The activity of two enzymes which metabolise the OPs paraoxon and chlorpyrifos were measured. These OPs are believed to have a pattern of metabolism similar to diazinon and propetamphos. Individuals were then categorised into slow, medium or fast metabolisers of paraoxon.

It is known from previous studies that approximately 50% of the population are slow metabolisers of paraoxon. Results in the survey accord with these findings. Although some subjects fell into this category there was no correlation with the measurement of cholinesterase and urinary dialkylphosphates, nor symptoms. It is only, however, at doses resulting in cholinesterase inhibition or cholinergic symptoms, that such a relationship would be demonstrable. The doses encountered in the survey were much lower than these levels.

Dip Samples

The dips were made up following the manufacturers' recommendations. Samples of dip taken before dipping started showed wide variation. One possible reason for this was inadequate mixing of the dip before the sample was taken. If the sample had been collected after the passage of one or two sheep, a more representative sample may have been achieved.

Samples taken at the end of the dipping day were expected to vary but to be above a minimum concentration. The replenishment rate of the dip is set to maintain the active ingredient at a level which ensures sufficient residual activity in the fleece. Again, concentrations varied and fell below the minimum.

Although the method of sampling was consistent throughout the survey no attempt was made to sample in accordance with MAFF procedures.

The results are summarised in the following table:

PRODUCT	Target Conc ppm	Actual Conc (Range) ppm	Target Minimum Conc ppm	Actual Minimum Conc (Range) ppm
Top Clip Gold Shield	400	116-264	100	113-286
Bayer Diazadip	400	241	100	168
Coopers Powerpack Winter	250	166	100	104
Youngs Ectomort Dip	320	74-148	125	44-62
Youngs Jason Winter Dip	280	111-169	125	20-42
Youngs Summer Dip	320	149	125	61

These analyses would not have identified impurities.

RECOMMENDATIONS

1. The design of some dips, particularly the older swim through type, could be modified to ensure that the forcing pen is separated from the dip. We noted several instances where the dipper almost fell into the dip as a result of sheep escaping from the pen and trying to run past the dip.

A forcing pen separated from the dipping tub would be ineffective.

2. Several operators experienced difficulty in reading the instructions on the tin as a consequence of both print size and spillage or rust on the outside of the tin. It would be beneficial if, in addition to the instructions printed on the tin, a separate water resistant card containing identical information was supplied by the manufacturer. This also would reduce the need to handle contaminated tins.

No mention of Safety Data Sheets. The manufacturers did not include them in the packs but were told by the VMD that this was a requirement.

3. Some manufacturers also supply protective clothing "free" with their dip pack (gloves and/or apron). In our experience the gloves supplied do not provide the required protection. If with all the resources available to them, a major chemical company proves unable to select appropriate protective equipment, what hope is there for an end-user? Manufacturers must make clear, specific recommendations for protective clothing; the use of bland, standard phrases such as "use protective gloves" is totally inadequate.

4. Some manufacturers provide measuring jugs with the dip concentrate, it would be helpful if all manufacturers adopted this policy.

5. Design of the 5 litre and 25 litre containers should be improved to reduce the "glug" factor which causes splashing.

The problem of can design has still not been overcome despite the products having been taken off the market twice for the matter to be dealt with. More seriously, the HSE makes noi mention of the fact that diazinon was packed in tinned steel, contravening WHO recommendations. Tin is a bivalent metal. Bivalent metals promote the formation of the highly toxic diazinon breakdown products. There was published evidence of serious poisoning being the consequence.

6. The introduction by one manufacturer of a system to automatically meter the dip concentrate during replenishment has obvious benefits for the operator. The process could be taken one stage further to enable the dip to be made up without the need to handle the concentrate.

Other manufacturers should be actively encouraged to develop similar systems.

7. Because diazinon gradually breaks down to other products during storage, it is advisable to use a fresh supply of concentrate for each dipping period. This is, however, not a practical option, as the dip is expensive and rarely packaged in volumes matching the requirements for smaller flocks.

If they are concerned about breakdown products in the can, why have they no analysed the contents? Are they going to recommend smaller cans or not? The issue of cost is a matter between the manufacturers and their customers.

8. To ensure even distribution of the dip concentrate throughout the dip bath the water-concentrate mixture is invariably stirred with the dipping stick before dipping starts. Because most dipping sticks have wooden handles, dip is absorbed along virtually the entire length of the handle and represents an immediate source of contamination for the dipper. If the stick is used in this way it should be rinsed off before further use. A better solution would be to utilize a metal handled dipping stick which will not absorb dip and can be more easily decontaminated.

9. Splashing to the lower body could be reduced for all dip types by the installation of a solid barrier between the dipper and the dip bath, which could take the form of a sheeted hurdle. Other advantages would be that the dipper would be less likely to overbalance and fall into the dip and it would prevent the booted foot being used in lieu of a dipping stick. The barrier should not be permanently fixed but hinged at one end to allow it to be released and moved quickly aside should emergency aid be required by an animal in the dip.

10. Dips are generally filled and replenished from a natural farm supply; however, when supply is limited, river water may be used. River or stream water carries the risk of leptospirosis particularly if used to wash out eye splashes or open wounds and therefore should not be used for this purpose.

11. Most operators claimed to have a supply of running water at the dip. In many cases this was a hosepipe, either connected directly to an automatic delivery system or submerged in the dip. We recommend that an independent supply be brought to the dip to ensure that clean water is always available. Uncovered water tanks near the dip are not suitable reservoirs for washing water as they become rapidly contaminated.

12 The results from our cholinesterase inhibition detection badges demonstrated that OP contamination of the skin was effectively removed by washing with soap and water. This reinforces the importance of rinsing off skin contamination when it occurs during dipping and during post-dip handling operations.

Where is the evidence for this conclusion? The conclusion is contradicted by the later CVL study.

13. On pre-dip interview, several farmers volunteered information regarding occasions when they had received eye splashes whilst dipping (as opposed to measuring the concentrate). During the actual survey we witnessed several eye splashes causing extreme pain and irritation. Despite this, most operators simply wiped their eye with their sleeve and carried on, even if water was available.

This is an observation, not a recommendation. Are they going to recommend that farmers should wear some sort of helmet and face-shield?

14. The presence of highly irritant substances such as phenols and epichlorhydrin in the dips and the propensity for eye absorption of other constituents, such as OPs, require appropriate facilities for eye washing to be available. Farmers should be advised regarding both the importance and correct use of this facility.

This, like most of the recommendations, is cosmetic. Eye-washing is too late for OP exposure as penetration is virtually instantaneous.

15. A major frustration and source of contamination for the mobile dip operator is the reluctance of the sheep to leave the vehicle after dipping due to the steepness of the exit ramp. These ramps could either be lengthened to provide a more gradual descent or additional battens added to provide better grip for the sheep.

16. The design of mobile dipping systems results in operators working in relatively enclosed spaces, the sides of these vehicles being constructed of sheet metal. To prevent the build-up of vapour, additional ventilation should be provided. This could be achieved by either replacing some of this solid sheeting with weldmesh or by installing suitable exhaust ventilation. Wherever possible the vehicle should be parked on an open exposed site. Adequate ventilation is particularly important in the case of mobile dippers as they drive their vehicles back to base after a long day's dipping, sometimes long distances.

17. The comments concerning ventilation are equally valid for anyone dipping inside a shed. The scope for installing a fixed ventilation system is much greater here than in the mobile situation. Because of the large area involved a dilution system, rather than a captive system would seem preferable.

Sheep should not be dipped inside a shed but MAFF was keen to offer grant-aid for dipping facilities that had unsatisfactory ventilation.

18. New dipping facilities should be constructed, as far as possible on open sites which allow the prevailing wind to disperse vapours away from the operators.

So dipping should take place only when the wind is in the right direction! Presumably dipping complexes should be equipped with weather-cocks and farmers should cease dipping if the wind changes.

19. Disposal of spent dip and cleaning the dipping bath are significant sources of exposure. In solution, diazinon degrades into other products. It is probably better, therefore, to dispose of the dip from the bath on completion of the dipping operation. Retention until the next dipping period would increase operator exposure to any breakdown products present.

"In solution, diazinon degrades into other products". *So, what's happening in the dip and on the sheeps' back? The HSE is worried about these products but does not specify what they are(TEPP etc) and does not analyse for their presence.*

Personal Protection

I The use of cholinesterase inhibition badges showed that normal splashing readily penetrates single layers of clothing - (shirt, trousers) whilst two layers appear to resist penetration. These results were confirmed by gauze patch estimations.

Badge tests were negative on skin protected by wellington boots.

These tests were also negative on the inside surface of leggings.

Tests on an apron 12 weeks after dipping gave a positive reading. The apron had, however, been used when lambing in the intervening period.

When dipping, the following items of protective clothing may be appropriate:

1. Wellington Boots

2. Personal Clothing (shirt and trousers) covered by:

i) waterproof leggings (polyurethane on nylon) with a short fisherman·s smock worn over the shirt

OR ii) overall (boiler suit) and apron

The use of leggings are probably more effective than an apron, particularly if the operator has to enter the draining pen, as they protect the entire leg. Bib and brace type leggings would provide better protection as they reach chest rather than waist height.

All waterproof protective clothing should be rinsed regularly during use and washed at the end of the day.

In view of the fact that virtually all of the compounds used in OP sheep dips, it might be more appropriate that all PPE should be used only once.

II A face shield is recommended by manufacturers when handling the dip concentrate. Some operators tried to dip while wearing a face shield because they had experienced eye splashes of dilute material. They found that vapour from the dip collected behind the visor so discontinued the practice.

Eye protection is recognised as advisable and, whilst there is resistance to wearing goggles, the use of chemical safety spectacles is a practical alternative. We have already mentioned the need for suitable eye washing facilities.

III The manufacturers of sheep dip recommend the use of protective gloves but do not specify the type. Gloves made of nitrile material resist penetration by petroleum distillate type solvents better than gloves made of either butyl rubber or neoprene. When tested against phenol in solution, neoprene and butyl rubber gloves provide greater protection. It is because of its greater resistance to solvent penetration that nitrile is recommended by glove manufacturers for use with sheep dip. Thicker nitrile will give greater protection, but there will come a point at which flexibility will be compromised.

The protection afforded by gloves is dependent not only on their being made of the correct material, but also on their correct use; ie, being rinsed off after use, internal contamination being avoided and being discarded when damaged or their recommended life is exceeded.

The majority of farmers use gloves, usually of the incorrect type, when handling the concentrate. The gloves are then put down near the dip until they are required for the next addition of concentrate, offering ideal conditions for contamination. It may be preferable if gloves were not worn, but the container and measuring device rinsed with clean water and hands washed after addition of the concentrate.

During dipping, the hands do not appear to become more contaminated than other parts of the body. They are more likely to be washed at intervals, hence there would seem to be no need to wear gloves during the dipping operation, unless a wooden handled dipping stick is used.

Four people were observed during the survey wearing gloves for dipping. Three were using the wrong type (natural rubber). One operator continued to wear a glove when it was badly torn, thus trapping dip inside, allowing continual absorption for the remainder of the day.

IV Four operators wore respiratory protection. Of these two wore disposable preformed masks (3M Farmer's Lung Mask) and two powered helmets (Racal Airstream).

In most circumstances this type of protection is not necessary. Atmospheric monitoring results show that dilutional factors at open sites should be sufficient to disperse the vapours present around the dip. At enclosed sites (mobile and covered dips) the problem should be tackled at source.

Disposable masks, of the type worn, are ineffective against volatile products. Tests on one mask showed it to be contaminated with OP, possibly as a result of splashing. This suggests the mask could act as an additional source of contamination. The only disposable mask which may provide some protection is one containing activated charcoal. This type is however recognised as being difficult to breathe through and therefore unsuitable for heavy manual work.

The powered helmets worn were fitted with activated charcoal filters. These are recommended only as "odour filters for non-hazardous substances below the OES". One of the operators commented that he could detect odours even when a new filter had been fitted, indicating breakthrough, and that on occasions he felt unwell after dipping (battery pack fully charged).

Future Investigations

1. We recommend that the summer dip 1991 be used to follow-up the survey for the following reasons:

   (i) The majority of people complaining of symptoms state that they experience them at this time.

   (ii) The atmosphere will contain a greater concentration of volatile substances.

   (iii) Other OPs; for example, chlorfenvinphos, will be used. This compound is more volatile than either diazinon or propetamphos. It is absorbed into body fats and is slowly released creating a more prolonged effect.

   (iv) Different delivery systems may be used which result in greater operator exposure.

2. We propose the following strategy:

   (i) To use the same population as that used for the autumn study.

   (ii) Urine samples for dialkylphosphate measurements to be collected from diazinon and chlorfenvinphos users at:

      (a) the end of the dipping day
      (b) 24 hours after the post dip sample
      (c) 48 hours after the post dip sample

      Whilst 24 and 48 hour total collection would be ideal it is impractical in all but one or two cases.

(iii) Atmospheric monitoring for volatiles using:
  (a) static tenax at dip
  (b) pumped and passive personal samplers in parallel at same sites (exposed and enclosed)
  (c) if appropriate, to use alternative adsorbent materials, or different gas chromatography columns

(iv) Blood sampling may be carried out to test for the presence of solvents and to analyse for specific esterases which may determine the rate of metabolism of the OPs in use.
Cholinesterase estimations are not proposed.

(v) To determine the presence of breakdown products of diazinon in dips which have remained in the bath since the previous dip; in partially used tins of concentrate

(vi) To review bulk dilutions using recognised sampling techniques at appropriate intervals.

(vü) To use the opportunity to discuss the feasibility and effectiveness of protective clothing recommendations with individuals and assess the provision of eyewash facilities.

As far as is known, these recommended further studies did not take place. That is not surprising in view of the fact that there was already evidence that the products were unsatisfactory. It would have unethical to carry out further studies. They have even concluded that the observers were be affected by their minimal exposures.
In addition, the HSE should have observed that its methods were inadequate.

CONCLUSIONS

Environmental monitoring demonstrated low levels of atmospheric contamination near dipping baths.

None of the results of biological monitoring were in the significant range.

The fact that no dramatic results were forthcoming does not indicate the absence of a problem, rather that the cause is more complex than originally envisaged.

This survey has carried out the basic work required in order to set targets for the future. It has also enabled new laboratory techniques, eg. dialkylphosphate estimations to be tested under field conditions and facilitates further work on esterase phenotyping.

The fact that exposure to OPs during dipping occurs at a relatively low level makes monitoring much more difficult. The limitations of certain techniques have been illustrated.

This report highlights other factors which may contribute to the symptoms reported by sheep dip users.

It has been inferred that problems associated with the use of sheep dip chemicals are encountered only in the South West of England. Reports of ill-health, however, are distributed throughout the country. Certain factions of the farming industry are convinced that compulsory dipping for sheep scab will cease in the near future. Whether or not this proves to be the case, sheep will continue to be dipped for other purposes as well as reactively when a scab outbreak occurs.

Scientists indicate that resistance may be developing to the OPs in use. This, would, in fact, give rise to the demand for stronger, more potent dips, which will demand stronger, more positive answers.

Resistance, a decade ago, but diazinon and propetamphos remain in use.

APPENDIX 1
LABORATORY METHODS

References are given for published methods where appropriate, in other cases a resumee is given.

1. Blood Cholinesterase
   Lewis P E, Lowing R K, Gompertz D. Automated discrete kinetic method for erythrocyte acetylcholinesterase and plasma cholinesterase. Clinical Chemistry (1981) 27. 926-929.

2. Serum Esterase Activities
   Three specific esterases were analysed:

   (i) Serum carboxytesterase
   Sterri S H, Fonnum F, Johnsen B A. A radiochemical assay method for carboxylesterase, and comparison of enzyme activity towards the substrates methyl [1-14C] butyrate and 4 - nitrophenyl butyrate. Biochem Pharmacol (1985) 34 (15), 2779-85.

   (ii) Serum Paraoxonase
   W H O Technical Document
   Organophosphorus Pesticides: An Epidemiological Study. World Health Organisation. Copenhagen, 1987 (Env Health series no 22), 103-107

   (iii) Serum chlorpyrifos-oxonase
   Furlong C E, Richter R J, Seidel S L, Motulsky A G. Role of genetic polymorphism of human plasma paraoxonase/arylesterase in hydrolysis of the insecticide metabolites chlorpyrifos oxon and paraoxon. Am J Hum Genetics (1988) 43, 230-238

3. Blood Solvents

Solvents were released from the blood samples and absorbed using Tenax tubes. These were analysed using thermal description, capillary gas chromatography and an ion trap detector.

4. Urinary Phenols and Dialkyl Phosphates

Evaluations were performed by extraction - analysis using capillary gas chromatography and flame ionisation of flame photometric detectors.

5. Tenax Passive Samplers

Wright M D. A dual capillary column system for automated analysis of workplace contaminants by thermal descorption. Analytical Proceedings (1987) 24, 309-311

6. Bulk Dip Samples

Branchflower W J, Rice D A, Hamilton J T G. Determination of propetamphos and diazinon residues in sheeps wool. Analyst (1987) 112, 1761-1763.

REPORTS OF SYPTOMS

	< THIS DIP								>	< PREVIOUS DIPS																				>
SUBJECT CODE	X2	B1	A1	A2	L2	L4	O1	O2	Q1	T	C	B1	E	A2	D	H1	G3	X2	L1	N1	B2	O1	Q1	W2	S1	T	U	V	W1
SYMPTOMS																													
HEADACHE		X							X		X	X	X				X		X						X	X	X	X	
THIRST			X						X					X									X						
TIREDNESS		X									X	X	X	X															
PARAESTHESIEAE				X										X															
SORE THROAT					X	X	X	X																					
NAUSEA																				X	X								
VISION														X															
CHEST	X														X														

The above table did not scan and has been re-typed.

APPENDIX 3

1. Richard G Ames et al, Protecting Agricultural Applicators from Over-Exposure to Cholinesterase-Inhibiting Pestides: Perspectives from the California Programme. J Soc Occup Med (1989) 39, 85-92.

2. Guidance Note MS 17 from the Health and Safety Executive, Biological Monitoring of workers exposed to organo-phosphorus pesticides.

3. Shuyang Chen et al, An epidemiological study on occupational acute pyrethroid poisoning in cotton farmers. British Journal of Industrial Medicine (1991) 4H, 77-81.

4. H J Mason, P J Lewis, Intra-individual Variation in Plasma and Erythrocyte Cholinesterase Activities and the Monitoring of Uptake of Organo-phosphate Pesticides. J Soc Occup Med (1989) 39, 121-124

5. C A Franklin et al, Correlation of Urinary Pesticide Metabolite Excretion with Estimated Dermal Contact in the Course of Occupational Exposure to Guthion, Journal of Toxicology and Env Health, (1981) 7, 715-731.

The table of results for the total population would not scan and has been omitted.

OVERVIEW

It has taken ten years for this report to emerge. It was seen on the first page that the report was regarded as secret by the HSE.

There is reason to believe that this report did not reflect the HSE's concern over OP sheep dips. The report is dated May 1991 but it is known that by March 1991 the HSE had called a meeting with the manufacturers and had drawn attention to the concern over the solvents and phenols. That was the start of the process that led to these products being withdrawn from the market and replaced with altered versions. It is also known that one of the authors of the report approached subjects of the study to say that there were serious problems with the OP dips, that these related to the solvents and phenols and that the formulations would have to be replaced.

The methods used in the study are shown to be inadequate.

The HSE study was based on conventional assumptions that camouflage the hazard from OP impurities, which is another matter over which the HSE has been secretive. Documents drawing attention to the presence and toxicity of diazinon impurities have been published since the 1950s but this information seems not to have been taken into account. The hazard from OP impurities was drawn to the attention of Government in 1989 but seems to have led to no immediate action.

By 1995 there was a clear trail of evidence indicating that around 1991/2 the Government had concluded that the OP dips were unsafe because of the inerts ***and*** *the OP impurities and had ordered that the formulations be changed.*

However, The Government repeatedly denied this and claimed that the products had not been monitored. If that claim were true then the Government had failed to properly apply the Medicines Act, which required that products be satisfactory in ***quality****. Despite its denials, the Government has recently been forced to admit to a study of the impurities carried out around 1992. The Government will not reveal the contents of the report on that study.*

It is also clear that at about the same time, around 1992, it was recognised that post-dipping exposure was probably more dangerous that exposure at dipping. The VMD ordered a study of post-dipping exposure which was carried out in 1993. The decision to withdraw the current formulations had already been taken but current formulation was used ***with the phenolics removed****. The VMD concluded from the results of this study that there had been a high exposure to diazinon from handling just six sheep after dipping. If that diazinon had contained the high-toxicity impurities then the exposure would have been even more hazardous.*

Below are appended:

1. A recent HSE Alert on Phenols
2. A Current HSE web-page

This is an incomplete scanned version of a recent HSE Alert on Phenols. Some of the contents are highly questionable. Eg the claim that phenols are not manufactured in the UK. The OP sheep dip certainly contained crude mixtures of phenolics that were UK manufactured.
The HSE has withdrawn the previous OES for phenols but does not explain why? Presumably the HSE has evidence to suggest that the old OES was unsafe. Exposure to the pre-1994 OP sheep dips resulted in an exposure above the old OES because those products had a strong phenolic smell and the threshold for being able to smell phenol is 5 ppm, ie the same as the old OES.

CHEMICAL HAZARD ALERT NOTICE - PHENOL
JUNE 2000
This guidance is issued by the Health and Safety Executive. Following the guidance is not compulsory and you are free to take other action. But if you do follow the guidance you will normally be doing enough to comply with the law.
This guidance provides information on the health effects associated with exposure to phenol at work. It also gives advice on good practice, which employers, users and suppliers may find helpful in considering what they need to do.

Why issue a chemical hazard alert notice?
Phenol currently has Occupational Exposure Standards (OESs) of 5 ppm (20 mg.m3), 8-hour time weighted average and 10 ppm (39 mg.m□3),15-minute short-term exposure limit (STEL). These limits were established some years ago, and have recently been reviewed by an independent committee of experts in occupational health. Because of the information now available on the health effects of phenol, the committee could no longer identify a level which is both safe and practicably achievable. HSC is therefore consulting on the withdrawal of the current OESs from 2001.

For substances where no exposure limit is set, employers should determine their own working practices and in-house standards for control so that repeated exposure does not cause ill-health. Because no safe exposure limit for phenol could be identified, the Health and Safety Commission's Advisory Committee on Toxic Substances will consider, in due course, setting a maximum exposure limit (MEL). A MEL places a duty on the employer to reduce exposure to as low as is reasonably practicable, and in any case below the MEL. It takes some time to set MELs. Once any MEL is set, the Control of Substances Hazardous to Health (COSHH) Regulations 1999 will clearly identify responsibilities. This guidance provides interim advice and information to suppliers, employers and users.

http://www.hse.gov.uklpubns/chan20.htm#3

What is phenol? - Phenol is a white crystalline solid which liquefies on contact with water.
It has a characteristically acrid odour and a sharp burning taste.

Where is it used? - It is used as a starting material for the production of a variety of chemicals, the most important being phenolic resins. It has a minor use in specialised paint strippers. Phenol is not manufactured in the UK.

What is the key health hazard? - Phenol is corrosive and diluted preparations of phenol solutions may also burn or irritate the skin. Recent evidence indicates that phenol can be genotoxic, which means it can cause changes to the genetic material in the body.

How does it get into the body? - Information from humans and animals shows that
phenol is well absorbed when swallowed, breathed in or in contact with the skin.

What should suppliers do? - You should ensure that the information contained in this notice is passed on to your customers as required by the Chemicals (Hazard Information and Packaging for Supply) Regulations 1994, as amended. You should take steps to review your safety data sheets to reflect the new findings.

What should employers do? - You should give priority to preventing your employees being exposed to phenol by any route (ie breathing in dust, mist or vapour, contact with the skin and swallowing).
Where preventing exposure to phenol is not reasonably practicable (e.g. by using a different substance), then you should adequately control exposure by a combination of engineering and process control measures. HSE recommends that, although the legal obligation is to reduce exposure to the OESs while they remain in force, it would be prudent for you to control exposure to as low a level as is reasonably practicable below the OESs.
Once the OESs are withdrawn, your legal obligation under COSHH remains to
achieve adequate control. Since a safe level of exposure cannot be determined it
remains our recommendation that you should control exposure to as low a level as is reasonably practicable.
In dealing with exposure, whether before or after the OESs are withdrawn, you should try to reduce the number of people exposed and the length of time each is exposed as required by good hygiene practice.
You must give all your employees who are, or who may be, exposed to phenol
sufficient information, instruction and training to understand the potential problems and the precautions they need to take.
You should make sure that employees, safety representatives or representatives of employee safety are aware of this information and consult on any action that you propose to take as a result.

What should employees do? You must co-operate with your employer in using the control measures (such as ventilation and PERSONAL PROTECTIVE EQUIPMENT) provided and reporting any defects found in the control measures.
You may wish to seek the advice of your safety representative or representative of employee safety.

Further information is contained in the next issue of EH64 available from HSE Books.
Further help: Contact HSE's InfoLine Tel: 0541545500
http://www. hse.Gov.uk/pubns/chan20.

This is a text version of a current HSE web-page. It states that the HSE and local authorities have responsibility for safe use of veterinary medicines, including sheep dips. But local authorities do not have the right to know the formulation of licenced medicines and it would appear that until 1991 the HSE did not know what OP dips contained. It is clear that the HSE failed to discharge its responsibilities in respect of OP dips. The list of relevant literature includes MS17, which the HSE had kept secret, even from its own area offices. The other two documents mentioned would be of interest.

Veterinary medicines (including sheep dips) Issue:
HSE's role in ensuring the safe use and disposal of veterinary medicines. Current legal Base and any legal developments:
Veterinary medicines are products which have been authorised by Agricultural and Health Ministers under the Marketing Authorisations for Veterinary Medicines Regulations 1994 (which implements EC single-market legislation) and the Medicines Act 1968. The Veterinary Medicines Directorate (VMD), an Executive Agency of MAFF, has primary responsibility for the authorisation scheme for veterinary medicines; and for their supply. Responsibility for their use at work falls to HSE and local authorities through the Control of Substances Hazardous to Health (COSHH) Regulations and the Health and Safety at Work etc Act.
Key Messages:
HSE ensures that its views are fed into the authorisation system through liaison with the VMD and by offering occupational hygiene advice to the Veterinary Products Committee, which advises licensing Ministers on applications for veterinary medicine marketing authorisations. HSE also seeks to control the risks arising from subsequent occupational use - the main HSE activities contributing to this are: a) research funding - a programme of HSE-sponsored research on veterinary medicines is co-ordinated through HSE's Pesticides and Veterinary Medicines
Research Subgroup.
b) Guidance - a comprehensive leaflet AS29(rev2) 'Sheep Dipping' gives simple step-by-step advice on all aspects of safe dipping and how to comply with COSHH.
Leaflet AS31 'Veterinary medicines - Safe use by farmers and other animal handlers' gives guidance on how to comply with COSHH and work safely with all veterinary medicines. Leaflet INDG141(rev1) 'Reporting incidents of exposure to pesticides and veterinary medicines', advises people what to do if they think that people, animals or the environment have been harmed by exposure to veterinary medicines (or pesticides). HSE videos giving advice about using veterinary medicines safely include: 'COSHH in Agriculture' UK 4114, which in a 'real life' setting shows how to carry out a risk assessment and gives practical advice on complying with COSHH; 'Sheep Dipping' UK 4223, an instructional video on safe dipping; and 'Staying Healthy' UK 4378, an award-winning video which highlights the main health risks to people working in agriculture. These videos, together with other display materials which help veterinary medicine users protect themselves, are frequently featured on HSE stands at agricultural shows and can be bought or hired from HSE videos (see below for the address).
c) Advice - HSE inspectors routinely give advice during visits to workplaces, including farms, on how to comply with COSHH and the measures necessary to protect health. Where necessary, Inspectors use their enforcement powers to ensure risks are properly controlled.
Sources of Further Information: Abstract of Medical aspects of work-related exposures to organophosphates.HSE Guidance Note MS 17.
Epidemiological study of the relationships between exposure to organophosphate pesticides and indices of chronic peripheral neuropathy and neuropsychological abnormalities in sheep farmers and dippers - overarching summary.
The Veterinary Medicines Directorate Website:
http://www.open.gov.uk/vmd/vmdhome.htm
The HSE publications listed, and all other HSE publications, can be obtained by
Mail Order from: HSE Books, PO Box 1999, Sudbury, Suffolk CO10 6FS.
Tel: 01787 881165 Fax: 01787 313995
The HSE videos listed, and all other HSE videos, can be ordered from:
HSE Videos, Dept HV, PO Box 35, Wetherby, West Yorkshire LS23 7EX.
Tel: 0845 741 9411 Fax: 01937 541083