Chemical Hazards and Poisons Report

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Contents

Editorial

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In this issue, our incident response section focuses on some recent significant incidents and toxicological issues of concern:

- A recent bonemeal fire incident in the East Midlands that generated considerable local public concern.

- Carbon monoxide is a major toxicological cause of death in the UK. A case study reports carbon monoxide poisoning at an indoor go-karting track. A second article on carbon monoxide considers two different types of oxygen therapy for carbon monoxide poisoning.

- Lead poisoning is a significant toxicological issue. Two articles provide further data on this: the first revolves around lead toxicity in adults and unexplained anaemia illustrated by two case-studies reported to the Chemical Hazards and Poisons Division by a consultant haematologist and the second reviews public health and lead poisoning.

- To continue the theme of haematological concern a review of the public health implications of methaemoglobinaemia is related to the management of a paediatric case.

As always, emergency preparedness issues are again identified as important. The articles presented in this issue cover two exercises, one focusing on COMAH site planning and the other exercising the multi-agency strategic response to a chemical, biological, radiological or nuclear (CBRN) incident and the recovery stage of such an incident.

Environmental issues are of note and in this issue we continue our series of articles on air pollution with: 'Air Quality Strategy for England, Scotland, Wales and Northern Ireland: new approach to the regulation of fine particles' and 'Particles as Air Pollutants: (I) The ambient aerosol'. Further articles in this series will appear in subsequent issues. This section also provides a report on the use of Geographical Information Systems (GIS) for exposure assessment. Risk communication to the public is always complex. A paper is included related to issues surrounding public communications during water-related chemical incidents.

A series of conference and training reports are included. Of particular interest are Traumacare 2006 that took place in Agra, India in September; the 5th Air Quality Pollution Forecasting Seminar; and the European Educational Programme in Epidemiology (EEPE) 19th summer course in Epidemiology.

The next issue of the Chemical Hazards and Poisons Report is planned for April 2007. The deadline for submissions for this issue is February 1st 2007. Please do not hesitate to contact us about any papers you may wish to submit. Please contact us on chap.report@hpa.org.uk, or call us on 0207 759 2871.

We are very grateful to Professor Gary Coleman for his support in preparing this issue.

Incident Response

The case of a smouldering bonemeal fire: consistent complications and the power of public concern

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Introduction

Bonemeal is used in power stations as fuel; having the calorific value of brown coal. Stable under normal conditions, temperatures above 46°C lead to self-heating and spontaneous ignition above 67°C. Gaseous combustion products may pose a human health risk; potentially containing: hydrogen cyanide, organic nitriles, polycyclic aromatic hydrocarbons (PAHs), dioxins and carbon monoxide.

On the afternoon of Thursday 1st September 2005, East Midlands Health Protection Unit (HPU) North, Grantham office was contacted by the Local Authority (LA) for advice concerning a spontaneous fire in an aircraft hangar at a disused RAF airfield near Market Rasen, Lincolnshire. The hangar was filled to the ceiling with 24,000 tonnes of bonemeal. Smoke had first been seen escaping from the eaves of the building six days previously and a changeable light breeze was now blowing from the southwest towards residential properties 500 metres downwind.

A media report from the previous day revealed that the Environment Agency (EA) was monitoring the situation and that a LFR (Lincolnshire Fire & Rescue) spokesman had announced that the fire posed no danger to the public. As there were no visible flames and the location of the seat of the fire was unknown, LFR would return if necessary when the smoldering bonemeal was isolated and removed, which would be coordinated by the owner.

Box 1: Rendering

The process of rendering is used to sterilise animal waste by crushing animal by-products, heating to remove water, and separating into fats (tallow) and solids. Bonemeal is formed from the ground solid component and was used in the chemical industry and as a constituent of soaps, cosmetics, and particularly animal feed. Following the BSE crisis, regulations prohibited the use of animal protein within animal feed and restricted the disposal of animal by-products to landfill. The market value of bonemeal subsequently declined and in 2000 an estimated 470,000 tonnes remained in storage. Currently, DEFRA estimates that 60,977 tonnes of bonemeal from BSE-related slaughter schemes is in stores nationwide[1].

Acute Phase

Initial concern centered on Bovine Spongiform Encephalitis (BSE) risk. Investigation via the Department for Environment, Food and Rural Affairs (DEFRA) and local State Veterinary Service (SVS) established that

this was negligible as no 'Category 1' material (animals suspected or confirmed as BSE infected) was present. The local HPU sought information and advice from the HPA East Midlands Chemicals and Environmental Team (CET) regarding potential emissions, particularly of dioxins and particulates.

The following day, Friday 2nd September, the local general practitioner (GP) contacted the HPU with the news that 6-7 patients had complained, by telephone, of sore throats and eye irritation. The LA reported steadily escalating public concern and dispatched an Environmental Health Officer to assess the site. The regional HPA press office was briefed, and after consultation between the HPU and CET/Chemical Hazards and Poisons Division (CHaPD), a CHEMET (plume forecast produced by the Met Office) was requested. The LA and LFR assessed the site as posing no public health risk; a holding press statement was agreed by the HPA and issued, which reiterated the low risk to public health and adopted 'watching brief' stance.

The weekend passed without incident, with no health complaints nor contact with on-call resources. The site, watched by LFR and the LA's Environmental Health and Trading Standards officers, showed no apparent change on Monday 5th, with a light plume of smoke now drifting away from residential properties. However, public complaints and press interest directed at the LA were unrelenting, and there was a loss of press and public engagement due to the delayed arrangement of an inter-agency incident meeting, which was finally scheduled for the coming Thursday, 8th September.

Attendees included representatives from: LFR, West Lindsey District Council, Lincolnshire County Council, the HPA/PCT, the EA, and the owner. The LA chaired the meeting, although no agency took lead responsibility for the incident. The public health risk was reviewed and three main options were discussed:

- Moving material off-site to locate and remove the seat of the fire,

- Damping down with water, or

- Taking no action, as the fire posed no risk to the building.

LFR estimated that the fire could burn for 8-12 months, but felt that the risks of intervening (i.e. flaming, or dust explosion) outweighed the risks in leaving the fire smoldering.

The LA indicated that, despite the number of complaints, they felt that statutory nuisance was not substantiated, but they would facilitate direct communication with residents.

The group retained a 'watching brief' stance and a joint press release and live radio and television interviews followed this first incident meeting; stating that there was no evidence of risk to the public from the fire. A follow-up meeting was scheduled for the following week.

Despite direct contact with both residents and the media, press interest in the incident continued and the local population remained active, with interviews held outside the hangar itself. Public concern over a perceived lack of action grew, to such an extent that allowing the fire to burn was considered untenable by the time of the second incident meeting on Thursday 15th September.

In view of the endurance of the fire, LFR considered that it was unlikely to resolve itself if left, and agreed that action was required. The only feasible option was to remove enough un-burnt material from the front of the hangar to create room to dig to the seat of the fire, and extinguish burning material after dispersal on the hangar floor. The owner agreed to source transport and arrange removal so that work could begin the following Monday (19th September). Shortly after the incident meeting, the LA granted short-term planning permission for the removed bonemeal to be stored at RAF Binbrook. A joint press statement was released the following day explaining that, although the multi-agency group had decided to act, the fire was not considered to present a significant risk to public health.

As the original public health risk assessment was based on a smouldering fire it did not evaluate implications of the anticipated remedial actions. The risk of flaming and practicalities of removal and material transport between sites were discussed by the incident team. Communication issues meant that, at this point, no environmental monitoring had yet been undertaken to confirm the original public health risk assessment.

CET/CHaPD arranged a site visit for 10:30, Tuesday 20th September, a day after work commenced to remove material. Details of a sampling regime were finalised and occupational health monitors were requested for the owner's operatives. The owner was pressed to provide security personnel to prevent public access to the site.

Events then escalated rapidly:

- 14:15: a worker informed LFR that flames had been seen on the summit of the bonemeal stack. The site was now controlled by LFR as a fire incident.

- 14:50: the HPU were informed by a fire officer that a swathe of bonemeal near the rear of the hangar was burning. As smoke appeared unchanged, LFR were not issuing sheltering advice.

The hanger as seen from the nearest residences (500m north-northeast) ©LFR, 2005

- 16:00: a side panel was removed from the hangar to allow smoke to vent, but the rear of the hangar was inaccessible; hampering LFR's operation.

- 16:15: contractors were employed to remove the hangar's rear panels and dig out the concrete immobilising the rear doors. Fire fighting was scheduled for the following day, whilst the increased activity on-site led to queries from concerned residents.

Sampling results (presented six days later) showed levels of benzene and toluene that exceeded the WHO air quality guidelines at the hangar entrance, but were very low 400m downwind, supporting the HPA's risk assessment. Operations were temporarily suspended whilst advice was sought from the Health and Safety Executive (HSE) after concern had been raised over appropriate personal protective equipment (PPE) for the owner's operatives, who were removing bonemeal on-site.

As with all incidents, added complications were encountered by the multi-agency group. A residents' enquiry under the Freedom of Information Act obtained conflicting letters from SVS/DEFRA; one of which stated that the material stored in the nearby hangar was 'Category 1' (animals suspected or confirmed as BSE infected). After intensive consultation, this later proved to be an unfortunate secretarial error. Further misfortune was encountered when explosions and smoke caused further public concern, but proved to arise from ill-timed testing at a nearby munitions factory.

The next incident meetings held on Wednesday and Friday (21st & 23rd September) covered ongoing issues, and addressed recent concerns raised by the public. LFR expressed concern that operations could be hindered by the absence of arrangements for disposal of wet bonemeal; a problematic issue that was discussed at great length and indeed did cause delays. It was subsequently decided that wet material would be landfilled and drier material would be mixed with dry bonemeal and incinerated. Planning permission for the temporary storage site at Binbrook was extended by the LA and it was agreed that the incident be declared closed once the damp material had been removed from the incident site and Binbrook had been cleared.

Box 2: Bonemeal Disposal

Legal disposal solutions were either rendering or incineration. Whilst available for undamaged bonemeal, the capacity for wet, or burnt, bonemeal was extremely limited and it appeared that disposal to landfill was the only realistic option, which would require a Minister-cleared decision by Defra. This pre-necessitated that all other options be explored and documented evidence of capacity problems produced.

Defra's final response placed the onus on the group to justify decisions on disposal: advising that the authorities take a pragmatic approach to enforcement of regulations, whilst also requiring continued searching for normal disposal routes.

In the interests of both the owner, and the agencies involved, the amount of landfilled material was minimised, but for a proportion of the cleared material no alternate disposal methods existed.

LFR recommended their response on the 28th of September and the fire was finally declared out on September 30th. It had burned for a total of 35 days.

The incident closed on December 21st after the action plan devised at the last incident meeting (on October 7th) went smoothly, with a less-than-anticipated 800 tonnes of material eventually sent to landfill. Media misinformation continued throughout the disposal phase; remaining an issue to be addressed on an ongoing basis.

Outcomes

In the wake of the incident, Defra committed to consult the rendering industry and examine best practice guidelines (including proper bay storage).

Lessons identified / Points to consider

Historic concern over the storage of BSE-related bonemeal led to a high degree of local sensitivity regarding potential health impacts. The past presence of a residents' action group who had opposed planning permission for the hangar's use meant that local residents were organised, and vocal, from the outset of the incident.

The delay in arranging the first inter-agency meeting and subsequent delays in circulating action points and points of agreement may have contributed to a loss of control over the public reaction to the fire.

However, in the earliest stage of the incident, LFR had released a statement that the fire posed no risk to the public, which was subsequently accepted by the HPA and other agencies.

Public reaction fuelled media coverage that became disproportionate to the actual risks.

Following local provision of information by the LA and a statement of the multi-agency group's intent to act, both complaints and media coverage diminished significantly. Direct action fully addressed the concerns of disaffected residents, which was not the case in the first weeks of the incident when a watching brief stance was adopted; leading to vocal public interaction with the media.

Thus, in locations where there is pre-existing public anxiety over the possibility of risk to health, a relatively minor incident may trigger a disproportionate public reaction that is antagonised by perceived inaction and can only be sated by a reactive response.

As no agency had a remit for complete control, management of the incident did not conform to the accepted hierarchy of a 'typical' acute incident. When the individuals responding were impelled to act, the scope of their actions was restricted to those that fell within the remit of their organisation. Thus, resolving the incident required both considerable negotiation and in-depth awareness of the roles and responsibilities of partner agencies.

Wider issues may be important. In this case the past management of a highly politicised situation (the BSE crisis and subsequent storage of bonemeal) was intrinsic to the incident's complexity. The direct association between Bonemeal and the BSE crisis clouded many aspects of the incident's management: from public and media interaction, to the complications of material disposal.

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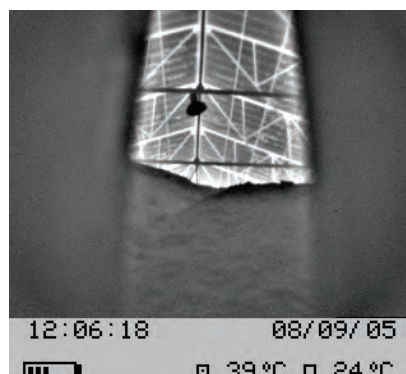
Thermal image showing uniform temperatures below 40ºC on the outer surface of the bonemeal pile   ©LFR, 2005

Removal of bonemeal to find the seat of the fire

© HPA-EM, 2005

Smoke-obscured bonemeal at the site of the removal operations

© HPA-EM, 2005

Carbon monoxide poisoning at an indoor go-karting track

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Incident Summary

In December 2005, seven cases of carbon monoxide (CO) poisoning were reported to the Leeds Health Protection Team. All the cases had been admitted to hospital following a birthday celebration at an indoor go-karting track. Patients carboxyhaemoglobin levels were measured at between 14.1– 18.3% on admission to hospital. Reported symptoms included headache, nausea, vomiting, dizziness, disorientation, slurred speech and 'shaking'. All cases were observed overnight, treated with 100% oxygen and discharged the following morning. Further investigation of the incident revealed that 34 attendees at the event were potentially exposed and many had experienced symptoms although only seven had presented to medical services. A health questionnaire was sent to all 34 known attendees at the event, to which 20 (59%) responded; 19 (95%) of the responders experienced some symptoms consistent with carbon monoxide poisoning. A site visit found that the exposure was due to high levels of CO being emitted from new Liquid Petroleum Gas (LPG) karts that were being used for the first time. The karts were withdrawn immediately from use to prevent further exposure. Anecdotal evidence suggests that this is not an isolated incident at karting tracks and there appears to be a lack of awareness of the dangers of CO poisoning among regular karters and track owners.

This incident summary has been written to help to alert colleagues in health protection to the risks of CO poisoning at indoor go-karting tracks and for them to have the opportunity of sharing this with colleagues such as Environmental Health Practitioners, Emergency Planning Officers and local Emergency Departments.

Background – indoor karting

Go-karting became popular during the 1990s and is seen as both a hobby and a sport that can be a route to professional motor racing. It has also become increasingly popular for corporate events and parties.

Description of incident and investigation

In March 2005, a Local Authority Health and Safety inspection at the site included trackside CO measurements. A build up of CO during the course of a race was identified. At certain CO levels, re-burning of CO rich air can lead to a rapid increase in concentrations. The company were advised to monitor CO levels and given advice about reducing CO levels by increasing ventilation.

Shortly before December 2005, the karting venue purchased some LPG karts which used removable gas cylinders. The 4th December 2005 was the first time that the new karts had been used.

On December 5th a medical Senior House Officer (SHO) called Leeds HPU to report that 7 people had been admitted to hospital overnight and had been discharged that morning. The Local Authority Health and Safety team visited the premises on the 6th December and served notice on the company prohibiting them from racing without monitoring CO levels.

From contact with four of the seven cases that were admitted to hospital, it emerged that a larger number of people had been affected during the event. The race, which was an 'endurance event' (one long continuous relay race), began at approximately 18:00 hours and was stopped early by the staff at approximately 20:00 hours. The majority of those attending displayed symptoms of vomiting and dizziness, one person was said to have collapsed in the canteen area and several had difficulty walking. The group that attended hospital were not regular karters.

The track owners were contacted and a list of participants was requested. Names and contact details were obtained for 34 individuals of whom only 5 lived within the local area, 7 lived within the wider West Yorkshire area and the remainder were widely dispersed in North Yorkshire, South Yorkshire, Humber, Lancashire and Derbyshire. Other local Emergency Departments were contacted but no further admissions or attendances from this event were identified.

A further 6 people were contacted by telephone to establish their symptoms and whether they knew of others who were seriously affected. It became clear that most of the remaining people who attended were regular karters who did not consider the health effects they experienced at the event to have been particularly serious. Most described feeling similar symptoms before at other karting events across the country. While all reported symptoms, none had sought medical attention and it seems that most people drove home afterwards. The karting centre identified one man as having been the worst affected and he was contacted to check on his wellbeing. He had recovered fully after initially experiencing severe incoordination, dizziness and confusion. He was driven home by a worker at the karting centre after the end of the race.

Health questionnaires were sent to all 34 of those who attended the event on the 7th December and also members of staff present who might also have been affected; 20 of the 34 responded giving a response rate of 59% (17 males, 3 females with an age range of between 15 and 54 years). All respondents said that they were otherwise fit and well and only 5 of the 20 were smokers.

Notes were retrieved for four of the seven cases admitted to hospital. These showed that they arrived at the Emergency Department at 21:30 hours, following the end of the race at approximately 20:00 hours. Blood samples were taken between 22:00 and 23:00 hours. Carboxyhaemoglobin levels of between 14.1% and 18.3% were measured. This was approximately 2-3 hours after the race ended and therefore the time at which the exposure can be reasonably thought to have ended. While it is not possible to calculate the likely peak concentrations of carboxyhaemoglobin, earlier levels would obviously have been higher.
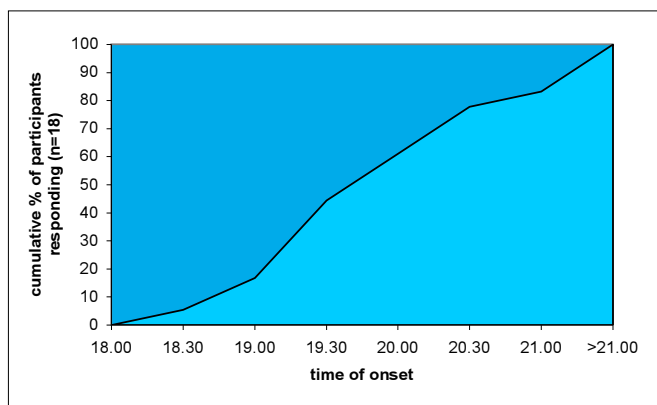
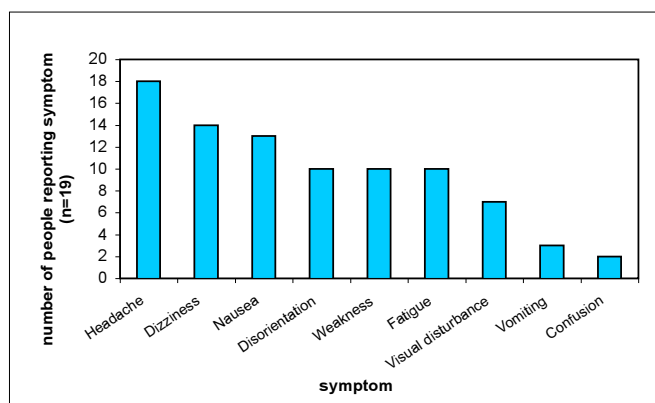Figure 1: Graph to show onset of symptoms reported

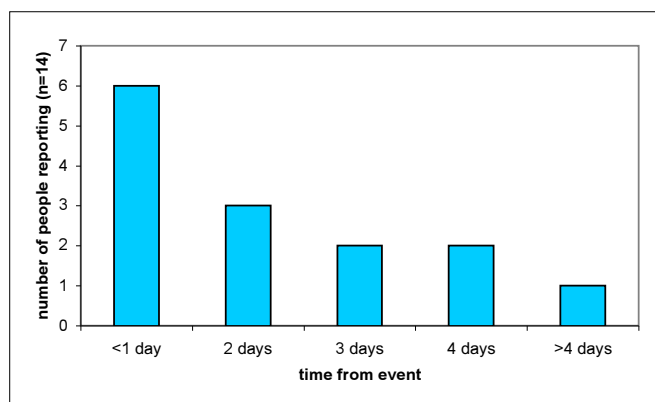Figure 2: Graph to show frequency of symptoms reported

Figure 3: Graph to show duration of symptoms reported

The onset of symptoms varied greatly (Figure 1) although there may have been a failure to recognise symptoms early on especially whilst driving. Headache, dizziness and nausea were the three most commonly reported symptoms (Figure 2). Some respondents made reference to 'adrenaline' helping to maintain concentration whilst driving and awareness of feeling unwell occurring when drivers stopped. People arrived at the venue from 16:00 hours onwards but there was no apparent association with time of arrival at the venue and either time of onset of symptoms or severity and duration. This suggests that significant exposure did not occur until after the start of the race.

Most driving teams had four people. One team had only two members and therefore spent more time driving than individuals in other teams. Both of these people reported the most severe symptoms, indicating that driving was associated with the highest exposure. One respondent mentioned that he had experienced a similar incident at a different venue

a few weeks prior to this one. Telephone follow up with some of the more regular karters indicate that a certain level of symptoms is commonplace and may have led to under-reporting. Loyalty to the race venue and unwillingness to cause difficulties for the venue may have encouraged some regular karters not respond to the questionnaire or to under-report symptoms. None of the respondents claimed to have been the person who was said to have collapsed in the canteen area afterwards.

The duration of symptoms did appear to be related to initial severity in most cases although this was not consistent (Figure 3). Three of the respondents who completed the questionnaire thought that they had still not fully recovered from the incident at the time of writing. Despite this, no other karters had either attended a hospital on the day or sought subsequent medical attention.

Following discussions with the kart suppliers and the gas system designers, it appeared that the fault was due to a modification in the gas cylinders that was allowing liquid petroleum to be drawn into the engines as the karts decelerated into the corners. This was leading to incomplete combustion and the production of high levels of carbon monoxide. A further modification was necessary to correct this and the karts were then tested to ensure that emissions were normal.[1] The standard used for CO is taken from workplace exposure limits for use with the Control of Substances Hazardous to Health Regulations 2002 (as amended). The standard for CO is an 8 hour time weighted-average workplace exposure limit of 30 parts per million (ppm) and a short term exposure limit (15 minute reference period) is 200 ppm.[2] It is advised that alarms are set to sound at a concentration of 200 ppm. If the alarm sounds all drivers should turn their engines off and ventilation should be increased until levels normalise.[3]

Carbon Monoxide poisoning

CO poisoning causes a multitude of effects due to inhibition of cellular oxidation, resulting in tissue hypoxia and cellular poisoning. Clinical symptoms of mild poisoning are non-specific and may mimic those of a non-specific viral illness (Box 1). Unintentional poisoning by inhalation of carbon monoxide is a well recognised problem, causing 148 deaths in England and Wales and 534 medical admissions in 2000,[4] although this may represent a significant underestimate of the true numbers.[4] Sources of CO include domestic heaters, outdoor stoves, barbeques and motor vehicles. CO poisoning may also result from the occupational use of various power tools and equipment in enclosed spaces, and diving equipment.[5]

CO binds irreversibly to haemoglobin resulting in the formation of carboxyhaemoglobin. Although carboxyhaemoglobin levels may not correlate well with symptoms and signs of toxicity, headache usually occurs at around levels of 20%. Dizziness, nausea, weakness and collapse occur at around 30%.[6] Once levels of carboxyhaemoglobin exceed 40% impaired judgment or collapse are likely to render a person unable to recognise or escape from the cause[5]. Exposure to concentrations of 500ppm of CO for five hours or more can result in blood carboxyhaemoglobin levels of around 50%.[5] At this level, unconsciousness and death is likely to occur.

Interpretation of carboxyhaemoglobin levels is also confounded by delays in obtaining blood samples and therapeutic intervention with oxygen. Clinical features may persist or begin long after the disappearance of measurable carboxyhaemoglobin, which has a half-life of only four to five hours when clean air is breathed.[7]

Box 1: Symptoms associated with carbon monoxide poisoning[6]

Mild toxicity

Minor exposures result in a throbbing temporal or frontal headache, fatigue, dyspnoea on exertion, light-headedness and dizziness. Patients with cardio- or cerebrovascular disease may experience exacerbation, such as myocardial ischemia or infarction, or stroke.

Moderate toxicity

Moderate exposures may produce severe headache, weakness, dizziness, nausea, vomiting, syncope, tachycardia and tachypnoea followed by bradycardia and bradypnoea, flushing, cyanosis, perspiration, decreased vigilance, diminished manual dexterity, impaired sensorimotor task performance, prolonged reaction time, difficulty thinking, impaired judgement, blurred or darkened vision, ataxia, loss of muscular control, tinnitus or roaring in the ears, sleepiness, hallucinations and cardiovascular toxicity.

Severe toxicity

Severe exposures produce syncope, seizures, confusion, disorientation, convulsions, involuntary evacuations, skin bullae, cardiovascular toxicity, ventricular dysrhythmias, cardiorespiratory depression, pulmonary oedema, respiratory failure, stupor, loss of consciousness, coma, collapse and death.

Delayed effects (usually seen in severely poisoned patients. Comatose patients with abnormal head CT scans appear to have a worse prognosis than those with normal scans)

Neuropsychiatric effects can appear several days after exposure. These include vegetative state, akinetic mutism, Parkinsonism, apraxia, agnosia, visual impairment, amnesic/confabulatory state, depression, dementia, psychosis, paralysis, chorea, cortical blindness, peripheral neuropathy and incontinence. Personality changes may also occur, with increased irritability, verbal aggression, violence, impulsivity and moodiness.

Discussion

This incident highlights the dangers of CO poisoning that may result from the use of internal combustion engines in enclosed spaces. Staff and some of the racers may have experienced some of the mild symptoms of CO poisoning in the past that may have led them to under-estimate the potential seriousness of the incident. Many people drove home, putting themselves and other road users at risk unnecessarily. While many of these shortfalls are those of the staff at the centre, none of the regular karters sought medical attention signifying a general lack of awareness of the dangers.

This incident may not be the first of its kind, although it may be one of the first to come to the attention of the HPA. While Health and Safety at the local council responded appropriately, they did not seek to involve the HPA. This raises issues of general communication with other organisations especially the circumstances in which the HPA should be informed and their role.

The National Karting Association (NKA) is a body that promotes karting provides advice and support regarding safety, training and the environment. The NKA also offers track inspections for those affiliated based on a 50-60 point safety plan, although it concentrates on track safety to minimize accidents.[8] There is an opportunity to add some training on the dangers of CO poisoning and appropriate actions that operators should take. However in general, there is a lack of awareness about CO poisoning. Teaching material and suitable leaflets that cover sources other than domestic heaters would help reduce the frequency of incidents and their resulting morbidity and even mortality.

This incident highlights a number of improvements that could be made in order to reduce the risk of CO poisoning at indoor go-karting tracks:

- Identifying those at higher risk of CO poisoning and advising them appropriately or excluding them from racing (including pregnant women).

- Informing racers and spectators of the symptoms of CO poisoning and giving appropriate advice about seeking medical attention and driving.

- Monitoring of trackside CO levels by staff that are trained to recognise problems early and take remedial action including increasing ventilation of suspending the race.

- Contacting other agencies in the event of an incident including medical services, health and safety and the HPA.

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Hyperbaric or normobaric oxygen therapy for carbon monoxide poisoning?

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Background

Carbon monoxide (CO) is a colourless and odourless gas that is generated by the incomplete combustion of carbon-based compounds. CO poisoning remains an important cause of both intentional and accidental injury worldwide and is one of the commonest intoxications confronting clinicians and toxicologists. Professor John Henry has noted that CO causes headache and confusion in both patients and doctors alike.[1] Diagnosis can be difficult because of the non-specific symptoms and deciding on the best treatment strategy is not always easy.

CO has an affinity 240 times greater than oxygen for haemoglobin. As a result, it displaces oxygen interfering with its transport in the body and can cause hypoxic damage in a variety of organs.[2] Acute poisoning can cause a variety of symptoms, including headache, nausea, irritability and weakness, and may lead to impaired consciousness, respiratory failure, cerebral oedema and metabolic acidosis. The type and intensity of symptoms is related to the level of exposure to CO and the corresponding concentration of carboxyhaemoglobin (Table 1).

Table 1: Carboxyhaemoglobin levels and symptoms

COHb %	Signs and Symptoms
10	No symptoms. (Heavy smokers can have up to 9% COHb)
15	Mild headache
25	Nausea & serious headache. Treatment with oxygen &/or fresh air usually followed by rapid recovery
30	Symptoms intensify. Potential for long term effects especially in babies, children, the elderly, those with heart disease & pregnant women
45	Unconsciousness
50+	Death

Chronic CO poisoning can have non-specific features, and is frequently undiagnosed. Most cases of CO poisoning recover uneventfully, but others develop neuropsychiatric features, such as memory impairment, disorientation, apathy, inability to concentrate, personality changes and Parkinsonism. Fortunately, the majority of these cases recover fully within a year.

There is no widely accepted treatment beyond supportive care and the use of supplemental normobaric oxygen (NBO), now considered the 'standard of care', although this has never been properly validated. Hyperbaric oxygen (HBO) has been widely used as adjuvant form of therapy. However its use in CO poisoning still remains controversial.

HBO is used to increase the partial pressure of oxygen above normal fully saturated levels by markedly raising the concentration of dissolved oxygen in the plasma. This in turn displaces CO from the haemoglobin molecule as a result of mass action. HBO has also been suggested as a form of therapy in many diseases leading to tissue hypoxia or ischemia, such as traumatic brain injury, acute coronary syndrome, acute ischemic stroke, late radiation injury therapy and thermal burns. HBO is also used in the treatment of neurological conditions where there is damage to distal nerves, such as multiple sclerosis, malignant otitis externa, idiopathic sudden sensorineural hearing loss and tinnitus and in the radiotherapy of malignant tumours. However, randomised control trials to evaluate these uses are few and with great methodological limitations. The Cochrane Collaboration Group has conducted extensive reviews of HBO treatment for these conditions but has only found some benefit in acute coronary syndrome, where it reduces adverse events and produces more rapid pain relief, but has no effect on overall survival.[3]

What is the evidence?

A recent review of the literature surrounding the evidence for HBO for the treatment of CO poisoning, has added little but rather sparked the debate again. Buckley et al., reviewed six randomised clinical trials.[4] Of these, two were incomplete publications (an interim report and an abstract). The treatment protocols used and follow - up varied greatly in the six trials. Because of this, it was not possible to combine the studies to do a meta-analysis. The clinical studies included in the review appear to have limitations that compromise their validity and applicability, including significant failure to follow-up, exclusion of patients with severe CO poisoning, lack of blinding and using HBO at - different treatment levels. For neurological symptoms, only two of the trials found positive outcomes, while the other four found no difference in outcomes.[5,6] Not surprisingly, the authors of this review concluded that there are insufficient data to support the benefits HBO over NBO therapy and that further research is needed to better define the therapeutic role in the management of CO poisoning.

What does this mean in practice?

Despite the review not being conclusive with respect to the efficacy, inefficacy or harm of HBO in the management of CO poisoning, further review of these findings by five toxicology experts highlighted consistent themes.[1,7-10] Two of the three trials that were more carefully conducted, show a benefit and as a result the experts consulted suggest that in the current situation it is best to treat, especially in selected patients. They suggest a series of possible clinical criteria to aid decision and as expected, different experts have different clinical criteria. These criteria have been summarised by Brent [9] but, while they remain a useful guide, they cannot be considered as a consensus guideline.

In summary, current evidence indicates that HBO is best reserved for selected patients, particularly those presenting with loss of consciousness, neurological impairment and cardiovascular dysfunction, or pregnant women. However, as Buckley suggested, there are insufficient data to support using HBO therapy as a standard of care, since the benefits of HBO in the management of CO poisoning are likely to be small.[4]

Summary

- CO poisoning is one of the commonest form of intoxication
- HBO therapy remains controversial
- Evidence on the effectiveness of HBO comes from a small number of studies with a variety of limitations compromising their validity
- Data are insufficient to consider HBO therapy as the standard of care for CO poisoning.
- Expert advice suggests it is best reserved for patients that:
  - are pregnant
  - have neurological impairment
  - have loss of consciousness
  - have cardiovascular dysfunction

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Anaemia and lead toxicity

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Introduction

Lead toxicity has been reported as a cause of anaemia. We report two patients with anaemia in whom lead toxicity was a significant contributing factor.

Case 1

In June 2000, Mrs HT, a 57 year old British-Indian woman was referred to the Haematology out-patients with an anemia with Haemoglobin (Hb) level of 8.8g/dl (normal range: 12-16g/dl). She was prescribed a course of iron tablets which she had been taking for two months prior to her attendance at the out-patient clinic. She was generally well apart from a painful right ankle. Blood tests at her first out-patient visit showed a Hb of 9.4g/dl, normal white blood cell count and platelet count. On microscopy, the blood film showed abnormal features in the red blood cells including basophilic stippling. Initial investigations showed normal haematinics and blood chemistry. Markers of haemolysis were negative as was a Coomb's test. A provisional diagnosis of myelodysplastic syndrome was considered and Mrs HT had a bone marrow examination under local anaesthesia. The morphology of her marrow was unremarkable and cytogenetics normal. By August 2000, her anaemia had improved to 10.5 g/dl. However, she continued to attend the haematology clinic periodically as there was no clear diagnosis for her illness.

In February 2002, her anaemia worsened (Hb 8.5 g/dl) temporarily. Red cell morphology continued to show abnormalities seen on previous occasions. Mild reticulocytosis was noted and HT mentioned that her sister, living in Ilford was being investigated for unexplained anaemia. She had further tests to investigate the possibility of a congenital haemolytic anaemia. These were normal.

In September 2003, we were asked to see HT urgently by her GP. Her anaemia had worsened further (Hb 7.3 g/dl) and she now complained of abdominal pain with anorexia and irregularity of bowel movements. On examination, her abdomen showed generalised tenderness. She was transfused and further blood tests sent for assessing complex causes of haemolysis and lead levels. This showed a blood lead level of 7.6mmol/l. HT was chelated with a course of DMSA tablets. Lead levels improved to 2.12 mmol/l in November 2003 but worsened again to 3.15 mmol/l in February 2004 necessitating a further course of DMSA. Her lead levels in November 2005 measured 1.69 mmol/l but she was still mildly anaemic with a Hb level of 11gm/dl.

A review of her history showed that Mrs HT was born in Kenya and has lived in UK since 1960's. From 1968 to 1972 she lived with her family in a Victorian terraced house in Stoke Newington. Since 1975, she has lived with her husband in a purpose built flat (built in 1970) in Hounslow. Despite her ethnic background she has no obvious risk factors for lead poisoning. She shopped and worked at Sainsbury's, and used standard cooking techniques in cookware procured locally. She travelled infrequently outside Europe and visited Goa as a tourist for two weeks in 2000. She denied using make-up or indigineous medication.

Case 2

Her husband Mr JT, aged 64 yrs was already attending Haematology out-patients since September 1999 for essential thrombocythaemia (ET)- a myeloproliferative disease that was being treated with cytotoxic chemotherapy. He was being reviewed every three months. His Hb levels were usually maintained at levels slightly below normal (10-12 g/dl) and this was attributed to the cytotoxic medication. From time to time, the anaemia was worse (Hb 7.5-8.5 g/dl) and an elevated reticulocyte count was noted. A leukaemic transformation of ET was suspected and Mr JT underwent another bone marrow examination. This showed no deterioration in his myeloproliferative disease. At clinic reviews, he complained of intermittent upper abdominal pain, anorexia and intermittent constipation. He was referred to the Gastroenterology department in 2002 where gastro-intestinal (GI) endoscopy and radiologic investigations were performed. These were normal. Further to the detection of lead toxicity in his wife, his blood lead levels were investigated in September 2003. This showed a result of 9 mmol/l. He was treated with DMSA. Within three months of DMSA (chelation) treatment, his abdominal pains improved and his Hb improved to normal levels.

The couple have two children aged 26 and 29 years. Both have normal Hb and lead levels.

Discussion

Chronic fluctuating anaemia in HT and JT was associated with severe lead toxicity. There are no obvious explanations as to the source of the lead. Diagnosis of lead toxicity in both the patients was complicated and lead toxicity was one among other causes of the anaemia in both patients.

Patients suspected to have lead poisoning are investigated by assessing blood lead levels. This investigation is usually requested if a combination of anaemia, abdominal pain and constipation occurs in patients who might be at a risk for lead poisoning. Our patients did not initially have tests undertaken for lead poisoning as the clinical and laboratory problems did not indicate lead toxicity in the first instance. In the case of HT, it may be that lead toxicity worsened an anaemia that still remains unexplained in that treating the lead toxicity has improved but not normalised her Hb. In the case of JT, his abdominal symptoms have improved and he is able to tolerate his chemotherapy better.

Unexplained anaemia comprises 30% of anaemia detected in >65's in the community.[1] Of those anaemias that have been categorised, anaemia of chronic disease (ACD) is the second commonest, next only to iron deficiency anaemia.[2] Data cited in the two references has been published from studies conducted in USA and given the difference in the nature of migration and demographics, does not readily lend itself to extrapolation to UK. ACD may vary in intensity and Hb levels usually range from 8.0 to 10 gm/dl. Some patients with ACD will be found to have other contributory factors to anaemia such as coexistent iron deficiency, renal failure. Thus a sizeable number of patients with anaemia may have no clear explanation of the cause of the anaemia or may have more than one reason to be anaemic.

Toxicologic causes of anaemia are uncommon. Lead and aluminium (in dialysis patients) are commonly cited as causes of anaemia. Lead toxicity can remain undetected for long periods and contribute to anaemia. Data from USA,[3] show a prevalence of elevated lead levels in 8.2% per 100,000 population in 2003. 94% of these individuals had excess lead because of occupational exposure. This data was collected from asking employers to perform blood lead testing at the workplace. Only employed individuals over 16 years of age were included in this survey and hence this data is considered to be an underrepresentation of the actual prevalence of abnormal lead levels in the community.

In UK, there is plentiful anecdotal data on lead toxicity and the causes of toxicity in these cases have been attributed to social, cultural and environmental factors at domestic sites or the work place. Thus the prevalence and distribution of this condition is likely to differ from that observed in USA. However, there is a lack of data regarding its prevalence in different geographic areas and among different ethnic/ gender/ age groups. Given the above, a proportion of patients with unexplained anaemia in UK will be found to have lead toxicity. Further, lead toxicity may not be the sole cause of anaemia. The paucity of data in UK renders it difficult to estimate the likelihood of having abnormal blood lead levels in individuals.

Recommendations:

1. It is recommended that patients with unexplained anaemia should be tested for lead toxicity

2. In UK, there is an urgent need for obtaining data concerning the prevalence of lead toxicity in different ethnic/ gender/ age groups. Such data will enable a better understanding of the natural history and size of the problem. Data from such a study will enable clinicians to investigate and treat patients more appropriately and for health protection actions to be implemented to minimise harm from lead exposure.

3. Haematologists, toxicologists and the Chemical Hazards and Poisons Division, HPA should collaborate to conduct studies to address these recommendations

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The public health impact of lead and the role of health protection

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The previous case reports illustrate the complex role played by lead in anaemia and the difficulty that such cases may present in identifying the source of exposure for individual patients.

The following report summarises the toxicology of lead and common exposure pathways, outlines the evidence for its impact on public health and why this may be underestimated, and makes recommendations for reactive and proactive health protection action to address this.

Introduction and historical context:

Lead has been widely used by man for several thousand years as its low melting point and malleability made it one of the first metals to be smelted and used by human society and it is widely found in the earth's crust[1]. The health effects of lead toxicity are well recognised; the Greek physician Dioscorides in the 2nd century BC, describing the now well-recognised neuropsychiatric effects of lead, observed that lead makes the mind 'give way' and it has also been proposed that lead toxicity may have been in part responsible for the decline of the Roman empire[2]. In the UK, the Industrial revolution of the 19th century resulted in lead poisoning being recognised as a common occupational disease and death was not an infrequent outcome. Although encephalopathy, the most extreme manifestation of lead poisoning, has now become rare, cases of lead poisoning continue to occur.

Globally, although public health interventions in many countries have been effective in reducing the number of extreme cases of toxicity, lead continues to be an important and possibly under-estimated cause of morbidity and mortality. This is illustrated by a recent World Health Organisation (WHO) report which estimated that the global disease burden due to lead-induced mental retardation is equivalent to 9.8 million disability adjusted life years (DALYs), and from cardiovascular diseases (due to elevated blood pressure) was equivalent to 250 000 premature deaths and 3.5 million DALYs [3]. In a sample of nearly 10, 000 adult participants in the US, blood lead levels as low as 5-9ug/l were associated with an increased risk of mortality from all causes, cardiovascular disease and cancer.[4]

Toxicology and Exposure pathways:

Sources of lead
The range of possible sources of human exposure to lead is extensive and diverse and can be grouped into those resulting from environmental sources, e.g. air pollution, occupational sources e.g. lead smelters and miscellaneous sources e.g. exposure from contaminated cosmetics. Globally, mass balance studies have estimated that the combustion of alkyl lead additives in motor fuels accounts for the major part of all lead emissions into the atmosphere, thus influencing all

compartments of the environment[5]. Contamination of domestic drinking water via lead water-pipes or 'plumbism' is particularly relevant in areas with soft water can also be a source of exposure.

Routes of exposure:

Ingestion
This is an important route of exposure for children during the developmental stages where there is excessive mouthing or in children with pica which may be secondary to behavioural problems such as autism. It may also be relevant in adulthood, for example, through the ingestion of traditional medicines[6] which are being increasingly recognised as an exposure source. A study from the US looking specifically at Ayurvedic medicines found that around 20% are contaminated with lead[7]. It is important to recognise that, in this context, the term contamination may be misleading as there is a belief within some health systems in the therapeutic properties of heavy metals such as lead.

Inhalation
Children are particularly susceptible to lead during the renovation of old houses as they may be exposed to dust particles containing inorganic lead from the layers of paint which pre-date the 1970s. Organic lead compounds in petrol are also readily absorbed through this route and this was the rationale behind the removal of these anti-knock compounds in most developed countries.

Dermal
There is limited evidence that inorganic lead may be absorbed by the skin; although dermal absorption of organic lead may occur in sufficient quantities to produce toxicity[8].

Foetal exposure
Transplacental transfer of lead is recognised as a route of exposure for the foetus as it readily crosses the placental barrier throughout gestation; subsequently, mothers may also expose the infant to lead via breast milk. This can occur in the absence of continued exposure due to the release of lead from the storage compartment of the skeletal system into the blood and so may be important for women who have been exposed as children in countries with high levels of environmental exposure.

Toxicology, pathophysiology and clinical presentation.

Distribution:
The distribution of lead is complex and can be considered as consisting of at least three compartments – blood, bones and soft tissues. Bone can be thought of as the storage compartment and the blood and soft tissues as the more labile and exchangeable compartment. With continued exposure however, the lead gradually becomes fixed to bone, probably as inert and insoluble lead phosphate[9] and the biological half-life for lead in this state is around 10-20 years[10]. Lead deposits in bone can be reabsorbed and released to blood during normal bone

remodelling, during periods of enhanced bone resorption as occur in certain disease states and in response to normal physiological processes such as pregnancy, lactation[11] and ageing. As a result, bone lead stores represent a potential source of soft-tissue lead exposure, even with declining environmental exposures.

Biomarkers of exposure:
Blood lead levels are the principal measure of lead exposure in clinical practice. The biological half life of lead is around 36 days[12] therefore blood lead levels are only a good indicator of recent lead exposure. Other biomarkers of exposure that may be more practical to use for children include the measurement of lead in milk teeth in order to assess previous exposure.

Toxicodynamics:
Lead is a complex toxin exerting numerous pathological effects across several physiological systems and it does this through the inactivation of enzymes either through binding to the SH-groups of their proteins or via displacement of other essential metal ions. For this reason many organs or organ systems are potential targets for lead, and a wide range of biological effects of lead have been documented. These include effects on the haematological, nervous, renal and reproductive systems in addition to cardiovascular, hepatic, endocrine and gastrointestinal effects. The often vague nature of the clinical presentation may mean that the diagnosis may not be immediately apparent.

Neurotoxicity:
The mechanism of the impact of lead on both the central and peripheral nervous system is also complex and operates through a variety of mechanisms including influences on neuro-transmitters, abnormal myelin formation and endothelial damage.

Although encephalopathy is the most serious and potentially life-threatening manifestation, at the other end of the clinical spectrum there is an increasing awareness of the large numbers of children with subtle behavioural and cognitive effects from blood lead levels that were previously thought not to be significant[13]. A study of blood lead levels in children in Mexico indicated that children's neurodevelopment is inversely related to their blood lead levels even in the range of <10 μg/dl and that there was a supralinear relationship between blood lead levels and neurobehavioral outcomes[14]. Children have a number of risk factors which make them more susceptible to the neuro-toxic effects of lead. The debate as to the health effects of blood lead levels that were previously thought to be safe is summarised in a recent Centers for Disease Control and Prevention publication[15].

Haematological:
Chronic exposure to lead is associated with anaemia[16] and there is evidence that iron deficiency may result in increased gastro-intestinal absorption of lead which may enhance this effect.

Renal:
Although there is mixed evidence for renal effects and difficulty in discounting the effect of confounders that may also cause renal dysfunction such as cadmium, several studies have reported nephropathy characterised by albuminuria, glycosuria and renal tubular acidosis. [16]

Cardiovascular:
There is evidence from some population studies that lead can cause a rise in both the systolic and diastolic blood pressures.[16]

Clinical presentation of lead poisoning in children:

Clinical manifestation	Typical blood lead concentrations (ug/dl)
Severe: Central nervous system: Encephalopathy (coma, seizures, ataxia) Gastro-intestinal tract: Persistent vomiting Haematological: Anaemia	> 70 – 100
Mild / moderate: Central nervous system: Hyperirritable behaviour, intermittent lethargy, decreased interest in play, "difficult child" Gastro-intestinal tract: Intermittent vomiting, abdominal pain, anorexia	50 - 70
Asymptomatic / vague symptoms: Central nervous system: Impaired cognition Peripheral nervous system PNS: Impaired fine motor coordination Miscellaneous: Impaired hearing, growth	0 - 49

Goldfrank, Flomenhaum, Hoffman, Houland, Lewin, Nelson, Toxicological Emergencies, 8th Edition 2006

Public health impact and policy context

Global epidemiology:
The disease burden from exposure to lead resulting in mild mental retardation (due to IQ point decreases) and cardiovascular outcomes (due to increases in blood pressure) was estimated at a global level by the WHO. This estimated that mild mental retardation (MMR) and cardiovascular outcomes resulting from exposure to lead amount to almost 1% of the global burden of disease and this estimate can be used to assess the magnitude of the benefits that could be accrued by increasing the global coverage of lead-reduction programs[17]. The report confirms that the greatest burden falls on developing countries. They estimated that the loss of IQ points and increase in MMR (defined as having an IQ score of between 50 and 69) may affect 1.1 persons per 1,000 population and 0.5 children (aged 0-4) per 1,000 population[18].

The rapid and often poorly regulated industrialisation that is being witnessed across many low and middle-income countries may result in increasing exposure and subsequent public health impact that can be attributed to lead. A recent example of this occurred in September 2006 when approximately 900 people were reported to have been affected from emissions from a lead smelter in western China. More than a quarter of those affected were children and it was reported that many of these are likely to have suffered permanent neurological impairment as a result of this exposure[19].

UK Epidemiology:

In the UK between 1981 and 1996, there was one reported death due to lead poisoning in a two year old girl who died of encephalopathy[20]. However, whilst this marks a dramatic improvement from the situation up until the 1970s, the cases that continue to be identified represent preventable hospital admissions, intellectual impairment and morbidity.

There have been two large population based surveys which have looked at the distribution of blood lead levels in the UK. The Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) measured the blood lead levels of 585 children in 1995 and this showed a mean of 3.44ug/dl at $2^1/_2$ years of age[21]. As part of the 1995 Health Survey for England, blood lead levels were measured for a sample of 6,868 people of whom 97% had blood lead levels less than 10µg/dl.[22] Despite these both indicating mean levels less than the current WHO reference level, there is concern that this masks a small proportion of the population with unacceptably high blood lead levels (3% of the HSoE participants and 5% of the ALSPAC children had levels greater than 10ug/dl) and, in view of the recent published evidence of possible health effects below this level, this may have resulted in the belief that lead no longer presents a risk to health in the UK.

Link with inequalities:
The link between deprivation and increased exposure to lead is important from a public health viewpoint as it may widen existing health and income inequalities. The US Centers for Disease Control and Prevention observed that children enrolled in Medicaid had a prevalence of elevated blood lead levels three times higher than those not enrolled[23]. Although blood lead levels in children in the US have fallen[24], in part to due policies that have raised awareness and encouraged screening, in developing countries lead toxicity remains a significant problem. Studies have estimated that more than half the children in India have blood lead levels >10ug/dl[25,26] and that relatively low blood lead levels in children were significantly associated with elevated risk of moderate and severe anaemia.

In the UK, demographic factors such as an increasingly diverse population, who may have increased risk factors for exposure to lead, means that cases of lead toxicity are likely to continue to be made and this may compound existing health inequalities within these groups.

Statement from the G8 group in 1997:

Lead poisoning is a major environmental hazard to children and our countries have taken many successful actions to reduce children's exposure to lead. Our countries continue to support the reduction in risks from exposure to lead. We call for further actions that will result in reducing blood lead levels in children to below 10 microgrms per decilitre. Where this blood lead level is exceeded, further action is required. We acknowledge the importance to child health of maternal exposure to lead and agree to reduce maternal exposure. We commit to fulfil and promote internationally the OECD Declaration on Lead Risk Reduction[27]. We commit to a phase-out of the use of lead in gasoline, the elimination of exposure to lead in products intended for use by children, the phase-out of the use of lead in paint and rust-proofing agents, the restriction of lead in products that may result in ingestion in food and drinking water and to set schedules and develop strategies for elimination or reduction of lead from these sources. In addition, we agree to conduct public awareness campaigns on the risks to children from lead exposure and to develop scientific protocols and programs to monitor blood lead levels in children to track our progress in this important effort[28].

1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health

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Discussion

Despite an awareness of the toxicity of lead for more than two millennia, recent estimates show that it remains an important environmental risk factor and is still responsible for an unacceptably high proportion of the global burden of disease. Unprecedented levels of global migration means that, despite public health policies in the UK to reduce blood lead levels, certain communities within the U.K. are likely to continue to be exposed to lead either through behaviour that exposes them to lead or from endogenous exposure due to body burden of lead within the skeletal system.

Harnessing the current interest in factors such as diet on children's behaviour and IQ and given the accumulating evidence that blood lead levels that were previously considered to be safe may now have adverse effects on cognition[29], could mean that it would now be timely to attempt to map the distribution of lead in children in the UK. The undertaking of such a population based survey may also serve to raise awareness of the importance of lead. It is important, however, that the effect on IQ of lead in children is seen within the context of other major influences such as deprivation and parenting.

The response to and effective management of patients with lead toxicity is likely to involve several agencies and may include acute trust staff such as physicians and medical toxicologists, local authority staff such as environmental health officers (EHOs), primary care staff such as health visitors, community paediatricians and GPs and water companies. This multi-faceted approach is needed in order to provide a response which should attempt to encompass the toxicological, clinical, environmental and social factors. Anecdotal reports have demonstrated the effectiveness of joint home visits consisting of health protection professionals and EHOs in undertaking this assessment and the use of questionnaires to guide the identification of the source of the exposure is also useful. These cases are often complex and it is important that the social aspects are fully considered. For example, whilst it is not ideal for a family to continue to reside in a house with lead paint if a child has been identified as having a high blood lead level, the health impact of them becoming homeless may be even worse and this may be a real risk if they leave rented accommodation and housing staff may be able to advise with this. It is important to ensure that all the necessary components of the management of the case are delivered and clinical expertise should be sought from toxicologists.

Recommendations:

1. **Epidemiology and Surveillance**
a) **Case notification**
   There is currently no system in place for notifying cases of lead poisoning in adults or children. Active surveillance of cases could help to estimate the size of the problem of clinically significant cases and subsequently help to target prevention strategies. One possible mechanism for this is that paediatric cases could be notified to British Paediatric Surveillance Unit (BPSU) [30] system which is a joint collaboration between the Royal College of Paediatrics and Child Health, the HPA and the Institute of Child Health.
b) **Population based surveys**
   An up to date estimate of the prevalence of toxicity in children in the UK is needed, as outlined by the G8 statement on Children's environmental health. In addition, population level surveys to look at the distribution of blood lead levels in adults could be undertaken using existing vehicles such as the Health Survey for England[31].

2. Case management and identification of source of exposure

a) Early identification of cases

Early identification of cases is crucial in order to be able to initiate early clinical management, which may include chelation therapies and symptomatic management, and also to reduce ongoing exposure. It is recommended that cases are referred early to the local HPU so that they can coordinate the environmental risk assessment with support as required from CHaPD.

b) Case management

Effective case management requires the joint action of several agencies. CHaPD are working with HPUs to look at developing resources such as guidance on the joint management of cases, which can be adapted according to local need and arrangements, and questionnaires to aid in the identification of the exposure source. The aims of this guidance will be to facilitate the management of these cases and ensure a consistent approach.

3. Prevention

Health education and awareness raising

In the UK, various groups are working with ethnic communities to raise awareness of the risk of using certain substances such as calabash chalk[32] which may contain high levels of lead. Other targeted health promotion programmes that could be considered include highlighting the potential risks of traditional medicines with health professionals.

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Public health review of methaemoglobinaemia

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Introduction

Environmental investigation of methaemoglobinaemia will require public health input. In this paper we present a case report, describe the subsequent investigation and provide an overview of the public health aspects of methaemoglobinaemia.

Case Report

A CCDC in Teesside was contacted on the 7th of April 2005 by a Paediatric SHO at the local hospital. The previous week an 11-month old boy had been admitted to hospital with a two week history of progressively increasing 'greyness'. On admission on the 31st of March, it was reported that he was not eating properly and was not sleeping, was irritable and had lost weight.

On examination the child was noted to be alert but dusky in appearance. His lips, hands and feet where slightly cyanotic and he had mild periorbital oedema. There were no heart murmurs. His pulse was 134, temperature 36.6°C, and oxygen saturation was low (78-80% measured at the toes and 83-89% measured at the fingers).

He had no past medical history of illness apart from several upper respiratory tract infections, was not on any medication (prescribed or otherwise), nor had any known allergies. He was delivered normally at term. His physical development was following the 9th percentile on the growth chart and he was up-to-date with his routine immunisations. He had no respiratory problems, dyspnoea, syncope, seizures, jaundice, urinary symptoms, pica of any description or abdominal complaints.

The child had a 20 year old half-sister (from his mother's previous marriage) living at home, and a 5 year old sister. His parents were born in Pakistan. Everybody else in the household was fit and healthy. There was no known family history of hereditary disease, though his father (currently living abroad) had diabetes and hypertension. There were no glyceryl trinitrate or other nitrates in the home. The family were non-smokers.

Routine haematology, biochemistry, chest x-ray, and ECG were all normal. The child's methaemoglobin level was 47.5% and he was given a diagnosis of moderate to severe methaemoglobinaemia.

The child was considered to have chronic rather than acute methaemoglobinaemia as he was apparently tolerating a relatively high level and because he had no history of acute exposure to chemicals or drugs. It was also felt that his presentation could fit with inherited enzyme deficiency. Because of the potential complications of treatment with methylene blue in the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency (a potential association of methaemoglobinaemia), the child was given vitamin C treatment. An exchange blood transfusion was considered but it was decided to see how well he responded to more conservative treatment.

On the day after admission, his methaemoglobin level had fallen very slightly to 47%, and the day after that to 44%. On the third day following admission, levels had fallen significantly to 25.4%. The G6PD levels came back as normal, as did the haemoglobin electrophoresis. The plan for the child's discharge was for methaemoglobin levels to be monitored (initially on a weekly basis) at the paediatric day unit.

The CCDC was asked to investigate possible environmental causes of this boy's methaemoglobinaemia. On discussion with the CCDC, the mother and half-sister revealed that the child would not eat or drink properly. He teethed at 5 months, but his mother did not use teething gels or other topical local analgesics/anaesthetics. He took - solids from about $3\frac{1}{2}$ to 4 months of age and ate dry food such as cereals, vegetables and desserts. At 7 months he began to reluctantly eat 'proper foods' such as mashed potatoes, boiled vegetables, rice, porridge, chapattis, meat, yoghurt, cheese and butter. Up to the age of 9 months he was fed Cow & Gate formula milk, but this was mixed using water boiled from the kettle.

The family moved into their present house 4 months before the patient was born. The house had a mains water supply. Prior to moving in a new boiler and double-glazing were installed, and the electrics and the central heating checked. There were no water features such as ponds or fish tanks in the home or garden, no weed killers were used, and no food preserving undertaken. No home-grown food was eaten. The child had not visited parks or farms. The mother habitually sprayed air freshener after changing the child's nappy.

The child had no history of pica of any kind (including paint, matches, wood, wallpaper, etc) and did not chew his toys. The gas fire had thermal bricks with imitation charcoal. The child slept with a bedspread which was washed regularly. There are no dyes or bleaches used for his bedding. Cleaning products were locked up above the child's reach. Paints and other materials are stored in an outdoor storage facility. The neighbours have not been noted to do any spraying in their garden. The child had not had other care outside the home. There were no pets or animals in the house. There was no pest control undertaken.

The local water company was contacted and water sampling requested. Analysis of the tap water showed a nitrate concentration of 1.5 mg/l and < 0.0024 mg/l nitrites, well within UK drinking water standards. Summaries of mains water analyses from the previous 15 months were supplied to the Heath Protection Agency which indicated compliance with drinking water regulations.

Subsequently laboratory results from the King's College Hospital in London and the National Haemoglobinopathy Reference Laboratory in Oxford showed that this child had an NADH methaemoglobin reductase deficiency. His mother also had reduced NADH methaemoglobin reductase activity, and was thought to be 'a heterozygote', or carrier. At his last appointment on the 16th of September 2005, methaemoglobin levels were measured at 9.7% (normal range is 0-3%).

Discussion

Methaemoglobin is formed when the ferrous iron (Fe^{2+}) component of haemoglobin is oxidized into the ferric form (Fe^{3+}). Methaemoglobin (MetHb) cannot bind with oxygen. The oxidation of one of the iron atoms distorts the haem structure so that the nonoxidised subunits bind oxygen avidly but release it less efficiently.[1]

Clinical presentation:
Methaemoglobinaemia classically manifests as cyanosis of the lips, tongue, nose and inner eyelids. Small quantities of methaemaglobin are normal but are kept at levels of approximately 1% by a methaemoglobin reductase enzyme. Below levels of 15%, methaemoglobin is unlikely to have any clinical effects (Table 1).

Table 1: Clinical Features of Methaemoglobinaemia

Levels (%)	Clinical features
0-15	Clinical effects unlikely
15-30	Mild effects- cyanosis, fatigue, dizziness, headache
30-50	Moderate effects- weakness, tachypnoea, tachycardia, mild dyspnoea
50-70	Severe effects- stupor, coma, convulsions, respiratory depression, cardiac dysrhythmias, acidosis
>70	Potentially fatal

Adapted from: Methaemoglobinaemia: Causes and Management. Guy's & St Thomas' Poisons Unit. December 2003

Incidence of methaemoglobinaemia

Most countries do not report rates of methaemoglobinaemia but reported incidence per 100, 000 population is 0.26 in Hungary, 0.56 in Slovakia, 0.74 in Romania and 1.26 in Albania.[2] There is little data on the incidence of acquired methaemoglobinaemia in the UK. Between 2002 and 2005 there were a total of 17 full consultant episodes (FCEs) for non-congenital methaemoglobinaemia in England (acquired/toxic methaemoglobinaemia[a]- 8 FCEs, methaemoglobinaemia of unspecified cause[b] – 9 FCEs) and 10 for congenital methaemoglobinaemia[c].[3] As 1% of the England and Wales population have private water supplies hospital episodes statistics may underestimate the true incidence of this condition. In addition, lower levels of methaemoglobin do not manifest clinically.

Environmental causes of methaemoglobinaemia

Ingestion of nitrite or nitrate contaminated water is the most common cause of acquired methaemoglobinaemia. Microbial action in soil or water decomposes wastes containing nitrogen into nitrite and nitrate.[4]

Nitrite is easily broken down into nitrate which is the predominant compound found in ground and surface waters. Nitrogen containing fertilisers as well as human and animal waste also results in elevated levels of nitrate in water.

WHO recommends that levels of nitrate in drinking water should not exceed 50 mg/l and 3 mg/l for nitrite.[5] Many European countries report high nitrate concentrations in drinking water (Table 2) and it is estimated that one third of the population of Europe is exposed to nitrate concentrations higher than the WHO standard.

Table 2: European countries reporting high nitrate concentrations in drinking water

England	France	Slovakia
Wales	Germany	Slovenia
Austria	Malta	Turkey
Belgium	Moldova	Ukraine
Croatia	Netherlands	Czech Republic

Source: Lack T. Water and Health in Europe: an overview. BMJ 1999; 318: 1678-1682.

In the US, shallow rural domestic wells are the most likely source of nitrate-contaminated water particularly in areas where there is widespread use of nitrogen-based fertilisers.[4] Drinking water quality is closely regulated in the UK and in most developed countries but regulation of private supplies is problematic. About 1% of the population of England and Wales have private water supplies including boreholes, wells, springs and rivers.[6] This approximates to about 50,000 private water supplies.

Nitrate concentrations in ground water have been increasing in the UK since the 1940s due to the growing use of nitrogen fertilisers, organic manure and larger tracts of land being cultivated each year.[7] In 2004, 29% of rivers in England had nitrate concentrations greater than 30mg/l. These rivers are found mainly in areas of intense agriculture, predominantly in the Midlands, Anglian and Thames regions.[7]

Areas in which nitrate levels in ground or surface water exceed 50mg/l are designated Nitrate Vulnerable Zones (NVZ). A total of 55% of England is designated as a NVZ (Figure 1). In NVZs where the high levels of nitrate are due to agricultural practices, farmers are required to control their application of nitrate to the soil. Rivers in eight of the NVZ receive one or more discharges from large sewage treatment works. In these areas, water companies are required to increase the level of treatment of sewage so that the nitrate discharged is reduced to an acceptable level.[7]

Dietary Nitrate in Food and Water

Over 70% of nitrates in the diet come from vegetables such as cauliflower and broccoli; approximately 21% comes from drinking water and the remainder from meat products (e.g. sausages) in which nitrates have been used as a preservative.[4] Sodium nitrite is used commercially as a colouring agent, food agent and anti-corrosive. Inadvertent ingestion of nitrites can also occur as a result of consuming water in pipes and tanks that have been contaminated with sodium nitrite containing corrosion inhibitor solutions.[9]

Bottle-fed or weaned infants under three months have the highest risk of developing methaemoglobinaemia and the risk decreases with age. Infants with diarrhoea or other enteric infections who are dehydrated with metabolic acidosis are susceptible to developing methaemoglobinaemia without exposure to exogenous nitrates. The most common cause of methaemoglobinaemia is the ingestion of infant formula prepared with water with a high concentration of nitrates, most commonly from well water.[10] Consumption of vegetables high in nitrates e.g. spinach, green beans and squash occasionally cause methaemoglobinaemia in infants less than three months.[11] Infants are at high risk of developing methaemoglobin for a variety of reasons:[12]

- A higher gastric pH which allows bacterial invasion of the stomach and hence an enhanced conversion of ingested nitrate to nitrite
- Greater fluid intake relative to body weight
- A higher proportion of foetal haemoglobin which is more easily oxidised to MetHb than adult Hb
- Lower levels of the enzyme that converts methaemoglobin to haemoglobin

Breastfeeding infants whose mothers consume water with high levels of nitrate do not have an elevated risk of nitrate poisoning because nitrate concentration does not increase significantly in breastmilk.[10] Levels of methaemoglobin increase during pregnancy from 0.5-2.5% up to 10.5% during the 30th week of pregnancy. As a result pregnant women may be more susceptible to development of clinical methaemoglobinaemia by nitrates/nitrites. [4]

A comprehensive list of chemical causes of methaemoglobinaemia is given in Box 1.

Occupational causes of methaemoglobinaemia

Most work-related causes of methaemoglobinaemia are exposures to aromatic nitro and amino compounds. These compounds are widely used in the synthesis of aniline dyes, as accelerants and antioxidants in the rubber industry, and in the production of pesticides, explosives, plastics and varnishes.[13] From 1967 to 1980, chloroaniline, p-toluidine, nitrobenzene, and nitrochlorobenzene were the most common industrial causes of methaemoglobinaemia in the UK. Dermal exposure was a more frequent route of toxicity than inhalation with these compounds."[14]

The American Conference of Governmental Industrial Hygienists (ACGIH) divides methaemoglobin inducers into two classes: industrial chemicals for which methaemoglobin formation is the principal cause of toxicity and industrial chemicals for which methaemoglobin formation is not the principal cause of toxicity. (Boxes 2 and 3)

Congenital causes of methaemoglobinaemia

Hereditary methaemoglobinaemia can either be due to a structural abnormality of the haemoglobin (known as haemoglobin M because of its association with methaemoglobin) or due to a deficiency of the MetHb reductase enzyme (nicotinamide adenosine dinucleotide cytochrome reductase - NADH).[15] There are several variants of Haemoglobin M; in most tyrosine replaces the histidine residue, which binds haem to globin. This displaces the haem moiety and permits

oxidation of the iron to the ferric state. Haemoglobin M is more resistant to reduction by the methaemoglobin reductase enzymes previously described.[15] Several variants of haemoglobin M have been described, including haemoglobin Ms, haemoglobin M$_{Iwate}$, haemoglobin M$_{Boston}$, haemoglobin M$_{Hyde Park}$, and haemoglobin M$_{Saskatoon}$ which are autosomal dominant conditions. There are 4 types of methaemoglobinaemia due to NADH deficiency, these are autosomally recessive in nature and heterozygotes have 50% enzyme activity.[15]

Other causes of methaemoglobinaemia

Other acquired non-occupational causes of methaemoglobinaemia include the use of certain medications such as silver nitrate used for topical antibacterial prophylaxis of burn wounds, antimalarials and topical anaesthetics (see Box 1).
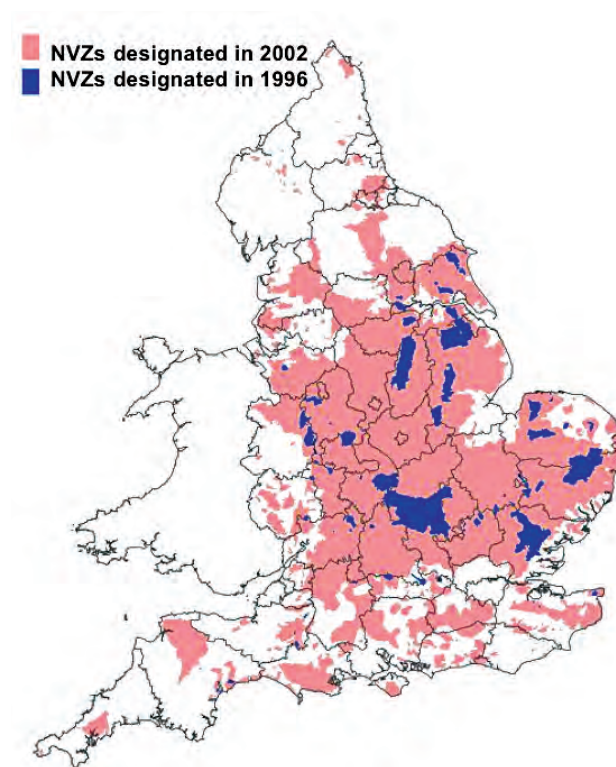
Figure 1: Nitrate Vulnerable Zones   © Crown Copyright
http://www.defra.gov.uk/environment/water/quality/nitrate/maps.htm

Management of acquired methaemoglobinaemia

Methaemoglobinaemia should be suspected in an otherwise healthy patient who presents with acute onset of cyanosis and in whom the respiratory distress is less than that which would be expected with the severity of cyanosis.[16] A history of possible exposure to MetHb-inducing agents including diet, source of water, occupation and medicines ingested or applied should be obtained.

Immediate management includes removal from the source of exposure and ensuring a clear airway. In general, mild cases of MetHb (plasma levels <15-20%) do not require any treatment other than avoiding the oxidising agent. Patients with MetHb levels >30% should have

methylene blue administered but patients with cardiorespiratory illness or anaemia may be considered for therapy at lower levels.[17] Methylene blue should not be administered to patients with glucose 6 – phosphate dehydrogenase deficiency as it can cause a severe haemolytic anaemia. Ascorbic acid (vitamin C) may also be used to reduce MetHb to haemoglobin but is of limited use in acute intoxications.[16]

Conclusion

Hospital episode statistics are likely to underestimate the true incidence of acquired methaemoglobinaemia. Only patients with levels above 15% are likely to present clinically so a significant proportion of cases will not be diagnosed. Public health input is required in the investigation of methaemoglobinaemia. A detailed history to ascertain possible exposure to a wide range of chemicals in the home and workplace and environmental sampling may be necessary. The most common cause of methaemoglobinaemia is ingestion of water with high levels of nitrates so where history is nonrevealing, drinking water should be considered as a potential source particularly if the supply is private. Bottle-fed infants have highest risk of developing methaemoglobinaemia particularly if formula milk is prepared with well-water. As illustrated in this case, methaemoglobinaemia can be due to congenital enzyme deficiencies. Investigation will require consultation with clinicians and possibly water companies and the local environmental health department.   A case investigation form is available from the Chemical Hazards and Poisons Division (email chemicals.london@hpa.org.uk).

Box 1: Inducers of methaemoglobinaemia

Agent	Source/Use
Inorganic nitrates/nitrites	Contaminated well water Meat preservatives Vegetables—carrot juice, spinach Silver nitrate burn therapy Industrial salts Contaminants of nitrous oxide canisters for anesthesia
Butyl/isobutyl nitrite Amyl nitrite Nitroglycerin	Room deodorizer propellants Inhalant in cyanide antidote kit Pharmaceuticals for treatment of angina
Aniline/aminophenols Nitrobenzene Sulfonamides Phenazopyridine Antimalarials Sulfones p-Aminosalicylic acid Naphthalene Copper sulfate Resorcinol Chlorates Combustion products Local anesthetics	Laundry ink Industrial solvents; gun-cleaning products Antibacterial drugs Pyridium Chloroquine; Primaquine Dapsone Bactericide (tuberculostatic) Mothballs Fungicide for plants, seed treatment Antiseborrheic, antipruritic, antiseptic Matches, explosives, pyrotechnics Fires Benzocaine; lidocaine; Propitocaine; Prilocaine

Source: Hazmap[a]

Box 2: Chemicals for which methaemoglobinaemia is the primary toxic effect

Aniline 2-Chloroaniline 3-Chloroaniline 2-Chloro-m-toluidine Dichloroaniline (mixed isomers) Dimethylaniline m-Dinitrobenzene o-Dinitrobenzene p-Dinitrobenzene p-Dinitrosobenzene	Dinitrotoluene, all isomers Diphenylamine N-IsopropylanilineMBOCA Methyl nitrite Monomethyl aniline Nitric oxide m-Nitroaniline p-Nitroaniline	Nitrobenzene o-Nitrochlorobenzenep-Nitrochlorobenzene Nitrogen trifluoride 2-Nitronaphthalenem-Nitrotoluene o-Nitrotoluene p-Nitrotoluene	n-Propyl nitrate Propylene glycol dinitrate m-Toluidine o-Toluidine p-Toluidine 2,4,6-Trinitrotoluene Xylidene, mixed isomers

Source: Hazmap Occupational Exposures to hazardous agents (http://hazmap.nlm.nih.gov)

Box 3: Chemicals for which methaemoglobinaemia is the secondary toxic effect

2-Aminophenol	Cyclohexylamine	Methylhydrazine	Otto fuel
4-Aminophenol	Dapsone	Metolachlor	Pentaerythritol tetranitrate
Ammonium nitrate	1,1-Dimethylhydrazine	Monolinuron	PERCHLORATES
o-Anisidine	1,2-Dimethylhydrazine	Monuron	Perchloryl fluoride
p-Anisidine	Dinitolmide	Naphthalene	Phenol
Arsine	Dinitro-o-cresol	alpha-Naphthylaminebeta-Naphthylamine	p-Phenylene diamine
Benzidine	Dinitrophenol	NITRATES and NITRITES	Phenylhydrazine
Catechol	1,2-Diphenylhydrazine	Nitrogen dioxide	Propanil
Chloramide	Ethylene glycol dinitrate	Nitrogen tetroxide	Pyrogallol
CHLORATES	Hydrazine	Nitroglycerin	Resorcinol
Chlordimeform	Hydroquinone	5-Nitro-o-toluidine	Silver, metal and soluble compounds
Copper	Hydroxylamine	2-Nitrophenol	Sodium hypochlorite
Copper sulfate	Isobutyl nitrite	4-Nitrophenol	1,2,3,4-Tetrahydronaphthalene
Cresol, all isomers	Methyl formate	2-Nitropropane	Tetranitromethane
Cyclohexanone peroxide	Methyl mercaptan		
	Methylene blue		

Source: Hazmap Occupational Exposures to hazardous agents (http://hazmap.nlm.nih.gov)

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'FAST' thinking for binge drinking

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Introduction

Binge drinking is essentially 'drinking to get drunk'. It is often defined as intake over twice the daily guidelines in one day (8+units for adult males and 6+ units for adult females).[1] Alcohol is toxic and chronic heavy drinking can harm every body system. But young people, who reassure themselves that their drinking behaviour is unlikely to continue in the long term, are still putting their immediate health at risk. High consumption of alcohol, however infrequent, affects judgement and coordination, putting the individual at serious risk of accidents and trauma. Alcohol can contribute to all the major causes of unintentional death, for example, road traffic accidents, fires, falls and drowning.[2]

A fire service investigation has found that alcohol is the largest contributory factor in fire deaths, proposing the careless use of cigarettes, matches and cooking appliances (for example chip pans) as the main causes.[3] The report estimates that 87% of fire victims had been drinking excessively themselves or had died as a result of someone else's drinking.

Binge drinking affects an individual's ability to make judgements about other risky behaviours, for example unprotected sex and the increased risk of sexually transmitted infections and unwanted pregnancy.[1] Alcohol misuse is also linked to domestic and stranger violence. There are an estimated 75,000 violent incidents in London and 4000 call-outs to the London Ambulance Service each year, related to alcohol misuse.[4,5] The consequences are often seen in accident and Emergency Departments, where over half of admissions at peak times are known to be related to alcohol consumption.

It is not possible for an individual to gauge when alcohol consumption becomes a risk to health. Blood alcohol levels of less than 500mg/l will give a feeling of wellbeing and sociability.[6] 500-1000mg/l will affect emotional control, and the individual might notice slurred speech, poor coordination and balance. Intoxication occurs with a blood alcohol level of 1000-5000 mg/l, which can result in coma and convulsions. Respiratory depression, poor airway protection and hypothermia are associated with very severe intoxication (>5000mg/l). However every individual is different, hardened drinkers may get few of these symptoms, whereas teenagers unaccustomed to drinking alcohol may become unconscious with blood levels as low as 1000 mg/litre.[6]

Figure 1: FAST alcohol screening test

For the following questions please circle the answer which best applies.				
1 = ½ pint of beer or **1 glass of wine** or **1 single spirit**				

1. **MEN**: How often do you have **EIGHT** or more drinks on one occasion?
   WOMEN: How often do you have **SIX** or more drinks on one occasion?

0	1	2	3	4
Never	Less than Monthly	Monthly	Weekly	Daily or almost daily

2. How often during the last year have you been unable to remember what happened the night before because you had been drinking?

0	1	2	3	4
Never	Less than Monthly	Monthly	Weekly	Daily or almost daily

3. How often during the last year have you failed to do what was normally expected of you because of drinking?

0	1	2	3	4
Never	Less than Monthly	Monthly	Weekly	Daily or almost daily

4. In the last year has a relative or friend, or a doctor or other health worker been concerned about your drinking or suggested you cut down?

0	2	4
No	Yes, on one occasion	Yes, on more than one occasion

FINAL SCORE = [   ]   **If final score is more than 3, please offer advice and booklet**

One evidence-based approach to tackling binge drinking is the provision of proactive, opportunistic screening and brief interventions in the primary care or Emergency Department setting.[7] The brief intervention may be as short as five minutes with a health care practitioner giving brief advice and counselling. One of the drawbacks to this approach is the time commitment required by medical staff in, what are already busy settings. The screening and delivery of brief interventions, by nurse practitioners in the Emergency Department has not however, been fully explored. This is a role the nurse practitioners working in the Emergency Department of Queen Mary's Hospital, Sidcup (LB Bexley), in Southeast London, are keen to develop.

A pilot study was performed for one month, in November 2004, in which the nurse practitioners screened patients for hazardous drinking patterns and delivered brief advice. This paper reports the pre-publication results.

Methods

All consenting, fully conscious patients, over 16 years of age, treated by a nurse practitioner in the Emergency Department, were screened using the Fast Alcohol Screening Test (FAST) alcohol-screening questionnaire. All those with a positive result for hazardous drinking behaviour were offered a brief intervention by the nurse practitioner, an information booklet and contact numbers for further local and national help.

Figure 1 illustrates the FAST alcohol-screening test used in the study. Details of the original test development are given in a Health Development Agency manual.[8]

Demographic details; age, gender, postcode, ethnicity, date of attendance, diagnosis and outcome, were retrieved from the Emergency Department information system and analysed in FAST positive and negative groups, using STATA version 7.0.

Results

Over the one-month study period, the nurse practitioners in the Emergency Department treated 584 patients over 16 years of age. 58% of these were male, 42% female. This was a young population of patients, with 47% under 35 years of age.

Of the 584 patients treated by the nurse practitioners, 303 patients were asked if they would consent to screening. 274 patients agreed and 29 declined (90% of those asked, consented; 47% of the total treated by a nurse practitioner were screened). The nurses acknowledged that there were busy times when they either forgot or felt unable to spare the time to consent patients. 63% of the 274 people screened were male. The mean age of the screened population was 38 years, range 16 – 83 years.

Thirty-five percent of patients screened were FAST positive (a score of three or more from the screening questionnaire, Figure 2). Forty-one percent of males and 24% of females were positive. There were a significantly greater proportion of males in the FAST positive group compared to the FAST negative group (p = 0.004). 42% of those under 35 years of age were FAST positive (Figure 3).

The majority (88%) of patients treated by the nurse practitioners during the study period placed themselves in the 'White' ethnic
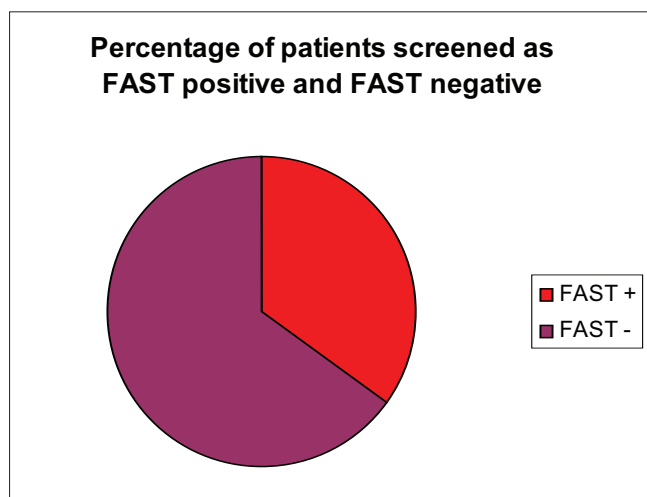

Figure 2: The percentage of all patients screened who scored 3 or more on the FAST alcohol screening test and are therefore likely to be binge drinkers
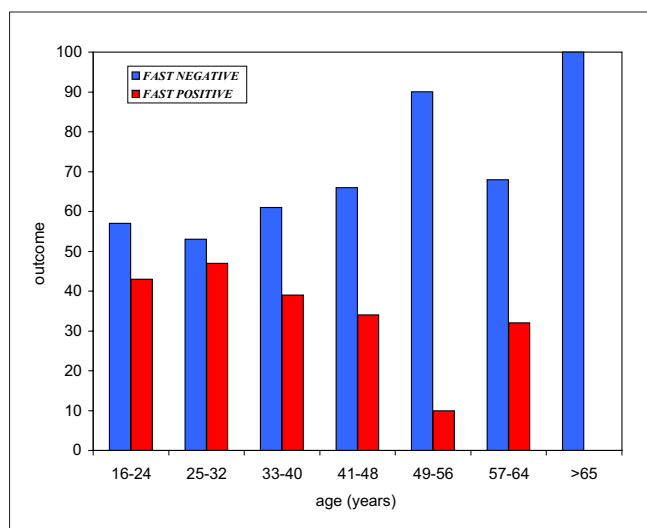
Figure 3: Outcomes of FAST screen per age band

category. There were too few patients screened in all non-White ethnic groups for any analysis to be made regarding the outcome of the screening test in individual ethnic groups.

There were a wide variety of diagnoses in both the FAST positive and negative groups. However, over two thirds (69%) of all patients presented with a fracture, a clinically suspected fracture, a soft tissue injury or a soft tissue wound. The number of individual injuries or conditions thought to be commonly associated with alcohol misuse for example, fractures of the hand (9 patients), fractured mandible (1 patient), dislocations (2 patients), emergency contraception (7 patients) and burns <15% (8 patients), were too small for comparison between FAST positive and negative groups.

50% of those with a fracture or a clinically suspected fracture were FAST positive. 34.8% of those patients with a soft tissue injury were FAST positive. 41.9% of those with a soft tissue wound were FAST positive.

Although it had been an original intention of the study to compare the occurrence of specific injuries in the FAST positive and negative groups, this was only possible for 'all fractures' (confirmed and

clinically suspected), 'all soft tissue injuries' and 'all soft tissue wounds', irrespective of injury site.

A null hypothesis was generated under the assumption that there was no change in the likelihood of sustaining a fracture, soft tissue injury or wound, irrespective of drinking habits. There were a significantly greater proportion of fractures than expected in the FAST positive group compared to the FAST negative group (Chi squared statistic = 4.3; p = 0.038). Controlling for gender (using logistic regression for binary outcomes) weakened the association between FAST outcome and fracture diagnosis (p = 0.055). This implies that part of the association found was explained by the gender differences in the FAST positive and negative groups and that there is only a marginal independent relationship between FAST outcome and fracture diagnosis.

If the proportion of fractures in the FAST negative group (10.1%) is expected to be the same as in the FAST positive group, it is possible to estimate that there were an excess of 8 (8.4) fractures in the population screened, which might be attributable to hazardous drinking. It can be extrapolated that within the population of 8431 patients treated by nurse practitioners in the year July 2004 – June 2005, there might have been an estimated excess of 258 fractures associated with hazardous drinking. This assumes that the results from the sample population can be generalized to the population of patients usually treated by nurse practitioners in Emergency Departments.

The mean number of attendances for all of the patients screened in the study was 2.9 in the period since the computerized record system was introduced in the Emergency Department in 1995. This was essentially equal in the FAST positive and negative groups (2.8 and 3.0 attendances respectively).

Discussion

This study demonstrated that 35% of patients screened by a nurse practitioner whilst attending for treatment in the local Emergency Department, tested positive for hazardous drinking behaviour using the FAST alcohol screening questionnaire. The inferred tendency to binge drink was more prominent in young, male patients. This may underestimate the true figure, as it may be that those who refused to participate were heavy drinkers or they were less approachable for screening. Similarly, this may be an overestimate, the nursing staff being reminded to ask the screening questions, if they suspected that the patient misused alcohol.

In this study there were a wide variety of diagnoses in both the FAST positive and negative groups. Even with the small numbers involved, it was possible to demonstrate significantly more fractures in the FAST positive group than expected compared to the FAST negative group.

The sample chosen was not representative of all people attending the Emergency Department during the study period. Many of the problems associated with screening for alcohol misuse in Emergency Departments, for example, acute intoxication, severity of injury and altered level of consciousness; all preclude screening and delivery of a brief intervention. The sample was therefore targeted to offset many of these difficulties and to maximise the likely health promotion benefit. The sample taken is also distinct from those *admitted* to hospital with alcohol related problems.

Prior to the study, the nurse practitioners at Queen Mary's Hospital,

Sidcup, voiced concerns regarding the number of patients that they treated with injuries related to alcohol. The nurse practitioners were very motivated to become involved in exploring ways to tackle the problem. They felt they were well placed to offer advice and highlighted that they already found themselves engaged in discussions regarding alcohol misuse with their patients. The sample represented a group of the population who are at risk and who are vulnerable, a group in which brief interventions have been shown to be effective.[9] This is also a population who tend to be discharged from the hospital with no other follow-up (as confirmed by the results of this study). The age and diagnostic profile of these patients suggests that they might not be regular attendees at a general practice surgery and so other opportunities for brief interventions might be scarce.

In this small sample, taken over one month, there were 8.4 more fractures sustained than expected, if the proportion of fractures in the FAST negative group was taken as the baseline rate. This will have implications for service provision for this group. Treatment for fractures involves radiology services and re-attendance at fracture/orthopaedic outpatient clinics for follow-up. In a much larger, prospective study of patients attending the Emergency Department of the Royal Liverpool University Hospital it was shown that for every 100 patients attending the Emergency Department with an alcohol related problem, at least 31 new patients would be seen in outpatients, attending 144 appointments over an 18 month period.[10,11]

The numbers of patients with other injuries, which might be suspected to be associated with alcohol misuse, were too small to detect differences in occurrence in FAST positive and negative groups. These include burns <15%, fractures of the base of the fifth metacarpal, minor head injuries, inversion/eversion injuries of the ankle and emergency contraception, for example. Alcohol has been linked to unsafe sex or abusive sexual activity, as it reduces self-control and impairs judgement. One study performed in 1989-90 in the north of England showed that 35% of students aged 19 years, had sex without contraception after drinking.[1]

Importantly, this study illustrated that nurse practitioners working in the Emergency Department were able to successfully administer the FAST alcohol screening questionnaire. Overall the nurses managed to screen between 38 and 56% of the patients they treated.

A post study questionnaire demonstrated that the idea of identifying people with hazardous drinking behaviour patterns and offering a brief intervention was supported by all of the nurse practitioners in the Emergency Department. A previously published postal survey of senior medical and nursing staff in Emergency Departments in Scotland demonstrated similarly positive results, with 82% of staff agreeing that it was worthwhile trying to identify such patients.[12] It was rare for the nurse to report that they had encountered difficulties when asking people about their alcohol consumption. The screening test was acceptable to both patients and staff. Three of the seven participating nurse practitioners did report that there were times when there was insufficient time to administer the test and the brief intervention. Self-administration of the test has been demonstrated as effective and will be considered for future service development. It was also acknowledged that consenting patients and explaining the study had taken a significant amount of time during the study period, which would not be necessary on a day-to-day basis. The four questions, which make up the FAST test, could all be easily incorporated into a normal conversation with the patient during the

consultation. They are all questions, which should legitimately be asked of almost every patient attending for treatment in Emergency Departments. It has been suggested that it is in fact negligent, not to ask these questions as this implies that the root cause of an injury is not fully investigated.[13]

A postal survey in Scotland revealed several important barriers to providing support for alcohol dependent patients in the Emergency Department setting.[12]. These included the mental state of the patient; the timing of attendance (which is often late); the brief period for which the patient is often in the department, making the building of a rapport difficult; and a lack of longer-term and community support and poor out-of-hours support for patient and staff. It is hoped that by screening only those patients seen by nurse practitioners in the Emergency Department, many of these issues are minimised.

The nurse practitioners felt that one of their strengths was their ability to form a rapport with patients in their autonomous, assessment and treatment capacity. Suitable models will vary for different Emergency Department settings across the country but it would seem that this model would work well within the Emergency Department at Queen Mary's Hospital in South East London.

Conclusions

1. This project has demonstrated that there is a significant problem with binge drinking amongst the population of people treated by a nurse practitioner at the Emergency Department of Queen Mary's Hospital, Sidcup, LB Bexley. 41% of males and 24% of females screened were positive using the FAST alcohol-screening questionnaire.

2. The project demonstrated that it is possible to screen a targeted, at risk population (all people attending a nurse practitioner in the Emergency Department). It has been previously demonstrated that brief interventions, delivered in the Emergency Department are effective.

3. The study demonstrated that the local nurse practitioners are willing and able to take on this health promotion role of screening and providing brief interventions in the Emergency Department setting.

4. The project has demonstrated that the characteristic profile of injuries and illnesses varies between those patients screening FAST positive and negative. There were significantly more fractures in the FAST positive group.

5. The project did not demonstrate any difference in the average previous attendance at the Emergency Department between FAST positive and negative groups. However, the differing profile of injuries might suggest an increase in the burden on some services from the FAST positive group.

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Exercise Merlin Aware

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Introduction

Exercise Merlin Aware was designed to test the multi-agency strategic response to a chemical, biological, radiological or nuclear (CBRN) incident and to give further consideration to the recovery stage of such an incident. This report outlines the initial planning process within the new resilience framework. It describes the lessons identified from the exercise and how these will be integrated back into the planning process.

Exercise Planning

Within County Durham a Joint Emergency Liaison Group (JELG) sits beneath the Local Resilience Forum structure, with representatives from all Category 1 and Category 2 responders. The Joint Emergency Liaison Group (JELG) has a number of working groups that are tasked to engage in various workstreams i.e. business continuity, emergency planning as outlined within the Civil Contingencies Act. An Exercise & Training Sub-Group was formed as part of this structure to develop multi-agency exercises to test specific risks across the area.

An initial planning team meeting identified that there was a requirement for the Local Authority to test one of their pipeline plans. Various possible scenarios were discussed. All agencies were keen to test their response to an incident that would have a huge impact on the area. The team was also keen to move the exercise on from consequence management to the handover of responsibility to the Local Authority for recovery issues. Agreement was reached to have a CBRN incident involving both a chemical and radiation component at different locations across the area.

Participants included:

Military	Atomic Weapons Establishment
Police	Fire Service
Ambulance Service	Health Protection Agency (including Regional/Health Emergency Planning Advisers, Chemical Hazards and Poisons Division, Radiation Protection Division & the Co Durham & Tees Valley Health Protection Unit)
Primary Care Trusts	Environment Agency
Network Rail	Northumbrian Water
Government Office North East	Local Authority
Northern Electric Distribution Ltd.	National Grid Transco

Exercise Summary

Exercise Merlin Aware ran over two days, the 14th and 15th October 2005. Its aim was to test a multi-agency strategic command's consequence management of a CBRN incident. This was the first multi-agency exercise within County Durham that would give consideration to, not only the initial impact and response, but also to the recovery. Although the simulated incident impacted on County Durham, flank players from Northumberland/Tyne & Wear and Cleveland were involved in recognition that mutual aid arrangements would need to be deployed.

This was the first exercise within the North East that tested the setting up of the Regional Civil Contingencies Committee (RCCC) arrangements at Government Office North East (GONE), a Health Gold and Joint Health Advisory Cell/Health Advisory Team (JHAC/HAT). This exercise took place during a transitional period when JHAC was moving on to the new HAT arrangements set out within the new Department of Health Guidance (2005). Under the old guidance a JHAC would be formed to deal with a deliberate release. A HAT is constituted to advise strategic command on both deliberate or non-deliberate incidents involving chemicals, biological, radiological or nuclear release.

Although primarily a strategic command level exercise, the scenario was also used to test the tactical response of the health service, through the activation and running of a Primary Care Trust (PCT) incident room at Easington and a Hospital Control Team at Darlington Memorial Hospital.

The scenario began with an explosion at Chilton, near Newton Aycliffe, which ruptured a pipeline at a gas compressor station. This threatened to engulf a neighbouring industrial premises which held significant quantities of toxic chemicals (chromium, cyanide and cadmium). As the scenario escalated, a dirty (radioactive) bomb was detonated in Durham Cathedral.

Health Gold/JHAC
Comments from the Regional Health Emergency Planning Adviser/ Health Emergency Planning Adviser:
- "The general structure of the JHAC/HAT worked very well and the support from Radiation Protection Division was both timely and helpful as was information and support from Chemical Hazards and Poisons Division (CHaPD).
- The two scenarios tested the JHAC/HAT structure and if these incidents had happened for real the formation of the RCCC would have been necessary at an earlier stage.
- There were long periods that JHAC/HAT Chair was away from the JHAC/HAT team and although a deputy was appointed there was a continuity problem with major health issues needing to be addressed whilst the multi-agency gold was sitting. The updated DH guidance recommends that 'The

Public Health Adviser will not usually fulfil the role of chair of HAT, but represent the team at the Strategic Coordinating Group (SCG) meeting' (February 2006 CHaPD Report article by Verity Kemp, 'NHS Emergency Planning Guidance 2005').

- The introduction of the Recovery Group (RG) and the link with JHAC/HAT team was not fully explored, probably because the JHAC/HAT team was still dealing with acute medical issues whilst the RG attempted to develop a recover strategy. Although this would happen in reality, the end result was the JHAC/HAT team became increasingly stretched because of trying to advise both the SCG and the RG

- The NHS Gold representatives were located in the same room as JHAC/HAT significantly increasing the number of people sitting around the table which impaired identifying key issues relating to public health as opposed to NHS capacity issues. The NHS Gold and JHAC/HAT started off as separate groups but merged. This may have been in part due to the shortage of space made available.

- The success of the JHAC/HAT was in no small way due to the experienced Chair who managed the cell in an effective and professional manner.

- The ability of the health teams to access information was significantly compromised by the lack of telephone, fax and internet access and should be addressed as a matter of some urgency.

- Overall the JHAC/HAT did make a significant contribution to the multi-agency response of the incident."

Communications

Comments from the HPAs Regional Communications Lead.

The primary objective of the exercise was to test the capabilities of various agencies' 'gold' command systems to work together, the operation of a SCG and in particular the response to a Chemical, Biological, Radioactive, Nuclear (CBRN)/hazmat incident. This was a great success.

Interagency communication between JHAC/HAT members also worked well as did communications between HPU members of JHAC/HAT and CHAPD.

A significant communications achievement during the exercise from a health perspective was that early public health messages were delivered timely and effectively to the broadcast media within the first hour. This is the first time this has been achieved in any multi-agency exercises within the North East.

The pre-agreed messages had been developed over the last year by local health communications leads and the Regional Media Emergency Forum and consist of generic messages for two types of incidents:

- Chemical, biological, radiological, nuclear – deliberate or accidental
- Large scale accident – train/plane/rail

The CBRN messages given out during Exercise Merlin were issued in a phased way as follows:

- Unless you have been advised to leave the area, stay indoors, keep the doors and windows closed and don't go out until you hear more about the nature of the incident on local radio - regular updates will be broadcast ('go in, stay in, tune in')

- If you were involved in the incident and think you were affected by the explosion or fumes, take advice from the emergency services at the scene about where to go to for decontamination or treatment. This is in the interests of your own safety and also that of others who you may come into contact with. Please wait until you receive advice from the professionals at the scene. Do not attempt to go to either your local hospital or your GP surgery of your own accord. To do so may result in a delay in your receiving the help you need as quickly as possible and it may lead to others being contaminated.

- If you were outside when it happened and were not close enough to the scene to be affected by the explosion or fumes but start to feel unwell (for example, if you develop a headache, running nose or streaming eyes) contact NHS Direct, where a trained nurse will give you advice. The number to call is 08 45 46 47.

- If you think someone you know has been involved in the accident do not ring local health and emergency services for information. They will be too busy dealing with the incident and casualties to take your call. Instead, listen to local radio and watch television for details of a helpline to ring. The number will be advertised as soon as possible. The people operating this helpline will have the latest information.

Lessons Identified

The multi-agency debrief identified a number of learning points:
- The physical location of the health gold and JHAC/HAT was situated too far away from other agencies and the gold meeting room.
- The equipment within the room was not adequate. There was only one phone line and no fax machine, although a fax machine was identified in the main police building, many messages were not being passed between Health Gold/JHAC and Exercise Control in a timely manner.
- In the future there is the need to arrive at JHAC/HAT fully equipped with mobile phones, lap tops with wireless internet connections and fax receiving capabilities, portable printers and a large clock
- The JHAC/HAT chair arrived with pens, paper, flip charts etc but it would be easy in a real event for these essentials to be forgotten.
- There is the need to ensure adequate administrative support for taking minutes, compiling attendance lists, reasons for decisions made etc.
- Decisions on the advice to the public could have been made faster and more accurate if maps of the relevant areas and CHEMET plume modelling had been provided when requested by JHAC/HAT.
- It would be preferable to allocate two rooms for Health Gold and JHAC/HAT in order to minimise disturbance and for members of each group to focus on their relevant issues.
- The Food Standards Agency, DEFRA and Northern Water did not take part in the exercise but in a real incident would have been key players for both incidents.
- Early media messages were used in a phased way and, therefore, need to be revised to reflect this process.
- Communications representatives would have benefited from their own meeting room.
- Gold meetings were too frequent and too lengthy which resulted in delays in communication between JHAC/HAT and Gold Command.

- An incident involving multiple sites and/or different agents would cause problems for communications in getting out messages (specifics – what caused problems and what solutions were proposed).
- Gold command was situated close to the second incident involving a radioactive bomb which in a real event might have required the evacuation of the command post leading to considerable disruption; an eventuality not normally considered.
- The two incidents were often described by different names which for some groups proved confusing. In a real event agreeing the name a particular incident would be referred to early would help.

Conclusion

The (R)HEPAs are working with the local police to advise on what facilities are required for health representatives at Strategic Command.

As part of the on-going training by the Department of Health (facilitated by the HPA) the NHS is undertaking Strategic Command Training.

The Regional Emergency Media Forum meets on a regular basis to discuss and implement changes to communication/media strategies.

New emergency planning guidance was published by the DH at the end of 2005. This will see the implementation/activation of a Public Health Advisor and a Health Advisory Team (rather than JHAC). The lessons learned from Exercise Merlin will be used to inform the planning process of a national HPA exercise to be run in June 2006 which will test the new arrangements and revisions implemented to procedures following Exercise Merlin.

Reflections from the perspective of a trainee in Public Health:
- "Unlike a Dan Brown book emergency plans are not the most gripping documents to read; however knowing you are about to take part in a major incident of unknown type really does focus the mind and need to understand the plans in detail such as the role of JHAC/HAT members, which emergency pods we have in the area, what is in them, and how we use them.
- I have previously been involved in table top exercises but none have come close to the reality of what it would be like in a real event. Throughout the two days of the exercise the experience felt "real" even though I knew it was not. Interestingly some of those who were involved in the exercise with me felt that the atmosphere was typical of the real Golds that they had participated in previously.
- I learnt a lot about chemical contamination and radiation and the different pathways that each can endanger human health, and the key roles of the other agencies in the JHAC/HAT.
- When my trainer was summoned to a press conference I found myself collecting and assimilating information on chemical contamination and the effects on human health and communicating this to the JHAC/HAT chair which is an experience I could never get in my day job.
- Having to assimilate information from different sources quickly, make decisions, and communicate messages to the public that are clear and unambiguous was a fantastic experience and excellent training for a real event.
- I also learnt the need to continuously reinforce the same health advice messages.
- I found the lack of provisions in terms of phones, computers, Internet connections and even a working clock surprising and quite disabling, rather like being asked to do your day job but without the tools.
- As someone who enjoys my food I would arrive at a real event with supplies of water and food as there is no one to look after you and what we were offered was definitely not in the healthy eating category."

Exercise Second Refine

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Introduction

On Sunday 4th December 2005 Exercise Second Refine ran on site at a solvent recovery plant in East Sussex. It was run as a live, 'real time' exercise, with a simulated emergency services response. The plant is the only site in East Sussex listed under the Control of Major Accident Hazards (COMAH) regulations. These regulations require on-site and off-site emergency plans which should be revised and tested at least once every three years.

The aims and objectives of the exercise were:

Aim

- To exercise the elements of the COMAH on-site and off-site emergency plans under simulated live conditions.

Objectives

- To exercise the co-ordination and integration of the emergency response

- To exercise the tactical and operational elements of the response

- To exercise the notification and communications arrangements for the responding agencies

- To examine procedures for warning and informing the public

Exercise Summary

The exercise was organised as follows:

The operational emergency services (bronze) were present nearby in a holding area, prior to the start of the exercise.

The tactical command centre (silver) was situated in a porta-cabin within the main plant area. Fire and rescue, ambulance, police and coastguard services were represented in this room.

In the more distant 'local authority control room', in telephone contact with silver command, were the Health Protection Agency (HPA), local authority and Environment Agency players. This room also had telephone contact with players situated off site at Bexhill and Rother Primary Care Trust.

The simulated emergency service response was commenced through the '999' call system. The HPA response was triggered through the usual 'out of hours' arrangement.

The initial scenario was that a lorry driver on site suffered a heart attack and crashed into a tanker loading methyl acetate. It transpired that the first lorry had an illegal tar burner, which ignited vapour from the tanker. Seven casualties from the accident needed to be rescued.

The fire caused ignition of a nearby tank of dichloromethane, heating the chemical to decomposition and causing the release of a 100m plume containing phosgene.

The main issues discussed were:

- The potential risks of the chemicals involved

- The need for sheltering versus evacuation in the plume area: CHaPD gave clear advice to shelter for those in the plume area

Exercise Second Refine

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- The need for personal protective equipment for rescuers

- Whether decontamination was needed for those exposed to the plume

- The health care response to children at a local primary school, who were potentially in the plume area

- Risks and actions with regard to boating activity in the local harbour

- The environmental consequences to local water supply, sheep farming and nature reserve.

Box 1 Phosgene

Phosgene is a colourless gas with an odour like musty hay. Inhalation may be fatal. Phosgene and its decomposition product hydrochloric acid cause direct irritation to mucous membranes, including the eyes and respiratory tract.

Following initial symptoms there may be an asymptomatic period before the onset of non-cardiogenic pulmonary oedema with severe breathlessness and a productive cough. These features may be delayed 24 (or rarely 48) hours.

Lessons learned by the HPU

- Overall the exercise provided an ideal opportunity to visit the site, meet key people and test the response using the facilities available.

- The Health Protection Unit (HPU) team needed to have more direct contact with silver command to ensure effective communication. This was a particular issue with regard to advising silver command about sheltering or evacuation.

- The information from the Chemical Hazards and Poisons Division (CHaPD) was provided rapidly by phone. However there were local difficulties during the exercise in receiving faxes or emails.

- The only fax machine was located at silver command, remote from the HPU team. Hence in future the HPU will take its own equipment to incidents and exercises, where necessary, to improve communications.

- The constraints of the exercise resulted in a compressed timescale and the local authority team were unable to fully consider the environmental impacts and actions.

- COMAH sites have chemical data sheets in their plans and wherever possible the HPU should be familiar with these chemicals. However this site was a re-processing plant which dealt with large numbers of chemicals. During the exercise the advice of the chemist employed by the COMAH site was particularly useful.

- The monitoring equipment held by the COMAH site was in the area of the plume and so unusable. Advice on environmental monitoring can be provided by CHaPD in the event of a real incident.

- The Health Protection Unit needs to be aware of the current Health and Safety requirements of staff going on site.

Reference
www.hpa.org.uk/infections/topics_az/deliberate_release/chemicals/phosgene.pdf accessed 13/07/06

Particles as Air Pollutants I: The ambient aerosol

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Introduction

Airborne particles are ubiquitous air pollutants. Concentrations in urban areas of the UK are now low in comparison with the days of the coal-smoke smogs, but recent epidemiological studies have shown, beyond reasonable doubt, that both long-term exposure to these particles and short-term exposure to raised concentrations damage health. In addition, it seems that no safe threshold of exposure can be defined: all concentrations appear to be capable of producing damage to health – at least to the health of some people. These findings have led to an explosion of interest in ambient particles and to concern about control of their sources. The Air Quality Strategy for England, Scotland, Wales and Northern Ireland deals extensively with particles and several National Advisory Committees have produced detailed reports on this air pollution.[1,2,3,4,5]

In the past ten or so years the suggestion that very small, ultrafine particles might be especially damaging to health has attracted support and, in the past few years, concerns about the toxicity of man-made nanoparticles including materials of nanoscale dimensions produced for a range of industrial and commercial uses has added to interest in this area.[10,11] Perhaps most surprisingly it has been recognised that the cardiovascular rather than the respiratory system seems to be the main target for the toxicological effects of the particles[6] It seems that though particles are inhaled and are either deposited in, or pass through the lungs into the circulation, damage to the lung is not the main problem except perhaps as a cause of signals, chemical and neural, which cause damage to, or cause perturbations of, the functioning of the heart and blood vessels. These are remarkable and unexpected findings: none of this was suspected as little as ten years ago. These findings have led both to increased concerns and misunderstandings. Despite the surge in research in this area, especially in the UK, the USA and in Europe, the exact mechanisms by which ambient particles damage health remain unknown – though there is no lack of plausible hypotheses. Distinguishing between assertions that rest on firm evidence and those that do not, has become important, as a number of effects have been attributed to particles without satisfactory evidence. Of course, it is difficult to prove that an effect is not produced by particles: often we lack evidence either for or against an asserted effect.

This short series of papers has been prepared with the intention of setting out what is known, or what should be known by those commenting on the effects of particles on health. We begin, in this paper by summarising some of the principles of aerosol science as applied to particulate air pollution.

The ambient aerosol

Physical scientists define an aerosol as:

> *"a colloid system in which the continuous phase (i.e. the dispersion medium) is a gas".*

This precise definition, modelled on that applicable to a liquid colloid suspension (a hydrosol) is perhaps less useful to the biologist than the definition provided by Muir (1972):

> *"The word aerosol is a general name referring to any atmosphere containing particles which remain airborne for a reasonable length of time...".* [9]

Green and Lane (1964) proposed a requirement that the size of the suspended particles should be sufficiently small to:

> *"confer some degree of stability, at any rate as far as sedimentation is concerned".*[7]

Fog, smoke, "smog", fume and the ambient air itself are all examples of aerosols. Aerosol science is a complex branch of physics and no attempt to deal with this subject in any extended way will be made here. Standard sources include Hinds' outstanding work *"Aerosol Technology"* and Green and Lane's seminal work *"Particulate Clouds: Dust, Smokes and Mists"*.[7,8] Muir's small but excellent book *"Clinical Aspects of Inhaled Particles"* is also helpful regarding the applied aspects of the subject.[9] Here only a short summary under a series of subheadings is provided.

How large are the particles in ambient air?

Particles in ambient air range from a few nanometres to the size of sand grains i.e. perhaps 0.5 mm in diameter. The smallest form as a result of condensation of small numbers of molecules of involatile substances and are typically produced by combustion processes (see Figure 1).
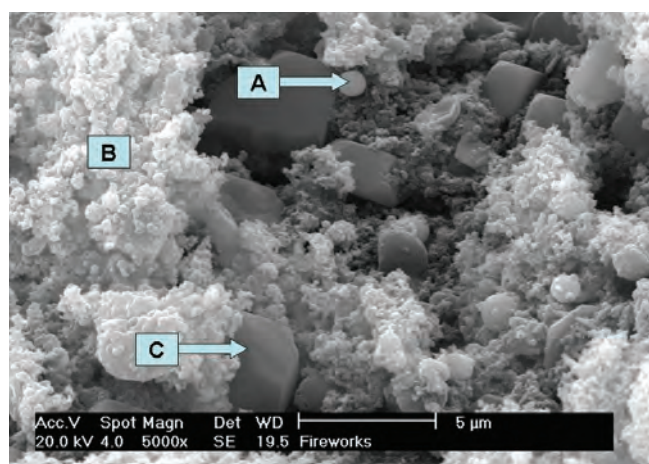
Figure 1: Electron micrograph of airborne particulate material
**A = Fe particle, B = Soot, C = Ammonium sulphates**
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Very small particles (or droplets – the terms are used interchangeably here) are also produced from gas phase reactions, for example, the formation of sulphuric acid from sulphur dioxide. The initial stage of forming such particles is described as nucleation, the deposition of material on the "nuclei" is described as condensation. Particles of less than about 10 nm in diameter are thus described as nucleation mode particles. Such particles though numerous weigh very little and contribute to only a small fraction of the mass concentration (see below) of the ambient aerosol.

Particles in the nucleation mode collide with one another as they move erratically through the air as a result of Brownian motion. When they collide they tend to stick together and to grow into larger particles. Growth in this way, and aided by further condensation on their surfaces of substances such as sulphuric acid, leads to particles in the 50 nm to 1 μm diameter range. This is described as the "accumulation mode". Particles in this size range make up a significant fraction of the total mass concentration, are long lived in the air and travel long distances: hundreds to thousands of kilometres.

Particles larger than about 1 μm diameter are described as "coarse mode" particles and are produced by physical processes such as the crushing of rocks, the lifting of sea spray from waves and the resuspension of dust for example by motor vehicles. Because these particles are comparatively heavy they have short lifetimes in the air – though they may be carried for long distances by winds – Saharan sand is not infrequently deposited in the UK. In actual fact most of the sand particles that are deposited in the UK are of less than 2.5 microns diameter: larger particles having been deposited en route to the UK. Coarse mode particles may contribute a good deal to the total mass concentration though compared with smaller particles the number of such particles per m³ is small. In terms of mass, coarse and accumulation mode particles may contribute about equal amounts to the total mass concentration of particles in the air.
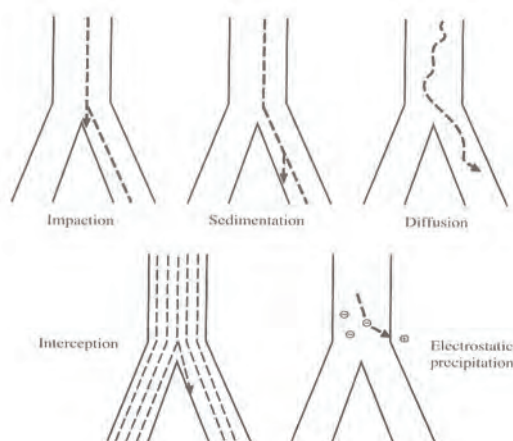
Figure 2: Mechanisms of Particle Deposition in the Respiratory Tract. Department of Health, 1995 © Crown Copyright.

Airborne particles leave the air by a variety of mechanisms (Figure 2). Three mechanisms: diffusion, sedimentation and impaction apply to particles both in the atmosphere and in the air taken into the respiratory system with each breath. Diffusion of particles – their movement in random directions through the air – is caused by molecules of gases bumping into the particles. Gas molecules are, of course, very small with tiny momentum and their impact would not be expected to move

large particles by very much. But small particles are displaced: this displacement is known as Brownian motion after the botanist Robert Brown who first observed the effect in 1827 amongst pollen grains suspended in a drop of water on a microscope slide. The rate of diffusion of a particle is inversely proportional to its diameter.

Gravity affects particles: they fall towards the earth's surface. As they fall they encounter resistance due to friction with molecules of gases and reach a so called terminal velocity. This may seem to contradict Galileo's famous demonstration of dropping two balls of lead of different sizes and noting that they reached the ground simultaneously. But, of course, large masses like lead balls are little affected by friction as they fall: a feather, on the other hand, is affected and feathers do not fall as fast as lead balls! The terminal velocity of a feather is less than that of a lead ball. Similarly, the terminal velocity of a particle of dust is less than that of a lead ball. The rate at which particles deposit by sedimentation is proportional to the square of their diameter.

Particles travelling in air streams continue in their original path for short distances when the air stream is deflected. This leads to impaction on surfaces. Here too the square of the particle diameter is a controlling factor. The combination of these three effects, Brownian motion, sedimentation and impaction (and interception – ignored for the moment) explains both dry deposition of particles from the air onto the ground, onto plants and buildings. They also explain deposition in the respiratory tract and on filters. It should be noted that coarse particles deposit efficiently by both sedimentation and impaction and that very small particles deposit efficiently as a result of diffusion, but particles of about 500 nm diameter (too big for diffusion and too small for impaction and sedimentation to be efficient) are only very slowly deposited. This in part explains the long life time of accumulation mode particles in the air and their low percentage deposition in the respiratory system. We shall return to this point later.

## Removal of particles from the air by rain

Particles grow in the air by the condensation of water vapour on their surfaces. Particles are also removed from the air by rain droplets: particles are "picked up" by the raindrops as a result of particles colliding with them under the forces of diffusion, impaction and interception. We would expect from the above discussion that particles in the 0.05-2.0 μm range to be least efficiently collected by rain droplets and this is the case. A proportion of these accumulation mode particles are, however, collected as a result of their forming nuclei from the condensation of water in clouds. Removal by rain is described as wet deposition. Wet and dry deposition remove particles from the air at different rates on wet and dry days: taking all days together, about 7% of 1 μm diameter particles and 15-30% of 3-5 μm particles are removed from the air per day.

Measurement methods applied to the ambient aerosol

Many methods for measuring the concentration of particles in the air have been developed. Some set out to measure the mass of particles in a given volume of air, others to measure how many particles occur in a given volume of air. Until recently, emphasis has been placed on mass concentrations. Methods also focus on different size ranges of particles. If we are interested in effects on health it is clearly sensible to collect only those particles capable of entering the airways. Large particles, between 10 and 100 μm in diameter enter the airways via the nose and mouth

but are efficiently deposited in the nose, mouth and pharynx and few reach the airways of the lung. Particles of less than about 10 µm in diameter reach the airways of the lung and a proportion are deposited there. As the inhaled air moves through the branching airways, the larger particles (within those less than about 10 µm in diameter) are deposited and thus the composition of the aerosol changes. By the time the air has reached the small airways and the gas exchange zone (the respiratory bronchioles, alveolar ducts and alveoli) rather few of the larger particles (within the fraction of the original aerosol that is < 10 µm in diameter) are left. A fraction of these small particles deposit in the small airways and gas exchange zone. A sequential removal of particles depending on size thus takes place as air moves from the nose and mouth towards the terminal airways and gas exchange zone.

Work by the International Standards Organisation (ISO) and the International Commission on Radiological Protection (ICRP) has led to a series of definitions of different size functions of the ambient aerosol. These are expressed as size distributions and are generally plotted as a series of cumulative distribution curves. Formidable equations have been fitted to these curves. The original work that lies behind the curves involves the use of dummies (mannequins) designed to represent the upper airways (nose, mouth and pharynx), physical models of the branching airways and mathematical models of the airways that include details of the length, diameter and branching angles of all the airways in the lung. Such mathematical models are based on studies of casts of the airways. Let us examine the standard curves.
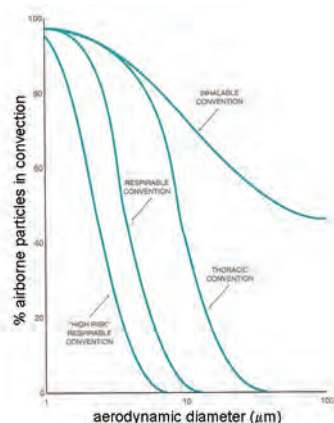
Figure 3:
ISO Health-Related Particle Sampling Conventions IS 7708 (1995)[12]
(reproduced from Department of the Environment,
1996, © Crown Copyright)[1]

Figure 3 shows the four "conventions": inhalable particles, thoracic particles, respirable particles and the so-called "high risk" convention for respirable particles. Let us look at the "thoracic convention". The curve represents a distribution of particle size. Note that the curve is S-shaped, or sigmoid. This sort of cumulative curve is derived from a bell-shaped curve: a similar shaped curve could be derived from the bell shaped (normal) distribution curve of, for example, heights of people in the UK. The curve shows that very few particles of diameter greater than about 20 µm get into the thoracic convention but that nearly all particles (close to 100%) less than 1.0 µm in diameter do. These cumulative distribution curves may at first glance, be confusing. It is important to remember that the curve shows the percentage inclusion of particles in the convention in terms of diameter.

The cumulative curves for the particle conventions are similar to those that can be derived from ordinary normal distribution curves, for

example, of people's height, but note that because the logarithm of the diameter is being used we speak of the average as the geometric mean rather than the ordinary arithmetic mean. If we are interested, as we are, in the shape of the distribution around the geometric mean we use the geometric standard deviation and not the ordinary arithmetic standard deviation (see Annex 1).

Let us look further at the thoracic convention curve. The geometric mean diameter is 10 µm and the geometric standard deviation is 1.5. Samplers have been designed to sample particles with almost the same size distribution as specified by the thoracic convention. These are described as PM_{10} samplers. The PM_{10} convention includes few particles of greater than 10 µm diameter. This has led to particles of less than 10 µm diameter being described as PM_{10}s. This is a loose usage of the term PM_{10}. The correct usage is: PM_{10} denotes the mass concentration of particles collected from the air by a device with an inlet designed to sample particles of 10 µm diameter with 50% efficiency and particles larger and smaller than 10 µm in accordance with a standard acceptance or transmission curve. Thus, PM_{10} refers to a mass concentration and it is more correct to say that the PM_{10} on Tuesday was 20 µg/m^3, than that the concentration of PM_{10}'s was 20 µg/m^3. In practice, PM_{10} is used both to denote the mass concentration and the particle size.

This rather long explanation applies, equally, to the other conventions. $PM_{2.5}$ represents, however, something of a complication. It was introduced as a modification of the "respirable convention" and is said to apply to children and the sick and infirm. In recent years, epidemiological studies have focused on $PM_{2.5}$ rather than PM_{10}. This is based on the perception that particles reaching the terminal airways and gas exchange zone of the lung may better represent the toxicologically active component of the ambient aerosol than PM_{10}. But it should be noted that PM_{10} will always include $PM_{2.5}$. Assertions that $PM_{2.5}$ can exceed PM_{10} are simply wrong. To conclude our examination of the "conventions" the following table should be examined. (Table 1).

Table 1: Inhalable, Thoracic and Respirable Fractions*

Aerodynamic Diameter (µm)	Inhalable Fraction	Thoracic Fraction	Respirable Fraction
0	1.00	1.00	1.00
1	0.97	0.97	0.97
2	0.94	0.94	0.91
3	0.92	0.92	0.74
4	0.89	0.89	0.50
5	0.87	0.85	0.30
6	0.85	0.81	0.17
8	0.81	0.67	0.05
10	0.77	0.50	0.01
15	0.70	0.19	0.00
20	0.65	0.06	0.00
25	0.61	0.02	0.00
30	0.58	0.01	0.00
35	0.56	0.00	0.00
40	0.55	0.00	0.00
50	0.52	0.00	0.00
60	0.51	0.00	0.00
80	0.50	0.00	0.00
100	0.50	0.00	0.00

*(Hinds, 1995, used with permission) 8, * ACGIH (1997)[13]*

Deposition of particles in the airways

The conventions discussed above tell us about sampling of particles: they do not tell us about the deposition of particles in the airways. This is important. We need to recall that the respirable convention tells us about those particles that can reach the terminal airways but not about the fraction of those particles that will be deposited there. The first thing to know about deposition is that fewer particles are deposited than actually reach the zone being considered. Figure 4 shows the ICRP curve for particle deposition in the lung. Look first at the total
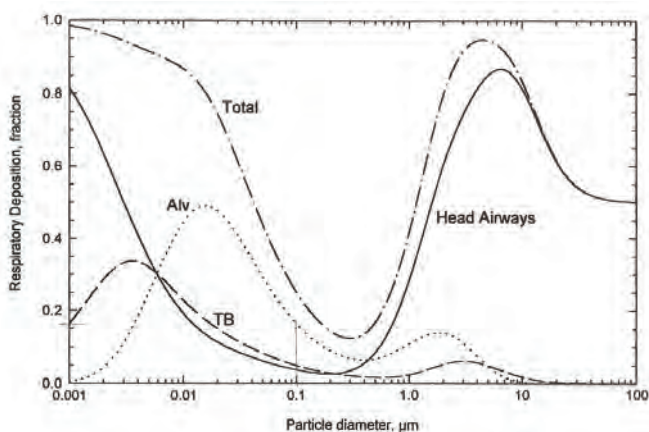
**Figure 4: Predicted Total and Regional Deposition for Light Exercise (nose breathing) Based on ICRP Deposition Model
(Hinds, 1995, used with permission)[8]**

deposition curve. This shows a prominent dip at about 0.5 μm particle diameter. Why is this? The answer is that large particles deposit efficiently by impaction and sedimentation and that very small particles deposit well by diffusion. None of these processes is very effective with particles of about 0.5 μm diameter and thus these do not deposit efficiently anywhere in the airways. Look next at the Head Airways
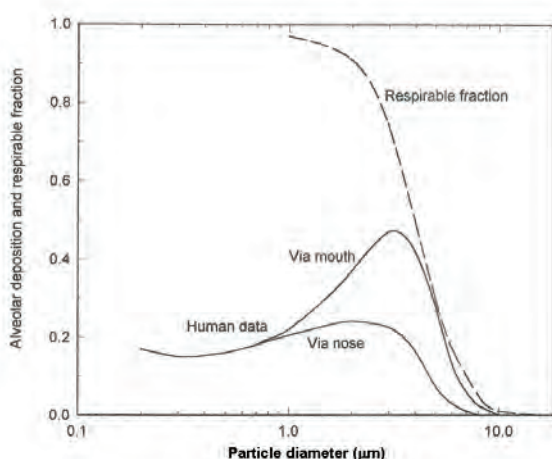
Figure 5: Comparison of Experimental Measurements of Alveolar Deposition and ACGIH Respirable Fraction (Hinds, 1995, used with permission)[8]

curve. Large particles deposit efficiently but so do very small particles. The latter may come as a surprise! Particles of less than about 10 nm (0.01 μm) in diameter deposit efficiently, by diffusion, in the upper airways of the head and few reach the lungs. Turn now to the curve for alveolar deposition. Note, firstly, that few particles of >10 μm diameter

are deposited in the alveolar region – this is because few reach the alveolar region: they are being efficiently removed by the upper airways of the head. Note, next, that there is a small hump at about 2-4 μm and a much bigger hump at about 20 nm diameter. Lastly, look at the tracheobronchial (TB) curve: deposition is rather inefficient at all diameters above about 0.1 μm.

We can now see how the "convention" curves have been derived. Figure 5 taken from Lippmann's work shows that the respirable fraction (defined by the respirable convention) will "catch" all the particles that could be deposited at the alveolar level during mouth or nasal breathing. The alveolar deposition fraction during mouth breathing is greater than that during nose breathing: the nose is an efficient particle filter.

Methods for measuring particle concentrations

Older methods

Black Smoke measurements are seen by some (especially in the United States) as an especially British method of measuring the mass concentration of particles in the air. Indeed, Black Smoke is sometimes referred to as British Smoke – both terms being abbreviated to BS. The method is simple: air is drawn through an inlet and then through a piece of filter paper. Particles are trapped on the paper which becomes blackened. The degree of blackening is later assessed by measuring the filter's capacity to reflect light. A calibration curve based on a gravimetric method is needed to convert the reflectance results into a mass concentration. The whole system imposes a size sampling on the particles reaching the filter: the method can be thought of as a special way of measuring the mass concentration of particles less than about 4 μm diameter. Of course, very black particles such as are found in diesel exhaust darken the filter more effectively than paler particles and the calibration and thus the results will be less than ideally accurate across a range of different aerosols. The ambient aerosol is affected by site (location) and by season and thus a mass concentration of 20 μg/m^3 measured in Cornwall may be a less good indicator of the actual mass concentration measured with the Black Smoke method in, say, Belfast. But Black Smoke measurement has a long history in the UK and runs of data lasting for >50 years are available at some sites. These provide a valuable resource for epidemiological studies and maintenance of at least some Black Smoke sites seems sensible.

Less well known in the health community are the M (metal) and S (sulphate) samplers first used in the UK in the 1970's. These samplers also collected particles on filters but the size distributions of the particles collected differ from that of the Black Smoke system: the M and S samples were, in fact, fairly close to a PM$_{10}$ sampler. Filters were weighed and the system was useful for determining long-term (e.g. annual) average concentrations.

Newer methods

Modern methods focus on PM$_{10}$ or PM$_{2.5}$; very recently, particle counting systems for the determination of number concentrations have been introduced at a few sites. The sampling devices in use can be divided into a number of groups.

(a) Gravimetric, cumulative samplers

Here, both high flow rate (e.g. Graseby Anderson PM$_{10}$ HiVol sampler) and low flow rate (e.g. Casella PQ167 Portable PM$_{10}$ Sampling Unit) instruments are available. All can be fitted with a sampling head that accords with the PM$_{10}$ sampling standards. The Graseby Anderson dichotomous sampler provides a two-stage approach with both PM$_{10}$

and PM2.5 being collected on Teflon membrane filters. All gravimetric cumulative samplers depend on weighing of the filter with its accumulated particles.

(b) Direct reading instruments

Three methods are available: optical, oscillating microbalance and beta attenuation. Optical methods rely on particles interacting with light and the extent of this interaction will depend not only upon the number of particles present but also upon their size distribution, shape and refractive index. Thus, calibration against a standard is necessary before mass or number concentration can be reported. Several monitors are available, including the Data RAM Portable Real-Time Aerosol Monitor and the Grimm Stationary Environmental Dust Analyser. The oscillating microbalance (Rupprecht Pataschnick Tapered Element Oscillating Microbalance or TEOM) is in widespread use in the UK as a method for providing real-time monitoring of PM10. A size selective head selects the particles – those admitted are deposited on a filter mounted on a tapered oscillating glass tube. The frequency of oscillation of the tapered element is dependent on the mass added to the filter and, critically, the change in frequency is proportional to the change in mass on the filter. The frequency is recorded and thus a continuous output reflecting the rate at which particles are deposited on the filter is obtained. Data on mass concentrations are collected every few seconds and combined to provide 15 minute and hourly data. The latter are made available to the public and archived. From these data the 24-hour average mass concentrations can be easily derived. Some problems occur: the air inlet is heated to 50 ºC to remove water bound tightly to particles. This process also removes a proportion of semi-volatile inorganic and organic substances and thus, in comparison with a gravimetric method, the TEOM "under-reads". A correction factor of 1.3 is applied to allow for this systematic error. Data from the Defra Automatic Urban Network sites are recorded with TEOM monitors (Figures 6a and 6b).

Beta attenuation devices depend on the reduction in intensity of beta particles passing through particles deposited on a filter. The beta particles are absorbed by the aerosol particles and the mass concentration of the aerosol is proportional to this absorption. Calibration using the mass absorption coefficient for quartz is used. But this leads to inaccuracies as the ambient aerosol is not pure quartz and the mass absorption coefficient of the ambient aerosol can vary by up to ± 20%. Several devices based on these principles are available: they are more widely used in some other European Countries than in the UK.
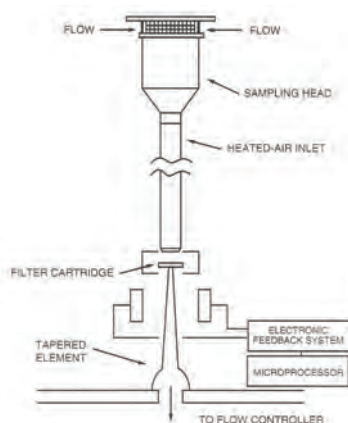

Figure 6a: Schematic Diagram of Rupprecht and Patashnick TEOM Ambient Aerosol Monitor (Department of the Environment, 1996, © Crown Copyright)1
Figure 6b: Photograph of a TEOM (© ERG, King's College, 2006)

Conclusion

The ambient aerosol comprises a complex mixture of particles of varying sizes and compositions. Particles taken into the lung are deposited by well understood mechanisms and sampling devices have been developed to mimic the capacity of the airways to restrict particle entry by size. PM10 and PM2.5 are commonly used metrics for the mass concentration of particles in the air. It should be understood that of the particles reaching any part of the airways of the lung, only a fraction are deposited. Deposition curves are available to predict regional deposition along the airways. Particles are collected and their mass or number concentrations determined using a range of devices. Of these the TEOM instrument is most widely used as a means of determining PM10 and PM2.5. The Black Smoke method is also in fairly wide use and long runs of data provided by this method are available.

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Annex 1

Aerosol science involves a good deal of advanced physics and mathematics and thus presents a challenge to the biologist. In this section a few of the fundamental terms are discussed using only basic mathematics.

Means and Standard Deviations

Let x represent the diameter of a particle.

For a set of N numbers, $x_1, x_2, x_3 \ldots \ldots x_N$
the arithmetic mean (m) is given by:

$$m = \frac{\sum_{i=1}^{N} x_i}{N}$$

The standard deviation (lower case sigma) is given by:

$$\sigma = \sqrt{\frac{\sum_{i=1}^{N} (x_i - m)^2}{N}}$$

The geometric mean and geometric standard deviations are given by:

$$m_g = anti\log \frac{\sum_{i=1}^{N} \log x_i}{N}$$

$$\sigma_g = anti\log \sqrt{\frac{\sum_{i=1}^{N} (\log x_i - \log m_g)^2}{N}}$$

respectively.

Consider two values of x, x_a and x_b. Let 68% of values of x fall between x_a and x_b on a normal distribution curve.

$$\sigma = m - x_a = x_b - m$$

Consider two values of log x, log x_a and log x_b

Let 68% of values of log x fall between log x_a and log x_b on a log-normal distribution curve.

$$\sigma_g = anti\log(\log m_g - \log x_a) = anti\log(\log x_b - \log m_g)$$

Recall that:

$$anti\log(\log A - \log B) = \frac{A}{B}$$

$$\sigma_g = \frac{m_g}{x_a} = \frac{x_b}{m_g}$$

$$\sigma_g^2 = \frac{m_g}{x_a} \cdot \frac{x_b}{m_g} = \frac{x_b}{x_a}$$

$$\sigma_g = \sqrt{\frac{x_b}{x_a}}$$

Calculation of the Inhalable Fraction (IF) and the Deposition Fraction (DF) for particles of given diameters

Formidable equations have been fitted to the empirically derived curves for the inhalability and deposition fractions for particles of specified size. Details may be found in Hinds' account (Hinds,1995).

For windspeeds of less than 4 m/second the inhaled fraction can be calculated from:

$$IF(da) = 0.5(1 + \exp(-0.06da))$$

da = aerodynamic diameter

The equations for the deposition fraction are more complicated. Remember that they have been fitted to the rather irregular curves we looked at earlier and thus cannot be expected to be simple equations. One example only, is given. Consider deposition in the alveolar region of the lung (DF_{AL}).

$$DF_{AL} = \left[\frac{0.0155}{dp}\right]\left[\exp(-0.416(l_n dp + 2.84)^2) + 19.11\exp(-0.482(l_n dp - 1.362)^2)\right]$$

This equation may be easily solved for a particle of given diameter *dp*. It needs to be attacked in 3 parts i.e. as:

$$DF_{AL} = A[B + C]$$

A, B and C have been added above and below the equation. Consider a particle of diameter 0.7 µm.

A is easy:

$$\frac{0.0155}{0.7} = 0.0221$$

B and C are more complicated, but still easy to work through on a pocket calculator.

One piece of mathematics needs to be recalled. The logarithms we are working with are natural logarithms i.e. logarithms to the base e.

Imagine that y = *ln x*

We could write antilog to the base *e* y = x but this is never done, instead we write *exp y = x*.

On a pocket calculator exp y is evaluated by entering y, then pressing "INV" and then the "*ln x*" key. Your calculator may not have an INV key, in this case it probably gives exponentials directly.

From the equation given above take B first. Begin with *ln dp*. This is -0.3567. Then proceed through the inner bracket, adding 2.84 to get 2.4833, squaring this to get 6.1669, multiplying by 0.416 to get 2.5654, changing the sign yielding -2.5654 and then pressing "INV" and then "*ln x*" to give 0.0769.

Now evaluate C to give 4.6789 and multiply by A to give 0.1029.

Thus the alveolar deposition fraction for particles of 0.7 µm diameter is 10.29%.

Computer programs are available to perform these calculations! Details can be found in Hinds' account.

Aerodynamic diameter

The aerodynamic diameter of a particle is defined as the diameter of a spherical particle of unit mass that has the same terminal velocity as the particle in question. The aerodynamic diameter *da* of a spherical particle is given by:

$$da = dp\sqrt{\frac{pp}{po}}$$

where dp = actual diameter of the particles, *pp* = density of the particle and *po* = density of water = 1 g/cm³.

The Air Quality Strategy for England, Scotland, Wales and Northern Ireland: New approach to the regulation of fine particles

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Introduction

The last issue of Chemical Hazards and Poisons Report explained that the Air Pollution and Noise Unit, led by Dr Robert Maynard, recently transferred to the Health Protection Agency (HPA). A previous article[1] set out recent work from the Committee on the Medical Effects of Air Pollutants (COMEAP) to which the HPA now provide the Secretariat. This article covers another major aspect of the unit's work – providing health advice to the Department for the Environment, Food and Rural Affairs (Defra), the government department in the lead on air pollution policy.

The Air Quality Strategy

The Air Quality Strategy sets out the policy of the UK Government and the devolved administrations for reducing levels of air pollution. It sets objectives for nine key air pollutants (Box 1). Local authorities are required to assess air pollution levels in their areas against these objectives and to draw up action plans to reduce air pollution levels if the objectives are unlikely to be met.

Box 1 Pollutants included in the Air Quality Strategy

- Benzene
- 1,3-butadiene
- Carbon monoxide
- Lead
- Nitrogen dioxide
- Ozone
- Sulphur dioxide
- Polycyclic aromatic hydrocarbons
- Particles (PM$_{10}$)
- Particles (PM$_{2.5}$) (proposed)

Developments over time – from standards to slopes

The Air Quality Strategy was first published in 1997.[2] At that time[a], the starting point for each objective was an air quality standard recommended by EPAQS – the Expert Panel on Air Quality Standards.[3] These standards gave a specific level of an air pollutant which should not be exceeded and were based only on health considerations. The strategy would then take into account feasibility and costs in setting an objective with a timescale and agreed exceptions. For example, the EPAQS standard for ozone is 50 ppb (100 μg/m^3) daily maximum 8 hour average. The objective states that this standard should not be exceeded more than 10 times a year and should be met by December 2005.

A major change in thinking occurred in 1998 with the publication of a report from COMEAP.[4] This report -'Quantification of the Effects of Air Pollutants on Health in the UK'- recommended concentration-response functions for quantifying the effects of changes in levels of air pollution on health. For example, the report suggested that a 10 μg/m^3 reduction in PM$_{10}$[b] was associated with a 0.8% reduction in respiratory hospital admissions[c]. These concentration-response functions were derived from epidemiological studies of the general population and so could be used to predict effects in the general population. This report paved the way for a more quantitative comparison of costs and benefits when setting objectives for the air quality strategy. The 1998 report has since been supplemented with a statement on cardiovascular admissions[5], a 2001 report[6] on concentration-response functions for the effects of long-term exposure on mortality and a recent interim statement[7] updating the 2001 report.

Another important issue is the fact that the epidemiological studies do not appear to suggest a threshold – the concentration-response function is compatible with a linear relationship down to the lowest concentrations examined. This is not to suggest that all individuals are affected at all concentrations, only that some individuals in a population will be affected. Some individuals are more susceptible than others and some individuals will have higher personal exposures to air pollutants than others (the studies use outdoor air pollutant concentrations as a surrogate for the distribution of personal exposures in the population).

This epidemiological evidence did not sit easily with the earlier approach that was strongly driven by whether or not a particular concentration (standard, objective or limit value) was exceeded. This meant that attention was concentrated on reducing pollutant levels in small 'hotspot' areas of the country above the standard (e.g. roadsides in London) which would generate small overall public health benefits. On the other hand, little attention was paid to further reducing levels in other areas which were already below the standard. The latter approach would generate larger public health benefits as it applies to a greater proportion of the population. If costs and feasibility are considered as well, it is often much more difficult and expensive to reduce pollution in the 'hotspot' areas than to reduce pollution more generally.

All of these considerations have led to proposing an 'exposure reduction' objective of a 15% reduction in PM$_{2.5}$ concentrations between 2010 and 2020 in the most recent review of the strategy.[8] This objective provides a policy driver for ensuring continued reductions in the country overall. This exposure reduction objective is supplemented by a proposed 'concentration cap' of 25 μg/m^3 on equity grounds. This means that any areas of the country above 25 μg/m^3 have to reduce concentrations to this level in any case. The concentration cap is set at a level which it is considered reasonable to achieve without great difficulty, i.e. if it is not achieved, it is considered that the population in that area of the country are being unfairly exposed to higher levels. This concentration cap is not the same as a standard or limit value as it is not the main policy driver for improving health: it is the exposure reduction objective which will drive levels down further below the concentration cap.

Highlights from the recent review of the Air Quality Strategy

The Air Quality Strategy has been reviewed several times to ensure it keeps up to date with the latest scientific evidence. A consultation document8 on the latest review of the Air Quality Strategy was published in April 2006. Highlights from this document are given below.

- Summaries of current levels of air pollutants and projected levels in 2020 are presented. This shows that good progress is already being made (Figures 1a and 1b) although more needs to be done.

- The average loss of life expectancy from current levels of man-particles on those born in 2005 (assuming that they are exposed to the same level for the rest of their lifetimes) has been estimated as a loss of up to 8 months. The uncertainty involved in this calculation is acknowledged[9]. This average loss of life expectancy is predicted to drop to $5\frac{1}{2}$ months or less by 2020.

- A detailed assessment of the costs and benefits of 14 possible measures to further reduce air pollution has been performed. This involved using the slopes described in the previous section to quantify the benefits. The consultation document proposes implementing, or strongly pursuing in international fora, the following measures based on their favourable benefit to cost ratio:

    - 3 variants of new European standards for vehicle engines (one likely to require particulate filters)

    - incentives to increase penetration of low emission vehicles into the vehicle fleet

    - a national road pricing scheme (although only one possible illustrative variant was considered)

    - reducing emissions from small combustion plants

    - reducing emissions from ships

- A package of measures covering new European vehicle standards, encouragement of low emission vehicles and reductions in emissions from small combustion plants was also considered (taking optimum timing as well as costs and benefits into account). This was the package used to justify the exposure reduction objective of 15% for $PM_{2.5}$.

- The consultation document proposes retaining almost all the current objectives but suggests replacing the previous PM_{10} objective for 2010 with the new exposure reduction objective for $PM_{2.5}$ described above. This is the first time an objective for $PM_{2.5}$ has been included in the strategy – this is in accordance with the developing evidence that finer particles are more harmful to health.

- Three new objectives for protection of ecosystems from oxides of nitrogen, sulphur dioxide and ozone are proposed.

- A 'long-term vision' chapter considering possible trends in air pollutant concentrations out to 2050 suggests that continued reductions in particles and nitrogen dioxide should be possible. Ozone reductions are considerably more challenging and would need large reductions in ozone precursors and global co-operation.

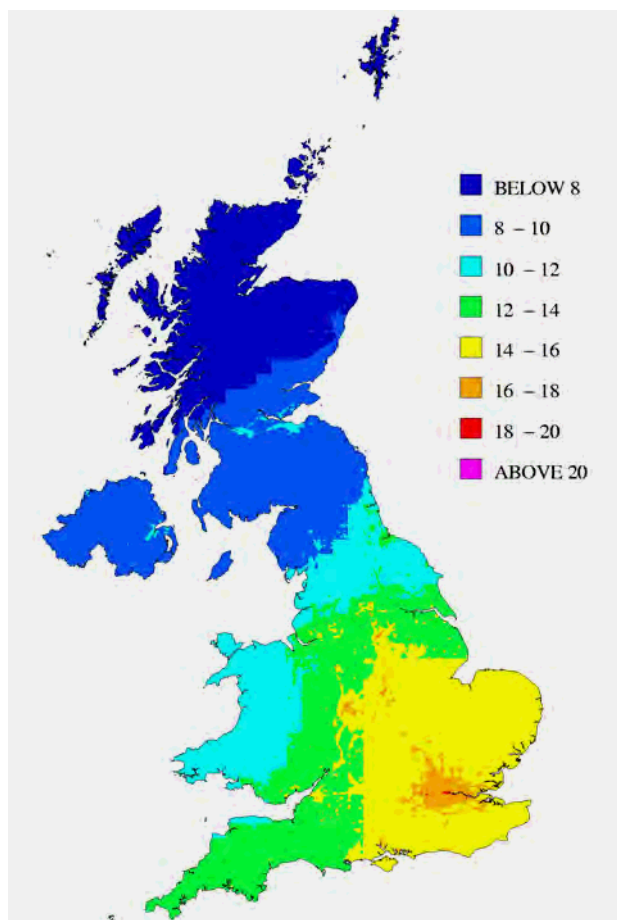**Figure 1a: Annual mean background PM$_{2.5}$ concentrations, 2003 (µg/m^3, gravimetric)**
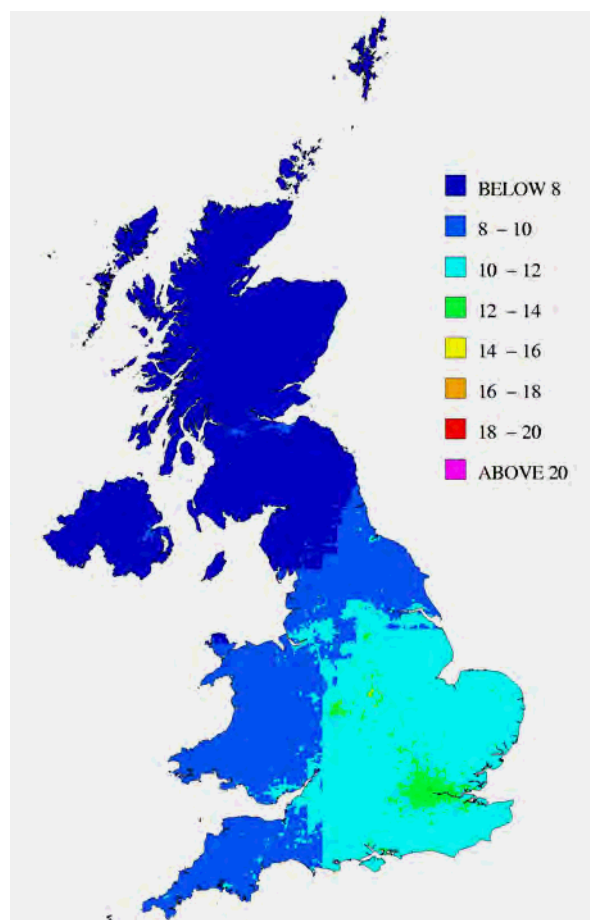

Figure 1b: Annual mean background PM$_{2.5}$ concentrations, 2020 (µg/m^3, gravimetric)

(Taken from 'The Air Quality Strategy for England, Scotland, Wales and Northern Ireland – A consultation document for further improvements in air quality Volume 1 April 2006' with permission from Defra. Crown Copyright)

Summary

Developments in the health evidence are an integral part of the development of the Air Quality Strategy from the original standard setting process to the more recent moves to more sophisticated cost-benefit analysis and shifts in the regulatory approach towards an exposure reduction objective. The Air Pollution and Noise Unit of the Chemical Hazards and Poisons Division of the Health Protection Agency will, with the advice of COMEAP, continue to ensure that changes in the understanding of the health evidence are taken into account in future policy developments.

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a   Over time, limit values from European directives, based on WHO guidelines, have been added to the strategy, in addition to, or instead of, objectives based on EPAQS standards.

b PM_{10} is defined as particulate matter which passes through a size-selective inlet with a 50% efficiency cut-off at 10μm aerodynamic diameter. $PM_{2.5}$ is similarly defined for a 2.5 μm aerodynamic diameter.

c   The time-series studies used to derive the concentration-response function simply shows that fewer respiratory hospital admissions occur on days with lower particle concentrations compared with days with higher particle concentrations. There is no information as to whether the respiratory hospital admissions are shifted in time (i.e. they will still occur but later than would otherwise be the case) or whether the respiratory hospital admissions have actually been prevented.

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The use of Geographical Information Systems (GIS) for exposure assessment

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Introduction

The unique ability of Geographical Information Systems (GIS) to integrate different spatial data sets has meant that it has been used for exposure assessment to help with environmental health studies. There have been several comprehensive reviews of this topic (Briggs and Elliott, 2005) (Briggs, 2005) (Nuckols *et al*., 2004) (Jerrett *et al*., 2005) (Dent *et al*., 2000) (Briggs, 2003). The purpose of this article is simply to outline the different ways in which GIS can be used for exposure assessment and to highlight the advantages and disadvantages of these approaches. It is based on a literature review completed as part of an Engineering Doctorate (EngD) based at the University of Surrey and the Health Protection Agency (HPA).

Exposure assessment

Exposure in environmental epidemiology or occupational epidemiology is usually defined as any contact between a substance and the surface of the human body (Nieuwenhuijsen, 2003). Exposure assessment is the study of the distribution of substances or factors that affect human health. It is usually carried out to try to establish the nature of the relationship between any exposure and its possible health effects. Exposure assessment can be used for epidemiological studies, risk assessments or health surveillance. In developed countries, the health risks associated with environmental exposure to chemical hazards would be expected to be relatively low. This means that, in order to detect risks when and where they exist, exposure assessments need to be very refined to enable resolution of small differences in exposure as accurately as possible. For the above reasons exposure assessment plays a crucial role in environmental health research.

GIS

A GIS is essentially a database system that allows large quantities of information to be analysed and viewed within a geographical context (Vine *et al*, 1997). This enables data to be manipulated, integrated and graphically displayed on maps. Essentially, GIS has the ability to integrate data, as illustrated in Figure 1, and this makes it useful for exposure assessment. GIS allows for the source - pathway - receptor model to be represented in both space and in time, meaning that the two spatial patterns of interest in exposure assessment, the distribution of pollutants and the distribution of people, can be linked together.
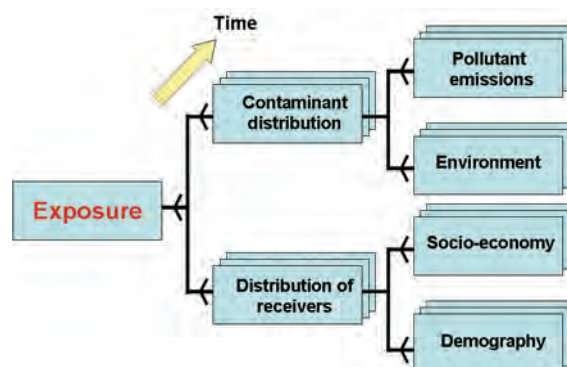

Figure 1 How GIS enables information to be integrated both in space and time to assess exposure (adapted from Briggs, 1992)

Use of GIS in health studies which use a proxy variable for exposure

Following the development of GIS in other fields throughout the past few decades, many researchers have used the technique for exposure assessment. However, because data about exposure to pollutants are not always readily available, many researchers have simply used distance from place of residence to the source of pollution as a proxy for exposure. This has particularly been the case for landfill sites, whose emissions are notoriously difficult to characterise (South West Public Health Observatory, 2002). For example, the Small Area Health Statistics Unit (SAHSU) study used a distance-based technique to investigate cases of congenital anomalies around landfill sites in Great Britain between 1982 and 1997 (Elliott *et al*, 2001). The study used a database that contained the postcode of every landfill site in Great Britain. Using GIS, all postcodes that were within 2 km of a landfill site postcode were classified as exposed, and those that were more than 2 km away were classified as unexposed. Similar or somewhat more sophisticated approaches have been used by other authors for exposure assessment (Briggs, 2005).

Using GIS to integrate dispersion modelling results with population data to estimate exposure

There are numerous software models available for estimating the distribution of pollutants in the environment (Defra & Environment Agency, 2002) (James *et al*, 2004) (Carruthers *et al*, 1992). Models are used because adequate environmental monitoring is rarely available for an entire area and time period of interest. The models are widely used for regulatory purposes; however, by using GIS and considering other variables that may affect exposure, they can also be applied for exposure assessment (Scoggins *et al*, 2004) (Walker *et al*, 1999) (Brody *et al*, 2002) (Brody *et al*, 2004) (Maslia *et al*, 1994) (Kinra *et al*, 2005).

In a case control study of stillbirths, Ihrig *et al,* (1998) investigated adverse reproductive outcomes around an industrial plant in Texas (USA), which released arsenic into the atmosphere (Ihrig, Shalat, & Baynesm, 1998). Information was collected on 119 cases and 267 controls between 1983 and 1993, with exposure estimated using an air dispersion model, the fugitive dust model that was developed by the United States Environmental Protection Agency (USEPA). The model took account of the amount of arsenic emitted from the plant, the prevailing wind direction and other atmospheric conditions, the velocity of the emissions, the height of the stack and the size of the particles emitted. The model was used to predict arsenic concentrations for a 16-year period from 1973 to 1989. Exposure levels were estimated for residential addresses of study subjects at the time of delivery of their babies. This study was superior to some others because estimating exposure using predicted levels of pollutants should be more realistic than using a proxy measure such as distance. The modelling was not, however, validated by comparison with measured data and obtaining suitable measured data for such purposes remains a problem for many studies.

Following a major fire at a plastics factory, Kinra *et al*. (2005) carried out an epidemiological study to compare health outcomes in people who sheltered during the fire and those who were evacuated. The Numerical Atmospheric dispersion Modelling Environment (NAME) code was used to model atmospheric emissions from the fire. The output from the modelling was linked to postcoded population data using GIS (Figure 2). Using this exposure assessment, the epidemiological study showed that evacuation conferred no protective advantage over sheltering and that there was some evidence of adverse health effects associated with evacuation.
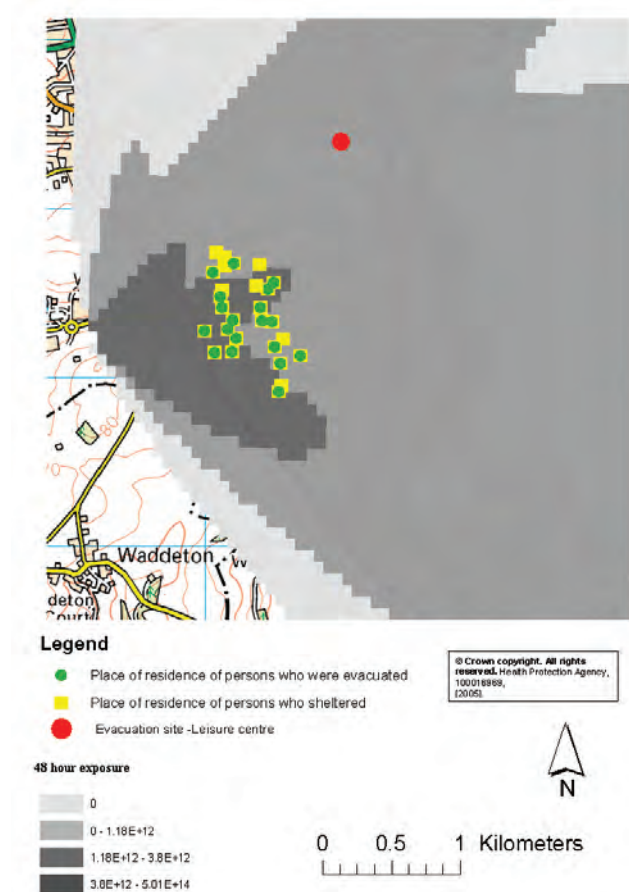

Legend

- 🟢 Place of residence of persons who were evacuated
- 🟨 Place of residence of persons who sheltered
- 🔴 Evacuation site -Leisure centre

48 hour exposure

	0
	0 - 1.18E+12
	1.18E+12 - 3.8E+12
	3.8E+12 - 5.01E+14

0   0.5   1 Kilometers

Figure 2 Estimated relative 48 hour exposure from a fire in a plastics factory (Adapted from Kinra *et al*., 2005)

Use of GIS for data interpolation

As well as the ability to integrate data, GISs also have some special capabilities that make them extremely useful for exposure assessment. Perhaps one of the most useful of these is the ability of a GIS to interpolate and extrapolate geographical data. This approach is often used for estimating concentrations of pollutants at locations where there are no monitoring sites. Prior to the development of GIS, interpolation was typically carried out using a simple approach known as the 'point in polygon' technique, which assumes that data from one monitoring point can represent the surrounding area - a simple approximation that does not require computer software. An example of this approach is demonstrated by Dockery *et al,* (1993), who investigated the relationship between air pollution and mortality in six cities in the USA. The study measured ambient air concentrations of total suspended particulate matter, sulphur dioxide, ozone and suspended sulphate at a centrally located monitoring station in each city, with data from this point taken to represent exposure across the entire city. Clearly, this can be a very imprecise assumption.

GIS computer software has enabled the use of more sophisticated techniques for estimating pollution levels between data points. One of the simplest of these is Inverse Distance Weighing (IDW), which is based on the principle that a particular variable (z) at a particular location will be influenced more by locations closer to it than by those further away. IDW works by weighting those points closer to a particular point more than those further away. In some cases, a simple inverse function of distance (d) can be used, for example, $1/d$. It is, however, more common to use some form of inverse power function (for example, $1/d^2$ or $1/d^3$), thereby placing greater emphasis on sites close to the pollutant source. Other interpolation techniques, that include the use of Triangulated Irregular Networks (TINS) and kriging, are described in detail elsewhere, for example, (Briggs, 2003).

The techniques mentioned here are some of the many GIS tools available to help with exposure assessment that can be applied with most GIS software systems; e.g. the Spatial Analyst or Geostatistical Wizard extensions in ArcView. Application of these tools can be rapid and can potentially provide extremely useful information for exposure assessment. Users should, however, ensure that they have appropriate training and guidance and can use the tools correctly, and that they are aware of the limitations associated with them.

Interpolating concentrations of pollutants using intelligent interpolation models

The application of GIS data interpolation using the statistical techniques mentioned above can be helpful for estimating concentrations of pollutants at unmonitored locations. However, such methods may be of limited use due to the fact that the spatial patterns of pollution are extremely complex and determined by many environmental factors. A better way of estimating pollution values is to use what Briggs (2005) describes as "*intelligent interpolation models*", which makes use of information not only from monitored pollution values but also from other variables that affect pollution concentrations (Briggs, 2005). The latter include factors that are related to pollution and have a geographical attribute, e.g. traffic volumes on a road or housing density. Techniques such as co-kriging, which involve the use of covariates to estimate pollution values, have been successfully applied in several studies (Briggs, 2005) (Cyrys *et al*, 2005) (Hoek *et al*, 2002).

An example of the above approach is the work carried out by Cyrys *et al.* (2005), who applied a form of stochastic modelling using monitored levels of NO_2 and $PM_{2.5}$ pollutant levels from 40 locations, along with information on traffic intensity and population density. The study involved the use of a regression model to predict exposures at unmonitored locations, which were then linked to population data to estimate exposure. In addition, atmospheric dispersion modelling was applied to sources of pollution using a Gaussian dispersion model and the results were linked to population data. To compare the two measures of exposure the modelled concentrations were classified into three categories: high, medium and low, based on tertiles so that there were equal distributions in each category. The two exposure assessments showed a good agreement, with 70 % of the study subjects classified into the same exposure category in both studies.

Using GIS to integrate personal monitoring data for exposure measurement

Predicting the spatial distribution of pollutants in the environment is a very complex task; however, it is only one aspect of exposure assessment. Estimating the distribution of receptors is equally important and, unfortunately, just as complicated. Many epidemiological studies or risk assessments make the false assumption that individuals spend all their time at their place of residence and that indoor exposure is equal to the exposure outside. However, in reality, people continuously move from location to location, thus changing their exposure constantly. Most people spend a considerable amount of time away from their place of residence each day, and most move house several times during their lives, making it very difficult to assess long term exposure. In addition, exposure is known to vary significantly between indoors and outdoors and even between rooms in the same building (Briggs and Elliott 2005). Therefore, even if it were possible to predict pollution concentrations accurately for every single location in the area of interest, ignoring population movement would lead to inaccurate estimates of exposure. As a consequence, there have been several attempts at including population movement in exposure assessment (Kousa *et al*, 2002) (Bartonova *et al* 1999). The techniques are termed integrated modelling, described by Briggs (1992) as "*the modelling and mapping of actual levels of exposure by intersecting geographical models of pollutants and human distribution*".

Bartonova *et al.* (1999) used the air dispersion model EPISODE to model pollution from different traffic scenarios in Oslo, Norway (Bartonova *et al* 1999). The study aimed to determine the impact that the construction of a traffic tunnel had on personal and community exposures. The dispersion modelling results were linked to diary-recorded information about where people spent their time. Using geocoded address data and GIS, it was then possible to estimate exposures. The results showed that constructing the new road tunnel had the effect of increasing the total traffic volume, though it also had the effect of reducing peak exposures and exceedences of regulatory air quality values. While integrated exposure assessment has very clear advantages over other methods of exposure assessment, it is resource intensive and has therefore not been used for many epidemiological studies. In addition, some diseases, for example, cancers, have long latency periods, making integrated exposure assessment even more difficult for this application of environmental epidemiology. Obtaining data over an appropriate timescale also remains a challenge.

Availability of software and data

There are a number of different GIS software packages available, although the two most common are ArcMap® provided by ESRI and MapInfo® (ESRI, 2006) (MapInfo, 2006). The costs obviously depend on what functionality is required (for example, extensions for carrying out data interpolation etc) but would typically be several thousand pounds. It should also be noted that obtaining environmental monitoring or modelling information for the exposure assessment can also be expensive. However, HPA staff have access to a large amount of Ordnance Survey digital mapping data under the Pan Government Agreement (PGA).

Conclusions

GIS is simply a tool for manipulating, integrating, interrogating and displaying geographical data. It cannot on its own provide answers to questions about the effects of the environment upon human health. GIS effectively provides easy access to a range of sophisticated statistical analysis techniques, making it an enormously useful tool for analysing data that describe the environment and human health - yet is worthless without the data. It must also be remembered that while GIS has the potential to enhance the understanding of the relationship between exposure and health outcomes, many of the existing problems with environmental epidemiology and risk assessment still remain. Even the most elaborate GISs still require input data. A lack of accurate data remains one of the main reasons why the effects of the environment on human health are not better understood.

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Risk communication to the public during water related chemical incidents

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"The biggest problem with communication is the illusion that it's been accomplished"

George Bernard Shaw

Introduction

In countries such as the United Kingdom although sophisticated water treatment, supply and distribution systems exist to ensure that the consumer is provided with safe drinking water, no water supply system is immune to chemical or microbiological contamination. Once a chemical or microbiological incident occurs in a water supply system, a complex organisational response is needed to protect public health. But while the technical and engineering aspects of managing a drinking water contamination incident are at the core of effective risk management, the important role of risk communication to the public during water contamination incidents, chemical as well as microbiological ones, has been widely neglected.

However, only in rare cases will chemical contamination of drinking water be detected, identified and removed early enough without posing a potential threat to public health. Chemical contamination of drinking water and the related risk management decisions are interlinked and rely on effective and timely risk communication to the public. A well-planned and effective risk communication strategy that goes beyond ad hoc measures can save life and reduce adverse public health impacts.

Failure to communicate with the public in a timely and appropriate way during a chemical incident related to drinking water, not only potentially puts people's health at risk, but can also become a public relations problem that affects people's trust in public health institutions and the credibility of water authorities/companies and government agencies for years. The chemical contamination incident in the Lowermoor treatment works in Camelford in 1988, where a water supply was accidentally contaminated with aluminium sulphate, is a good example for all the things that can go wrong in risk communication to the public. The water authorities/companies and government agencies reacted too slowly to consumers' complaints about strange taste, odour and 'sticky touch' of the water. Not only did people consume - after being reassured about its safety - contaminated water, but public perceptions about the event and adverse health outcomes differ significantly from official assessments.

Do we know more today about effective risk communication with the public since Camelford? This study started with the questions: does an evidence base exist for risk communication with the public during chemical incidents related to drinking water? What are the legal grounds for public notification in the UK? Do any standardised guidelines exist? How do other governments regulate risk communication with the public and public notification during drinking water contamination incidents?

Methods

A comprehensive thematic literature review has been carried out in order to identify evidence-based literature on risk communication during the management of chemical incidents related to drinking water. The aim of this literature review was to establish an evidence base, by collecting a summary of experiences with risk communication during water contamination incidents. However, because it was anticipated that the review would reveal only small numbers of peer reviewed papers on risk communication to the public during chemical incidents related to drinking water, examples from evidence-based literature on risk communication during microbiological incidents were also included. Even though chemical incidents in drinking water differ significantly to those of microbiological incidents, the results of studies after a microbiological incident yield important insights into the failures of risk communication with the public. Additionally, background articles and the grey literature were also sought in order to review different international guidelines in such events.

The Integrated Catalogue of the British library, Pub Med, Aqualine (1960 to present), the Risk Abstracts database (1990 to present) were searched for the terms "Risk communication OR Public Notification AND (drinking) water", and "Risk communication OR Public Notification AND chemical* AND water", and "risk perception AND (drinking) water". Additionally the reference lists of publications were also been searched. No restrictions were made on the publishing year. Publications were only selected if they were directly relevant to the context – risk communication to the public during acute drinking water contamination incidents. Selection was made by reviewing the titles, abstracts or the full document.

Results of the literature review

Risk communication can be broadly defined as "*any purposeful exchange of information and interaction between interested parties regarding health, safety, or environmental risks*"[1], and has been recognised as an important component of risk and emergency management since the 1980s. No single theoretical framework exists, rather the respective problem concepts from different disciplines and fields were applied. However, while a wide range of literature about risk communication in general can be found, very little has been published with regard to drinking water.

In PubMed the search for 'risk communication OR public notification' produced 16709 results and in Risks abstracts database 227, which was reduced to 32 and 3 respectively when the search was limited to 'drinking water'. After reviewing the papers, only one evidence-based article on the management of a chemical incident related to drinking water that addressed risk communication could be identified.[2] Four studies assessed the effectiveness of 'boil water' notices and advisories after microbiological incidents in drinking water.[3] One of these studies looks into the differences in awareness and compliance rates among people in an emergency situation and in an ongoing drinking water problem.[4] The following table provides an overview of the articles.

Table 1. Overview of literature on risk communication during a drinking water contamination incident

Article	Type of incident	Risk communication Measure	Awareness - Compliance –	Source of information	Results - Lessons learned
Winston *et al*. 2002 (journal)	Chemical (spillage of ammonia in urban water supply, Tel Aviv/Israel)	"do not drink" and "boil water" order	89% of the population had been aware of the order. 82% had complied with the instruction given in the advisory.	Television and radio broadcasts.	- invocation of precautionary principle did prevent further harm - toxicity testing was done late in the chain of events - need to develop protocols & directives for civil emergencies - need for better coordination & distribution of alternative water supply
Angulo *et al*. 1997 (journal)	Microbiological (Outbreak of salmonellosis in community water supply, Missouri/USA)	"boil water" order	Only one person out of the 92 surveyed had not been aware of order. But 9 only became aware after drop of additional leaflet. 31% did NOT comply with the instructions given in the advisory.	Not known	- people did not recognize severity of situation due to insufficient information. No information about associated illnesses had been given -reasons for non-compliance: 44% not remembering, 25% disbelieving.
O'Donnell *et al*. 2000 (journal)	Microbiological (sewage entered distribution network, Wigan and Bolton/UK)	"boil water" order	All but one of the 241 surveyed households had been aware of the order. 62% who received and read notice did not comply in some way.	85% had become aware through leaflet, 10% by word of mouth & reading the newspaper.	- even though notice was received early & awareness was high, compliance was low - elderly & disabled persons had difficulties in reading & understanding the message - similarity of leaflet to other mail was criticised - residents wished to be continuously informed about incidents & repairs - other methods of notification had been suggested by residents
Harding *et al*. 2000 (journal)	Town A. Microbiological (ongoing filtration problem, Oregon/USA) Town B. Microbiological (E. coli detected in water supply, Oregon/USA)	A. Notification about supply of unfiltered & untreated water that was only chlorinated B. "boil water" order	A. 86% were aware and took actions. B. 70% were aware and took actions. A. 35% did drink boiled water. 64% used bottled water. B. 57% did drink boiled water. 77% used bottled water.	- residents (74%) relied mainly on newspaper as source of information about drinking water problems, followed by mail, flyer from utility, family & friends and local health department - mail from utility considered most reliable source	- community with a long-term drinking water problem responded differently to public notification - Wording of notification was significant. Residents in town B had received specific information about how to boil water. Notification for town A did not contain any specific remedial action. - residents preferred bottled water
Halperin 2004[5] (presentation at workshop)	Microbiological (power outage)	"boil water" order	Less than 50% of those aware of the notice followed the instructions. Women and younger respondent were most likely to comply.	Television was prime source of information.	- difficulties in reaching customers without powers for their televisions & radios - difficulties in providing alternative potable water supply - elderly customers least likely to have been aware - confusion among residents who had to boil water & how long - need to improve communication plans and advisories

Comparison of national legislation and guidelines on risk communication to the public

The UK, Australia and the USA were chosen as examples, because in each country the water sector is organised in a different way and different approaches to regulate water companies are applied.

	United Kingdom	USA	Australia
Regulating authority	Drinking Water Inspectorate (DWI)	U.S. Environmental Protection Agency (EPA)	State health agencies
Federal mandatory drinking water standards	Yes.	Yes.	No. Australian Drinking Water Guidelines (ADWG) serve as a reference.
Legally binding legislation on public notification	No. Under the Water Industry Act 1991 it is mandatory for suppliers to notify relevant – but not the consumer -authorities in any event that is likely to give rise to a significant risk to consumers.	Yes. Safe Drinking Water Act (SDWA) states public notification requirements relating to violations of national drinking water standards.	No. Health authorities are responsible for notifiable incidents guided by response protocols
Standardised guidelines on timing of public notification.	No.	Yes. In case of a Tier 1 violation which is classified as maximum contamination level violation (MCL), treatment failures and violations of a variance, and might cause acute illnesses among consumers require notification within 24 hours. In case of a Tier 2 violation, which include MCL that may have a potentially serious but not immediate adverse health effect, notice is required within 30 days. For all other violation and situations a notice is required within 12 month and may be part of the annual report of the water company. [1]	No. But recommended: - When supply is interrupted - When water quality does not meet the guidelines, and there is a possible health risk - When treatment fails or a reticulation system is compromised - When monitoring is not carried out at the recommended frequency, and hence there is no assurance that the supply is safe - When monitoring is not carried out using the recommended testing procedures - When adopted levels of service are not met (e.g. because of floods, cyclones etc.)
Standardised format for advisories	No.	Yes. Notices must entail following information: - Description of the violation or situation. - When the situation occurred. - Any potential adverse health effects including standard health effects language. - The population at risk. - Whether alternative water supplies should be used. - Action consumers should take (if any). - What the system is doing to correct the problem. - When the system expects to resolve the problem. - The name, business address and phone number of the system owner or operator. - A statement to encourage distribution of the notice to others.	No.

The comparison of national legislation and guidelines on risk communication in the UK, Australia and the USA displays great differences. While in the US risk communication and public notifications are incorporated into federal laws and comprehensive guidelines are in place, guidelines on risk communication in Australia are only provided on a legally non-binding basis, and in the UK no legislation or guidelines on risk communication exists at all. These differences arise, in part, from differently structured water sectors, as well as different mentalities towards costumers. In the US, a customer-oriented approach has a long tradition, as well as the enshrinement of the 'right-to-know' principle in the national legislations and guidelines. In the UK, on the other hand, the privatisation of the water sector has consequently caused consequently the separation of the regulation of the industry from its operational business. In Australia, where states and territories enjoy considerable autonomy, a highly regulated approach towards risk communication would not be possible.

In perspective, however, the absence of any kind of guidelines and legislation on risk communication in the UK seems striking. In the absence of guidelines, water companies operate in a legal and normative vacuum. The appropriateness of their measures is solely judged by DWI on a case-to-case basis. On the other hand the DWI has little evidence against which to judge water companies' risk communication practices.

Discussion

One of the major obstacles for effective risk communication to the public, is the fact that in spite of many theoretical risk communication models and the constant claim for a science-based approach, little empirical evidence has been generated so far. The shows that no evidence base for risk communication to the public during chemical incidents related to drinking water exists. However, public health professionals and water utilities would benefit from a more comprehensive documentation management of contamination incidents in drinking water.

The few studies on the effectiveness of risk communication during a water contamination event yield, even though each setting is different, similar results. Awareness about advisories is usually high among the affected population, but compliance rates are low. Unfortunately, not much research has been done on why people fail to comply and how the communication could be improved. While experts in risk communication emphasise in their theoretical approaches, the danger of public outrage and people's tendency to exaggerate hazards and risk, the opposite seems to be the case. People might often not be scared enough. This might be due to a lack of information on associated illnesses and risks as a study showed.[6] Currently the 'boil water' and 'do not drink' advisories of a major water company in the UK, do not contain any further information than just advising people to boil water or not to use. The strict US legislation on what kind of form and content such advisories have to have, accommodates for the fact that people need more information than just to be told what to do and what not to do. People need information to make informed choices. Even though people might view hazards and risks differently than experts, be irrational about their fears and have a tendency to be scandalized by the media, none of the studies reported public outrage. People become anxious and turn to alternative sources of information, if they feel unsatisfied with official messages. In that case unofficial sources such as from the media and other experts can conflict with the official message.[7] The public uproar in case of the incident in Camelford and Exeter can be partly explained by that, where the findings of the official investigations differ greatly from public perceptions about the incident.

Taking into account that the studies discussed involved "only" 'boil water' notices (except Winston *et al*. 2002), the shortcomings in risk communication and the poor compliance rates should trigger great concern among water companies and health authorities. In case of a chemical contamination that would require a 'do not drink' or 'do not use' order, equally poor compliance rates could result in a major public health problem.

Recommendations

Several recommendations regarding risk communication which apply to water companies and health authorities can be made:

1. There is a need for a more comprehensive documentation of experiences with risk communication to the public during management of chemical water contamination incidents. A science-based approach in risk communication is only possible with a comprehensive documented evidence-base and empirical results. Water companies as well as health authorities would benefit from shared experiences, since knowledge about management of chemical contamination in drinking water is still limited. The CHaPD's "Chemical Hazards and Poisons Report" could serve as a medium and platform for such sharing.

2. Closer collaboration between water companies, the local health authorities and academia is necessary in order to investigate the effectiveness of risk communication to the public and public notifications. Academia has taken up the challenge to do further research in that field without compromising their independence. Water companies have to become more transparent about their risk communication strategies in order to allow a comprehensive evaluation of current practices and view further research in that field, not as a threat to their image, but as a way forward to improved customers relations. Health authorities, with advice from CHaPD, could play a central facilitating role in that collaboration.

3. There is a need to develop standardised guidelines on risk communication and public notification procedures. Guidelines would not only assist water companies in incorporating risk communication and public notification standards in their crisis management strategy, it would also provide guidance to public health doctors involved in the management of an incident whose experience of drinking water incident risk communication may be fairly limited.

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From IPPC Information to Environmental Public Health Bulletin

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Introduction

The Integrated Pollution Prevention and Control (IPPC) Support Unit is based within the Centre for Public Health at Liverpool John Moores University. We work closely with Health Protection Agency (HPA) colleagues within the Local and Regional Services (LARS) as well as colleagues within the HPA's Chemical Hazards and Poisons Division. The unit is part of a wider environmental public health team which has developed over the last ten years. The team provides environmental public health support to the Regional Director of Public Health at Government Office North West (GONW) and to the Director of the Health Protection Agency North West. We undertake original research and also provide environmental public health education and training within academic programmes at the University and for various health protection capacity building events.

This paper describes the development of the IPPC bulletin that we have been producing for the last two years into a general environmental public health bulletin for the region.

Background

The IPPC Support Unit was established to support health authorities prepare their responses to the health consultation process under the Pollution Prevention and Control Regulations (PPC) 2000. As a consequence of the abolition of health authorities in 2003, Primary Care Trusts (PCTs) are the Statutory Consultees for the regulations and our service is available to all 42 PCTs in the North West of England.

Since September 2004 we have produced a quarterly IPPC bulletin; this aims to increase awareness of the IPPC process by summarising the work carried out by the IPPC Support Unit and informing colleagues of areas of interest within the IPPC agenda. The bulletin is distributed electronically to North West PCTs, Local Authorities (LAs), colleagues at the Environment Agency (EA), local HPA units and public health colleagues at GONW. It is distributed nationally to environmental public health colleagues within other academic institutions and other IPPC support units within the HPA.

The bulletin was produced for several reasons. We recognised that there was a need to promote the health consultation within the PPC regulations to a wider audience, including regulatory bodies (EA and LAs) and we particularly wished to demonstrate an overview of the process. This was intended to highlight the range of industrial processes covered by the legislation; furthermore, as the location of industrial processes is often dependent upon geographical features, a single PCT often receives a number of similar applications and therefore doesn't experience the breadth of environmental public health issues revealed by the PPC consultation.

Current Bulletin

Each quarter, we provide an update of the number of IPPC applications that the unit has received for comment. We display total numbers and a breakdown of the number and types of process received and alert readers to the forthcoming applications (by sector). In several of the bulletins we have expanded on one of the industrial sectors that have featured in our recent work, for example we assessed a considerable number of landfill sites from around the region and presented a précis of the types and location of the sites as well as the main health issues raised through the consultation process. We included a summary of the outcome of the PPC permit process, i.e. the number of sites that had received a permit, those that had been refused (and why) and the number still to be determined. This proved to be a useful exercise as we were able to interrogate the public register and ascertain how the comments received by the regulator had been integrated into the decision process prior to the permit being determined.

We have a regular 'What's On' column in the bulletin, where we display details of events and training held by our partner agencies and other stakeholders, both locally and nationally. We also provide an opportunity for staff within the IPPC unit to 'write up' any events that
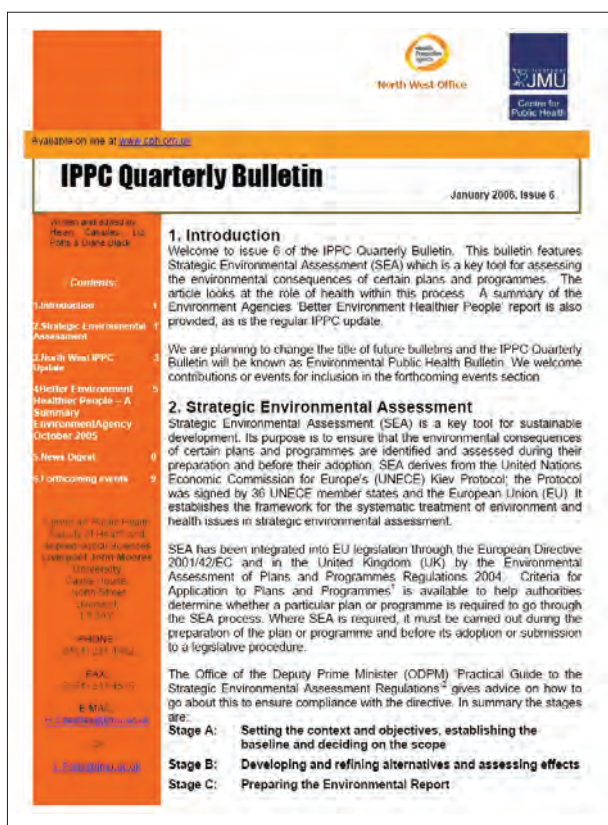
they have attended, this encourages a systematic appraisal of training and shares information gleaned from events.

Each issue of the bulletin has a topical editorial; previous bulletins have included a feature about 'Bonfire Night' and air quality (October), information regarding carbon off-setting to coincide with the summer holiday period and recently, discussion about Strategic Environmental Assessment. As the bulletin has become more established these short editorials have become more relevant to our audience and with the bulk of IPPC consultations ending in 2007, we are in the process of developing the bulletin into an environmental public health bulletin.

Proposed format

Changing the focus of the bulletin from IPPC to general environmental public health, albeit with a North West flavour, is expected to encourage a wider readership and help to maintain the relationships built up during the IPPC consultation process. It will provide an opportunity for stakeholders within the North West to communicate on issues of common interest and strengthen existing networks. Whilst we will continue to include IPPC within the bulletin, our intention is to have a theme for each issue and include local data to support our discussions. We are planning to select an environmental public health hazard and, for example discuss the risks arising from the hazard, methods of control (regulatory as well as physical methods) and local effects in terms of environmental and health impact.

As the bulletin is in the public domain it is also anticipated that members of the public will find it useful either as a source of information or to share information; we are including an article from one of our local interest groups in our first new format bulletin. We currently receive a number of requests for environmental public health advice and information from the public, either through our own website, the Northwest Public Health Observatory or the Regional Office of the HPA; we generally deal with these in an ad hoc manner in conjunction with colleagues at the health protection units. The new bulletin will enable us to pre-empt some of these requests, pass on lessons learned from dealing with these requests and identify issues which require taking forward.

Conclusion

IPPC has provided a unique opportunity to include public health at the centre of pollution prevention and control. The production of a regular bulletin has enabled us to communicate to a wide range of stakeholders, including the public and regulators and it is imperative that the relationships developed as a consequence do not disappear as the permitting process concludes.

The first edition of the Environmental Public Health bulletin was published in May and focused on carbon monoxide as an environmental public health hazard; it was well received and is available at www.cph.org.uk/publications. Our second is due imminently and is introduces the topic of risk perception and environmental hazards. We welcome suggestions and contributions for future bulletins; should you wish to be on our mailing list, please contact d.black@ljmu.ac.uk

IPPC bulletins can be downloaded from
http://www.cph.org.uk/publications.asp

Traumacare in India

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TraumaCare 2006 (TC2006), one of an annual series of conferences organised by the International Trauma Anesthesia and Critical Care Society (India), took place in Agra between 8th and 10th September. With over 500 delegates from many specialities and from all parts of India, this event is one of the largest of its kind in the sub – continent, where the impact of the modern trauma epidemic is increasingly being felt as a result of increasing industrialisation and increase in mechanised road transport.

TC2006, like previous conferences, recognised that trauma as such should not be limited to the conventional physical variety, and several presentations were devoted to injury from toxic exposures, now termed 'toxic trauma.' The catastrophic release of methyl isocyanate in the central Indian city of Bho Pal in 1984 which lead to over 5000 deaths was the subject of a key note presentation by one of the original responding doctors. The essential need for support for life support measures, particularly emergency ventilation for large numbers of lung – damaged casualties was a continuing lesson from this incident.

In a special session, the continuing care of patients following organophosphate (OP) poisoning, a common event in India was discussed. Lakshman Karalliedde and David Baker, both from CHaPD, London, gave presentations on the clinical relapse and re – paralysis that occurs in a significant proportion of patients following recovery from the classic early OP – induced cholinergic syndrome, a condition first described by Senanayake and Karalliedde in 1987 [1] and termed Intermediate Syndrome (IMS). Management of this condition requires several days of intensive care and places a serious burden on health care facilities throughout India. Lakshman Karalliedde reviewed the original work that lead to the description of the condition and its wider recognition in the years since [2]. IMS is now widely recognised in between 20 and 40 % of patients admitted following ingestion of OP pesticides.

It is becoming apparent from laboratory studies on changes in the acetyl choline receptors in the neuromuscular junction that in IMS the re – emergence of cholinergic paralysis may be due to genetic susceptibilities and that there may be links with other genetically determined muscle weakness diseases such as myasthenia gravis. The key question that remains in IMS is why some patients are affected following OP poisoning and not others.

Early diagnosis of developing IMS remains a key issue in planning the clinical management and DB presented an overview of the clinical neurophysiological changes that have been recorded in patients with IMS. In essence the acute cholinergic syndrome of OP poisoning is associated with a depolarisation paralysis of the type most commonly seen following the use of muscle relaxants such as suxamethonium (widely used in anaesthesia for endotracheal intubation) whereas IMS is associated with electrophysiological signs typical of non – depolarising paralysis produced by curare – like agents. This indicates that a reduction in the number of functioning acetyl choline receptors in the neuromuscular junction may be the cause of IMS. There have been indications using the sensitive single fibre electromyography technique that this is the case in sub - clinical exposures to OP [3] and a new clinical project is being planned in India to use the technique for identification of developing IMS.

In parallel with the Agra conference there was a course in trauma management (the Comprehensive Trauma Life Support Course) which has been specially developed to improve trauma management training in that country. A new section of the course to cover management of mass toxic trauma is being developed by David Baker. Much of the material to be included parallels that being developed for the current Department of Health hot zone working initiative in the UK (HART) to provide specially trained and equipped medical and paramedical personnel to provide essential life support and antidote therapy inside a contaminated zone following a chemical agent release. This marks a continuing acceptance of the need to provide medical care ever further forward in chemical incidents to avoid potential fatalities from chemically – induced respiratory failure.

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5th Air Quality Pollution Forecasting Seminar: Air quality impact of the Buncefield Oil Depot explosion 22nd June 2006

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This event was hosted by Netcen on behalf of the Department for the Environment, Food and Rural Affairs (Defra) and the devolved administrations at Culham Science Centre. The conference was attended by a range of professionals including health professionals, local authority staff, environmental consultants, academics and representatives of different government agencies. Presenters at the conference included the Health Protection Agency (HPA), Defra, the Met Office and representatives of those who operate the automatic air quality monitoring networks.

The calm stable meteorological conditions coupled with the high plume buoyancy (caused by the heat of the fire) meant that the smoke travelled to a high level in the atmosphere. This resulted in pollutant concentrations at ground level being very low, even at small distances away from the immediate scene of the fire in residential areas. The HPA used information from national, regional and local air quality monitoring network to assist with the acute incident response. Information from the regional and local air quality networks operated by King's College London Environmental Research Group (ERG) indicated that there were a number of peaks in Particulate Matter with a diameter of less than 10µm (PM_{10}) concentrations possibly related to the Buncefield fire. However, pollution levels still remained low or moderate bands (Committee on the Medical Effects of Air Pollutants, 2006).

The Met Office outlined how they had undertaken atmospheric dispersion modelling using the Numerical Atmospheric dispersion Modelling Environment (NAME) model in the initial part of the incident response to determine the spatial distribution of the plume. After the initial response to the incident, more sophisticated modelling was conducted using the best available information about the source term. Analysis of air trajectories by the Met Office indicated that some of the peaks in PM_{10} monitored by the air quality monitoring networks could indeed be due to the plume from the Buncefield fire. The Met Office also presented the results of some initial dispersion modelling showing what would happen if the incident had occurred in different meteorological conditions.

During the Buncefield incident it was also very fortunate that the joint Met Office/ Natural Environment Research Council (NERC) Facility for Airborne Atmospheric Measurements (FAAM) aircraft was in the UK. This gave a huge amount of extra data which proved invaluable for exposure assessment and also helped to validate the atmospheric dispersion modelling. The plume itself could be sampled using the aircraft and an estimate of the height of the plume was also obtained.

Overall, the conference highlighted the huge strain that the Buncefield Oil Depot explosion and fire placed on those responding to the environmental and health impacts of the pollution it caused. It also highlighted how fortunate it was that the favourable meteorological conditions resulted in low ground level pollutant concentrations (and therefore low risk to health). The conference also highlighted a number of lessons identified for responding to similar incidents in the future.

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European Educational Programme in Epidemiology (EEPE) 19th Residential summer course in epidemiology Florence, Italy, 26 June – 14 July 2006

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I attended the 19th European Educational Programme in Epidemiology (EEPE) residential summer course in epidemiology held in the CISL 'Studium' centre which stands on the hills close to Florence, towards Fiesole, close to San Domenico. The course has been successfully running since 1988 under the directorship of Professor Rodolfo Saracchi (National Research Council, Pisa, Italy) and is sponsored by the International Epidemiological Association (IEA), the WHO Centre for Environment and Health, and the Italian Association of Epidemiology.

In the first two weeks, the course offers five general modules on epidemiological study design, including: rates and risks, confounding, cohort and case control study design, cross sectional studies and bias; and statistical analysis of epidemiological data, including analysis of cross-sectional and case-control studies using logistic regression models. The morning and afternoon sessions included lectures, computer based analyses (using the "Stata" package), exercises and group discussion sessions. The teachers came to Florence to teach for one week each only, so that participants were exposed to a wide variety of expertise and teaching styles. The epidemiology teachers in the first week included Professor Neil Pearce (Director of the Centre for Public Health Research, Massey University, Wellington, New Zealand); Professor Franco Merletti and Dr Lorenzo Richiardi (University of Turin) and Professor Rodolfo Saracchi. In the second week, the lectures were given by Professor Rodolfo Saracchi and Professor Jørn Olsen (School of Public Health, UCLA, USA and the University of Aarhus, Denmark). The statistics modules were taught in the first week by Dr Bianca De Stavola from the London School of Hygiene and Tropical Medicine (LSHTM), UK and Dr Laura Ciccolallo from the Istituto Nazionale dei Tumori, Milano, Italy; and in the second week by Professor Simon Cousens (Professor of Epidemiology and Medical Statistics from LSHTM).

In the third (final) week, a choice of seven special modules was available, ranging from 'cancer epidemiology' and 'cardiovascular epidemiology' to 'the impact of changes of global climatic environment', 'local and occupational environment and health', and 'social environment and health', to 'advanced statistical analysis of follow up studies' using "Stata" or the R package.

In the third week of the course, I attended the three environmental epidemiology modules. '**Global climatic change and health**' was taught by Professor Anthony McMichael who is the director of the Australian National Centre for Epidemiology and Population Health at the Australian National University in Canberra and is heavily involved with the assessment of health impacts for the Intergovernmental Panel on Climate Change. He lectured on the science of global climate change, epidemiology and systemic environmental changes, scenario-based health risk assessment, and the handling and communication of uncertainty.

'**Local and occupational environment and health**' was taught by Professor Josep Antó and Dr Jordi Sunyer, both from the Municipal Institute of Medical Research (IMIM) in Barcelona, Spain This module focused on epidemiological design and application for environmental epidemiology, the use of biomarkers, and ecological and individual exposure assessment.

'**Social environment and health**' was taught by Dr Bruna Galobardes who is from the Department of Social Medicine, at the University of Bristol. Her classes revolved around socio-economic indicators in epidemiological research (socio-economic position) and the lifecourse approach to investigating health.

In order to take the course, students were expected to possess some knowledge of epidemiological and statistical methods at introductory level. Students came from all over Europe, and as far as Australia, New Zealand and Bangladesh, with variety of academic and public health backgrounds. The teachers were equally international and stemmed from academic institutions across Europe, Australia, New Zealand and the States. The days were heavily packed and started at 08.30 and finished at 19.30, with some study time at lunchtime. As a student, the vast amount of information being absorbed was somewhat overwhelming resulting in a sharp learning curve, but the course offered fantastic networking opportunities with other students and teachers (who were excellent) came from a wide mixture of backgrounds and cultures. I would highly recommend the course to others interested in sharpening up their knowledge and skills in epidemiology.

Environmental public health training: evaluation and future developments

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Introduction

The increasingly diverse nature of public health protection has increased the need for training in environmental public health for public health professionals. Within the East Midlands, and indeed nationally, there has been a long-standing demand for development in chemical and environmental issues for HPA Consultants in Communicable Disease Control, nurses, and Specialist Trainees/Registrars. As a result, the Health Protection Agency East Midlands (HPAEM) and the Chemical Hazards and Poisons Division (CHaPD) have collaborated with the Trent Multi Professional Deanery to develop a five-day environmental public health module delivered at the University of Nottingham. A pilot was held in June 2006; with spaces limited to 20 delegates.

The module's aims and objectives are:-

- To provide a broad ranging foundation in environmental public health and emergency response to Consultants, Nurses, Public Health Trainees, people on the voluntary register, and other people working in public health.
- To provide basic health protection knowledge across the fields of chemical, radiation, environmental hazards, and emergency response.
- To provide an understanding of multi-agency working in a dynamic environment.
- To encourage recognition of the type of health advice required for casualty care and consideration of the long term need for health surveillance.

The module design has purposely been focused on a case study approach to maximise the opportunity for problem-based learning and enhance the practical application of knowledge in relevant scenarios (Table 1).

Speakers from a wide range of organisations and fields of expertise contributed to the pilot:

- HPA-EM Chemicals and Environmental Team
- HPA-EM Consultants in Communicable Disease Control
- HPA Chemical Hazards and Poisons Division (London & Cardiff)
- HPA Radiation Protection Division
- Nottingham University
- Fire Service
- Ambulance Service
- Environment Agency
- Local Authorities
- Government Office East Midlands

Specialist areas were illustrated using scenarios/case studies, encompassing: air quality, radiation, land contamination, and water pollution (drinking & surface waters).

Scenarios and case studies were linked to exercises, which provided attendees with the opportunity to employ newly-gained knowledge. These involved: emergency response (chemicals) – both acute (fire/spill/flooding) and chronic (e.g. linked to land contamination), chemical and control of major accident hazards (COMAH) plans, cluster investigations, sources of information/specialist advice, and occupational health and understanding of legislation.

Table 1: Environmental Public Health Training Core Subjects

Core Subject	Speaker's Organisation/Agency	Case Study	Exercise
Environmental hazards and environmental risk assessment models	HPA East Midlands CET	Emissions from a chemical plant	Buncefield incident
Toxicology	CHaPD (London)	Heating oil contamination	
Environmental protection legislation	Centre for Public Health, Liverpool John Moores University		
Environmental epidemiology	CHaPD (London)	Broomfield coal tip fire	A coal tip fire
Environmental monitoring	Ashfield District Council	Bonemeal incident	Assessment of local air quality
Biomonitoring	CHaPD (Chilton HQ)		Tanker spill exposure
Land Contamination	CHaPD (Cardiff)	Castlegate/Durham Road Developments	
Radiation	Medical School, QMC	The Watras incident	Transportation
Incident roles and Responsibilities	CET, CHaPD, Local Authority, Ambulance service, Fire Service, Environment Agency	Pyramid Products factory fire	Fire/COMAH/Occupational health incidents

Figure 1: Environmental public health training evaluation
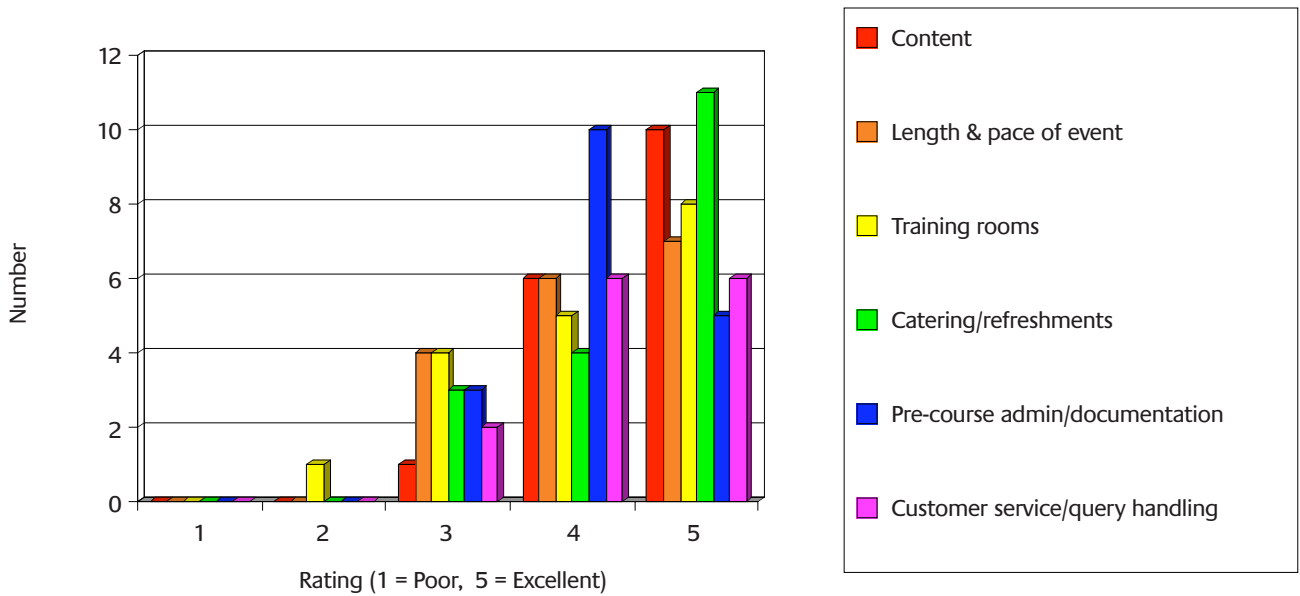
Evaluation

A comprehensive evaluation of each session, and the course overall, was undertaken by the delegates. Each participant was asked to comment upon the content of each session, the quality of the speaker and their teaching materials and the quality of the facilities and course administration.

Overall, the content, length and pace of each session scored highly, with the vast majority of scores in the good/excellent category (Figure 1).

In 4 areas of assessment, 91% of individual sessions received good/excellent ratings. On average, for individual sessions, 93% of attendees indicated that their objectives had been met.

In summary, 94% of responders confirmed the course was good/excellent in meeting their objectives.

Comments

"The course is an excellent introduction to environmental public health and covers a wide breadth of topics and has a useful balance of lectures and interactive sessions. The interdisciplinary coverage gives a good overall picture of environmental public health."

"The course provided me with all the info I was expecting and gave the chance for applying it in scenarios. I very much enjoyed it."

"I feel more confident to take on an incident... [I] feel able to support new/other nurses in incident management."

"The importance of the media has been stressed several times during the course. [I] need to be more aware of this resource and work proactively with the media."

"I am involved in an alleged disease cluster related to a tar pit. I hope to use some of the learning from the contaminated land session and risk communication principles... I hope to be able to use the principles and learning during the public health on-call."

Group work on air quality

(© HPA-EM, 2006)

Real-life incident case studies were used to illustrate theory

(© HPA-EM, 2006)

Learning Application

Research has shown that "the integration of new knowledge and skills requires transition time and focused effort on application." Delegates were asked to detail how they would apply the knowledge that they gained from the module in their day-to-day work. A second, more focused, evaluation document has been released to the delegates (2 months after the course) to assess the extent to which the module has contributed to their work. This will help further assessment of whether the course objectives have been met and the suitability of the course for different occupations.

Future

The course will continue to run and will next be staged in 2007. The comments received during the pilot will ensure that the course evolves to meet, and exceed, the expectations of future attendees.

It is anticipated that future iterations of the course will encompass a final day assessed group exercise in order to assess the level of practical knowledge gained by delegates over the period of the course.

Contaminated land was one of the specialisms covered

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One Day Symposium

Tuesday 5th December 2006

Richard Ley Development Centre,
Upper Fforest Way, Swansea,
SA7 0AN

Health Protection Agency

Major Industry, Chemical Incidents and Public Health: Buncefield and Beyond

The workshop will take the form of a number of presentations from key individuals, organisations and agencies, highlighting issues that emerged from Buncefield. The lessons learnt will be applied to emergency planning in Wales and will culminate in a series of conclusions and recommendations and form the basis of future collaboration. The seminar will appeal to all individuals with a role to play in chemical incident management, including first line responders, analytical and environmental scientists, health care professionals, government agencies and organisations, as well as policy makers.

Objectives:

- To provide a forum for debate centred around emergency planning, preparedness for major chemical incidents and subsequent response and recovery.

- To absorb lessons learnt from the Buncefield incident and to translate these in to the Welsh model as appropriate.

- To probe current Welsh planning and preparedness.

- To fortify, augment and reinforce current networks and practices.

- To use the workshop as a springboard to strengthen regular multi-disciplinary and multi-agency communication and collaboration.

If you have any questions about the event please contact us:
Email: buncefieldconf@hpa.org.uk
Telephone: 029 2041 6852

Registration fee: £50.00

To see the detailed programme and to book your place online please visit:

www.hpa-events.org.uk/swansea

In partnership with:

National Public Health Service for Wales
Gwasanaeth Iechyd Cyhoeddus Cenedlaethol Cymru

Wales Centre for Health
Canolfan Iechyd Cymru

Llywodraeth Cynulliad Cymru
Welsh Assembly Government

Training Events

Events organised by other HPA centres

**HPA East Midlands and Trent Multi-Professional Deanery
Environmental Public Health Module**
20-22 & 25-26 June 2007
University of Nottingham

This course aims to provide basic health protection knowledge across the fields of chemical, radiation, environmental hazards and emergency response. The content of this course will include, Toxicology, Environmental Epidemiology, Environmental Risk Assessment Models, Environmental Monitoring/Bio-monitoring, Air Quality, Land Contamination, Water Pollution, Radiation and other topics.

For Applications and further information from the Health Protection Agency (HPA Staff only), please contact Sarah Mottershead, Training Co-ordinator, 0115 9601412.

For Non HPA Staff further information and applications are available from Trent Multi-Professional Deanery, please contact Kim Platkiw, Public Health Secretary, 0115 8467117.

Closing Date: 20 April 2007
Some Cancellation Charges may apply, see contacts for further details.

If you would like to advertise any other training events, please contact Karen Hogan (chemicals.training@hpa.org.uk).

Training Days for 2007

The Chemical Hazards and Poisons Division (CHaPD) considers training in chemical incident response and environmental contamination for public health protection a priority. The 2007 programme is being developed to offer basic and more detailed training, along with the flexibility to support Local and Regional Services initiatives as requested.

How to Respond to Chemical Incidents

- 31st January, Holborn Gate, London

- 26th April, Holborn Gate, London

For all on the on-call rota including Directors of Public Health and their staff at Primary Care, other generic public health practitioners, Accident and Emergency professionals, paramedics, fire and police professionals and environmental health practitioners

The general aims of these basic training days are to provide:

- An understanding of the role of public health in the management of chemical incidents
- An awareness of the appropriate and timely response to incidents
- An understanding of the interactions with other agencies involved in incident management

These training days also have specific educational objectives. These are, to be aware of:

- The processes for health response to chemical incidents
- The type of information available from CHaPD, London to help the health response
- The resources available for understanding the principles of public health response
- The training needs of all staff required to respond to chemical incidents

A maximum of 40 places are available for each course.

Planned training events for 2007 include:

- How to Respond to Chemical Incidents (additional days)
- Contaminated Land Training Day
- Contaminated Water Training Day
- Contaminated Air Training Day
- Environment and Public Health Day
- Essentials of Toxicology for Health Protection (5 days)
- Introduction to Environmental Epidemiology (5 days)

Planned seminars/conferences for 2007 include:

- Environmental and Occupational Epidemiology (3rd one day UK & Ireland meeting)
- Chemical Hazards and Poisons Conference 2007

Please see the CHaPD Training Events web page for regular updates: http://www.hpa.org.uk/chemicals/training.htm

Booking Information

Those attending CHAPD (L) courses will receive a Certificate of Attendance and CPD/CME accreditation points.

The cost of the training days are £25 for those working within the Health Protection Agency and £100 for those working in organisations outside the Health Protection Agency. Places will be confirmed as reserved upon receipt of the fees. These charges are to cover lunch, training packs and administration costs.

For booking information on these courses and further details, please contact Karen Hogan, our training administrator on 0207 759 2872 or chemicals.training@hpa.org.uk

CHAPD (L) staff are happy participate in local training programmes or if you would like training on other topics, please call Virginia Murray or Karen Hogan to discuss on 0207 759 2872.

Chemical Hazards and Poisons Division Hotline: 0870 606 4444

Available to Government Departments, allied Agencies and Organisations, First Line Responders, the NHS and other HPA Divisions