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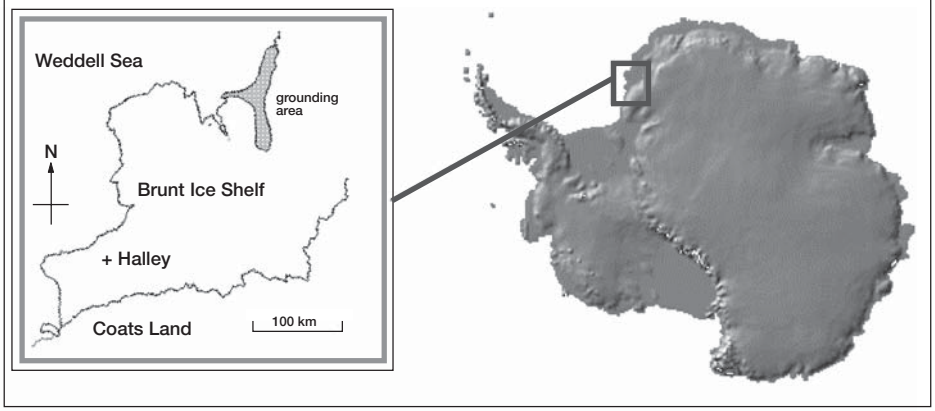

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(from March 2004)

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Chairman's report

The repercussions of arsenic contamination of drinking water in Bangladesh have been described in several past issues of the *ECG Bulletin*. This human tragedy has drawn together scientists from many disciplines in an effort to understand the geological circumstances of this disaster, how the drinking water might be purified, the mechanisms of arsenic toxicity, and the likelihood that other countries could become similarly affected. In this issue of the *Bulletin*, these themes are further explored:

- First we are grateful to the editorial staff of the *Journal of Environmental Monitoring* for allowing us to reproduce a review article by Mike Sharpe on the global aspects of arsenic contamination.

- We have featured previously the British Geological Survey's (BGS) work on monitoring for arsenic contamination in the UK (*ECG Bulletin*, July 2003). Now we report a survey by the BGS of a disused arsenic and copper mine in Devon.

- The latest in a series of proceedings from the SEGH* International Conferences on Arsenic Exposure and Health Effects was published in 2003, and Leo Salter takes the opportunity to review this book for the *Bulletin*.

- The 22nd European Meeting of SEGH was held this year at the University of Sussex. Arsenic contamination and remediation were the topics for many of the papers and posters at this meeting, and we are grateful to Professor Mike Ramsey from the Centre for Environmental Research at Sussex University for permission to reproduce some of the posters. (The selected posters accompany the version of this *Bulletin*, which appears on the RSC Website).

- For next year's Distinguished Guest Lecture, we will expand further on the relationship between health and exposure to metals and metalloids in the environment with a presentation by Professor Jane Plant from the BGS.

BRENDAN KEELY,
University of York,
June 2004

* Society for Environmental Geochemistry and Health

Deadly waters run deep: the global arsenic crisis

This article was written by Mike Sharpe (MS Consulting), Contributing News Editor for the Journal of Environmental Monitoring (JEM) and was first published in JEM Volume 5, issue 5, 2003, pages 81-85N.

As any reader of detective stories knows, arsenic is the murder's poison of choice. While deliberate poisonings may be rare, we nevertheless have a killer in our midst. Over recent years, the growing global population and lack of safe drinking water have led to the exploitation of groundwater resources in many parts of the world. In so doing, we have inadvertently tapped–literally–into another problem: arsenic-rich waters from deep-water aquifers. The implications will be with us for many years to come.

Arsenic is a relatively common element found throughout the earth's crust. It is introduced into water through the dissolution of minerals and ores, and in some areas concentrations in groundwater are elevated as a result of erosion from local rocks.[1] Commercial uses, such as in alloying agents and wood preservatives, may result in environmental releases and industrial effluents also contribute arsenic to water. Combustion of fossil fuels is a source of arsenic in the environment through atmospheric deposition.

The greatest threat to public health from arsenic is through drinking water.[2] Exposure at work and mining and industrial emissions may also be significant locally. Inorganic arsenic can occur in the environment in several forms but in natural waters, and thus in drinking-water, it is mostly found as trivalent arsenite (As(III)) or pentavalent arsenate (As(V)). The arsenite form is more mobile and toxic for living organisms. Organic arsenic species, abundant in seafood, are very much less harmful to health, and are readily eliminated by the body.

Health effects and health standards

Chronic arsenic poisoning, as occurs after long-term exposure through drinking water, is very different to acute poisoning.[2] Immediate symptoms of an acute poisoning typically include vomiting, oesophageal and abdominal pain, and diarrhoea. Long-term exposure causes cancer of the skin, lungs, bladder, and kidney, as well as other skin changes such as pigmentation changes and thickening (hyperkeratosis). Increased risks of lung and bladder cancer and of arsenic-associated skin lesions have been observed at drinking water arsenic concentrations of less than 0.05 mg l^{-1}. Absorption of arsenic through the skin is minimal and thus hand-washing, bathing, laundry, etc. with water containing arsenic do not pose a human health risk.

However, the symptoms and signs of chronic exposure appear to differ between individuals, population groups and geographic areas. Thus, there is no universal definition of the disease caused by arsenic, which complicates assessment of the health burden. Similarly, there is no method to differentiate internal cancers caused by arsenic from cancers induced by other factors. Following long-term exposure, the first changes are usually observed in the skin: pigmentation changes, and then hyperkeratosis. Cancer is a late phenomenon, and usually takes more than 10 years to develop.

The relationship between arsenic exposure and other health effects is still unclear. For example, some studies have reported cardiovascular and pulmonary disease, diabetes, and neurological and reproductive effects.[1] According to one recent study, long-term exposure to arsenic in drinking water is directly related to the development of atherosclerosis in the arteries leading to the brain.[3]

Research published in 2001 claims to have found an underlying mechanism to at least partly explain cancer effects.[4] It demonstrated that a human cell's own metabolic responses to arsenic exposure produce compounds capable of causing genetic damage. A joint US–Canadian team, led by researchers from EPA's National Health and Environmental Effects Research Laboratory, studied the effects of methylated trivalent arsenic on human lymphocytes in culture and on isolated DNA. They found that methylated trivalent arsenic derivatives, which can be produced by the body in an attempt to detoxify arsenic, result in reactive compounds that cause DNA to break. The findings could be used to quantify genetic damage in human populations exposed to arsenic.

More recent research has claimed a further, indirect health mechanism.[5] Earlier this year, scientists at Dartmouth Medical School, US, reported that exposure to small amounts of arsenic in drinking water may inhibit expression of genes involved in a critical housekeeping function that enables cells to repair damaged DNA. The process, known as DNA repair, is considered a major biological defence in the body's ability to fight cancer. According to lead author Dr. Angeline Andrew: "This study supports the hypothesis that arsenic may act as a co-carcinogen–not directly causing cancer, but allowing other substances, such as cigarette smoke or ultraviolet light, to cause mutations in DNA more effectively." A similar DNA inhibiting mechanism has also been reported for another toxic metal, cadmium.[6]

The World Health Organisation (WHO) has set norms for arsenic in drinking water since 1958.[2] Since 1963, WHO's "Guideline Value" has been 0.05 milligrams per litre (mg l^{-1}) (or 50 ppb, parts per billion), and many countries have adopted this as the national standard or as an interim target. WHO's latest (1993) guidance set a "provisional guideline value" for arsenic in drinking water of 0.01 mg l^{-1}. Based on health criteria, the guideline value would be less than 0.01 mg l^{-1}, but this level represents the realistic limit of measurement.

The US experience

As a continental landmass where many communities rely on groundwater, the United States is affected by high levels of arsenic in some drinking water supplies.[7] Because small water systems typically rely on wells for drinking water, while the larger systems typically rely on surface-water sources, arsenic tends to occur in higher levels more often in water used by small communities. The average level measured in US groundwater samples is around 1 ppb, but higher levels are not uncommon. Compared to the rest of the US, Western states have more water systems with levels exceeding 10 ppb, and levels exceed 50 ppb in some locations. Levels exceeding 10 ppb are also found in parts of the Midwest and New England. According to EPA, 5.5% of water systems, serving 11 million people, exceed the 10 ppb level.

In 1986, arsenic was included on a list of 83 contaminants for which EPA was required to issue new standards under the Safe Drinking Water Act (SDWA).[8] The existing standard of 50 ppb had been set in 1975, but dates back much earlier. Having missed a previous deadline, in 1996 Congress directed EPA to propose a new standard for arsenic in drinking water by January 2000, and to issue a final standard by January 2001. Congress also directed EPA, with the National Academy of Sciences (NAS), to study arsenic's health effects to reduce the uncertainty in assessing health risks associated with low level arsenic exposure. In 1999, the National Research Council (NRC)–the research arm of the NAS–concluded that the existing standard was inadequate for EPA's public health goals and recommended a prompt reduction.[7] In June 2000, EPA proposed a revised standard of 5 ppb and projected that compliance could be costly for small communities.

Following the 2000 Presidential election, the Bush Administration reopened the topic to consider levels of 3 ppb, 5 ppb, 10 ppb, and 20 ppb and re-examine the risk and cost issues.[9] The final rule, setting the standard at 10 ppb, came into effect on February 22, 2002 and requires public water systems to meet the new standard by January 2006.

In developing standards, EPA is required to set a Maximum Contaminant Level (MCL), defined as the maximum allowable concentration using the best-available technology, treatment or other means, and taking costs into consideration.[8, 10] EPA's cost determinations are typically based on costs to systems servicing more than 50 000 people. Less than 2% of community water systems are this large, but they serve roughly 56% of the population served by community systems. The smallest systems, those serving fewer than 3300 people, will be exempt from the new standard for up to 9 years and EPA has announced financial assistance to help them comply with this and other SDWA rules.

EPA's revision of the arsenic rule has been hugely controversial. Critics say there is little evidence as to whether significant adverse health effects occur from ingesting arsenic at very low levels, and consequently the costs of the new rule for the American public utilities is not justified.10 Indeed, the NRC report stated: "No human studies of sufficient statistical power or scope have examined whether consumption of arsenic in drinking water at the current MCL [50 ppb] results in an increased incidence of cancer or noncancer effects." Subsequent studies, reviewed at the time of the 2001 reappraisal, failed to fill this gap.

The most contentious point in the scientific debate has been the assumption that the toxicity of arsenic increases linearly (i.e. uniformly) in proportion to increases in its concentration.[10,11] Virtually all known toxicological processes follow a sublinear model–i.e. increases in cancer risk are negligible at low doses. Critics say the NRC accepted that only sublinear models were plausible but was forced to opt for the linear model instead because it could not agree on which sublinear model was correct. This, they claim, led to a conclusion in favour of a lower arsenic standard that was not supported by the science.

Bangladesh: a country in crisis

One area where the health effects of arsenic exposure are beyond doubt is Bangladesh[12, 13] (see *Analyst*, 1994, **119**, 168N for one of the earliest reports on this).[14] In the mid-1990s it emerged that arsenic had contaminated well water in parts of the Bengal Delta. This is the coastal floodplain of numerous rivers, including the Ganges and is shared by Bangladesh and the Indian state of West Bengal. The latest surveys estimate that around 36 million people in the Bengal Delta are drinking contaminated water, and 150 million are at risk.[15]

Ironically, the crisis has its origins in development efforts to give the people of the region access to safe drinking water. Until the 1970s, most villages in Bangladesh and West Bengal had either dug shallow wells, or collected water from ponds or rivers–and regularly suffered cholera, dysentery and other waterborne diseases. Instead, development agencies advised local people to bore deep "tube wells" into the water aquifers to reach clean, pathogen-free water. Up to 20 million of these tube wells were dug. Unfortunately the drilling hit precisely the depth of arsenic-rich rock.

Hydrogeologists have determined that the source of the problem is rocks naturally rich in arsenic which were eroded from the Himalayas thousands of years ago and deposited by the region's rivers. The arsenic-bearing sediments became buried and lie about 50 to 75 m beneath the surface. Until relatively recently, however, arsenic was not recognised as a problem in water supplies and the standard water testing procedures did not include tests for it. The role of alluvial aquifers as a potential source of arsenic in groundwater is now much better understood [Box 1].[16, 17]

WHO experts predict the situation will get much worse and should be considered as a public health emergency. "It is reasonable to expect marked increases in mortality from internal cancers once sufficient latency has been reached," says Professor Allan H. Smith of the University of California, a WHO adviser.[18] Studies in other countries where the population has had long-term exposure to arsenic in groundwater indicate that one in ten people who drink arsenic-contaminated water may ultimately die from cancer. Dramatic increases in such deaths and cases have been reported in Taiwan, Chile and Argentina.

In Britain the issue has ended up in court, with 750 Bangladeshis suing the British Geological Survey (BGS), which assessed more than 50 wells in 1992.[15] BGS was paid by the UK Overseas Development Agency from development aid funds to conduct a hydrochemical baseline survey of the tube-well water quality in Bangladesh and assess its toxicity to humans. The claimants say BGS should have tested for arsenic, but BGS argues there was no indication in the scientific literature at the time that arsenic might be associated with river and delta plains.

In a statement outlining its case, BGS said: "Arsenic only occurs in a water-soluble form in certain hydrogeological conditions. It is one of a large number of

BOX 1: Mobilisation of arsenic in alluvial aquifers

In areas such as the Bengal Delta Plains, the source of arsenic in alluvial sediments is dependent on the geology of the source terrain, while the mobility of arsenic in groundwater is influenced by the sediments' geochemical and hydrogeological characteristics. Specifically, the retention and/or mobility of As within the subsurface environment under different redox conditions is controlled predominantly by the interaction of the aqueous phase with the different mineral phases of the aquifer sediments.

Redox conditions within sedimentary aquifers are known to be controlled by chemical processes such as adsorption–desorption, precipitation–dissolution of unstable As minerals, organic content, and biological activity. However, mechanisms governing the mobilisation of arsenic from the sedimentary aquifers are less well understood.

Recent investigations reveal that secondary Fe and Al phases play a key role. These Fe- and/or Al-phases are characterized by variable surface charge, negative at higher pH and positive at lower pH. At lower pH, these surface reactive phases attain net positive charge leading to significant adsorption of As(V) (arsenate) species. The occurrence of As in groundwater is a process driven by the changing redox conditions where the arsenic phases are selectively desorbed as a response to the reduction of Fe^{3+} phases to soluble Fe^{2+} species. High-As occurrences concomitant with the increased Fe contents in groundwater supports this hypothesis. Part of the As in the groundwater appears to be quantitatively related to the release of As phases, mainly as As(V) form adsorbed on the surface reactive Fe-oxides and hydroxides.

The researchers conclude that although the geological sources of As could be proved unequivocally in this case, more detailed research is needed to characterize the chemistry of the aquifer materials in order to understand the water–solid-phase reactions. Another important aspect of research was to highlight the need to develop low-cost geochemical techniques for the removal of As suitable for application in developing countries.
Adapted from: ref. 16.

trace elements which are therefore not routinely tested for in groundwaters unless there is independent evidence to suggest its presence. In 1992 such evidence did not exist in relation to Bangladesh, since alluvial plains of the sort which underlie much of Bangladesh were not generally recognised as posing an arsenic risk. We now know that a number of such areas of the world do have enhanced levels of arsenic in groundwaters."

The British High Court did not agree and in May 2003 dismissed BGS's application to strike out the claim. Aid agencies have a "duty of care" to those they aim to help, the judge said. The case is due to return to court early next year, when the BGS will have to answer why it failed to carry out the arsenic tests.

Meanwhile, new research across India's Ganges Basin suggests that the crisis in the sub-continent could extend much farther than previously thought. According to epidemiologist Dr. Dipankar Chakraborti of Jadavpur University, the Bengal Delta "may be only the tip of the iceberg".[19] Untold numbers of the region's 450 million residents could be exposed to dangerous levels of the element in their drinking water. He is calling for urgent regionwide water-well analysis. "The arsenic problem intensified during a period of long neglect. Our earlier mistakes must not be repeated," he says.

Tipped-off to a spate of cancer deaths and skin lesions in the village of Semria Ojha Patti in the Indian state of Bihar, Chakraborti's team sampled wells in the village. Half contained five times the accepted safe limit of arsenic; one in five wells had 30 times the safe level. Bihar is 500 kilometres west of the Bengal Delta and is geologically akin to much of the Ganges Basin. The research in Bihar has sparked fears that similar arsenic contamination in Vietnam, Thailand and Taiwan could also be more widespread.

Research for a global problem

The issue of arsenic in drinking water is now recognised as a global problem. Relevant research is being undertaken on many fronts.

Treatment technologies

A number of established technologies are effective in reducing arsenic in drinking water. These include:activated alumina filters, anion exchange, distillation, reverse osmosis, and nanofiltration.[1, 2] Also, as a safeguard against organic arsenic, granular activated carbon filtration may be used.

While many large water systems are equipped with these treatment technologies, they may be less amenable for use by small community water systems or individual households. As part of its Arsenic Rule Implementation Plan, EPA has committed to sponsor further research and development of more cost-effective technologies as well as technical assistance and training to operators of small systems to reduce their compliance costs.[20] Around US$20 million has been pledged for the period 2002/2003.

As well as research, a strong emphasis is being placed on demonstrations of low-cost treatment technology. Twelve sites have already been selected for practical demonstrations of technologies and treatment techniques, and an invitation for a further round was launched earlier this year.

Appropriate technologies

Even technologies designed for small-scale community treatment systems may not be suitable for developing countries, as they are moderately costly and require technical expertise. Hence, international donors and aid agencies are funding research into appropriate treatment technologies and techniques that could be deployed quickly and effectively in southern Asia and other affected regions.

WHO, for instance, has sponsored a technique called STAR (Stevens Technology for Arsenic Removal) as an effective and inexpensive method for filtering out arsenic from household drinking water supplies.[18] The system uses a mixture of iron sulfate, calcium hypochlorite and sand as a filtering agent. Another filtration agent is laterite, a local raw material found throughout the Indian sub-continent.[16]

Genetic engineering

Another field of interest is the use of genetic engineering to create plants that could clean arsenic from contaminated soil and groundwater. Phytoremediation–the use of plants to absorb chemical pollution from soils–is a well established technique, but few naturally occurring plants thrive on toxic sites.

By inserting two bacterial genes into thale cress, *Arabidopsis thaliana*, US researchers have created a plant that not only grows well in the presence of arsenic but is able to store the toxin in its leaves.[21] The genes, from the bacterium *Escherichia coli*, make enzymes that digest arsenic compounds so they can be absorbed. The arsenic-rich leaves can then be harvested relatively easily and safely incinerated, making it ideal for phytoremediation. Eventually plants could be developed that might clean a contaminated site in just two or three years. The technique may also be applicable to a wide variety of plant species able to grow in different environments.

Australian scientists are investigating a different genetic approach–harnessing bacteria to help purify arsenic–contaminated water. A team at La Trobe University, led by microbiologist Dr. Joanne Santini, is studying 13 rare bacteria isolated from gold mines in the Northern Territory and Bendigo, Victoria.[22] They have found one bacterium, NT-26, that is an arsenite-munching champion. It eats arsenite–the most problematic form of environmental arsenic–and excretes arsenate, which is easier to get rid of. Dr Santini's group has found the enzyme directly responsible for converting arsenite to arsenate and is working to identify the same enzyme in other microbes. The team is also hunting for other proteins and genes involved in eating arsenite. They hope to use their findings to set up a bioremediation system for cleaning up mining wastewater and also provide safer drinking water for areas such as Bangladesh and West Bengal.

Health studies

Finally, researchers continue to probe the health effects of prolonged low-level arsenic exposure. As noted above, recent

work has made important breakthroughs here, suggesting mechanisms both for direct DNA interactions and co-carcinogenic effects. Other research into biomarkers of exposure seems to confirm that, for the same level of exposure, some people run a higher risk of developing cancer than others [Box 2].

Such findings should help us to develop a better informed public health response to the arsenic issue in both developed and developing countries.

BOX 2: Biomarkers of arsenic exposure

While the health effects of exposure to high levels of arsenic are significant and well documented, the impact of low to moderate level exposures remains unclear. Researchers at the Harvard School of Public Health, led by Dr. David Christiani, have studied biomarkers of exposure to arsenic and heritable susceptibility in two of the worst affected areas–Taiwan and Bangladesh. These studies are designed to fill important research gaps in our understanding of arsenic and human health.

The researchers are utilising a population-based approach, incorporating markers of exposure (drinking water arsenic, toenail arsenic, and measures of inorganic arsenic in urine), susceptibility (genetic polymorphisms in metabolizing genes), and outcome (squamous cell carcinoma of the skin, bladder cancer, and non-malignant skin lesions). In addition to studying the impact on health status including cumulative arsenic exposure, age, gender, and diet, they will examine the effect of gene polymorphisms in the Glutathione S-transferase (GST) superfamily of enzymes, which plays an active role in arsenic metabolism processes.

Similar research to date on bladder and skin cancer in Taiwan has revealed that:

- A person's ability to metabolize inorganic arsenic into less toxic metabolites (monomethylarsonic acid [MMA] and dimethylarsinic acid [DMA]) is directly related to the risk of both bladder and skin cancer. Methylation of DMA to MMA plays an important role in lowering, but not eliminating, the risk of skin and bladder cancer. Methylation of inorganic arsenic to DMA exhibits a weak effect in the opposite direction.
- In cases with similar methylation abilities and cumulative arsenic exposures, men had a higher risk of skin cancer, suggesting that additional behavioural or genetic factors may play a role.
- There is a significant relationship between smoking status, cumulative arsenic exposure, and increased risk of bladder cancer.
- The risk of developing skin cancer after long-term exposure to arsenic in drinking water was enhanced by certain genetic characteristics, most significantly by variation at codon 72 of the tumor suppressor gene p53.

These findings have important public health significance, suggesting that at the same level of exposure, some persons are at higher risk of cancer development than are others. Efforts to protect the more susceptible among us will ensure protection for all.
Adapted from: ref 23.

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MIKE SHARPE

News of the EHSC

The Environment Health and Safety Committee has recently responded to a Department for Environment, Food and Rural Affairs consultation reviewing the future of the UK Chemicals Stakeholder Forum on which the Royal Society of Chemistry has a representative. The Committee will shortly be submitting it response to the DEFRA consultation on the latest EU Chemicals Strategy known as REACH. This consultation is aimed at informing the Government's position on the proposed REACH regulation.

The Working Party on Notes, in keeping with its strategy to engage with the wider public has produced a 'message' note entitled 'Why do we worry about chemicals?' This note is aimed at promoting an understanding about risks associated with chemicals and that these risks can be controlled. WPN will shortly be publishing the note 'What is a poison?' as well as two revised notes, one on the 'Harmful effects of chemicals on children', the other a guidance note on the 'Safety of laboratory workers with disabilities'. The above submissions and notes will all be available on the RSC website.

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Arsenic bioaccessibility and speciation in soils at an abandoned arsenic mine in SW England

Possible sources of arsenic contamination have become a concern internationally. **Ben Klinck** and colleagues from the British Geological Survey describe in detail one source of arsenic exposure in the UK – a disused arsenic and copper mine in South West England.

In the mid 1800's the use of arsenic (As) as a pesticide became common and by 1869 the Devon Great Consols Mine near Tavistock in Devon was supplying half the world's arsenic from calcined arsenopyrite. Production ceased around about 1925.

Arsenic in soils is spatially widely distributed at Devon Great Consols. The As median concentration in mine soils is 2100 mg kg^{-1}, but ranges from 250mg kg^{-1} to 69000 mg kg^{-1}. Background soil samples taken from agricultural fields near to the village of Bere Alston and not affected by mineralisation or mining disturbance, have a median As value of 71 mg $kg^{-1}$and a range from 17 mg kg^{-1} to 172 mg kg^{-1}. A further set of soil samples collected from a farm to the south west of Devon Great Consol, with underlying, unworked mineralisation, show higher As concentrations than the background soils (As median concentration: 163 mg kg^{-1}).

The As bioaccessibility has been investigated using an *in vitro* test – a physiologically based extraction test (PBET) developed to simulate the leaching of a solid matrix in the human stomach and gastrointestinal tract. The term bioaccessibility is here used to describe the fraction of the total As concentration that is soluble in the stomach and gut and as a result is available for systemic uptake. The amount of As that is actually adsorbed systemically, the bioavailable fraction, is less than or equal to the amount that is bioaccessible. The median value of bioaccessible As for the soils on the mine site is 408 mg kg^{-1}. Much lower concentrations are measured for the agricultural soils over mineralisation (PBET As median value of 14 mg kg^{-1}) and for the Bere Alston background soils (PBET As median: 7 mg kg^{-1}). The results suggest that bioaccessible As is a better measurement than the total As content when considering risk assessment, and bioaccessibility data have clear implications for site-specific risk assessments.

Chemical sequential extraction data have been used to help elucidate the nature of the physico-chemical forms of As in the soils and mine waste material. Chemometric data processing allows characterisation of the matrix by resolving the number and composition of the physico-chemical components present. The most significant component contains mainly iron, As, and traces of sulphur, and is extracted in the last part of the test.

Further evidence of the Fe-As association comes from scanning electron microscopy, which shows As-rich, iron oxyhydroxides coating the surface of altered waste fragments and clastic grains (Figure 1). The coatings show various microfabrics from colloform iron oxyhydroxide to more crystalline coatings (fine-needle-like crystals).

X-ray absorption near edge structure (XANES) analysis indicates that As(V) is the dominant oxidation state in the mine waste materials and soils. Quantitative fits of EXAFS spectra using theoretical standards indicate As(V) in tetrahedral coordination with O and second and third -neighbour Fe atoms, ruling out the presence of major arsenopyrite. Second and third neighbour As-Fe EXAFS distances imply either adsorption of the As onto an iron oxide/hydroxide substrate or incorporation of the As into a mixed metal oxide phase. The two different As-Fe distances may reflect the presence of doubly oxo-bridged and singly oxo-bridged species.

Acknowledgments This article is published with the permission of the Executive Director, British Geological Survey. Dr Helen Taylor of the BGS helped in the acquisition of the XAFS/XANES data. Dr John Charnock, Daresbury, provided assistance with data interpretation. Tony Milodowski, BGS, carried out the SEM work.

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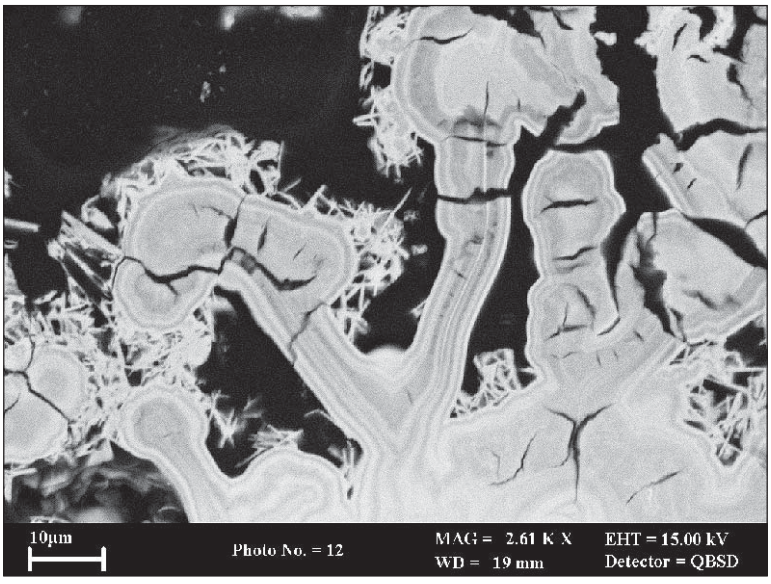

Figure 1 BSEM image showing detail of arsenic-rich iron oxyhydroxide cement coating the surfaces of altered waste fragments. It shows banded colloform oxyhydroxide gel material displaying shrinkage (desiccation) cracks. This is encrusted by more crystalline acicular oxyhydroxide.

Book review

Arsenic Exposure and Health Effects

W. R. Chappell, C. O. Abernathy, R. L. Calderon, D. J. Thomas (Eds.)
Elsevier 2003, pp 533, ISBN: 0-444-51441-4, £100.00

This book records the proceedings of the Fifth International Conference on Arsenic Exposure and Health Effects to be organised by SEGH (the Society of Environmental Geochemistry and Health), and which took place in July 2002 at San Diego, California. The genesis and history of these conferences from 1992 to the present day (the Sixth Conference is in July 2004) is admirably set out in the book's Preface which delineates the increasing US and global interest in arsenic exposure with a range of countries being represented (Bangladesh, India, Nepal, Thailand, China, Slovakia and others).

This volume includes contributions from many of these regions and is divided into sub-sectors covering Occurrence and Exposure, Epidemiology, Biomarkers and Animal Models, Mode of Action, Intervention and Medical Treatment, and Water Treatment and Remediation. So, in a way, something for everyone. My own interest was drawn by the several papers which examined the Mode of Action where a battery of biomolecular techniques had been focused on elucidating the mechanisms of arsenic toxicity – particularly in relation to carcinogenicity. The nature and consequences of oxidative stress induced by arsenic, the effects of antioxidants and signalling cascades induced by arsenic were clearly and authoritatively discussed. In particular, the paper by Kitchin and co-workers ("Some Chemical Properties Underlying Arsenic's Biological Activity") was illuminating.

The thread that links Occurrence (Eight papers variously from India, Nepal, Slovakia, Canada, California, Viet Nam) through modes of action and medical effects becomes uncertain when chronic low dose effects need to be demonstrated epidemiologically – and without such a demonstration legislation, intervention and remediation will be deprioritised. Although exposure in some areas of the world is apparent at a population level (for instance in Kshitish Saha's paper "Grading of Arsenicosis: Progression and Treatment" – photos not for the squeamish!), methods for identifying low levels of exposure and for monitoring the consequences of such exposure are difficult, expensive and fraught with complexity. The papers relating to these issues offer much of interest.

So, I am enthusiastic about this text if a little overwhelmed by the detail. The book is a massive source of literature references and the papers themselves are often introduced by reviews and summaries which would feed easily into a lecturing programme. I don't think it's the sort of book I will be lending.

Dr LEO SALTER,
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Meeting report: How useful will genomics, proteomics and metabonomics be to assess chemical risk in humans?

The RSC's Occupational and Environmental Toxicology Group (OETG) held a one day meeting in September 2003 at the Society of Chemical Industry, London to discuss the use of 'omic' technology in the risk assessment of chemicals. One objective was to compare the present states of technology with the promised potential. The targeted audience consisted of those people involved in risk assessment but not currently versed in the technological advances. This was reflected successfully in the background of registrants who mainly came from industry, occupational health orientated academia and government organizations. Speakers from academia and industry provided an excellent framework and stimulations for discussion. Not only were the technologies discussed but the problems in their potential use for human risk assessment processes. **Andy Smith**, Chairman of the OETG reports.

Can genomics contribute to the assessment of chemical risk in humans? **(Dr T. Gant, MRC Toxicology Unit).**

The current state of toxicogenomics was introduced by Dr T. Gant who described, firstly, the technological bases of gene arrays to detect changes in gene expression following experimental administration of drugs and chemicals. The importance of replicates, good study design and appropriate statistical analyses were emphasized. The ILSI/HESI (International Life Sciences/Health and Environmental Sciences Institute) collaboration was described which has aimed to examine the reproducibility of genomics on test toxicological samples between different laboratories and for various phenotypic endpoints. It has been clear that for some samples very different results have been achieved with different genomic systems. The more data the better was emphasized and the European Bioinformatics Institute in collaboration with ILSI was aiming to produce a toxicogenomics database. This would of course require a great deal of standardization of information acquisition across the toxicogenomic

field. Dr Gant described how patterns of gene expression with certain drugs and chemicals in experimental animals might be used to extrapolate to humans. Alternatively, the global gene expression changes could lead to greater understanding of the mechanisms of toxicity of particular drugs and chemicals. This could speed up drug development and could be taken in comparison with other data but not necessarily substitute for it. Whole networks of gene expression may be revealed not seen so far using other methods. In addition, the mechanistic bases of genetic variation in response to drugs and chemicals could now be explored in much more detail.

One of the challenges would be how to distinguish primary responses from secondary responses due to subsequent tissue damage. Temporal studies and model non toxic chemicals would be important approaches for comparison with human exposure responses at low doses. Failure to see lack of a changing profile would not in itself be conclusive proof that toxicity did not occur.

As far as studies on humans are concerned, obtaining appropriate tissue could be difficult. Rationales for extrapolating from available tissues such as blood to non-available tissues may be a way forward.

The role of proteomics in preclinical solutions
(Dr P. Camilleri, Biochemical Solutions)

Of course toxicogenomic data reveal patterns of gene expression changes or stability at the RNA level. What is more important, but technically at the moment more difficult, is to resolve complete patterns of protein changes in cells or whole tissues following exposure to drugs and chemicals. Dr Camilleri described the electrophoretic and mass spectrometric techniques constituting proteomics. As far as toxicity studies are concerned samples from liver, kidney, plasma and urine have usually been used to appreciate the responses of experimental models to drugs. Proteomics has now become an automated procedure in cutting and digesting protein spots from electrophoretic gels and their analysis and identification by mass spectrometry. By judicious choice of proteins, such as those involved in cell proliferation, it has been possible to distinguish between median and high doses the effects of which could be extrapolated to human doses. As with toxicogenomics, bioinformatics increasingly has an important role to play. So far the resolution and sensitivity is probably not as great as with toxicogenomics. However, important data can be obtained from readily available human samples such as urine and plasma that are not appropriate for genomic investigations.

Incorporation of toxicogenomics into predictive and mechanistic toxicology
(Dr T. Zacharewski, Michigan State University)

Dr T. Zacharewski was invited to the meeting especially for his experience in the USA. He was supported by a generous contribution from the RSC Angela and Tony Fish Bequest. Dr Zacharewski outlined how computational models were being developed to integrate DNA, RNA and protein interaction data which can be used to further elucidate mechanisms of toxicity as well as support risk assessments. Recently, blocks of SNP (single nucleotide polymorphisms) have been found to be inherited and these blocks might be used to generate data to rationalize drug dosing. Dr Zacharewski's present field of research is particularly concerned with the assessment of chemical endocrine disruptors that might be pharmaceuticals, industrial chemicals, phytoestrogens or environmental pollutants. Tiers of testing were described that increasingly use genomic molecular knowledge in the assessment process. For instance, searches for the distribution of dioxin responses elements in genes across three species were compared. As with a previous speaker, the importance of temporal studies were emphasised.

It was of interest to learn that both the US FDA (Food and Drug Administration) and EPA (Environmental Protection Agency) were encouraging the submission of toxicogenomic data. The FDA were developing guidelines on the use and submission of such data but it is not known exactly if those data will be used in decision making. However, data submitted by a sponsor might be used to make a case for a drug.

Metabonomics: The biochemical oracle
(Dr J. Shockcor, Metabometrix)

Although genomic and proteomic technologies are currently of high profile, in recent years there has been a steady development of nuclear magnetic resonance (NMR) to analyse plasma and urine, for instance, to identify changes in levels of natural metabolites in humans and experimental animals. Many of the bioinformatic and statistical techniques employed have much in common with those used in proteomics and toxicogenomics.

Dr Shockcor demonstrated how sensitive NMR techniques can be applied to analyse urine samples to detect metabolic responses to pathophysiological stimuli or genetic modification. Changes in levels of individual metabolites from many metabolic systems can be recognised in pattern recognition. This can be used not only for classification but also to identify new metabolites and probe for pathological mechanisms. Principle component analyses has proved a powerful statistical tool and can feed back information to other techniques such as genomics and proteomics. Most work has been done with clinical samples from drugs. Although sensitivity perhaps requires more development for application to some chemical risk assessment circumstances, it has the great advantage over the other techniques in only requiring urine samples and thus in human studies need not be invasive.

Identification of metabolic biomarkers using open and closed systems and the problems of cross species validation
(Dr C. Waterfield, GlaxoSmithKline)

In a continuation of the metabonomics theme Dr Waterfield illustrated its use in the safety assessment of peroxisome proliferator drugs following administration to test systems. In particular, metabolism of phospholipids and peroxidation were studied to understand mechanisms of action, identify potential biomarkers and investigate potential confounding factors. Findings could be fed back to proteomic studies for further investigation. It was important for safety

assessment to make cross species comparison if new biomarkers identified were to be of a use in human risk assessment.

Rational risk assessment in a changing world
(Professor A. Boobis, Imperial College)

So far much of the discussion had been of the potential of 'omic' technology but an important objective of the meeting was to discuss the current limitations for use in human risk assessment exercises. From his extensive experience Professor Boobis outlined his own view of present human risk assessment processes for chemicals and considerations for the use of the 'new' technologies in the light of evolving ideas and expectations. For instance, modest enlarged liver in rodents is no longer necessarily considered a toxic response but perhaps just adaptive. There are increasingly higher expectations of minimum risk but costs increase and there is great pressure for lowering animal usage. The genomics revolution and other advances in biomedical research may contribute to easier and more certain assessments. To identify new highly relevant biomarkers from 'omic' data much more mechanistic information will be required. However, resources can be limiting and often study designs and validation are unsatisfactory. In addition, biology is complex and there can be multiple interacting and parallel pathways leading to toxicity. Not all molecular changes are necessarily adverse or relevant and chemicals can have multiple unrelated affects. Species differences can be significant.

Professor Boobis pointed out that there must be quantitative relationships between any new potential biomarkers and adverse effects. Are there thresholds? We also have limited knowledge on what is normal variation within and amongst individuals. What governs homeostasis in an individual to maintain a constant environment, e.g. body temperature or fluid content, in response to chemicals? Finally, the role of risk assessment was to take on board advances in sciences but not to drive the development of science. ILSI/HES is trying to bring government and academic together with industry to develop the risk assessment process.

The number of issues raised in this last presentation stimulated a vigorous debate. It was clear that all three types of techniques were promising and provided complimentary information. Tremendous progress had been made in the last few years. Considerable developments and data generation were required, however, to reach the utopia of selectivity and sensitivity required for use in the field of non-drug chemical risk assessment for human studies.

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This report originally appeared in the *Occupational and Environmental Toxicology Group Newsletter*, Spring 2004. The ECG thanks the OETG for permission to reproduce this article.

Environment, Sustainability and Energy Forum

The Environment, Sustainability and Energy Forum (ESEF) recently carried out a consultation exercise to obtain views on a proposal to change reporting arrangements within the Royal Society of Chemistry (RSC). RSC specialist interest groups with an interest in environmental matters, namely the Environmental Chemistry Group (ECG), Water Science Forum (WSF) and Occupational and Environmental Toxicology Group (OETG) were invited to change their reporting requirements so that they report to ESEF in the future. We were delighted to hear that both ECG and OETG have decided with go with these new arrangements, and we look forward to working with them in the future on areas of mutual interest.

ESEF's portfolio of activities is growing and we are beginning to develop a presence both within the RSC and in the wider community to promote the central role that chemistry plays in environment, sustainability and energy issues.

Our activities are organised under three key initiatives:

(i) Chemistry of the Natural Environment. The Forum is developing plans for a workshop on the Chemical Aspects of Climate Change later in the year which will bring together environmental chemists and other key scientific disciplines (e.g. biologists) to understand the key challenges facing chemists in understanding forcings and feedback mechanisms in climate change and to explore the challenges at the interface of chemistry and other disciplines. This workshop will set the context for RSC action in this area and form the first of a series of workshops in the subject area

(ii) Sustainable Energy. ESEF is formulating new RSC energy policy which will outline the key priorities for chemistry and the chemical sciences which must be overcome to meet our future energy demands. We are also organising workshops and collaborating with other learned societies on energy conferences.

(iii) Green Chemistry. We are building on an RSC report published last year *Benign and Sustainable Energy Technologies* (http://www.rsc.org/lap/polacts/benign_report.htm) which makes several recommendations to further promote green chemical technologies. Activities here include organising workshops with organisations such as FIRSTFARADAY partnership on land and natural water remediation, workshops focussed on green products and developing links with the American Chemical Society.

ESEF is also active in developing RSC policy on topics which fall within its remit. For example, we are currently responding to the consultation on Defra's Sustainable Development Strategy. We are also formulating RSC position papers on current topical environmental issues such as air quality and many more.

If you would like more information about any of these activities then please contact **Dr Eimear Cotter**, Manager, Environment, Sustainability and Energy Forum on 020 7440 3333 or cottere@rsc.org

Tropospheric hydrogen peroxide and hydroperoxides

Data from the Halley Station, Antarctica and the Jungfraujoch, Switzerland

Sarah Walker from the School of the Environment, University of Leeds and colleagues explain the significance of measuring tropospheric H_2O_2 and ROOH.

Introduction

Oxidants in the troposphere principally control the levels of trace gases in the atmosphere. The presence of natural and anthropogenic pollutants e.g. carbon monoxide (CO), nitrogen oxides (NO_x) and sulphur dioxide (SO_2) in turn affect atmospheric oxidant composition. Peroxides are strongly oxidising species, so as well as being oxidation products, are important oxidants in their own right.[1] As such, they play an important role in the gas phase free radical chemistry of the atmosphere. They are formed *via* free radical chemistry involving the hydroxyl radical (·OH) so the presence of high H_2O_2 concentrations is symptomatic of atmospheric reactions involving the ·OH radical.

H_2O_2 also plays a key role in the atmospheric oxidation of SO_2 to sulphuric acid (H_2SO_4) and sulphate (SO_4^{2-}) aerosol[2] so its presence indicates the occurrence of aqueous phase chemistry leading to acid precipitation. Furthermore, there has been speculation that H_2O_2 in the atmosphere can lead directly to vegetation damage.[3] To summarise, peroxides are present in both unpolluted and polluted air and are useful in providing an indication of the chemistry occurring at a particular location. Measurements of peroxides in a 'pristine' environment such as the Antarctic, remote from the complicating factors of additional chemical pollutants, will improve our understanding of the chemical composition of the natural background atmosphere.

An automated instrument has been developed to measure the annual cycle of gas phase hydrogen peroxide and organic hydroperoxides in Antarctica. The ground-based measurements are being acquired at the new Clean Air Sector (CAS) Laboratory at the Halley Station, Antarctica (see p. 1). These measurements are part of the campaign: **Ch**emistry of the **A**ntarctic **B**oundary **L**ayer and the **I**nterface with **S**now (CHABLIS), which is due to culminate in an intensive summer campaign in 2005. We aim, through involvement in this project, to:

- Estimate a complete annual cycle for speciated peroxides in the Antarctic troposphere, investigate seasonal and diurnal variations, and facilitate the understanding of tropospheric chemical processes.
- Assist with the development of atmospheric chemical models, and test these models under extreme conditions.
- Help understand the chemistry of an unpolluted region in order to give real context to chemical processes occurring in polluted atmospheres.

This NERC-funded investigation is part of the Atmospheric Chemistry research programme within the School of the Environment at the University of Leeds (http://www.env.leeds.ac.uk/research/ias/chemistry/index.htm).

To prepare for CHABLIS, instrument field testing was undertaken in Switzerland at the Jungfraujoch High Altitude Research Station (46.55˚N, 7.98˚E, 3580 m above mean sea level), during the Free Tropospheric Experiment (FREETEX 2003) in February/ March 2003. A nebulising reflux concentrator sampled ambient air twice hourly, prior to on-site analysis by HPLC speciation, coupled with post-column peroxidase derivatisation and fluorescence detection. Hydrogen peroxide (H_2O_2) ranged between 0.92 ppbv down to below the detection limit (19 pptv) over a 14-day period. Methyl hydroperoxide (CH_3OOH) was also observed and ranged between 0.12 ppbv down to below this detection limit. No other organic hydroperoxides (ROOH) were detected. The peroxide data have been compared to other species and meteorological parameters that were measured during the campaign. (ppmv = parts per million by volume; ppbv = parts per billion by volume; pptv = parts per trillion by volume).

Background

Peroxides are produced predominantly by the self-reaction of hydroperoxy radicals (HO_2, reaction 1a) or by cross-reactions between HO_2 and other peroxy radicals (RO_2, reaction 1b). They are also produced *via* the ozonolysis of alkenes[4, 5] and are found within plumes as a direct result of biomass burning.[6]

The formation of peroxy radicals, discussed extensively by Lightfoot *et al*,[7] occurs predominantly *via* the photo-oxidation of carbon monoxide (CO) and volatile organic compounds[8] (VOC). Other sources of these radicals include the thermal decomposition of peroxy-acetyl nitrate[3] (PAN) and the ozonolysis of alkenes[9] *via* stabilized Criegee biradicals (R_1R_2COO). Peroxy radicals are relatively reactive species. This is because the presence of an H atom (or R group) weakens the O-O bond in O_2, so HO_2 (or RO_2) reacts much more easily than O_2 itself. Peroxy radicals can also react with nitrogen oxides, NO_x (NO and NO_2) *via* termination reactions forming nitric acid and organic nitrate, $RONO_2$ reactions 2a and 2b); these reactions compete with self/cross-termination reactions 1a and 1b, and thus suppress the production of H_2O_2 and ROOH.

Additionally, HO_2 (and RO_2) can react with NO in propagation reactions, producing NO_2 and hydroxyl radicals, ·OH (and RO; see reactions 3a and 3b). NO_2 can then be photolysed[10] to replenish NO (see reactions 4a and 4b) and thus further decreasing peroxy radical, and therefore hydroperoxide concentrations.

Gas phase formation of hydroperoxides is therefore dependent on NO concentrations. At low NO concentrations, reactions 1a and 1b dominate[11] and exhibit a second-order dependence[12] on HO_x concentrations. At higher NO levels, NO oxidation reactions 2a, 2b, 3a, and 3b become more important. At NO levels exceeding 100 pptv, the

production of hydroperoxides can be substantially suppressed[9] where by reactions 3a and 3b dominate, implying net photochemical production of O_3 (*via* 4a and 4b) is favoured over hydroperoxide production. At much higher NO levels (~2 ppbv), termination reactions 2a and 2b dominate giving increased free radical scavenging.

In contrast to the effect of NO, other pollutants can cause raised peroxide levels. High levels of VOC and CO cause H_2O_2 concentrations to increase (e.g. reactions 5a and 5b) due to the increased availability of free radical species. Hence, peroxide concentrations are enhanced when there is higher VOC:NO_x ratios. This is in contrast to ozone, whose concentration increases with CO, VOC *and* NO_x.

Production

	$HO_2 + HO_2$	→ $H_2O_2 + O_2$	(1a)
	$RO_2 + HO_2$	→ $ROOH + O_2$	(1b)

Effect of NO

	$HO_2 + NO$ (+ M)	→ HNO_3 (+ M)	(2a)
	$RO_2 + NO$ (+ M)	→ $RONO_2$ (+ M)	(2b)
	$HO_2 + NO$	→ $NO_2 + \cdot OH$	(3a)
	$RO_2 + NO$	→ $NO_2 + RO$	(3b)
	$NO_2 + h\upsilon$ (< 420 nm)	→ $O + NO$	(4a)
	$O + O_2$ (+ M)	→ O_3 (+ M)	(4b)

Effect of CO (and VOC)

	$\cdot OH + CO$	→ $CO_2 + H$	(5a)
	$H + O_2$ (+ M)	→ HO_2 (+ M)	(5b)

Effect of NO_2

	$NO_2 + \cdot OH$	→ HNO_3	(6)

Sinks

(a)	$H_2O_2 + OH$	→ $H_2O + HO_2$	(7)
(b)	$H_2O_2 + h\upsilon$ (< 350 nm)	→ $2\cdot OH$	(8)
(c)	dry and wet deposition		

The principal sinks for hydroperoxides[13, 14] are dry and wet deposition, reaction with ·OH radicals (reaction 7) and photolysis (reaction 8) at ultraviolet wavelengths between 190 and 350 nm.

Hydrogen Peroxide and Organic Hydroperoxides in Antarctica

Measurements of hydroperoxides in Antarctica have previously focussed on snow and firn,[15] (aged snow that is granular and compact and in a transitional state between snow and ice), Antarctic waters[16] and preserved H_2O_2 concentrations in ice cores.[17] The mechanisms controlling H_2O_2 and ROOH in the Antarctic troposphere, however, have not been investigated to such an extent. Jacob and Klockow[18] measured H_2O_2 concentrations in ambient air, in conjunction with snow and firn cores, at the German Research Station, Neumayer. Despite diurnal variation in temperature and relative humidity of 100%, sufficient for depositing gaseous H_2O_2 onto the snow surface, no significant diurnal cycle was observed. H_2O_2 was found to range between 0.1-1.1 ppbv.

Riedel *et al.*[19] conducted the first year-round Antarctic measurements, also at Neumayer. It was assumed that no other organic hydroperoxides would be present in such a remote marine atmosphere through previous measurements conducted by Weller *et al.*[20], where only CH_3OOH, alongside H_2O_2 could be detected. The data indicated the occurrence of seasonal variations. During polar night, H_2O_2 ranged from below the limit of detection (LoD) of 0.05 ppbv, up to 0.11 ppbv. CH_3OOH was also detected and ranged from below this LoD, up to 0.14 ppbv. As expected, higher mixing ratios were found during the sunlit period (see Table 1 on next page).

It is therefore important to implement highly sensitive and reliable techniques to measure the sub-ppbv concentrations present in 'pristine' Antarctic air, away from the complicating influence of anthropogenic pollutants. Only then can the natural seasonal and diurnal variation of these trace species, be deduced.

Field Testing at the Junfraujoch

Gas phase hydroperoxides were sampled twice hourly *via* an automatic nebulising reflux concentrator. Analysis was performed on-site by HPLC speciation (C-18 reversed-phase column) followed by post-column peroxidase derivatisation[21] and fluorescence spectrophotometric detection. Calibration was performed daily *via* serial dilution of a stock solution of H_2O_2 (previously standardised by titration against a known $KMnO_4$ solution) producing liquid standards. A schematic of the instrument is shown in Figure 1. The field tests for CHABLIS during FREETEX 2003 emphasised any technical modification needed for deployment in Antarctica.

Figure 1: Schematic diagram showing assembly of the gas phase peroxide instrument

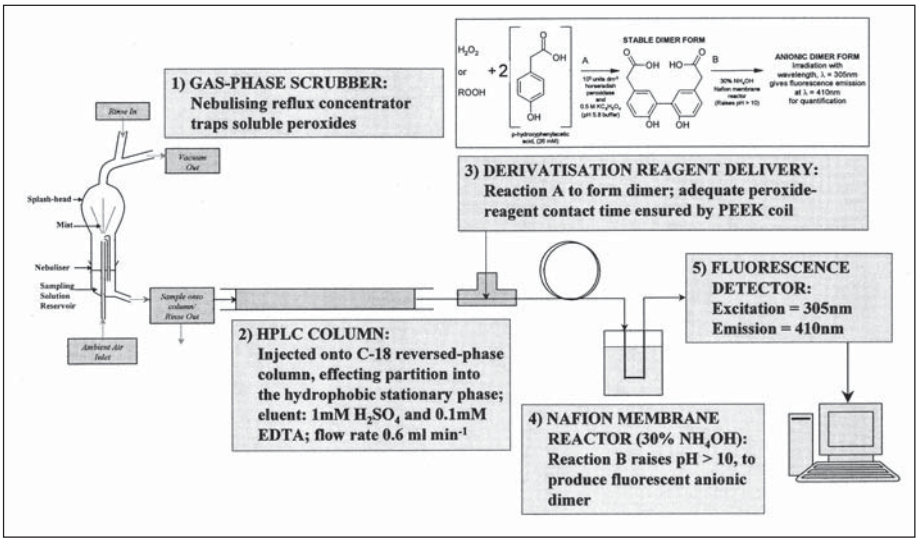


Table 1: Mixing Ratios for H_2O_2 and CH_3OOH at Neumayer during 1997 – 1998

Conditions	H_2O_2 Mixing Ratios (ppbv)		CH_3OOH Mixing Ratios (ppbv)	
	Mean	Range	Mean	Range
Polar Night	0.054 ± 0.046	< 0.03 - 0.11	0.089 ± 0.052	< 0.05 - 0.14
Sunlit Period	0.20 ± 0.13	< 0.03 - 0.91	0.19 ± 0.10	< 0.05 - 0.89

Table 2: Summary of results from the Jungfraujoch

Species	LoD (pptv)	Range (ppbv)	Average (ppbv)
H_2O_2	19	< 0.019 – 0.92	0.17
CH_3OOH	19	< 0.019 – 0.12	0.03
Other ROOH	19	< 0.019	not detected

Results from the Jungfraujoch

A summary of results is shown in Table 2. The LoD was based on three times the standard deviation of the baseline.

Figure 2 shows a time series for H_2O_2. The three maxima (1st, 6th and 10-12th March 2003) correspond to a *regional* wind direction change from NW to SW (based on 5-day back trajectories[22]). This implies NW air masses carry a *lower* peroxide/peroxide pre-cursor concentration than SW air, and suggests SW air masses had increased photochemical activity compared to NW air, agreeing with previous research[23, 24]. Average *local* wind direction[25] was 224°, 265° (SW), 312° (NW) respectively, so the third event (10th-12th March) occurred during a *local* north-westerly which may explain why it is less well-defined. Increased atmospheric pressure and low NO_x were also observed during this period, which could also enhance peroxide production (reactions 1a and 1b) whilst suppressing reactions 2 - 4 and 6.

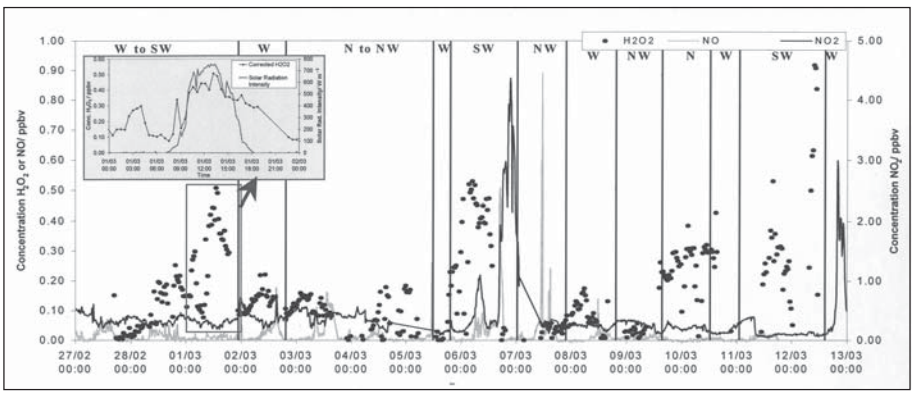
The diurnal cycle for 1st March (see inset, Figure 2) shows that peak H_2O_2 (0.51 ppbv) at 13:08 corresponded to maximum solar radiation intensity (~750 W m^{-2}). The earlier peak (0.30 ppbv) at 04:00 occurred during minimum solar radiation intensity, may be due to secondary factors e.g. rising water vapour levels that were experienced at this time.

Hydroperoxide formation is also sensitive to the rate of ·OH radical oxidation of NO_2 (reaction 6). During peak NO_2 levels, reaction 6 may exceed ·OH-initiated oxidation of CO and VOCs to produce HO_2 and RO_2 (reactions 5a and b), and thus hydroperoxides. Maximum observed NO_2 was 4.36 ppbv (21:20, 6th March), which corresponded to minimum H_2O_2 levels. Figure 2 shows H_2O_2 reducing to a minimum when NO concentrations exceeded 0.1 ppbv. This implies suppression of H_2O_2 production and agrees with previous research.[9]

Conclusion

A 14-day dataset was achieved at the Jungfraujoch where the instrument was tested for deployment in Antarctica. The instrument, currently at Halley, is equipped to measure peroxides until 2005. Datasets collected during CHABLIS are required to advance the understanding of how peroxides behave in the Antarctic troposphere, and hence enhance our knowledge of tropospheric photochemistry, acid deposition and the oxidative capacity of the atmosphere.

Figure 2: H_2O_2 time series with air mass classifications[22, 26] and NO_x data[25] for 27/02-12/03/2003; Inset: diurnal cycle for 1st March alongside solar radiation intensity[25]



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Web links:
Glacier glossary http://nsidc.org/glaciers/glossary/ (includes a definition of **firn**)

Halley & other Antarctic research stations http://www.antarcticconnection.com/antarctic/stations/halley.shtml

Problems with Plastics?

Media storm! That is probably the most appropriate term to describe the reception of our short article published recently in the journal *Science* [1]. There we reported our observations of the distribution of microscopic particles of plastic in the marine environment. In addition to worldwide TV coverage, publications as diverse as the *Wall Street Journal*, *The Guardian* and *Plastics & Rubber Weekly* all ran reports of our work – the result of a multidisciplinary study by ecologists and environmental and physical chemists.

In collaboration with Andrea Russell from Southampton University and some of our former students, and with funds from the Leverhulme Trust, we identified plastics and other particles – obtained by flotation from a variety of intertidal and subtidal sediments – using microscopy interfaced with Fourier Transform-Infrared Spectroscopy (FT-IR). Comparison of the FT-IR spectra with those in published databases allowed us to identify fragments of polythene, nylon and many other polymers in the sediments. These probably form from abrasion and mechanical degradation of larger pieces of plastics. We do not know the environmental consequences, if any, of these minute fragments, but in laboratory trials we were able to show that the particles were ingested by barnacles, lugworms and amphipods. Furthermore, when we studied archived planktonic samples collected by the Sir Alister Hardy Foundation for Ocean Science's Continuous Plankton Recorder since the 1960s, we were able to show how the amounts of microscopic plastics in the oceans have increased with time.

We would like to know the fate of any chemicals associated with these plastic particles. It is known that some hydrophobic pollutants are strongly sorbed by different polymers. For example, it is possible that the plastics act in the same way as solid phase extractants (SPEs) when SPEs are used to isolate pollutant analytes from water. Also, many additives and processing agents are used in plastics' manufacture, e.g. as pigments, biocides, plasticizers *etc.*, and many of these chemicals are designed to leach to the surface of the plastics. We now need to establish whether such chemicals can be transferred from the plastic to marine organisms. We hope that our current research programme, again funded by the Leverhulme Trust, will soon provide answers to these urgent environmental questions.

STEVE ROWLAND and RICHARD THOMPSON,
University of Plymouth,
June 2004

[1] Richard C. Thompson, Ylva Olsen, Richard P. Mitchell, Anthony Davis, Steven J. Rowland, Anthony W. G. John, Daniel McGonigle, Andrea E. Russell. Lost at Sea: Where does all the plastic go? *Science,* 2004, **304**, 838.

Web links:

The Sir Alister Hardy Foundation for Ocean Science (SAHFOS)
www.sahfos.org

R. C. Thompson *et al*, 'Lost at Sea: Where does all the plastic go?' *Science,* 2004, **304**, 838 http://www.biology.plymouth.ac.uk/staff/Thompson/Thompson.pdf

The RSC's Library and Information Centre – An environmental chemistry knowledge centre

The foremost repository of chemical knowledge in Europe, the Royal Society of Chemistry's Library and Information Centre (LIC) at Burlington House is also an excellent source of information on the environment and environmental chemistry. The LIC has accumulated an impressive array of resources for chemists over 160 years – from rare journals to books covering all aspects of the chemical sciences. These days it also subscribes to electronic journals and databases. **Nazma Masud** from the LIC explains how the library's collections can benefit environmental scientists.

One of the latest additions to the LIC's electronic database collection is **Knovel** (http://www.rsc.org/lic/knovel_library.htm). RSC members have free access to this web portal, which allows quick but thorough searches of a collection of 700 essential databanks and electronic books – including the *Environmental Contaminant Reference Databook*, the *Hazardous Chemicals Handbook* and *Patty's Toxicology*.

The LIC provides among its core services the Chemical Enquiries Helpdesk – a comprehensive, confidential, and largely cost-free service for RSC members. Relevant information resources – not always available in other libraries – are utilised by the Helpdesk's chemical information specialists to answer members' information enquiries. The Helpdesk can often supply that crucial piece of elusive information, provide further leads, or add a new perspective to a search strategy. There are regular enquiries in the area of chemical hazards, health & safety legislation, and environmental chemistry. For example:

- Levels of ammonia in East Anglian peat bogs;
- Toxicity of methyl bromide;
- Antibiotic and hormone content of sewage sludge and possible exposure from the spreading of sludge onto land near residential areas;
- Olfactory detection limits for ozone.

Resources used for this work include:

Chemical Abstracts

Handbook of Environmental Data on Organic Chemicals, 4th Edition

Dictionary of Substances and their Effects (DOSE), 2nd Edition

A reference book on chemicals and their impact on humans and the environment across the globe, the new edition from the RSC brings together data on over 4,100 chemicals and provides comprehensive information on mammalian and avian toxicity, occupational exposure, ecotoxicity, and environmental fate.

ECH&S (Environmental Chemistry, Health and Safety)

This RSC publication contains information on chemicals deemed to cause actual or potential problems to humans or the environment. It covers the environment not only from a scientific/technical standpoint, but also from business and legal perspectives.

TOXLINE

The US National Library of Medicine's toxicology database (http://toxnet.nlm.nih.gov) contains bibliographic information on the biochemical, pharmacological, physiological, and toxicological effects of drugs and other chemicals.

Croner's Waste Management

Croner Publications (Wolters Kluwer) regularly update this manual, which covers UK and European legislation, practical guidance to aid compliance, specific information on different types of waste, directory of contractors, consultants, local authorities and recyclers, and a list of statutes and official guidance publications.

ECETOC (European Centre for Ecotoxicology and Toxicology of Chemicals) Publications

Environmental Health Criteria (EHCs): Publications of the International Programme of Chemical Safety under the auspices of WHO

Provide risk assessments of the effects of chemicals on human health and the environment.

Members can visit the LIC at Burlington House in Piccadilly or at www.rsc.org/lic to see what other services we offer. The LIC is an invaluable benefit of membership, and the services of the Chemical Enquiries Helpdesk are a valuable knowledge resource. Send us an enquiry from http://www.rsc.org/lic/library3.htm or library@rsc.org to see what we can do for you or call us on +(44) 207 440 3373 for more information.

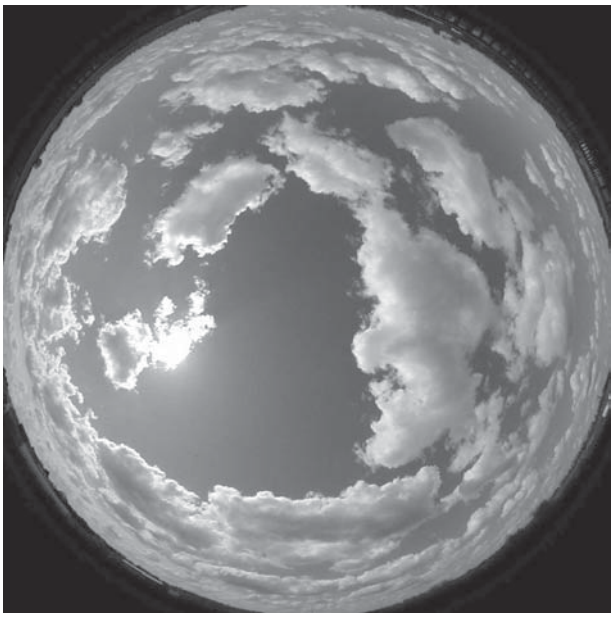
Forthcoming symposium

Faraday Division

Faraday Discussion 130

ATMOSPHERIC CHEMISTRY

University of Leeds, UK
11 – 13 April 2005



Introduction

Faraday Discussion 130 will be held exactly 10 years after the first Discussion on atmospheric chemistry and at a time when public awareness of the consequences of climate change and the health implications of air pollution has never been higher. Chemistry plays a key role in the removal of greenhouse gases, the production of secondary pollutants and the rate of recovery of stratospheric ozone.

Why Should You Attend?

An interdisciplinary approach has led to major advances in understanding atmospheric chemistry. A wide range of experimental methods, field instrumentation (from surface to space) and modelling tools have been developed to study all regions of the atmosphere in order to better understand links between chemistry and our changing atmosphere. This Faraday Discussion seeks to bring together chemists, physicists, meteorologists and atmospheric scientists, from diverse backgrounds in laboratory studies, field measurements, and the development of numerical models and chemical mechanisms.

The Science

New and unpublished experimental and theoretical work will be presented in the following areas:

- Gas phase spectroscopy, chemical kinetics and photochemistry
- Atmospheric processes at the air/liquid and air/solid interface
- Chemical field measurements in the atmosphere and instrument development
- Development of chemical mechanisms and interpretation of field data through modelling
- Satellite measurements
- Interaction of atmospheric chemistry and climate change

Call for Papers

Offers of papers related to the themes for discussion are now invited. Abstracts that fit most closely with the themes of the meeting will be selected for oral presentation. Authors of the selected abstracts will then be invited to submit their work as a full paper, which will form the basis of their short presentation at the meeting. The full paper must contain new, unpublished work and be submitted five months in advance of the Discussion. The papers selected for presentation and discussion will be refereed and then sent to all participants as preprints four weeks in advance of the meeting.

How to Submit a Poster Abstract

Abstracts of presentations should be e-mailed to Christine Hall as a Word attachment with the subject header: 05FD130 abstract.
Deadline: 4th February 2005.

Sponsorship and Exhibitions

Sponsorship and exhibition opportunities are available. Please contact the RSC Conference Office for details.

Request for Further Information on Faraday Discussion 130 *Atmospheric Chemistry*

The programme and application form will be circulated in Autumn 2004. All enquiries about attending the Discussion should be addressed to Christine Hall:
Telephone: +44 (0) 20 7437 8656 or +44 (0) 20 7734 1227
E-mail: conferences@rsc.org
By post: FD130, RSC Conference Office, Burlington House, Piccadilly, London W1J 0BA UK
Website: http://www.rsc.org/FD130

Meeting report: ECG DGL 2004 Environmental Chemistry from Space

The RSC Environmental Chemistry Group was fortunate in attracting three exceptional speakers for this year's Distinguished Guest Lecture and Symposium, which took place on March 3rd 2004 in the Meeting Room of the Linnean Society of London.

Dr Paul Monks (University of Leicester) opened the meeting with a presentation on *"Probing the troposphere from space"*. His description of the Earth System as a coupled multi-phasic land-sea-air system illuminated one of the salient reasons for satellite studies of the atmosphere – how can the Earth System be studied holistically unless from space? This is especially important in relation to long-range pollutant transport (twenty percent of any air pollution event is from a distant source) and for monitoring geopolitical initiatives such as Kyoto. However, Dr Monks also pointed out the difficulties associated with satellite measurements – the troposphere is a well-mixed layer some 10 km from the Earth's surface and there are problems with studying this layer through the whole bulk of the atmosphere from a satellite 800/900 km up in a Low Earth Orbit. He explained that the satellite systems are an integral part of a network of measurement techniques for studying tropospheric chemistry. Land stations such as those at Mace Head in Ireland and Cape Grim in Tasmania provide single site data (spatially limited) and flights by research aircraft such as the BAE-146 provide snapshot data (temporally limited) to a data repository and then comparisons can be made to the continuous global coverage from satellites – "… satellite measurements provide data which integrate the limited spatio-temporal scales of the each of the other techniques".

In the second talk of the afternoon – *"Remote-sensing of air-sea fluxes of CO_2: constraining the global $C0_2$ budgets"* – **Professor Jim Aiken** from the Plymouth Marine Laboratory (PML) described work at the Centre for Observation of Air-Sea Interactions and Fluxes (CASIX) in supporting NERC initiatives on the quantification and understanding of the Earth System. Data from satellites are used to study phenomena such as the air-sea fluxes of chemical species, surface plankta distributions, marine biogeochemistry, sea temperature and ocean circulation processes – but with a focus on carbon dioxide fluxes, the core carbon cycle and global climate. Models are needed to exploit these data and to quantify carbon fluxes; three-dimensional models with coupled biology are being used (North Atlantic Model, North-West European Shelf Model). Satellites provide data on winds, sea surface temperature, solar radiation, and ocean colour to modellers, and these are used as an input to model parameterisation and also to measure the success of the models' predictive capacities in relation to the carbon cycle. As always, the modellers seek more accurate data – which can only come from geostationary satellites.

Professor John Burrows (University of Bremen) closed the symposium with the **ECG 2004 Distinguished Guest Lecture** *"Viewing the Earth's environment from space: the challenges, the progress and the future."* He viewed the use of remote sensing via satellite as a means of obtaining objective data about the impacts of anthropogenic activities on the biogeophysical system, and delineated several crucial questions that needed a continuous programme of expansion in Earth Observation Systems. The enormous economic and social costs of climate change to the world community highlight the need for observations of emissions to the atmosphere – these are crucial for understanding the rate at which climate change is occurring and for detecting any successes in its amelioration. Ozone depletion was continuing to occur. The coupling of climate change with ozone depletion via phenomena such as the occurrence of polar meso spheric clouds meant that expectations of a post-Montreal Protocol smooth, continued, diminishment of the ozone hole might be confounded. Studies of NO_x sources in the stratosphere, the impact of meteorites and solar proton events on the Earth's atmosphere, the quantification of atmospheric aerosols, and surface temperature measurements are all dependent on satellite observations – because it is only via satellites (both Low Earth Orbit and Geostationary Earth Orbit) that data with adequate temporal and spatial resolution can be obtained.

Satellite measurements of the atmosphere began in the 1960s when the USSR attempted to measure ozone by UV spectrophotometry from space. Subsequently the NASA Nimbus series and other satellite systems improved and extended these early experiments. In 2002 the European Space Agency launched SCIAMACHY (Scanning Imaging Absorption Spectrometer for Atmospheric Chartography).

This satellite (together with the GOME (Global Ozone Monitoring Experiment) series) has extensively increased the understanding of global pollution processes. Tropospheric nitrogen dioxide has been tracked from sources such as industrial centres in Japan and China, burning in Africa, South America and Indonesia, and the success of vehicular emission reduction technology in the US and Europe is clearly demonstrated by observations of continent-wide reductions in nitrogen dioxide concentrations. Cloud formation and growth has been examined via aerosol studies, chlorophyll-a is being tracked across the oceans, and reactive species such as OClO and BrO (which are crucial in ozone depletion) are being quantified.

Professor Burrows completed his lecture with a clear exposition of the arguments for extending the range and accuracy of satellite data via the use of advanced technology detectors in geostationary satellites. More accurate data would provide continuous regional-scale information for policy development and regulatory purposes, reduce the need for ground stations (and avoid their limitations) and assist co-operative actions by the global community for the benefit of us all.

Dr LEO SALTER,
Cornwall College,
Pool, Redruth, Cornwall

This summary first appeared in *ESEF News*, Issue No. 2, Spring 2004. More detailed accounts of these three presentations will appear in the January 2005 issue of the *ECG Bulletin*.

Recent books on the environment and on toxicology at the RSC library

The following books and monographs on environmental topics, toxicology, and health and safety have been acquired by the Royal Society of Chemistry library, Burlington House, during the period January to June 2004.

Aldo-keto Reductases and Toxicant Metabolism
(ACS Symposium Series No. 865)
Penning, T. M.; Petrash, J. M. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238464

***sec*-Butanol**
(Joint Assessment of Commodity Chemicals No. 43)
ECETOC, Brussels, 2003, ISBN: 0773633943

***n*-Butanol**
(Joint Assessment of Commodity Chemicals No. 41)
ECETOC, Brussels, 2003, ISBN: 0773633941

Consumer's Good Chemical Guide: A Jargon-free Guide to Chemicals of Everyday Life
Emsley, J., Spektrum, Oxford 1994, ISBN: 0716745054

Environmental Fate and Effects of Pesticides
(ACS Symposium Series No. 853)
Coats, J. R.; Yamamoto, H. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841237220

Environmental Impact of Fertilizer on Soil and Water
(ACS Symposium Series No. 872)
Hall, W. L.; Robarge W. P. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238111

Food Factors in Health Promotion and Disease Prevention
(ACS Symposium Series No. 851)
Shahidi, F.; Ho, C-T.; Watanabe, S.; Osawa, T. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238073

Ionic Liquids as Green Solvents: Progress and Prospects
(ACS Symposium Series No. 856)
Rogers, R. D.; Seddon, K. R. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238561

Nutritional Aspects of Bone Health
New, S. A.; Bonjour, J. (Eds.), Royal Society of Chemistry, Cambridge 2003, ISBN: 0854045856

Oriental Foods and Herbs: Chemistry and Health Effects
(ACS Symposium Series No. 859)
Ho, C. T.; Lin, J. K.; Zheng, Q. Y. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238413

Pesticide Decontamination and Detoxification
(ACS Symposium Series No. 863)
Gan, J. J.; Zhu, P. C.; Aust, S. D.; Lemley, A. T. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238472

QSARs: Evaluation of the Commercially Available Software for Human Health and Environmental Endpoints with respect to Chemical Management Applications
(ECETOC Technical Report No. 89)
ECETOC, Brussels, 2003, ISBN: 0773807289

Tetrafluoroethylene
(Joint Assessment of Commodity Chemicals No. 42)
ECETOC, Brussels, 2003, ISBN: 0773633942

Utilization of Greenhouse Gases
(ACS Symposium Series No. 852)
Liu, C.; Mallinson, R. G.; Aresta, M. (Eds.), American Chemical Society, Washington DC, 2003, ISBN: 0841238278

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