COMMITTEE ON THE TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

REPORT OF THE COT SUBGROUP ON THE LOWERMOOR WATER POLLUTION INCIDENT

Introduction and Background

1. A copy of the draft Final report of the COT Lowermoor subgroup (LSG) is attached at Annex A for final approval by the COT.

2. The LSG was established in 2001 to advise Health and Environment ministers on the possible long term health effects arising from a 1988 water pollution incident in North Cornwall. The terms of reference of the Subgroup were:

> "To advise on whether the exposure to chemicals resulting from the 1988 Lowermoor water pollution incident has caused, or is expected to cause, delayed or persistent harm to health; and
>
> " To advise whether the existing programme of monitoring and research into the human health effects of the incident should be augmented and, if so, to make recommendations."

3. The Lowermoor water pollution incident occurred on 6 July 1988 at the Lowermoor water treatment works near Camelford, North Cornwall. A contractor's relief tanker driver put 20 tonnes of aluminium sulphate into the water supply at the works. Water supplies to an estimated 20,000 people were polluted with aluminium, sulphate and other metals dissolved from pipework and plumbing materials (copper, lead, zinc and iron). Flushing of the distribution system to remove the contaminated water resulted in the disturbance of old mains sediments, mainly deposits of iron and manganese oxides, leading to contamination of the water with raised concentrations of these metals.

4. Two previous investigations have considered the health implications of the incident. Their conclusions, and the background to the current investigation, are given in Chapter 2 of the LSG report under 'Historical Perspective'. Chapters 3 to 6 discuss the information available to the Subgroup on water contaminant concentrations and, hence, possible exposures, on the toxicity of the chemicals of interest, and on the epidemiological and other studies carried out on the local population. The Subgroup also conducted interviews with 54 people who were in the area at the time of the contamination and received written information from another 59 (see Chapter 5). Please note that paragraph 5.182 may require a little revision following receipt of two recent literature papers. If so, this will be presented at the COT meeting.

5. The conclusions are given in Chapters 7 and 8. Chapter 7 is a discussion of the implications for health of the contaminants in relation to modelled and measured levels of exposure. Chapter 8 gives the Subgroup's opinion on whether

the adverse health effects which have been reported by some individuals who received the water were caused by exposure to the contaminants involved. Recommendations for further work are given in Chapter 9.

6. The COT has discussed the report previously on two occasions. In April 2005 it discussed the draft report which was out to consultation. Few comments were made by Members. The Committee discussed the then draft Final Report in October 2007 as reserved business. Members were informed that there had been a severe case of confirmed congophilic angiopathy which occurred at an early age in an individual who was resident in Cornwall at the time of the contamination incident in 1988. Higher than usual levels of aluminium had been found in the brain. The case was referred to the West Somerset coroner who had opened an inquest into the death of the individual. The minutes of the 2007 meeting are attached at Annex B for Members' information.

7. Following correspondence with the Coroner, and receipt of legal advice, completion and publication of the Subgroup report was then deferred until the Coroner's proceedings were completed. The inquest finally ended in March 2012. Given the time since the COT last saw the report, it was considered appropriate to bring the draft Final version back to the COT for approval. The major changes since 2007 are as follows:

- The case of the individual who was the subject of the Coroner's inquest is discussed in further detail in paragraph 5.137 – 5.149, and the Coroner's verdict is included. This section also discusses work commissioned by the Coroner into the metal content and pathology of tissues from 60 postmortem brains donated as part of the MRC study on Cognitive Function and Aging.
- The Coroner's verdict is attached at Appendix 20.
- Chapter 6 and Appendices 26 - 27 undate the information on aluminium from the scientific literature from 2007 to May 2012. Chapter 6 refers to the recent JECFA assessments of aluminium and lead. The section on metal-metal interactions is also updated, with a review at Appendix 29.
- Chapter 7 is updated slightly to reflect the changes in Chapter 6.
- Chapter 8, paragraph 8.40 provides the Subgroup's comments on the Coroner's case.
- Chapter 9 and Appendix 30 provide detailed recommendations worked up with appropriate experts for the neuropsychological and neuropathological investigations recommended previously by the Subgroup.

8. Members will wish to know that the two local representatives, who were appointed to the Subgroup in 2002 by the local MP Paul Tyler (now Lord Tyler) resigned at the Subgroup's final meeting on 16 October 2012. Written statements have been received from both members.

Question for Members

9. COT members are asked whether they have any remaining comments on the Final Report and whether they are content for it to be published.

Secretariat
November 2012

COMMITTEE ON THE TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

REPORT OF THE COT SUBGROUP ON THE LOWERMOOR WATER POLLUTION INCIDENT

Draft of the final report of the COT Lowermoor subgroup (LSG)

Secretariat
November 2012

COMMITTEE ON THE TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

REPORT OF THE COT SUBGROUP ON THE LOWERMOOR WATER POLLUTION INCIDENT

Minutes of the 11th December 2007 meeting, COT discussion of the LSG Report (**Reserved Business**)

In 2007 this item was discussed in reserved session. The minutes will remain reserved business until the final report is published.

Secretariat
November 2012

Appendix 1: Membership of the Lowermoor Subgroup

Chairman

Professor H Frank Woods CBE BM DPhil FFPM FRCP (Lond & Edin) F Med Sci (Formerly Sir George Franklin Professor of Medicine, Division of Molecular and Genetic Medicine, University of Sheffield. Now Emeritus)

Members

Professor J Kevin Chipman BSc PhD FRCPath FBTS FSB (Professor of Cell Toxicology, School of Biosciences, University of Birmingham)
Dr Lesley Rushton OBE BA MSc PhD CStat (Principal Research Fellow, Department of Epidemiology and Public Health, Imperial College London)
Ms Jacquie Salfield BSc MSc MIFST CertEd (Public interest representative)
Professor Stephan Strobel MD PhD FRCP FRCPH (Director of Clinical Education, Peninsula Postgraduate Health Institute)
Dr Anita Donley (formerly Thomas) OBE MB ChB PhD FRCP (Consultant Physician, Plymouth Hospitals NHS Trust)

Two local representatives also served as members until 16 October 2012

Secretariat

Ms Alison Gowers BSc MSc (From 28 April 2003 to 2 April 2004)
Ms Sue Kennedy Administrative Secretary (From 8 December 2011)
Mr Khandu Mistry Administrative Secretary (Until 5 May 2007)
Mr George Kowalczyk BSc MSc DABT CChem FRSC (Until 11 October 2002)
Ms Frances Pollitt MA DipRCPath Scientific Secretary
Ms Helen Smethurst BSc MSc (From 10 December 2001 to 28 May 2004)
Mr Michael Waring MA MB BChir BA FRCS LRCP Medical Secretary (Until 31 October 2001: first meeting only)

Appendix 2: Current Membership of the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment[34]

Chairman

Professor David Coggon OBE MA PhD DM FRCP FFOM FFPH FMedSci

Members

Mr Derek Bodey MA (Public interest representative)
Dr Roger Brimblecombe BSc MSc PhD DSc FRCPath FSB Cbiol
Professor Janet Cade BSc PhD PHNutr
Dr Rebecca Dearman BSc PhD
Dr Mark Graham BSc PhD FBTS
Dr Anna Hansell MSc MB BCh PhD MRCP FFPH
Dr David Harrison MD DSc FRCPath FRCPEd FRCSEd
Professor Roy M Harrison OBE PHD DSc FRSC Cchem FRMetS HonMFPHM Hon FFOM Hon MCIEH
Professor Brian Houston BSc PhD DSc
Professor Justin Konje MBBS MB MRCOG Dip Ultrasound
Professor Brian Lake BSc PhD DSc FBTS
Professor Ian Morris BPharm PhD DSc
Dr Nicholas Plant BSc PhD
Professor Robert Smith BA MSc PhD (Public interest representative)
Dr John Thompson FRCP FBTS
Professor Faith Williams MA PhD FTox

Secretariat

Mr Jon M Battershill BSc MSc (Scientific Secretary - HPA) (to 26 September 2012)
Dr Diane J Benford BSc PhD (Scientific Secretary - FSA)
Ms Frances Pollitt MA DipRCPath (Scientific Secretary - HPA) (from 26 September 2012)
Ms Julie Shroff (Administrative Secretary)

[34] As at 11 December 2012

Appendix 3: Health and other professionals who provided information

Those who had meetings with the Subgroup

Professor Freda Alexander (Edinburgh University)
Dr Paul Altmann (Radcliffe Hospital, Oxford)
Mr Malcolm Brandt (Black & Veatch Ltd)
Dr Chris Buckingham (South West Water Authority Residuary Body)
Mr Rolf Clayton (Crowther Clayton Associates)
Dr Ian Coutts (Royal Cornwall Hospitals, Truro)
Professor Jim Edwardson (Newcastle University)
Professor Margaret Esiri (Oxford University and Oxford Radcliffe NHS Trust)
Dr Chris Exley (Keele University)
Dr David Harris (Aluminium Federation)
Mr Peter Jackson (WRc-NSF)
Dr Chris Jarvis (General Practitioner)
Dr James Lunny (General Practitioner)
Mr Richard Mahoney (Aluminium Federation)
Mrs Jenny McArdle
Professor Tom McMillan[35] (Glasgow University)
Dr David Miles (West of Cornwall Primary Care Trust)
Dr Anthony Nash (General Practitioner)
Ms Pat Owen (West of Cornwall Primary Care Trust)
Mr James Powell (Black & Veatch Ltd)
Professor Nick Priest (Middlesex University)
Professor Michael Rugg (University of California Irvine)
Mr Anthony Wilson
Mr Chris Underwood (South West Water plc)
Dr Neil Ward (Surrey University)

Those who provided written information to the Subgroup

Professor Jim Bridges (Surrey University)
Dr A Davies-Jones (Royal Hallamshire Hospital, Sheffield)
Dr David Gould (Royal Cornwall Hospitals Trust)
Professor ARW Forrest (University of Sheffield)
Dr Norah Frederickson (University College, London)
Dr Alan Foster (formerly Derriford Hospital, Plymouth)
Professor Irvine Gersch (University of East London)
Professor Martin Koltzenburg (Institute of Child Health)
Dr Richard Newman (General Practitioner)
Mr Norman Roberts (Royal Liverpool and Broadgreen University Hospitals NHS Trust)

[35] By teleconference link

Dr Tony Wainwright (St Lawrence's Hospital, Bodmin)

Appendix 4: Discussion of the quality and reliability of scientific data

http://cot.food.gov.uk/pdfs/lsgreportapp4.pdf

Appendix 5: Consultation Responses

Introduction

Most consultation responses are published in full below, with the consent of the author. Where permission to publish was not received, only a brief description of the content is provided.

In two cases the responses contained only personal medical information and these have not been published, for reasons of confidentiality. Published papers, articles or book chapters sent with some responses have not been reproduced for copyright reasons.

List of those who provided consultation responses

Date	From	Comments
1/2/05	Dr R Handy Plymouth University	Enclosed published book chapter and article.
17/2/05	Mrs C Wyatt Cornwall	Letter with personal medical information.
17/2/05	Mr T Chadwick Cornwall	Document with observations made at the time of the incident and comments on the remit of the investigation and the nature of the contamination after the incident and flushing programme.
22/2/05	Mrs S Joiner Cornwall	Attached
23/2/05	Mr R Bowler Cornwall	Enclosed published article: 'Probe overcomes hairy problem' (New Scientist, 1 April 1995).
24/2/05	Mrs J Young Cornwall	Enclosed published article: 'Clash over water analysis results' (Surveyor, 31 May 1990).
1/3/05	Black & Veatch Ltd Surrey	Attached
9/3/05	Mr P Stewart Australia	Attached
14/3/05	Mr P Stewart Australia	Attached
29/3/05	Mr P Stewart Australia	Attached
20/4/05	Mr P Stewart Australia	Attached
9/3/05	Mr and Mrs I Clewes Devon	Letter with comments on remit of investigation, draft report and

		presentations at public meeting.
14/3/05	Dr D Miles West of Cornwall Primary Care Trust	Letter with comments on paragraphs 5.47 to 5.50 of draft report.
28/3/05	Mrs E Sigmund Cornwall	Document with comments about personal experience of events and on Subgroup's failure to review medical records and undertake clinical investigations.
21/3/0	Mr D Cross Somerset	Attached
8/04/05	Mr D Cross, Somerset, and Mr P Smith, Cornwall	Attached
12/4/05	Ms S Hazell Cornwall	Letter with personal medical information.
13/4/05	Leigh, Day & Co Solicitors London	Attached
15/4/05	Dr C Exley Keele University	Attached
21/4/05	Mr C Buckingham Pennon Group plc	Letter with comments on the report.
21/4/05	Mr A Wilson Cornwall	Attached
21/4/05	Dr R Burnham Royal College of Physicians	Attached
214/05	Dr Bettina Platt University of Aberdeen	Attached
22/4/05	Mr E Jansson Department of the Planet Earth Washington	Attached
22/4/05	Dr M Waring Health Protection Agency	Attached
19/5/05 and 31/3/06	Dr W Rea Environmental Health Center - Dallas	Attached

01/03/2005

Frances

I have attached a list of typos/errors/comments for the consultation report. It mainly relates to our report (Appendix 10). There are four relatively important points which I have highlighted in red – these definitely should be amended. The other points are less important. I have made the amendments which relate to our report using track changes. Let me know if you would like a PDF version.

Regards

James

James Powell
Black & Veatch Ltd
69 Grosvenor House
London Rd
Redhill
Surrey RH1 1LQ

Ref	Page/Para/Line	Comments
A	181/7.29/3	I am not familiar with the 'Margin of Safety' concept, but I suspect the equation is incorrect. Surely it should be: $MoS = \frac{NOAEL}{Daily\ intake}$ i.e. higher intake gives lower margin of safety
B	196/6/2	Not sure if this definition of MoS is correct (see A)
C	Abbreviations and Glossary	Margin of Safety is not listed in glossary or abbreviations
D	Glossary	Definition of coagulant could cause concern as in water treatment terms, Aluminium Sulphate is a coagulant: In water treatment, the a coagulant is a substance which causes very fine particles to stick together (it is an ionic process)
Appendix 10 – BV report		
1	264/5/2	Minor Typo – Replace '*of*' with '*for*'
2	264/5/4	Minor Typo – Replace '*on*' with '*of*'
3	265/2/2	Minor Typo – Delete '*and*'
4	270/6/1	Minor Typo – Replace '*of*' with '*for*'
5	270/7/6	Minor Typo – Replace '*which*' with '*that*'
6	275 Fig 12	Zero minutes graphic is printed as black it should be blue
7	277/4/3	Minor Typo – Lower case '*aluminium sulphate*'
8	279/16/1	Serious Typo – Replace '*contact*' with '*clear water*'
9	281/1/2	Serious Typo – Replace '*12*' with '*24*'
10	281/3/3	Minor Typo – Replace '*affect*' with '*effect*'
11	281/3/8	Minor Typo – Replace '*affect*' with '*effect*'
12	283/1/2	Minor Typo – Delete space after 'Table 1'
13	284/1/29	Minor Typo – Replace '*with*' with '*within*'
14	284/2/6	Delete sentence *'BVCs has been unable to locate the fifth private supply, that titled "Mount Camel"* – Following discussions at the public meeting, we realise that we had already accounted for this sample
15	285 Fig 22	Dates are all one day too late i.e. should run 6 July to 9 July not 7 July to 10 July
16	286/5/	After paragraph 5 insert new paragraph "*One private sample was taken on the morning of 7th July and analysed by the Laboratory of the Government Chemist, Taunton in December 1988. The measured aluminium concentration of 28 mg/l is consistent with the modelled results.*" – see comment 14
17	290/2/1	Minor Typo – Insert '*the*' between '*for*' and '*area*'
18	293/2/3	Minor Typo – Replace '*for*' with '*of*'
19	295/2/14	Incorrect statement - Delete "*At worst the contaminant concentration would be no greater than the CFD modelling predicts. If the density of the sludge blanket was greater than the contaminant, the latter would be a layer above the sludge blanket and therefore dilution and dispersion would have been accelerated.*"
20	296 Point 5	On reflection we would prefer to remove the word '*serious*'. It is a bit emotive and our conclusion relies on the assumptions about the set up of the tank e.g. outlet level, the sludge issue. If these assumptions prove to be incorrect then the level of doubt would be reduced NB. This is also quoted on page 67 Para 3.70
21	Appendix 10 general	Throughout our report we have used the term 'Clear Water Tank'. We note that in the main report the term 'Treated Water Reservoir' is generally used (e.g. Page 29).

09/03/2005

Dear Sir,
Please find attached a case study report which supports the toxicity of aluminium. If you have any questions, please feel free to contact me.
Peter Stewart

A Case Study In Human Aluminium Toxicity.

Background.

The subject was employed in an aluminium remelting plant in the period from February 1996 until December 1998. The plant processed 1200 tonnes/month of aluminium by ingot melting, continuous casting, rolling reduction, annealing, drawing, winding, & scrap reprocessing.

Occupational contamination levels were not determined at the time of exposure; however, anecdotal evidence of exposure relates that after being in the plant for more than 20 minutes, a film of oil and metal dust would be present on the skin, and that the plant contained a visible haze.

In July 1996 the subject presented to the on-site medical centre complaining of fatigue. Blood glucose was tested and the random result was 4.91 mmol/l, a normal result with no indications of diabetes. In May 1998 the subject's health had deteriorated, so he attended a private hospital health assessment centre for a full check-up. The symptoms evident were gastro-intestinal (IBS), fatigue, and increased number of respiratory infections.

Blood test results indicated mildly elevated liver enzymes (GGT and ALT), characteristic of Non Alcoholic Liver Disease (NALD), and anisocytosis, reflected in a high RDW. The problem was incorrectly diagnosed as due to lifestyle factors, and no further tests were conducted.

At the same time the subject had been experiencing significant problems with neurological dysfunction, as indicated by short-term memory, decision making, mood, irritability, aggressiveness, and anxiety.

There is no history of any other exposure to aluminium. The local water authority has regularly tested for aluminium as per their quality assurance program, and low levels are maintained. There have been no incidences of chemical overdosing to the water supply, or use of Al-based antacids.

Discovery.

In 2003 the subject contracted cellulitis, which was taking a long time to heal. In the investigation of the reason for the delayed healing, a hair sample was taken on the 16th July 2003 and submitted for mineral analysis. The result reported for Aluminium was 248 parts per million (ppm).

The reference interval established by the laboratory in accordance with normal clinical laboratory protocol is less than 18 ppm. A recheck of the result was performed and confirmed prior to release of the data.

The test was conducted by Trace Elements Inc. (TEI), which is a licensed and certified clinical laboratory that undergoes regular inspections with Clinical Laboratory Division of the Dept. of Health and Human Services, HCFA, USA. Analysis is by ICP-Mass Spectrometry (Sciex Elan 6100) methodology for all trace element determinations. The laboratory is equipped with a trace element class clean room utilizing HEPA filtration systems. The level of Al in the range reported (248 ppm) is found in less than 0.04% within the population tested of over 27,000 patient samples (samples obtained in accordance with established collection protocol) processed by the laboratory.

Subsequently the initial hair tissue mineral analysis was supported by tests of other tissues including Toenail, Fingernail, Foot Skin, Semen, and Underarm Hair, with other laboratories, and over a significant period of time. Refer to Graph One for the results mapped over the duration of stage one chelation.
In order to confirm the likely source of exposure, semen from 2000 was tested and allowed the construction of the probable contamination curve. The aluminium levels are consistent with a biological half-life of 3 years, and indicate that the level of tissue contamination in 1998 would have been about three times the level detected in 2003. Refer to Graph Two for the backward projection of aluminium levels.

Exposure Estimate.

Exposure is estimated using actual and estimated physical conditions:

- Assumed airborne contamination Level, 15 mg/m3
- Elevated temperature environment due metal remelting
- High breathing rates due heat, activity, & level of contamination.
- Respiration, 10 breaths/minute
- Lung Volume, 4.5 litres at end, (5.7 litres at start)
- Duration, 10 hours/day, 50% presence, 1074 days
- Uptake, at saturation conditions, 15%
- Total uptake over 3 years = 32,622 mg.
- Half Life, (non-repeating exposure) = 3 years
- Calculated body burden 5 years after exp. = **10,765 mg.**

Exposure is compared to projections based on tissue test results:

- Tissue tests, (mean) = 102 mg/kg
- Body weight = 140 kgs.
- Calculated body burden from tests = **10,529 mg.**

Hypothetical Clearance of Sequestered Aluminium:

Based on possible biological clearance rates for aluminium, the following projection can be made for the reduction in sequestered tissue levels between 1998 and 2003:

- Av. Biological Fluids Al Content = 6.55 ppm, 5/9/2003
- Urinary Clearance at this Rate = 17,930 mg
- Sweat Clearance, (0.3 l/day) = 3,586 mg (impaired)

- Hair Tissue Clearance = 16.25 mg
- Nails Tissue Clearance = 15.00 mg
- Faecal Clearance, (from 3.1) = 3.3 mg/day = 6,022 mg
- Calculated Clearance over 5 years = 27,569 mg.
- Calculated body burden from hypothetical clce = 5,053 mg.

The clearance of sequestered aluminium from the body is consistent with the calculated half-life, the tissue test results, and the occupational exposure, with the hypothetical clearance being higher than the actual clearance due to the mobilisation at the time of fluids testing (5/9/2003).

The Estimated Occupational Exposure exceeds the Recommended Tolerable Weekly Intake (RTWI) of 7 milligrams per Kg body weight for all age groups, as set by the FAO/WHO Joint Expert Committee on Food Additives, (JECFA), by a significant amount, (being factor 3.37).

Adverse Health Effects.

Adverse health effects from aluminium, experienced by the subject both at exposure, and during chelation, and also reported in medical research are as follows:

- Psychological dysfunction, tested by QEEG 17/6/2003
- Cognitive Impairment, tested by ERP 24/10/2003
- CNS Balance Disturbance, tested 26/2/2004
- Cognitive Impairment, tested by Neuropsychological Series 6/8/2004
- Vareta Neurofunctional Assessment, evaluated 3/6/2004
- Neural Plaque, high-resolution Spect scan 8/4/2004
- Gastro Intestinal disturbance (IBS)
- Intestinal Flora Imbalance and Leaky Gut
- Anisocytosis and Haemolytic Anaemia
- Lymphocytopenia
- Chronic Fatigue Syndrome
- Sperm Hyperactivation Dysfunction, as per SPA 9/4/2004
- Sperm Morphology, Head Defects, as per SCSA 3/5/2004
- Renal Impairment
- Hepatic Impairment
- Solar Hyperkeratosis from sweat gland exudation of Al

Neuropsychological Testing revealed a pattern of dysfunction which had some similar findings to that of Altmann (19), in a retrospective study of people exposed to contaminated drinking water at Camelford, who found reduced performance on psychomotor speed relative to estimated premorbid IQ, which could not be attributed to anxiety.

Reproductive Effects.

Metals in semen appear to inhibit the function of enzymes contained in the acrosome, the membrane that covers the head of the sperm. The effect is to disrupt the acrosome reaction and inhibit capacitation. The enzyme acrosin, contained in non-reacted acrosome, is thought to have a role in digestion of the sperm path through the zona pellucida, or in the zona binding process. The acrosome reaction occurs following tight binding to the ZP3 receptor located on the zona pellucida of the oocyte and is a prerequisite for the fertilisation process. Sperm that acrosome-react prematurely will be unable to bind to the zona pellucida. Sperm which are unable to bind to the oocyte and/or unable to acrosome-react, will also be unable to fertilise the oocyte, Dana (74).

The study of Aluminium in Finnish Men, Hovatta (1), found a definite correlation between aluminium in the spermatozoa and motility and morphology of the spermatozoa, but there was no correlation between the concentration of aluminium in the seminal plasma and the semen analysis parameters. In addition, "The semen analysis of the three men with clearly the highest aluminium concentrations in their spermatozoa, (from 8.7 to 21.5 ppm), showed asthenozoospermia in all three cases, (A + B motilities from 16 to 46%)".

In this case the subject recorded an aluminium level of 16.7 ppm in 2000, which is of both semen and spermatozoa, and scored an A + B motility of 53% in 2004. Morphology in 2000 was unknown, and in 2004 was unusually low.

In a study of seminal plasma metal levels, Dawson (45), found an inverse relation between aluminium in seminal plasma and sperm viability. Apparently the presence of the metal in the seminal plasma exerts a toxic effect on sperm.

The subject's reproductive status is therefore consistent with aluminium toxicity in the period c2000 to the current day.

Current Situation.

The chelation of aluminium continues in a pulsed manner as allowed by the renal and hepatic capacity. Although the level is within the reference range, it rebounds after the chelation run, indicating that tissue stores still exist.

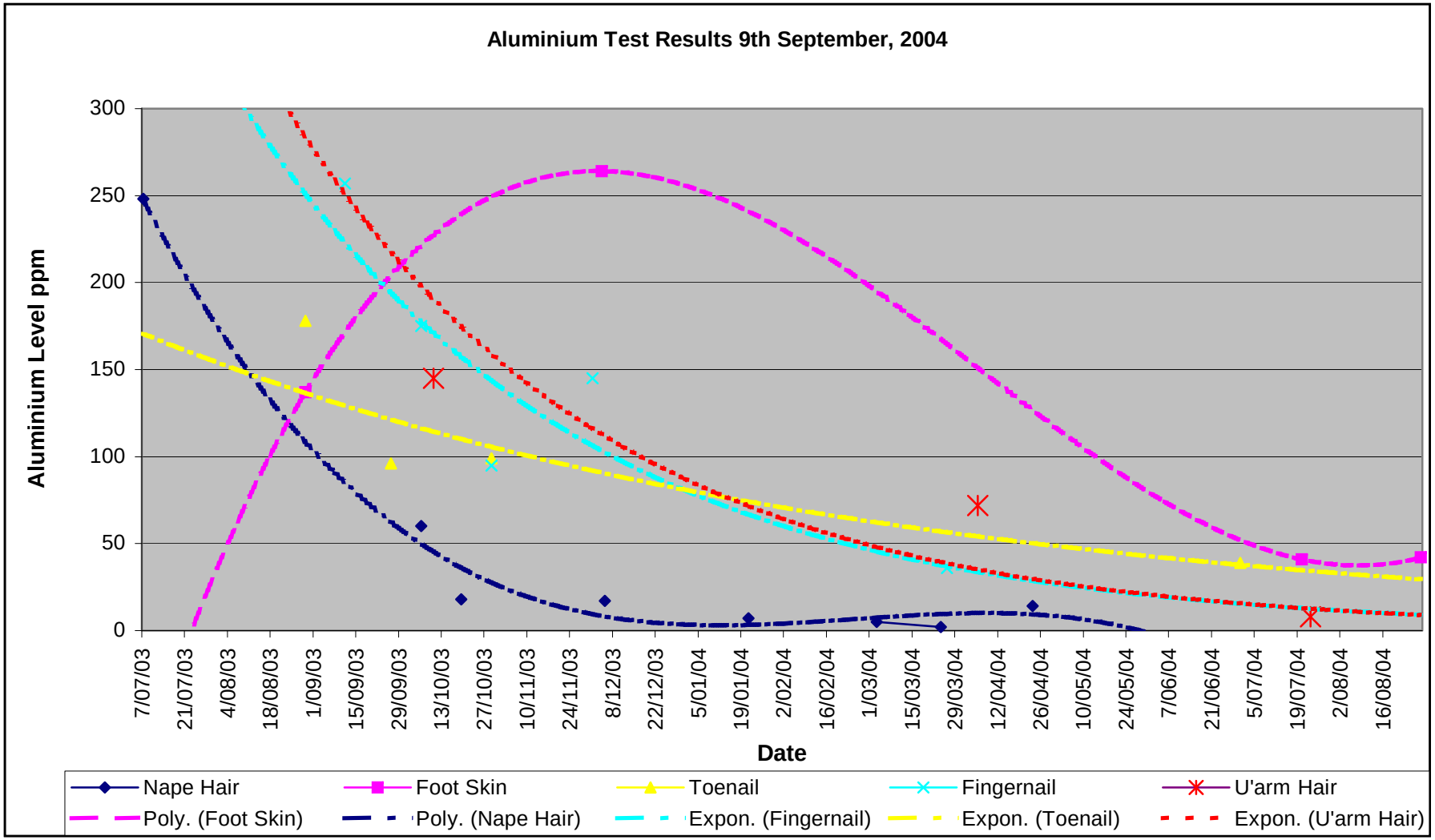
The test of removal will be the no-rebound test, plus a clear brain scan using the PET isotope PIB C-11, which is said to be capable of detecting neural plaques and is currently under clinical trials.

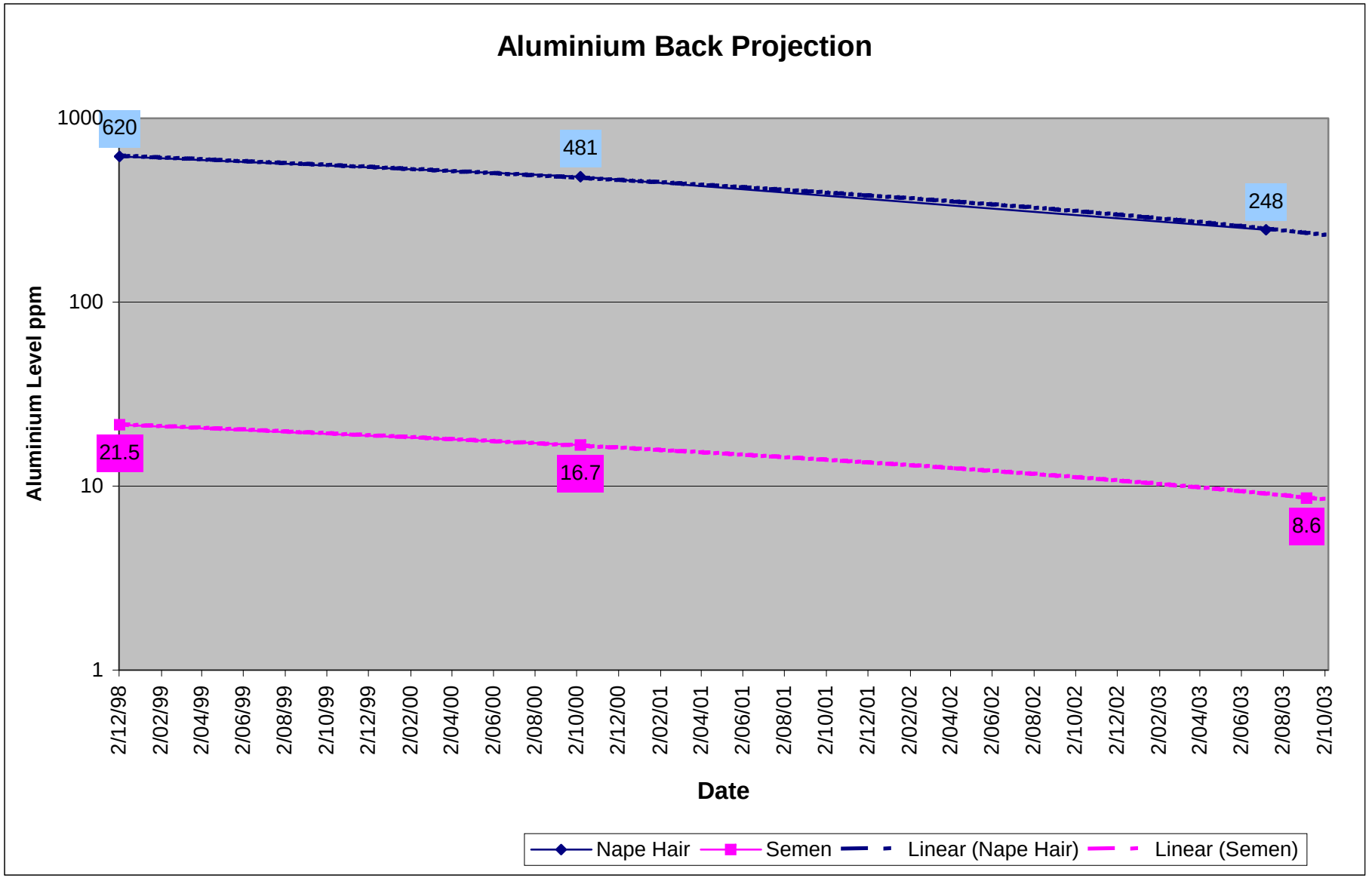
Peter Stewart.
8th March 2005.

Attached:

- Aluminium Tissue Test Results, 9th September 2004
- Aluminium Back Projection, to 1998

- Bibliography/References


Aluminium Test Results 9th September, 2004
Aluminium Level ppm
300
250
200
150
100
50
0
7/07/03
21/07/03
4/08/03
18/08/03
1/09/03
15/09/03
29/09/03
13/10/03
27/10/03
10/11/03
24/11/03
8/12/03
22/12/03
5/01/04
19/01/04
2/02/04
16/02/04
1/03/04
15/03/04
29/03/04
12/04/04
26/04/04
10/05/04
24/05/04
7/06/04
21/06/04
5/07/04
19/07/04
2/08/04
16/08/04
Date
Nape Hair
Foot Skin
Toenail
Fingernail
U'arm Hair
Poly. (Foot Skin)
Poly. (Nape Hair)
Expon. (Fingernail)
Expon. (Toenail)
Expon. (U'arm Hair)


Aluminium Back Projection
Aluminium Level ppm
1000
100
10
1
620
481
248
21.5
16.7
8.6
2/12/98
2/02/99
2/04/99
2/06/99
2/08/99
2/10/99
2/12/99
2/02/00
2/04/00
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2/12/02
2/02/03
2/04/03
2/06/03
2/08/03
2/10/03
Date
Nape Hair
Semen
Linear (Nape Hair)
Linear (Semen)

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2. Glutathione as a treatment for male infertility.
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   Romano, Santucci, Marrone, Gabriele, Necozione, Valenti, Francavilla, and Francavilla. Human Reproduction, vol 13, no 3, pp2115-2121, 1998.
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   Fuping Zhang, Li Yang, Shuping Bi, Jian Liu, Feng Liu, Xianlong Wang, Xiaodi Yang, Ning Gan, Yu Tsing, Jun Hu, Hongzhao Liu, and Tainming Yang. *Journal of Inorganic Chemistry*, 87, 2001, 105-113.
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DRAFT December 2012

Good Morning Khandu
Please add the attached “Hyperlinked References” to the case study, which I have submitted earlier.
Thank you.
Regards, Peter Stewart.

Aluminium Toxicity References (Hyperlinked)

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High serum Al & acute encephalopathy in a patient with ARF	Pubmed=1915506
Al from tissues causes sepsis, neurological dysfunction, & mortality	Pubmed=3208458
Increased IL-1 converting enzyme expression & activity in AD	Pubmed=10374748
AlOH induces Th2 associated IL-4 and IL-5 production	Pubmed=10586035
A study of the immunology of chronic fatigue syndrome	Pubmed=9576011
Cell mediated immune response in chronic liver diseases	Pubmed=9914713
CFS; clinical condition associated with immune activation	Pubmed=1679864
CFS research. Definition & medical outcome assessment	Pubmed=1322076
Absorption and disposition of Aluminium in the rat	Pubmed=3735104
Al ingestion alters behaviour & some neurochemicals in rats	Pubmed=8500814
Lipid composition & neuronal injury in primates after chronic Al exposure	Pubmed=9522055
Effects of Al on the progression of lead-induced nephropathy in rats	Pubmed=11140823
Al induced oxidative stress in rat brain; response to HEDTA & CA	Pubmed=12643979
Influence of Al on neurotoxicity of lead in adult male albino rats	Pubmed=15266904
Aluminium in AD; are we still at a crossroad?	Pubmed=15666086
Blood oxidative stress status in patients with macrophagic myofasciitis	Pubmed=15511609
A study of the dermal absorption of Al from antiperspirants using Al26	Pubmed=11267710
CNS disease in patients with macrophagic myofasciitis	Pubmed=11335699
Macrophagic myofasciitis lesions from vaccine derived AlOH in muscle	Pubmed=11522584
Macrophagic myofasciitis: a summary of Dr Gherardi's presentation	Pubmed=12184366

A 62 YO female with progressive muscular weakness	Pubmed=14997943
Nonalcoholic steatohepatitis: what we know in the new millenium	Pubmed=12425538
NAFLD in patients investigated for elevated liver enzymes	Pubmed=12560853
Serum leptin level a negative marker of hepatocyte damage in NAFLD	Pubmed=12768390
Spectrum of NAFLD associated with normal ALT levels	Pubmed=12774006
NASH and insulin resistance: interface between specialists	Pubmed=12836490
A natural history of NAFLD; a clinical histopathalogical study	Pubmed=14499785
Current biochemical studies of NAFLD & NASH; a new approach	Pubmed=14499793
Insulin resistance & ferritin as major determinants of NAFLD	Pubmed=14610526
Vitamin E & C treatment improves fibrosis in patients with NASH	Pubmed=14638353
No direct role for leptin in the pathogenesis of human NASH	Pubmed=14687831
Increased levels of hepatotoxic TNFa occur in ALD & NASH	Pubmed=14720457
NAFLD among patients with hypothalamic & pituitary dysfunction	Pubmed=15057893
NAFLD; a comprehensive review	Pubmed=15104027
Role of cytokine signaling suppressors in NASH in the mouse	Pubmed=15240880
Mechanisms of apoptosis induction in human SH	Pubmed=15330907
Epidemiology of nonalcoholic fatty liver disease	Pubmed=15331060
The clinical features, diagnosis, and natural history of NAFLD	Pubmed=15331061
NAFLD in individuals with severe obesity	Pubmed=15331062
NAFLD in the pediatric population	Pubmed=15331063
Mitochondria in NAFLD	Pubmed=15331066
TNF & its' potential role in insulin resistance and NAFLD	Pubmed=15331067
Lipid metabolism in hepatic steatosis	Pubmed=15331068
Histologic features and clinical correlations of NASH	Pubmed=15343508
A longitudinal study of repeat liver biopsies for NASH	Pubmed=15382171
Oxidative stress & depletion of LCPUFA's contribute to NAFLD	Pubmed=15454290
Elevated ALT may signify the presence of NAFLD	Pubmed=15492608
NAFLD is an early predictor of metabolic disorders	Pubmed=15505132
Non alcoholic fatty liver disease	Pubmed=15505593
The risk factors of fibrosis in NASH	Pubmed=15526543
NASH	Pubmed=15554595
NASH	Pubmed=15560051
NAFLD; a review	Pubmed=15625647
Progress in understanding the pathogenesis of NAFLD	Pubmed=15619243
Gastric mucosal calcinosis from Al based therapy	Pubmed=8447508
Complexation of aluminium with DNA (calf thymus)	Pubmed=3559548
Polynucleotide cross-linking by Aluminium	Pubmed=2560791
Comparison of DNA adducts from exposures & various human tissues	Pubmed=8319665
Treatment of thymic lobes with Al provoked T cell apoptosis	Pubmed=7621860
A study of calf thymus DNA complexation with Al and Ga cations	Pubmed=8723774
Spectroscopic & voltammetric study on binding of Al to DNA	Pubmed=12137370
Aluminium inhibition of hexokinase	Pubmed=4114525
The relaxing effect of Al & La on gastric smooth muscle in vitro	Pubmed=4715218
Al affects the gastro-intestinal smooth muscle via multiple sites	Pubmed=1156030
The dialysis encephalopathy syndrome. Possible Al intoxication.	Pubmed=1244532
Evidence of aluminium accumulation in renal failure	Pubmed=549003
Dementia, renal failure, and brain aluminium	Pubmed=434672
Metabolic balance of aluminium studied in six men	Pubmed=476923
Immunologic & nonimmunologic activation of macrophages	Pubmed=7391601
Metabolism and toxicity of aluminium in renal failure	Pubmed=7395774
Histoenzymatic study of the effects of Al phosphate on gastric mucosa	Pubmed=7243581
Al-containing dense deposits in the glomerular basement membrane	Pubmed=7072638

Hepatic Al accumulation in children on total parenteral nutrition	Pubmed=6438295
Hyperparathyroidism and bone Al deposits may coexist	Pubmed=3830569
Aluminium in tissues	Pubmed=3915959
Effects of hyperparathyroidism & Al toxicity on bone scan in HD patients	Pubmed=3991531
Al in precipitates of alveolocapillary basement membranes of uremic P	Pubmed=4050886
Pseudohyperparathyroidism syndrome assoc with Al intoxication & RF	Pubmed=4014298
Depressed erythroid progenitor cell activity in Al OL mice	Pubmed=8807624
Chronic Al intoxication in rats; dose dependent morphological changes	Pubmed=9500123
Al accumulation & neurotoxicity in mice after LT feeding with Al/citrate	Pubmed=8412742
Effect of Al on mice wrt infection; immune supression	Pubmed=12474775
Alzheimer's dementia and the Aluminium hypothesis	Pubmed=3412205
Effect of Al ind AD like condition on oxidative energy in mitochondria	Pubmed=10656181
Paired helical filaments of AD share antigens with normal NF	Pubmed=6620982
Dialysis encephalopathy with fractures & muscle weakness	Pubmed=7218657
Animal model of Al-induced osteomalacia	Pubmed=6842959
Renal osteodystrophy in diabetic patients	Pubmed=8355457
The effect of DFO on tissue Al in Al-loaded rats with renal failure	Pubmed=2980801
Al accumulates in experimentally induced carcinomas of rats	Pubmed=7804026
Interaction b/w antacid & gastric mucosa using an "artificial stomach"	Pubmed=1469628
Al in bone of a case of renal osteodystrophy & dialysis encephalopathy	Pubmed=6716213
AlCl is cytotoxic to cultured V-79 fibroblasts in vitro	Pubmed=11844038
Mineral metabolism of rats fed varying amounts of Al compounds	Pubmed=4067662
Al toxicity is altered by diet and kidney function in rats	Pubmed=1941183
SR-B1 may take up oxidatively modified lipoproteins & AB-apoE	Pubmed=11959156
31P NMR spectroscopy of brain in aging and AD	Pubmed=3316499
High-field 19.6T NMR of aluminated brain tissue	Pubmed=15388089
Gut Al permeability & bone deposition with normal renal function	Pubmed=908869
The compartmentalisation & metabolism of Al in uremic rats	Pubmed=3973462
Neither serum Al or DFO chelation reflect skeletal aluminium	Pubmed=4067379
Development & reversibility of Al induced bone lesion in the rat	Pubmed=3794513
Diagnosis of Al-associated microcytic anaemia in dialysis patients	Pubmed=2909650
Effect of CA & Maltol on Al accumulation in rat brain and bone	Pubmed=8445293
Al in the CNS, liver, & kidney of rabbits with atherosclerosis	Pubmed=2340946
Al salts interfere with the absorption of nutrients from the gut	Pubmed=8473881
The binding of Al to protein & mineral components of bone & teeth	Pubmed=9397572
The bioavailability of Al in man, including Al-26, a review	Pubmed=15152306
Al uptake by the parathyroid glands	Pubmed=479346
Evidence for a toxic effect of Al on osteoblasts	Pubmed=3213621
The evolution of osteomalacia in the rat with acute Al toxicity	Pubmed=2816514
Effect of Al & Cd intake on antioxidant status in rat tissues	Pubmed=11673849
Al induces lysosome damage to liver, spleen, and kidneys of rats	Pubmed=3624785
Uremia, dialysis, & Al; Al occurs in all organs and tissues	Pubmed=9422488
Al induces alts. in cell immune response in a dose dependent manner	Pubmed=11562064
Al influences cytokine production & depresses CD4+ immune response	Pubmed=8301020
Al induces IL-18 and can facilitate Th2 induction	Pubmed=12562321
Antigen dose defines T helper & cytokine response in mice	Pubmed=10841947
Hair as an indicator of Al exposure in dialysis; comp to bone & plasma	Pubmed=2785480
Effects of the combined exposure to Al & ethanol in the rat	Pubmed=1673624
Camelford water incident: serial neuropsychological assessments	Pubmed=8094970
Comparative Al mobilising actions of several chelators in rats	Pubmed=7908811
Effects of Al & F on enzymes in the jejunal mucus membrane of rats	Pubmed=1328029
Effect of Al on rat brain is enhanced by calcium deficiency	Pubmed=6479848

Dietary Al and renal failure in the koala	Pubmed=15168340
A study of the cytotoxicity of Al to cultured brain cells	Pubmed=8150659
Intestinal perfusion of dietary Al: association with the mucosa	Pubmed=7926905
Microparticles in human gut associated lymphoid tissue contain Al	Pubmed=8675092
Immunotoxicity of Aluminium chloride	Pubmed=15318624
Effect of oral Al citrate on tissue distribution of Al	Pubmed=8349199
Tissue Al distribution in various age rats & changes in metabolism	Pubmed=7590531
AlF affects the structure & functions of cell membranes	Pubmed=15110101
Influence of organic acids on Al absorption & storage in rat tissues	Pubmed=8647304
The influence of complexing agents on the kinetics of Al in rats	Pubmed=11407750
The competition of Fe & Al for transferrin = Al deposition?	Pubmed=9208284
The Al induced acceleration of the aging process in rat hippocampus	Pubmed=11226739
CT mediastinal lymph nodes after Al exposition	Pubmed=11305576
Aluminium: impacts and disease	Pubmed=12123643
Water content of aluminium, dialysis dementia, and osteomalacia	Pubmed=3908086
Metabolism and possible health effects of aluminium	Pubmed=2940082
Iron and aluminium homeostasis in neural disorders	Pubmed=7843099
Effects of ingested Al on essential metals, esp. zinc, in treated mice	Pubmed=3428182
Bone Aluminium content in Alzheimer's disease	Pubmed=7606282
Al & chronic renal failure: sources, absorption, transport, & toxicity	Pubmed=2647415
Diagnosis & treatment of Al bone disease	Pubmed=8840316
Al chelation by 3-HP-4-ones in the rat demonstrated by microdialysis	Pubmed=8862748
Al deposits in the brain & affects the cholinergic neurotransmission	Pubmed=9116693
The causes, diagnosis, & treatment of Al toxicity in CRF patients	Pubmed=9275645
The promotion of Fe-induced generation of ROS in nerve tissue by Al	Pubmed=8962602
Al toxicity may contribute to immunological impairment in CRF	Pubmed=8671818
Effect of AlOH on Al tissue distribution & localisation in liver	Pubmed=8882343
Al accumulation in tissues of rats with compromised kidney function	Pubmed=8737962
Distribution of Al in different brain regions & organs of rat	Pubmed=8773759
Bile is an important route of elimination of ingested Al by rats	Pubmed=8658541
Hormone rel. diffs. in the effect of Al on Ca tspt in the small of the rat	Pubmed=8644129
Status & future concerns of clinical & env. Al toxicology	Pubmed=8772797
Systemic Al toxicity: effects on bone, hematopoietic tissue & kidney	Pubmed=8772804
Age dependent Al accumulation in the human aorta & cerebral artery	Pubmed=8971367
Effects of Al on mineral metabolism of rats IRT age	Pubmed=9148276
Al-sensitive degradation of AB 1-40 by murine & human intracellular enzymes	Pubmed=8947944
The effect of age on Al retention in rats	Pubmed=9020501
ST oral 3-HP-4-one inc. Al excretion & reverses Al toxicity in the rabbit	Pubmed=9029049
Mechanisms of Al absorption in rats	Pubmed=9129475
Analysis of intestinal absorption & storage of Al in uremic rats	Pubmed=9249771
Myelin is a preferred target of Al-mediated oxidative damage	Pubmed=9264541
Al metabolism in rats by Al26 isotope	Pubmed=9316614
Interactions of AB's with the BBB	Pubmed=9329690
Uremia, dialysis, and aluminium	Pubmed=9422488
Relative roles of intestinal absorption & dialysis fluid exposure in HDP	Pubmed=9430871
Aluminium, Alzheimer's disease, and bone fragility	Pubmed=9462346
Al decreases the Mg conc of SC & trab bone in rats	Pubmed=9600675
Action of Al-ATP on the isolated working rat heart	Pubmed=9629673
Uptake of Al & Ga into rat tissues & the influence of antibodies	Pubmed=9630424
Silicon reduces gastrointestinal absorption in rats	Pubmed=9651136
Membrane comp can influence the rate of Al3+ mediated lipid oxidation	Pubmed=9677347
A comparative study of several chelating agents in rats	Pubmed=9677621

A cluster of equine granulomatous enteritis cases and the link to Al	Pubmed=9778770
Effect of Al & Al citrate on blood & tissue Al levels in the rat	Pubmed=9823440
Effect of Al on K-induced contraction in ileal longitudinal smooth muscle	Pubmed=9827026
Effects of Al, Fe, Cr, & Y on rat intestinal smooth muscle in vitro	Pubmed=9845462
Metabolism of aluminium in rats	Pubmed=9863066
Effect of Al on SOD activity in adult rat brain	Pubmed=9877534
Vit E protects against oxidative injury stimulated by excess Al intake	Pubmed=9891850
Low serum Al in dialysis patients with increased bone Al levels	Pubmed=9725776
Variations in Al concentration of caprine, bovine, & human bone samples	Pubmed=10328341
Permeability of rat epithelium to inhaled aluminium	Pubmed=10409396
Al and other metals in bone of ESRF patients	Pubmed=10471660
Behavioural effects of Al in mice: influence of restraint stress	Pubmed=10494050
Effect of Al induced AD condition on oxidative energy of mitochondria	Pubmed=10656181
Myelination of the SC in mice exposed to aluminium	Pubmed=10693976
Evaluation of deferiprone protective effect on Al toxicity in mice	Pubmed=10931505
Al mobilisation by chelating agents in al loaded rats	Pubmed=10987213
Effects of Al comps on tissue distn. & concs. of essential elements	Pubmed=10752672
Al toxicokinetics: an updated minireview	Pubmed=11322172
Changes in mouse brain tissues after prolonged Al ingestion	Pubmed=11393311
Daignostic utility of serum Al & the DFO test	Pubmed=11464651
Effects of Al & DFO on essential elements in Al exposed animals	Pubmed=11757400
The role of trace elements in uraemic toxicity	Pubmed=11904350
The effect of pleurodosis caused by AlOH on lung/chest wall mechanics	Pubmed=11976897
Al induces DNA synthesis in human fibroblasts in vitro	Pubmed=12002655
Effect of LT Al feeding on tissue cholinesterases	Pubmed=12127022
Effect of Al on activity of mouse brain AChE	Pubmed=12372547
Melantonin & pinoline prevent Al induced lipid peroxidation in rat synaps	Pubmed=12755500
Al-induced pro-ox effect in rats: protective role of exogenous melantonin	Pubmed=12823611
A model of Al exposure & lipid peroxidation in rat brain	Pubmed=14716098
Effects of Al on phosphate metabolism in rats	Pubmed=15221202
Comp study of intestinal absorption of Al, Mg, Ni, & Pb in rats	Pubmed=15235150
Molecular exchange of metal ions & tissular calcium overload	Pubmed=15235153
Selective binding of sucralfate to mucosal resection-induced gastric ulcer	Pubmed=15458285
Antioxidant effects of VitE & Se on lipid peroxidation in Al loaded rats	Pubmed=15487771

Peter Stewart.
14th March 2005.

TOX/2012/38

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Mulgrave, 3170, Australia
Phone: 9560 3992
Fax: 9560 3911
26th March 2005

The Managing Editor,
Hong Kong Medical Journal,
By e-mail: hkmj@hkam.org.hk

Re: "Use of hair analysis in the diagnosis of heavy metal poisoning: report of three cases", HKMJ 2004; 10: 197-200.

Dear Sir,

Please allow me to correct some misconceptions that are expressed in this article:

- The level of heavy metals in blood or urine will only reflect a recent exposure since the liver, kidney, and spleen perform an effective filtration function,
- An overload of a metal entering the bloodstream will be sequestered into tissues and organs, including the brain. The body will excrete the metal with tissues as they grow-out, but will not re-metabolise it into blood without chelation,
- In all cases of a significantly abnormal reading that is being used as the basis of chelation therapy, the result should be validated by a retest,
- In Case One involving dyslexia, there is a low-level overload of twice the reference range. It is noted; "the patient did not receive any treatment and developed normally thereafter", confirms the overload grew-out naturally,
- In Case Two involving epilepsy or autism, the actual metal levels are not cited. It is noted; "The child showed no signs or symptoms suggestive of metal poisoning, and gave no history of exposure to heavy metals". Autism itself is a possible indicator of metal poisoning, and the first check for a 3-yo would be to perform the same test on the mother,
- In Case Three involving feeding problems and drowsiness, the actual metal levels are not cited. They may not have been the main problem,
- A real case study is attached and is self-explanatory. The problem would not have been diagnosed without hair analysis. I suggest that all practitioners consider hair analysis as a valuable tool if used correctly, and recognise that blood and urine analyses are not suitable screening tools for past metal exposures.

Yours sincerely,

Peter Stewart.

TOX/2012/38
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26th March 2005

Mr Khandu Mistry,
Administrative Secretary,
COT: Lowermoor Sub-group,
DOH London.

Re: COT Report References, Yoshinaga (1990), (two papers).

Dear Sir,

Please note our comments regarding the research articles referenced:

1. **Interrelationship between the concentrations of some elements in the organs of Japanese with special reference to selenium-heavy metal relationships, Yoshinaga *et al,* 1990.**
    - In the elemental concentrations between organs, there is generally good correlation, with the following predictable exceptions:
        i. Cadmium accumulates in the liver and kidneys.
        ii. Copper accumulates in the liver.
        iii. Iron accumulates in the liver, and to a lesser extent, the kidneys.
        iv. Selenium accumulates in the liver and kidneys,
        v. Mercury accumulates in the liver and kidneys,
        vi. Zinc accumulates in the liver and kidneys.
    - There were also some non-predictable exceptions:
        i. Calcium was lower than the RR in all tissues except the kidney,
        ii. Potassium readings are all about 20 times normal hair RR,
        iii. Sodium levels are all about 10 times normal hair RR,
        iv. Phosphorus levels are all about 15 times normal hair RR,
        v. Zinc was lower than the RR in all tissues except as noted above.
    - The study purports to identify correlations between the antioxidants Zn & Se, and the toxic elements Hg & Cd in human organs, particularly the liver & kidney, and achieves this. However, there were some unusual levels obtained for essential elements that warrant further investigation. The causes of the deaths were not given in this paper. The study does show there is a good correlation between element levels in different organs, including brain, & also accumulation in kidney & liver.
2. **Lack of significantly positive correlations between elemental concentrations in hair and in organs, Yoshinaga *et al,* 1990.**
    - Original statistical data appeared in the study in (1) above. Rib data was added from another study which was not reviewed,
    - Please note that individual readings were NOT supplied, and abnormal readings do not appear to have been eliminated or validated,
    - Magnesium hair readings are consistent with tissue readings,
    - Phosphorus readings are consistent with normal hair RR, (tissues high),
    - Calcium readings for tissues are in fact low. The hair statistical result is high, due probably to a max reading of 7 times normal. Otherwise there is probably reasonable correlation between the hair and tissue readings,

- Iron hair readings are consistent with tissue readings,
- Copper readings are consistent, with some distortion by a high value/s,
- Zinc in hair is normal, although low in all tissues,
- Selenium is consistent with a mean of all tissues,
- The method of statistical analysis is totally inappropriate for the purpose of the study. Abnormal readings do not appear to have been filtered out or validated. Correlation analysis should be performed on each series, and then condensed. The analysis for this study should be repeated using a more appropriate methodology,
- Hair samples were taken of distal hair and the length of the hair sample was NOT CONSTANT for all subjects. Normally hair length may vary from 10mm in males to 300mm+ in females. At a growth rate of about 3-5 mm/week, the time delay between hair sample and analysis could be anything from 1.0 week to 1.5 years, compared to a notional delay of 1.0 week for tissue samples,
- Even though the hair elemental readings are consistent with the tissue readings (except calcium and phosphorus), and the quoted hair reference ranges, there are abnormalities in the data.
- Due to the faulty hair sampling protocol, this study is **fatally flawed** and should be discounted as a reference. Reworking of the analysis is not possible from the data supplied.

Yours sincerely,

Peter Stewart.

COT Lowermoor Subgroup Report Response

1. Major Reference Source

- Appendix 16 includes the document:
  "Report On Toxicity Of Aluminium.
  An update of the 1997 WHO IPCS Report with emphasis on neurotoxicity."
  This will be referred to by the designation RTA.
  In the RTA, some of the conclusions detailed in the original document, which is WHO EHC-194, 1997, appear to have been edited out.

  In **EHC-194**, the toxicity of aluminium is clearly acknowledged in certain risk populations, and those populations are as follows:
  - Impaired renal function
  - Occupationally exposed
  - Premature infants

  The definition of Risk Population is therefore determined by **the probability of an accumulation**, not by any inherent differences in the biokinetics of the metal or the exposure situation.

  In addition, it is acknowledged in 1.10, 8.6, and 11.1 that aluminium causes the following serious conditions:
  - Encephalopathy / neurotoxicity
  - Vitamin-D resistant osteomalacia
  - Microcytic anaemia

- There is NO indication that aluminium does NOT follow the normal dose dependent relationship for a toxin, ranging from the NOEL, to acute & toxic iatrogenic exposure during dialysis, causing encephalopathy.

- There is every indication in the research that the effects of aluminium are linear within the effective band between the NOEL and the saturation level.

- Therefore, the requirement for demonstration of the toxic effects of aluminium in any person is **the validation of elevated aluminium levels**, which will be in tissues rather than serum for delayed testing scenarios.

2. Implications For Lowermoor

- The Lowermoor incident reflects a poor evidence profile in regard to primary levels contributing to the exposure. This arose due to the incorrect analysis of the initial water problem, its' short duration & high intensity, the distributed nature of the effects, the operation of line flushing, and other factors.

- Given that there are no reliable blood or urine results that focus on the peak exposure period, the measure of exposure must be tissue test results.

- So, for example, the neuropsychological tests very clearly indicate abnormal results in exposed persons, & the effect is MCI (Mild Cognitive Impairment), which is on the damage scale from zero to dialysis encephalopathy.

- Therefore, the conclusion in 1.14 that; "It is not anticipated that the increased exposure to aluminium would have caused, or would be expected to cause,

delayed or persistent harm to health in those who were adults or toddlers at the time of the incident…" is not consistent with the evidence.

- In addition, the statement in 1.22 that; "On the basis of the available data, it is not anticipated that the combination of metals which occurred as a result of the pollution incident would have caused or would be expected to cause delayed or persistent additive or synergistic effects" is unsupportable, in that no epidemiological studies have tested or reported the particular scenario, and the Report has ignored the recent studies by Bush *et al*., and Exley *et al*., that identify iron, zinc, copper, and aluminium as contributors to the amyloid cascade in neurodegeneration.

3. Effects Of The Poisoning

- There is a serious lack of hard data in regard to the reported symptoms. The symptoms reported included neuropsychological effects, joint pains and/or swelling, nail problems, cancer, thyroid disease, malaise, tiredness, exhaustion, dry thirst, sensitization, skin problems, gastro problems, arrhythmia, diabetes, & reproductive problems. For example, tiredness and exhaustion are symptoms of haemolytic anaemia caused by aluminium poisoning, yet no data has been presented of actual test results. All of the symptoms listed are linked by research and/or reports to aluminium.

- It is stated in 1.26 that "There is no indication from the toxicological data that the estimated exposures to the contaminants which occurred after the incident can cause effects on joints, and it is not possible to conclude that there is a causal relationship between the joint pains and/or swelling reported and exposure to the contaminants." It is an undisputable fact that metals cause lipid peroxidation, and in so doing, interfere with the essential fatty acid cascade. Since it is this cascade that is responsible for prostaglandin production and inflammation on a large scale (and not related to infection), the association between aluminium and joint pains is highly probable.

- Research clearly identifies a link between aluminium and skin problems, which would give rise to a condition similar to eczema or dermatitis in certain individuals.

- In regard to cancer, the size of the aluminium dose may be such that the metal will grow out before it can initiate a cancer. Brain deposits of aluminium are a different problem, they do not grow out; they cause neural degeneration and premature ageing, and most certainly contribute in some way to the onset of dementias.

- Aluminium is associated with a decrease in lymphocyte count. I do not recall any results of lymphocyte subset testing in the Report. The onset of lymphocytopenia represents a severely depressed immune condition with a high risk of infection, and hence the link to leukaemia. The statement that "the pollution incident did not cause an increased incidence of infection" does not seem consistent with the patient feedback.

- The neuropsychological test results from Altmann are consistent with the toxicity profile for aluminium as stated in WHO EHC-194, and summarized in 5.111 "The

authors concluded that the pattern of abnormalities seen was similar to findings they had previously described in "aluminium loaded but asymptomatic patients undergoing dialysis" (Altmann *et al*, 1989; Altmann, 1991). The authors also concluded "these studies suggest the participants responded to (their) tests, as a group, in a manner compatible with the presence of organic brain disease and in a way similar to dialysis patients exposed to aluminium".

4. Estimation Of The Contamination

- The water sampling conducted by SWWL was flawed and the data is therefore not reliable. As stated in the Report:
    - In the case of the 2-minute flush sample, most of the contaminants from the domestic pipework (copper, lead or zinc) would have been flushed away before the sample was taken. Therefore, the monitoring data for these metals for water from the cold tap may not have revealed the highest concentrations that occurred after the incident.
    - The exact location of the sampling sites was not supplied to us as South West Water Ltd consider that they cannot supply the names of customers at the address from which the sample was taken or information which could identify the customers

- The data from other sources may be more reliable than that from modeling. As per the Report: "The highest aluminium and sulphate concentrations were recorded in a sample collected at a farm in Helstone near St Teath at 5.00 am on 7 July 1988. This contained 620 mg aluminium/l and 4,500 mg sulphate/l. This sample also contained 9.0 mg zinc/l, the highest concentration recorded in the immediate post-incident period."

- In contrast, it was stated: "BVCs concluded: "Given that this is the only major anomaly with the modeling results, it raises serious doubt about the validity of the sample."" Modeling had predicted that the peak outlet concentration entering the network would be 325 ppm.
- In the case of reservoir mixing, the following scenario would apply, which is consistent with the Helstone test. The capacity of the treated water reservoir is approximately 2,300 cubic metres (m3), but it was believed at the time to be about 60% full, (1,380 m3 water). Therefore, if all the added aluminium sulphate were completely mixed into this volume, the maximum concentration in the reservoir would have been approximately **615 ppm** of aluminium and 3,300 mg sulphate/l. (Crowther Clayton Associates, 2003).

- The WHO utilizes 2 litres/day as the standard consumption of drinking water. At the concentration derived above, the daily dose of aluminium would have been 615 ppm X 2 Litres/day = 1230 mg.

- The WHO recommended maximum daily allowance of aluminium is 7 mg/kg bw/ day, applied to a notional 60 kg person, equates to a RDA of 420 mg/day. Consequently, **the RDA has been exceeded by a factor of 3**, reflecting the sequestration into tissues and the elevated tissue test results.

- The ATSDR Minimal Risk Level Publication (MRL) Jan 2003, cites the MRL for aluminium as 2.0 mg/kg/day with an endpoint noted as "neurological". The

exposure **has exceeded the MRL by a factor of 10** and has resulted in adverse neurological consequences for many of those exposed.

5. Tissue Test Results

- Tissue test results details are generally not available. For example:
  - Taylor (1990)
  - Ward (1989)
  - Ward (1990)
  - Cross (1990) b
  - Powell (1995), & does not indicate they tested for aluminium
  - Howard (1993), but positive correlation concluded
- Other tissue test results have shown positive, but source data is not provided:
  - Eastwood (1990) reported positive and discrete bone staining in exposed healthy individuals
  - McMillan (1993) reported that the stainable aluminium had disappeared within 18 months in normal individuals

- In the Report, section 5.40, it is stated: " Dr Newman also reported that he arranged for "approximately 435" patients to give samples of blood, hair, nails and/or saliva for analyses by Dr Neil Ward of the University of Surrey (see paragraph 5.150). These were tested for concentrations of metals (Newman, personal communication, 2002)." This data was available in summary from only.

- The results are discussed in the Homeopathic Report, Appendix 4. Aluminium, copper, and lead all show elevated levels reflecting the contamination exposure, and the sequestration of elements into tissues.

- More detailed results are available from the tissue testing of pigs at the piggery at Treburgett, and are reported in the document:

  "Multielement tissue status of sows exposed to aluminium in North Cornwall as a result of the Lowermoor Water Treatment Works Incident", N. I. Ward, Dept of Chemistry, Univ of Surrey.

- The data from these tests is comprehensive and includes a control group with comparative testing. As anticipated, the following results were characteristic:
  - Aluminium, copper, and lead were elevated in the kidneys and livers of the pigs
  - The elevated aluminium and copper levels were reflected in the hair results in a consistent manner
  - The elevated aluminium level was reflected in the bone results in a consistent manner
  - Corresponding reductions in iron and zinc are typical of active metal disturbances

- The conclusion drawn by Dr Ward is totally consistent with the data, and is as follows:

  "In conclusion, these results support the case that the chemical nature of the contaminated water supplies following the North Cornwall Lowermoor Water Treatment Works incident is indicative of being responsible for the increased Al, Cu, and Pb levels found in the various body organs and tissues of these

affected sows. Moreover, the toxicological effects of such metals in a mixed cocktail of metal species caused imbalances in other essential metals (Zn and Fe) and thereby induced the various behavioural problems and health disorders of those affected sows."

- The adverse health effects reported for the sows associated with the exposure were reported as:
    - Failed matings
    - Decreased litter size
    - Higher post-natal mortality
    - Reduced growth rate
    - Reduced feed conversion efficiency
    - Increased culling of sub-standard breeders

  These effects are consistent with a toxic exposure to metals. The continuation of effects after the event had passed is also indicative of the sequestration of metals into tissues extending their effects.

- The toxic effects of metals is reported in the following references:
    - **Aluminium, lead and cadmium concentrations in seminal plasma and spermatozoa, and semen quality in Finnish men.** Hovatta *et al.,* Human Reproduction, vol 13, no 1, pp115-119, 1998
    - **Glutathione as a treatment for male infertility**. D Stewart Irvine. Review of Reproduction, (1996), 1, 6-12.
    - **A prospective analysis of the accuracy of the TEST-yolk buffer enhanced hamster egg penetration test and acrosin activity in discriminating fertile from infertile males**. Romano *et al*., Human Reproduction, vol 13, no 3, pp2115-2121, 1998.
    - **Analysis of the impact of intracellular reactive oxygen species generation on the structural and functional integrity of human spermatozoa: lipid peroxidation, DNA fragmentation and effectiveness of antioxidants.** Twigg *et al.,* Human Reproduction, vol 13, no 6, pp1429-1436, 1998.
    - **A Case Study In Human Aluminium Toxicity,** Stewart, 2005, Personal communication.

6. Validity Of Tissue Testing

- In 5.161 it is stated: "The scientific literature indicates that metal concentrations in hair are not a good quantitative indicator of exposure to metals (Poon *et al*, 2004; ASTDR, 2001; Yoshinaga *et al* 1990)." This assertion has no basis in fact. The references were reviewed and commentary is as follows:
    - In **Poon (2004)**, there is an attempt to correlate blood, serum, & urine metal levels with hair analysis, and of course there is NO essential relationship. Metals are filtered from the biological fluids and will appear normal within 30 days. Chelation was offered but not implemented. There may have been some correlation after chelation.
    - In **Yoshinaga (1990)** (1), heavy metals were analysed in internal organs to determine relationships, and it was found that there was a correlation between Se & Zn and Hg & Cd, especially in the liver and kidney. (Precursor to study 2).

- In **Yoshinaga (1990)** (2), it was intended to demonstrate that there was a relationship between internal organ element level and hair element level. Unfortunately they utilized distal hair of different lengths and consequently different time periods. They acknowledge this potential error, and the conclusions are subsequently invalid.
- In **Wilhelm** *et al*., Scalp Hair as an Indicator of Aluminium Exposure; Comparison to Bone and Plasma, Human Toxicology, 1989, Jan, 8(1); 5-9, there appear to be methodological problems with the measurement of hair aluminium during dialysis and its' interpretation.

- Other studies, which did not contain flaws, are supportive of tissue testing in the recording of a historical exposure and that has resulted in sequestration, and they are briefly reviewed as follows:
  - **A Case Study In Human Aluminium Toxicity**, Stewart (2005), (personal communication), indicates that hair analysis is reliable, accurate, consistent over a long period, and has good correlation to physical symptoms.
  - **Hair Lead Levels in Young Children From The F.R.G**., Wilhelm *et al*., (1989), it was stated; "we conclude that by using standardized conditions hair analysis is a valuable screening method."
  - **German Environmental Survey 1990/92 GerES II**, clearly profiles metal levels in human hair and relates them to environmental contaminants.
  - **The Nail and Hair in Forensic Science**, Daniel *et al*., J Am Acad Dermatology, Vol 50, No 2.
  - **Heavy Metal Poisoning and Its' Laboratory Analysis**, Baldwin & Marshall, Ann Clin Biochem; 1999; 36; 267-300.
  - **Hair as a Biopsy Material; Trace Element Data on one Man Over Two Decades**; Klevay *et al*., European J of Clin Nutrition, 2004, 1-6.
  - **Determination of Metal Concentrations in Animal Hair by the ICP Method**, Chyla & Zyrnick; Biol Trace Elem Res, 2000, V75, 187-194.

7. Report Recommendations

- In the Recommendations For Further Research it is proposed that there should be additional and appropriate neuropsychological testing. However, the other recommendations call for monitoring of problems only. This is a totally inappropriate level of response given the evidence of health issues brought forward to the Committee and documented in their Report. The issues associated with aluminium indicate the following should occur:
  - Tissue testing of exposed persons
  - Testing for haemolytic anaemia & thyroid for fatigue cases
  - Testing for lymphocyte subsets for infection cases
  - Testing for lipids and antioxidant status for inflammation
  - Bone mineralization and iPTH testing for tissue test +ve cases
  - Kidney & liver function testing for fatigue/+ve tissue cases
  - Testing of motility, SPA & SCSA for +ve tissue cases or cases involving reproductive problems

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- **Further Toxicological Studies are definitely required, however, it can be categorically stated that any individual who has significantly elevated tissue levels of aluminium above the reference range of 16-18 ppm has received an overload, and any such individual who has symptoms of illness has received a dose which is above the NOAEL for that individual, all other factors being equal. Those individuals require testing, treatment, and support until it is demonstrated that the effects are fully discharged.**

P Stewart

20th April 2005.

Dear Frances,

We recently were sent a copy of an email from you to Mrs. Sigmund, in which you stated that the study was a 'toxicological risk assessment' and expressed the view that examining patients' medical records would have been of no assistance in understanding the effects of the Lowermoor Incident. This has now been quoted by Dr. Exley, and must be regarded as being in the public domain.

We strongly disagree with these statements. I have been carrying out risk assessments on a professional basis for over 20 years, as an Environmental Impact Assessor, and these frequently include health issues. A risk assessment, by definition, is a predictive exercise designed to avoid future adverse effects. It is possible to interpret part of the Terms of Reference of the LSG study to include this, in the sense that we were asked to assess whether or not future developments could be anticipated.

However, the primary purpose of this study was to collect all available evidence on what had happened to people up to the time of our study. That cannot be described as a risk assessment. Nor did we anticipate that the study would be regarded primarily as a toxicological assessment, and based almost entirely on the scientific literature. As we have often said, this was a unique incident and there has been continual political opposition to the full investigation, and therefore publication, of any comprehensive accounts of it. The literature will inevitably be a highly defective source of data on which to rely.

The first use of the term 'toxicological assessment' in the context of the Lowermoor Incident that we have been able to identify was by Mr.Michael Waring, on 24th August 1988, in a letter to Dr. Grainger at Truro. In this he expressed the remarkable view that the Lawrence Report was " a toxicological assessment" of the incident that was "to the point and, I believe, accurate". The Lawrence Report was **not** a toxicological assessment. Its primary purpose was to investigate the handling (or perhaps we should say, mishandling) of the incident at the works, and estimate roughly what levels of aluminium might have existed in the water supply as a result.

On both counts the Lawrence Report was defective but, in the case of his highly conservative estimate of the resultant pollution levels, our own analysis of the SWWA water quality data has revealed just how inaccurate his assessment was. Yet Mr. Waring's idiosyncratic assessment of the value of the Lawrence Report as a basis to assess the toxicological risks arising from the incident appears to have formed the basis for all subsequent dismissive views expressed by the DoH ever since.

This highly unreliable conclusion has been incorporated into the Executive Summary of the Draft Report. It was also leaked to the media two days before the official release of the Draft, attracting a substantial amount of concern both amongst the people of North Cornwall and the scientific community, and compromising the reputations of the members of the LSG.

Peter and I are extremely disturbed with sections of the statements contained in the Executive Summary that we believe to be inconsistent with the evidence collected by the LSG. We note with extreme concern that the absence of adequate evidence or scientific understanding is, in a number of cases, assumed to imply not only the absence of risk that a future adverse effect may occur, but worse, to suggest that

existing symptoms reported by the public are almost certainly unrelated to exposure to the contamination.

It is our position that the Lowermoor Incident must be treated as a serious industrial accident, and investigated as such. After any such incident, examination of those involved must be collected and evaluated, thoroughly and impartially. In the case of chemical accidents, this always requires full medical investigation of those exposed to contact. Since this crucial and elementary response has still not been complied with by the health sector, it is essential that this study should now take on this responsibility.

We have recently received a communication from Dr. Exley expressing similar concern,. He indicates that even now suitable medical investigations could be carried out to identify those who may still carry a body burden of aluminium. Any that do may have an increased risk of developing aluminium-related pathological conditions in the future. We therefore consider that the current Consultation Period must not be regarded as the end of the road for this investigation, and that further direct investigations of both the medical records and of the people exposed to the incident should now be instigated. Until all of the remaining evidence - or at least enough to provide a valid assessment - has been collected and weighed, the health effects of this industrial accident will remain unverified.

In the light of these recent developments, we have prepared a review of the Executive Summary. We have identified those - generally very short - sections of the statements included in it on which we have reservations, and provided our perspectives on how these may require amendment in the final version. These comments are designed to be helpful, not destructive, and we trust that they will be accepted in that spirit.

External criticism of the Draft is liable to be based purely on what is included within it. For scientific assessments this is to be expected. But in identifying any perceived defects in the Draft, public concern is liable to include criticism that the LSG has taken a particular approach to the study that is perhaps inadequate. We consider that it is impossible to understand the context of the study adequately without a clear knowledge of the historic and current political constraints that operate upon the work of the LSG. We have therefore included a section that provides at least some of the background to the incident, identifying defects in the past response from the health sector that have led to the appalling shortage of monitoring date that has been such an obstacle to the work of the LSG.

Finally, we feel that the recommendations provided in the Draft do not go nearly far enough. Sociological impacts of this incident affect the mental health and well-being of a substantial number of people within the local population that we represent. We are concerned that there is no recognition of the need for social action, in addition to purely medical, in dealing with the effects of this incident. We have therefore provided some additional suggestions about what needs to be learned from this incident and its mishandling, so that lessons for future responses may be learned.

Doug Cross and Peter Smith

Doug Cross, Forensic Ecologist

TOX/2012/38

A Review of the Executive Summary of the Lowermoor Sub-Group's Draft Report of February 2005.

Doug Cross and Peter Smith - Local Representatives
CoT - Lowermoor Sub-Group

3rd April 2005

Statement by the authors.

It is not common practice for members of a specialist group to issue a dissenting version of a consensual document produced by the group. Past reviews of the effects of the Lowermoor Incident have been controversial, and the present study aimed to correct the deficiencies of the earlier studies and shed new light upon the incident and its medical effects. In the circumstances, we feel that the LSG has provided a far better assessment of probable exposure levels than has ever been made before.

However, in our view, the wording of the Executive Summary of the Draft Report of the Lowermoor Sub-Group (LSG) of the Committee on Toxicity (CoT) provides unacceptably optimistic conclusions regarding the long-term medical effects of the incident of July 1988. Whilst these statements reflect to some degree the arguments set out in the main text of the Draft, they nevertheless suggest a degree of unanimity of opinion regarding the interpretation of the evidence available to the LSG that is unjustified.

In reviewing the evidence, too much reliance has been placed by the LSG on the toxicological literature in order to assess whether or not the effects of ill health reported by the people are consistent with published reports on the toxicology of the relevant substances. As a unique incident, no literature sources are entirely relevant, and only clinical assessment of those still affected by medical conditions that they associate with exposure can provide conclusive data on which a true assessment of the impacts of the incident can be made. The LSG has effectively ignored this source of data, refusing to examine medical records unless specifically provided by those giving personal evidence. Even then, no critical debate of the validity and implications of such records has been held in LSG meetings.

As a result, where caution is merited, dismissive reassurances are provided. Where lack of knowledge prevents viable scientific conclusions being drawn, optimistic value judgements are offered. Where inconvenient evidence emerges, it is discarded as unrepresentative of predicted impacts and values.

Discrepancy between the study objectives and the approach of the Department of Health.

We believe that the Executive Summary was compiled by the DoH Secretariat. It contains generalizations that are incompatible with evidence provided to the LSG, but that reflect the adamant refusal of the DoH to acknowledge that this incident could have had any serious medical repercussions.

The stubborn refusal of the DoH to admit that any serious toxic threat was implied by the incident has a long history. The first reference that we have been able to trace in which this attitude is evident is a letter dated 24th August 1988, from Mr. Michael Waring, Senior Medical Officer at the DoH. Writing to Dr. C R Grainger at The Cornwall and Isles of Scilly Health Authority, he refers to the Lawrence Report of August 1988 as " a toxicological assessment" of the incident that was "to the point and, I believe, accurate". At around this

time he circulated a letter to the residents of the area assuring them that no adverse medical effects were likely, yet he appears not to have cited any toxicological authority to support this claim.

In fact Dr. Lawrence's Report was nothing of the kind. It investigated the incident in its context as an industrial accident, and provided an initial rough but extremely inaccurate estimate of the extent of the level of contamination, based largely on what few records were available from SWWA water sample analyses. The approach adopted by Mr. Waring is consistent with official DoH policy regarding the incident, at that time and since.

In the new Draft, this attitude re-emerges. It repeatedly offers value-laden judgements that are inconsistent with a disinterested assessment of the evidence presented to the LSG, as does the Press Release issued by the Department of Health (DoH) two days before the release of the Draft. Both assure readers that adverse health effects are not expected to occur. This implies that it is possible to judge the relative probabilities of adverse effects developing or not. LSG members have repeatedly stated that there are often insufficient data, or too much scientific uncertainty, to allow them to make any realistic assessments of the level of probability of the risk of delayed effects developing.

Such optimism is inappropriate in the face of evidence provided to the LSG over the past three years. Indeed, members have felt it appropriate to recommend renewed studies of selected groups of people exposed to the incident, particularly with respect to neuropsychological conditions and the achievements and welfare of children exposed to the contamination. In our view, claiming that the LSG holds opinions that imply a higher level of certainty than the data justify misrepresents the views of members. It is mischievous and unacceptable.

What was the real objective of this study?

Within the last few days the reason for the dichotomy between the intent of the original Terms of Reference for the Sub-Group's study and the approach adopted by the Secretariat in the preparation of the Draft Report's Executive Summary has become clear. In response to an inquiry from a member of the public the Secretariat stated,

"The committee took at face value the information which members of the public told them about their health. Members did not consider that there was a need to confirm what they were told by looking at medical notes or by commissioning medical assessments, neither was this the purpose of the investigation. The assessment made by the committee was a toxicological risk assessment, in which the key questions were:

1. *what levels of exposure were individuals likely to have had to the contaminants and,*
2. *given what is known about the toxicity of the contaminants, were they likely to have caused harm to health at these exposures.*

Medical notes and clinical investigations of individuals claiming persistent ill-health would not have assisted in this risk assessment."

(DoH Secretariat, 17th March 2005)

This is the first indication that we have had that the DoH considered this study to be a toxicological risk assessment. The statement bears a disturbing similarity to the ill-founded view expressed by Waring about the Lawrence Report almost sixteen years ago. However, the quote above is the key to understanding the root of our concern about the general direction of this Draft. The first Term of Reference for the LSG's work requires it to examine

whether or not the incident "caused, or was likely to cause, delayed or persistent harm to human health". The key issue was not, was exposure "likely to have caused harm to health", but did it **actually** do so in the past, and on the basis of that evidence, is it possible that it will do so in the future?

A risk assessment is predictive; except as a purely academic exercise it cannot refer to a historic event. Motorway crashes are not investigated as 'risk assessments', but as analyses of facts, based on verifiable forensic evidence collected about the incident. The Lowermoor Incident should be analysed in exactly the same way, and this is clearly implied by the first Term of Reference. Also, in order to carry out a risk assessment of the possible future implications of a historic event, the assessors must always draw upon the actual historic case evidence to validate its conclusions. In examining the effects of a chemical accident such as this, it is imperative that the medical condition of those exposed to its should be investigated by expert toxicologists in the relevant fields.

To ignore such evidence, particularly when the event under examination was unique, is bad science. Attempting then draw unvalidated conclusions about a historic event, and then to rely on these to extrapolate predictions as to what additional risks the exposed population may face in the future, is irresponsible.

We agreed to work as Local Representatives on the assumption that the LSG would examine all of the available evidence, to decide if the reported health effects were real or not. We did not agree to make a theoretical and retrospective analysis of the risk of a toxicological response developing, especially one that relied heavily on published literature sources but not the actual medical records of the exposed population. This incident was unique, and detailed analysis of it has been continually suppressed. Inevitably, the scientific literature cannot be an adequate source of data on which to assess the its medical risks. Dr. Exley's recent criticism of the adequacy of the literature cited in the Draft implies that there may be additional sources of toxicological data so far unexamined by the LSG. This in no way invalidates our perspective that without data specifically from this or a very similar incident, examination of the literature must always be subservient to examination of the people themselves.

The Sub-Group has in fact assembled a considerable amount of valuable data within the constraints of the scope of its brief and the inordinate delay in commissioning such work. But since no medical records have been examined, the Sub-Group has been forced to rely upon anecdotal evidence from individuals who think that they have experienced some sort of adverse response to their exposure. This is not to decry the claims of those giving evidence - some, indeed, have been examined by reputable medical specialists, and can demonstrate real medical damage.

But in strict evidential terms, verbal evidence from unqualified victims of the incident is an unsatisfactory basis on which to attempt to establish the damage done and what may occur in future, just as eye-witness accounts from bystanders at an accident must be treated with caution. Without detailed examination of actual medical records this approach cannot develop a rigorous and scientifically valid analysis. The validity of at least some (and therefore by association, all) of the medical records of people claiming medical damage has apparently been compromised, and the practice of 'flagging' is so fraught with defects and inadequacies. We therefore have serious reservations about the validity of any analysis or risk assessment (whatever it may be called) that fails to look in critical detail at the only tangible evidence available.

The DoH's extraordinary claim that

"Medical notes and clinical investigations of individuals claiming persistent ill-health would not have assisted in this risk assessment."

immediately raises a pertinent question. If this is so, then why did the Sub-Group spend so much time reviewing, and in many cases, rejecting or downgrading the validity of published data from those few such sources of direct medical and biochemical evidence as were accessible? We read the papers published by Altmann, Miles, Exley, Ward, Macmillan, and many others, and even interviewed them personally. Now it appears that we may all have been simply wasting time, engaging in displacement activity. We therefore stress that without professional (and now, independent) examination of those who appear to exhibit symptoms of a toxic syndrome, we believe that it is impossible to assess adequately the full extent of the actual medical damage caused by this incident.

On such a flawed basis, no risk assessment (retrospective or otherwise) can ever be prepared. Without evidence, science is powerless. Without science, there can be no interpretation.

Understanding the background of the Lowermoor Incident

For a balanced understanding of the impacts of the Lowermoor Incident, an informed knowledge of its political context is essential. An appreciation of the extraordinarily sustained political opposition to investigating the health effects and long-term implications of exposure to the contaminated water reveals how dramatically the scope of the study and its effectiveness have been compromised. The following notes therefore summarize this neglected aspect of the background to the study, because they reveal the constraints that exist on the work of the LSG in its attempts to obtain sufficient medical data to be able to draw viable conclusions.

In the second part of this document, we have reviewed the statements contained in the Executive Summary of the Draft Report, in an effort to direct attention to possible alternative, and perhaps more balanced, interpretations and the existence of confounding evidence. The dissenting opinions expressed are entirely our own, and may not be taken to represent those of any other member of the LSG. Where we dissent, we have provided the substance of our arguments and the evidence on which we rely. All of this evidence has been presented to the LSG, yet some does not appear in the Draft, despite being central to the interpretation of evidence presented in the Draft Report. In this review, we take the opportunity to place this additional evidence in the public domain, in the hope that others better qualified than we may find it helpful in drawing their own conclusions, and that the Final Report will provide a more reliable assessment of the health effects of this incident.

Our personal backgrounds.

The authors of this document were appointed to the LSG in October 2001, on the recommendations of Mr. Paul Tyler, MP for North Cornwall, and Mr. Michael Meacher, who was at that time Minister for the Environment. Peter Smith is a Registered Homeopath, and has practiced with the people of North Cornwall for many years. He is Chairman of the Lowermoor Support Group, a local self-help body established to communicate information amongst those affected by the incident and to others who wished to be kept abreast of the progress of investigations. (This privately-run self-help group should not be confused with the CoT Lowermoor Sub-Group, which is referred to throughout this document as the LSG)

Douglas Cross is an independent professional Environmental Analyst and Forensic Ecologist, who was a resident of Camelford at the time of the Lowermoor Incident. He has extensive experience the investigation of environmental and public health issues in water supply and water pollution incidents in many countries. He acts as a Team Leader for International Development Agencies preparing Environmental Impact Assessments - i.e., predictive risk assessments - of a very wide range of developments.

Our role as members of the Lowermoor Sub-Group

Our role has been to provide local knowledge of the incident and facilitate contact between the LSG and the local community. Both of us had, and continue to acquire, detailed knowledge of the incident and of the effects experienced by some, but certainly not by all, local people. Our experience of unacceptable water quality in the area extends back many years, to well before the date of the incident itself (6th July 1988). After the event we both worked to salvage data that the public sector persistently ignored, and for many years we have continued to collect new information as it became available. We have no financial or other compromising interests in relation to this independent investigative work, or that could affect our position as members of the LSG.

General comments on the political constraints on the Draft Report.

The Draft Report of the LSG was published in February 2005. It is not the final version, but a consultation document that aims to provide a comprehensive interim review of all available data on the Lowermoor Incident that occurred on 6th July 1988. This study began in October 2001, and 19 meetings were held in London and two in Camelford. In addition, four visits to Camelford were made between July 2002 and October 2003 to collect evidence directly from local residents. Evidence was taken orally at both venues, as well as from written submissions, and additional research evidence came directly from some authors and from the literature. We provided data from our own records, as well as new evidence based on practical investigations during the course of the study.

Political obstruction and the scope of the LSG study.

The consequences of the politicization on all investigations related to the Lowermoor Incident have been dramatic, and it is impossible understand this incident fully without a clear understanding of the political dimension within which all subsequent officially sanctioned follow-up work has been rigidly controlled. Members of the Lowermoor Support Group (that is, the local group, not the CoT sub-group) have been aware throughout the entire life of this incident of covert attempts to block or subvert independent investigations of the incident and of its medical effects. Such misdirection has continued during the course of the LSG's study.

The obstruction of adequate (or indeed, of any) comprehensive medical investigation of the medical effects following the incident has made it impossible to obtain properly researched and analysed epidemiological or clinical data on the exposed population. The Draft Report makes no significant comment on the effect of this obstruction on the availability of relevant data. Yet many people were surprised and alarmed that the original remit for the LSG's scope of work, that would have permitted it to take evidence on the handling of the incident, was summarily removed immediately after news of the study was first announced to the public.

This is not irrelevant: even now, sixteen years after the incident, we hear that legal actions initiated by some of those affected by the incident are being subjected to pressure to desist.

The question arises, precisely what (or whose) interests are threatened by providing victims of this incident with the comprehensive medical attention and legal support that should have been available to them? We deplore these attempts to prevent the exposure of unethical political objectives and of professional incompetence, especially as it appears to take precedence over determining the extent of the very real medical problems that some people exposed to the incident still experience.

It is evident that these spoiling activities originated at a very high level within the Department of Health. But it is also clear that the water sector was implicated; water was a Government-managed asset, and the sector was under the control of the then Minister, Mr. Michael Howard. It was planned to privatize the industry 18 months later, and the chaotic response to the incident within the Water Authority undoubtedly alarmed many in Government. Pressure to carry out a covert damage limitation exercise appears to have become a Government priority, through concern that exposure of the startling incompetence underlying the incident would damage the commercial value of the Water Authority.

International implications - not just 'a minor local affair'.

The exposure of the defects in the management of the Lowermoor Water Treatment Works has played an important part in alerting the water industry world-wide to the potential hazards of accidents involving aluminium sulphate. Yet despite the publicity surrounding the Lowermoor Incident there have been many subsequent instances of accidents involving the loss of, or environmental pollution by, this substance, right up to the present day. Even in February this year, spilled aluminium sulphate was recycled through the water treatment process at a facility in the UK, instead of being treated as a toxic, acidic or hazardous waste as is required by law. No formal risk assessment in advance of the decision by management to recycle this waste back into the food chain has been reported to us.

The need to ensure that all of the lessons implicit in the incident are fully appreciated remains as urgent and important today as it was sixteen years ago. Only by gaining a full understanding of the political dimensions that underlay attempts to conceal the effects of the incident can the limitations, constraints and defects of the Draft Report be adequately appreciated.

Until this perception is countered and corrected by a balanced and truly authoritative Report, industry will continue to regard accidents involving this substance as trivial and of little concern. After all, if this 20 tonne spillage directly into a water supply system really was innocuous, then any lesser spillage to a less sensitive environment will inevitably be viewed as being of no real consequence.

Scope and limitations of the LSG study

The new Draft Report is the third official attempt to publish an account of the evidence about the health impacts of the 1988 incident. Unlike the original study group, headed by Prof Dame Barbara Clayton, the LSG has obtained access to a wide variety of formerly unavailable records, and has interviewed a far greater number of people with direct experience of the incident and its health effects. In consequence, the LSG has been able to

compile a larger database of evidence than has ever been available to researchers into this affair.

The LSG's Draft Report issued in February 2005 is a working document, and not a definitive final report. It is intended that this document should be used as the basis for constructive peer review, and to encourage those with relevant expertise and experience to provide comment and new insight into the evidence and its implications. The Draft has assembled mainly anecdotal evidence on the immediate (acute) medical effects of exposure and the subsequent (chronic) medical complaints that have been attributed to it by people who were present at the time of the incident. This forms the substance of the first of the two Terms of Reference for the LSG's work, and we consider that the LSG has carried out that obligation as well as can be expected under the circumstances.

Indeed, the LSG has widened the scope of studies on this incident into fields not considered by earlier studies. For example, it has recognised the significance of secondary contamination of the water supply within domestic plumbing systems, caused by the solution of additional metals in the highly acidic water. By commissioning a hydraulic model of the water treatment and distribution system, for the first time it has been possible to demonstrate the timing and approximate peak concentrations of the primary contaminants, both within the Lowermoor Works and in the distribution system close to the Works. Sadly, even in this important work discrepancies have been allowed to enter the calculations, compromising the value of the model. This is examined in detail at the end of the Review section of this document.

Reliable new sources of medical information have become available, providing analytical data on human tissues (blood, bone, nails, hair) and urine. The LSG has also had access to a limited number of investigative medical reports produced after the event by independent specialists, and scientific understanding of the toxicology of the main contaminants has developed considerably during the years following the incident. With these innovations, the new Draft is unquestionably the most comprehensive compilation of still accessible evidence, and provides an invaluable resource for further analysis and debate.

Inadequacies in the medical data that obstruct the LSG's investigation.

But against this, in the sixteen years following this serious industrial accident there has been no planned and comprehensive medical assessment of the entire population on which the LSG could draw for medical data and analysis. It is an unfortunate fact that those few clinical studies that have been carried out after the event provide no more than very limited and almost random hints of the more complex issues underlying the incident. A group such as the LSG should have been convened much closer to the time of the Incident, when far more contemporary data would have been easily available. On the other hand, it is also true to observe that with the passage of time long-term and delayed effects that were not evident closer to the time of the incident have now become evident.

We deplore the failure of the health sector to take seriously the need to carry out professional quality monitoring of the health of the community in the wake of what is officially known as Britain's worst water poisoning incident. It is tragic that the absence of a comprehensive health monitoring system means that once more there is a real risk that medical information on the conditions of some of those affected may still be lost.

Critical comment has been made regarding the apparently limited references cited in the Draft. The literature review examined scientific publications up to 2003. The shortness of the

list of cited references is due purely to considerations of space; many more documents and sources were examined than have been noted in the Draft. However, the Draft should not be seen as a definitive assessment of the literature on the toxicology of the various primary and secondary contaminants involved in this incident. For such detailed information the reader should rely on a systematic review of all of the relevant literature.

The system of 'flagging' medical records. The Clayton Committee recommended that the local health authority should set up a post-incident health monitoring system. The health authority appears to have interpreted this as requiring it to compile statistics on cancer rates and hospital discharge rates. It relies on the system known as 'flagging' medical records, which identifies individuals whose records are of interest to some research or monitoring programme within the Health Service. The objective is that the local health authority will be alerted when a flagged individual develops a particular condition.

This system is seriously compromised. At present it relies on hand-annotated medical records: there is no computer-based system that automatically identifies any cluster or trend of unusual medical conditions that might become evident in the flagged group of records relating to the exposed population. It appears that flagging does not guarantee that the records of the considerable number of people who have subsequently moved out of the area are always reported back to the health authority in Cornwall. Nor does it appear to apply to those infants that were unborn but in the womb at the time of the incident.

Failure to collect pathological samples on the decease of people exposed to the incident. Following a serious incident involving any potentially toxic chemical, whenever any person from the flagged populace has died, pathological tissue samples should be retained and examined specifically to determine whether or not the deceased reveals any evidence of medical conditions that might be related to the exposure. Since metal toxicity has been linked with progressive neurodegenerative conditions, this requirement is especially relevant to the Lowermoor Incident. Only detailed examination of appropriate brain and spinal chord preparations post mortem will reveal possible cryptic toxicological indicators of exposure to any or all of the contaminants experienced during this incident.

We understand that no such investigative pathological examinations have been carried out by the health authority. There is therefore no reliable diagnostic evidence based upon prepared tissue specimens (as opposed to symptomatic evidence) on the incidences of Alzheimer's Disease and other neurodegenerative conditions within defined groups of people known to have been exposed to the contaminated water. Yet within the local community, the risk of developing such conditions is now an issue of paramount concern and anxiety.

In short, there is no evidence of willingness within the health sector to set up adequate monitoring of the full range of conditions that might develop as a result of exposure to the pollution. The scope of what ineffective health monitoring has been carried out was apparently founded on the inadequate investigations and placatory assurances issued by the Clayton Committee, and by DoH advisors with no professional expertise in the relevant toxicological fields. Because of the prolonged delay in setting up the present study, the range of possible follow-up investigations that could now be recommended has been severely limited.

We consider that the public health sector policy relating to monitoring this incident has been based on incompetent initial advice by unqualified advisers, reluctance to investigate the incident by adopting the precautionary principle, and reliance on a less-than-adequate

selection of published data from largely superceded scientific research. It is surely obvious that since the incident was unprecedented, inevitably there can be no clear published guidance on which to identify a specific range of resultant medical conditions that might be relevant.

Rejecting evidence from 'unofficial' tissue samples. In contrast to this lax approach within the public sector, human and/or animal tissue samples were collected by a few concerned health professionals, and by both of us. In a number of cases these tissue samples provided data on metal contamination levels that were significantly different from established 'normal' ranges. Remarkably, the relevance of the animal data has been summarily dismissed by the health sector, with the argument that animal models are inappropriate when dealing with human populations. This attitude rejects at a stroke the validity of all animal experimentation in the fields of medical and pharmacological research.

A similar bias is evident in the Draft. The largely worthless water sample data collected from samples taken far too late in the incident by SWWA are provided in exhaustive detail. In stark contrast, Cross's unique and robust data on the effect of drinking contaminated water by pregnant sows, the subsequent declines in pre- and post-partem survival of the fetuses, and on the growth rates and food conversion rates of the surviving piglets, are given only limited and passing mention. The concerned reader is left to attempt to recover the original material from relatively inaccessible sources.

Dismissing evidence from 'self selected' patients. Privately organised surveys have been dismissed by the health sector as 'self selected', as if the only randomized surveys carried out by professional epidemiologists can provide evidence of serious medical damage within a population. In any instance of a disease or environmental contamination affecting more than a single person, the only way that the medical sector is likely to become aware of it is through those people who are affected selecting themselves to report the problem to their local health care professional. Cross and Newman's 1988 survey of acute symptoms was purely aimed at alerting the health sector to the existence of widespread and severe health problems in the local community, and its main findings were replicated by subsequent more formal studies. Yet this original study has been repeatedly dismissed on the grounds that its data originated from 'self selected' sources. The results of other clinical studies - even by professional specialists such as Altmann et al - were dismissed for the same spurious reason by the establishment. We have repeatedly argued that the victims of any serious accident are not identified by random epidemiological surveys; they either refer themselves for medical assistance or are identified at the scene of the accident by emergency service investigators. It is a matter of deep concern to us that the DoH ordered the emergency response team at Guy's Hospital Poisons Unit not to attend this accident, or to have any contact with the people of North Cornwall.

Applying inappropriate statistical analysis. The majority of those few attempts at data analysis that have been made by establishment health sector professionals have themselves been severely flawed by defects in both the methodology and the statistical interpretation. Some have obscured possibly significant adverse effects by diluting specific sub-groups with overwhelming numbers of irrelevant and largely unexposed 'controls' - people who were not exposed to the worst (or even any) of the contaminated water. The North Cornwall cancer incidence analysis adopted this defective approach. The attempt to discount the Camelford School leukaemia cluster by this means is another example of confounding data from a specific sub-group by including large numbers of non-members. We find unacceptable in this Draft Report the apparently summary dismissal of the significance of these cases with the

claim that they could have been caused by infection. In the text of the Draft Report this was offered as no more than a hypothesis about the possible cause of leukaemias in general. But it was then inflated to justify the suggestion that these three specific instances of childhood leukaemia in a single class were caused by infection. This is a wholly spurious inference in the absence of any corroborative evidence.

The case for a Lowermoor Syndrome.

Although many people exposed to the contamination did not experience long-term adverse medical effects, a small group does appear to exhibit a common group of health problems that developed immediately after the incident. These include the acknowledged joint pain and swelling and 'neuropsychological' complaints. Within this group, the latter condition is often referred to as memory loss or confusion, but in a number of cases clinical investigation has revealed the existence of some form of what is generally referred to as 'brain damage' – either cognitive dysfunction or actual physical changes within the tissues of some part of the brain that has been demonstrated in CT and MRI scans. The sufferers are adamant that the symptoms that they experienced immediately or very shortly after exposure led to them seeking medical assistance that eventually led to the discovery of this damage.

Other symptoms that are common to this group are the loss of finger and toe nails, persistent skin rashes that are highly resistant to any form of treatment, a continuing reduced ability to carry out tasks and occupations that they were previously able to do with ease, and difficulties with tasks such as writing and dealing with numbers.

The Draft effectively separates such symptoms and deals with them – often dismissively – as if they are entirely unrelated. We consider this to be a serious defect, and suggest instead that these associated symptoms should properly be regarded as constituting a distinct syndrome caused by severe exposure or individual susceptibility to the toxic insult that they all experienced.

It is therefore worth recording that in the course of the investigations by the North Cornwall Homoeopathic Project, the epidemiological data gathered by Smith et al on 70 individuals clearly delineated such a syndrome. This was so widely shared that continuing to gather repetitive data seemed superfluous at the time of the study. These data were unaccountably omitted from the Appendices of the Draft Report when it was released, and yet the same syndrome emerged as a distinct feature when the LSG interviewed sufferers – many of them unknown even to the NCHP – to the extent that a member of the LSG used the expression " … pattern recognition …" to describe the repeated description of the condition by local people giving their evidence.

Conclusion.

The shortcomings and defects in the database of available medical evidence on which the LSG has been able to draw for its analysis have had a severe effect on the ability of the members to reach robust conclusions. The misrepresentation introduced into the Draft by the opinions expressed in the Executive Summary confound unfortunate ignorance with unnecessary bias and confusion. The failure of the health sector to recognise the common symptoms of sufferers exhibiting what we refer to as the Lowermoor Syndrome is reinforced by the LSG's division of individual symptoms and considering them as if they are unrelated.

Review of the Executive Summary

In the following pages, original text taken directly from the Electronic Version of the Draft report is in italics; those specific sections to which our comments refer are underlined. Comments follow each extract in turn.

"Who received contaminated water and how long was the water supply contaminated after the pollution incident?"

***"1.10** With the exception of those locations for which monitoring data exist, it is not possible to determine whether any particular point on the Lowermoor distribution network did or did not receive contaminated water because of a large scale flushing exercise which was carried out by the water supplier at different points in the distribution network. The extent and severity of the contamination can only be determined by the analysis of samples of water taken at a particular vicinity and time. Sequential water quality data are not available to enable a description of the progress of the aluminium sulphate as it travelled through the distribution system."*

Comment This is unacceptable. First-hand reports of contaminated water at locations for which no formal monitoring was carried out cannot be discounted purely on the grounds that no analytical data exist. Certainly the 'extent and severity' – presumably this means concentration – of the contamination remains unknown, but in some cases it is possible to say if such contamination existed at such locations. It is also a matter of concern that in several cases where SWWA water samples were analysed by other fully accredited Public Health Laboratories, the level of contamination of the critical components aluminium and sulphate were often found to be higher in the analyses of the independent laboratory than was indicated by the results obtained by SWWA.

***"1.11** The period of contamination with high concentrations of contaminants was short. Both water quality data and modelling of the passage of aluminium in the trunk mains indicate that the concentrations of this metal in the water supply fell rapidly from a high, initial peak. However, thirty per cent of samples taken up to the end of 1988 and 6% in 1989 remained above the 1984 WHO Guideline Value for Drinking Water Quality for aluminium. This value was set to avoid deposits in the distribution system and discolouration of water, not because of a risk of adverse health effects above this concentration. Concentrations of copper and lead were high for approximately a week after the contamination incident and very few water samples exceeded the 1984 WHO Guideline Value for zinc."*

Comment This only applies in the trunk mains. In branches of the distribution system highly contaminated water remained for much longer, and many areas were not flushed for weeks or, in some cases, for up to 18 months after the incident. The reiteration of the mantra that the Aluminium (Al) standard is set, in effect, for aesthetic reasons and not because of any health implications, deliberately diverts attention from the real medical issue. When this standard was set there was very little appreciation that aluminium was a potential toxin – the implication of the repeated use

of this phrase is that there was no relevant health consideration, and not that if any did exist then it could be discounted. This is an example of the use of assertion instead of informed opinion to divert public concern away from the issues before the LSG.

In fact, three out of six studies have found a statistical link between Al concentrations in drinking water and the prevalence of Alzheimer's Disease – hardly a robust assertion of an absolute lack of association between the two. With such uncertainty, the efficacy of the current standard for Al in drinking water to protect health interests must be considered unproven, and is indeed the subject of official concern elsewhere.

The study has been totally unable to interpret information that reveals that water quality in the area served by Lowermoor has been unreliable for a long period before the 6th July 1988 incident. For example: -

- In 1967 a discharge of sludge from the Lowermoor Works into the Tregoodwell Stream in Camelford resulted in a substantial fish kill. As the result of the research by Cross, for over twenty years the water quality regulators in the South West had the only environmental standard in the UK for aluminium in freshwaters designed to protect fish life.

- During the summer of 1986 a number of ducklings at Helstone died after drinking tap water, and the owner received a substantial compensation payment from SWWA.

- On 28th June 1988, only eight days before the Lowermoor Incident, mains water fed into a new plastic-lined swimming pool in Helstone was so acidic that adolescents who jumped into the pool quickly left it again, complaining of a stinging sensation. Shortly after they developed sores and blisters over their bodies. Attempts to neutralise the acidity of this water failed when the supply of sodium bicarbonate proved inadequate.

- On the morning of 6th July 1988, some hours before the Lowermoor Incident itself, a young woman at Treveighan, just south of Camelford, experienced ulceration of the mouth when drinking a cup of coffee.

The implication is that there have been persistent and repeated failures over the years to manage the water treatment process at the Lowermoor Works. Lime pump failure alone cannot account for the extreme reduction of pH in the swimming pool at Helstone shortly before the main incident, and it seems probable that there has been over-dosing of aluminium sulphate at least sporadically on a number of occasions in the past. Where this coincided with a lime pump failure, sporadic incidents in which severely acidic water was released to the mains distribution system may account for these earlier instances of acid water delivery.

***"1.12** Water quality data on the contaminants arising from the flushing exercises indicated that the proportion of samples with concentrations of manganese above the relevant 1984 WHO Guideline Value increased in the month after the incident but fell markedly thereafter. <u>The proportion of iron samples exceeding the relevant 1984 WHO Guideline Value rose in the month after the incident and remained high to the end of 1990."</u>*

Comment High iron concentrations indicate that the old scale lining the pipes had been either dislodged or even dissolved by the acidic water. Following flushing, some oxidation of the exposed pipe metal would have occurred. In fact high iron concentrations were recorded for years after the event.

Irrespective of this, however, the toxicological implications of the high levels of **uranium** (up to 0.2 mg per metre length of pipe) reported by Powell et al have never been discussed. (Powell J J et al. Assessment of toxic metal exposure following the Camelford Water Pollution Incident: evidence of acute mobilization of lead into drinking water. *Analyst March 1995 Vol. 120: 793-8)* Although this paper was quoted by the LSG in its discussions of the implications of the presence of lead, it entirely failed to pick up the authors' comment about the need to review the possible toxicological significance of the subsequent exposure of the population to uranium dissolved from pipe linings and entering the public water supply.

"On the basis of the toxicity data in the scientific literature and the estimated exposures, would the contaminants be expected to cause delayed or persistent harm to human health?"

***"1.13** This question is considered separately for each contaminant in Chapter 7. The possibility of additive or synergistic interactions is also addressed. For each contaminant, the implications for health of the worst case estimated intakes are considered in the context of the toxicological and epidemiological data in the scientific literature."*

Comment This is an unsatisfactory approach. Since this incident appears to have been unique, no relevant scientific studies have been published. The literature is therefore not a reliable source on which to base conclusions. The only relevant primary evidence is from the incident itself, yet this report tends to subjugate this in favour of published reports about largely non-comparable studies.

Also it is divisive – it attempts to deal with each of the individual symptoms reported by people providing evidence as if it were a discrete medical condition. The Draft in fact appears to use this technique to discount many of the symptoms revealed, and on that basis it then draws the overall conclusion that further medical developments are unlikely.

But in this incident, there has been a clear pattern of medical effects shared by a number of those who claim to have been most severely affected. In these cases, their symptoms should not be divided and examined individually. Instead, they should be treated collectively, as a discrete syndrome, and the evidence must be weighed in that context.

***"1.14** It is not anticipated that the increased exposure to aluminium would have caused, or would be expected to cause, delayed or persistent harm to health in those who were adults or toddlers at the time of the incident. However, the possibility of delayed or persistent harm to health, although unlikely, should be explored further in those who were bottle-fed infants at the time of the incident (i.e. below one year of age)."*

Comment This is a value laden statement, and should surely read 'Nothing in the literature appears to indicate this'. Such an optimistic conclusion is entirely unjustified. Similarly, 'although unlikely' implies an ability to judge the relative significance of potential for and against such damage. No adequate foundation for this claim exists.

As evidence of the uncertainty about this subject, recent literature records the development of amyloid plaque (a known associate of the symptoms of Alzheimer's Disease - AD) close to the cuproprotein alpha synuclein in the brain that changes its configuration in the presence of aluminium. Despite claims that aluminium does not enter the blood to any significant degree, there is clear clinically reliable evidence from the medical records of a number of Camelford residents that they experienced extremely high plasma aluminium concentrations for some time after the incident. Contrary to the assumed situation, therefore, exposure of their tissues to aluminium (and possibly even of brain tissue) was possible, and the rejection of any risks from this source is unjustified.

There may be genetic reasons for susceptibility to aluminium absorption in a small number of individuals. Nevertheless, there were thousands of potentially exposed people in the area at the time, and a possible consequential increase in the incidence of AD or other neurodegenerative or neurotoxic conditions cannot reliably be discounted. It has been reported that the cause of death of AD victims may be officially recorded as some other acute development. If this is correct, then the effectiveness of any local health authority monitoring system must be in serious doubt. This conclusion that any consequential effect is unlikely is therefore unsound.

Confining future monitoring to those who were below one year of age and bottle fed at the time is unjustified. Moreover, there is an additional reason for including all unweaned infants in future monitoring (see our notes on section 1.17, below)

Because there is doubt about the potential of an exposure in this incident to initiate delayed effects all exposed people who can be shown to have been at risk should be monitored. Those still exhibiting severe disability that they attribute to exposure should be offered comprehensive medical examination by specialists who are expert in aluminium (and related) toxicology. These experts must also be immune from Government pressures to conform with the politically-acceptable mantra that exposure presents little real health risk. This has prevented both local people and those who were visiting the area at the time from receiving the medical assistance to which they are entitled under the provisions of the Human Rights legislation.

***"1.15** The increased concentrations of copper in the first week or thereabouts after the incident probably contributed to acute, adverse gastrointestinal symptoms. <u>It is not anticipated that</u> they would have caused, or would be expected to cause, delayed or persistent harm to health."*

Comment Again, this is an unsafe conclusion. There are still no reliable data on the actual copper concentrations experienced by some people during this incident. Cross's research contribution to this debate suggests that investigations should be considering the possible effects of exposures of up to around 2000+ mg Cu/l, particularly through

dermal absorption, in some cases. The toxicological literature relating to copper is unhelpful in such instances, especially since uniquely in this case such exposure was contemporary with exposure to both aluminium and sulphuric acid. The significance of dermal exposure to some people with skin damaged by sunburn or other existing forms of dermatological damage should be reviewed.

Once again, genetic defects in some individuals may result in abnormalities of copper homeostasis that may make them vulnerable to changed configuration of the brain cuproprotein alpha synuclein. The role of encephalopathies associated with cuproproteins is now becoming of increasing research interest in neuropathological research, especially in the field of atypical Parkinsonism-like neurodegenerative conditions. As with aluminium, alpha synuclein responds to the presence of the cupric ion by changing its configuration; this time Lewy Bodies typical of the development of some forms of Parkinson's Disease are reported to form close to the site of the reconfigured cuproprotein.

These conditions are very often misdiagnosed, and the British (but not other) medical establishment is curiously unwilling to acknowledge the existence of similar neurodegenerative syndromes that result from exposure to copper, aluminium and pesticides, although they are well documented. These conditions can be induced at any time in the life of a person, but typically do not manifest until late middle age, when the damage becomes progressively intolerable, and ultimately fatal. With such a severe health risk, the adoption of the 'Precautionary Principle' is particularly vital.

Without far more effort being put into screening all deaths in the exposed population for all possible (even if improbable or rare) conditions, our understanding of the vulnerability of human populations to such exposures will remain uncertain. The total absence of detailed analysis of tissues taken from people exposed to the incident and who have subsequently died is unacceptable and demonstrates a serious procedural anomaly that merits investigation.

"1.16 The occasional high concentrations of zinc which occurred after the incident may have contributed to acute, adverse gastrointestinal symptoms. It is not anticipated that they would have caused, or would be expected to cause, delayed or persistent harm to health."

Comment This seems to be reasonable overall, but again, such apparent optimism is not justified. Dr. Neil Ward's work on induced zinc deficiency and metabolism seems to suggest otherwise.

***"1.17** It is unlikely that the potential brief period of increased exposure to lead, would have caused, or would be expected to cause, delayed or persistent harm to health. However, any additional exposure of young children to lead is undesirable and the possibility of a delayed or persistent effect should be explored further in those who were bottle-fed infants at the time of the incident, potentially the most highly exposed group. Inorganic lead compounds are considered to be possible carcinogens in humans and it is not possible to say whether the small additional exposures to lead will have any effect on cancer incidence.*

Comment The statement relies on the fact that the fluid intake of unweaned infants may be as much as 2.5 times as great for each unit of body weight as that of weaned

infants. It therefore identifies bottle-fed infants as having a far greater relative exposure level to contaminated water (assuming that their feeds were made with tap water and not bottled water). This is a dangerously flawed assumption.

In fact, Cross recorded a 20-fold increase in the concentration of aluminium in a batch of cows' milk made into ice cream at one dairy near Camelford, only four days after this incident. In considering the capacity of the body to eliminate absorbed aluminium, the Draft identifies no literature source in which lactation has been found to provide such a pathway; the observation of this additional excretory pathway is apparently unprecedented. In the absence of evidence to the contrary, it must be assumed that this indicates a potential new source of exposure for breast-fed infants.

Before this excretory pathway was revealed, the possibility of increased exposure to lead of breast-fed infants would have been rejected, yet it raises the question, can lead (and indeed, both primary and secondary pollutant metal ions involved in this incident) also be excreted via this pathway? If mothers were excreting even a part of any of their own excess lead (or other metal) body burden through lactation, the possible risk of the exposure of their infants to this indirect source of lead contamination is uncertain, but cannot be ignored.

Older children in the Camelford area have been reported to have shown signs of sudden and long-lasting changes in behaviour after the incident, a known effect of even very small increases in blood lead levels. Our comments above relating to possible repeated incidents of acidic water discharges from the Lowermoor Works also imply a need for a much more cautious approach to the claim that any long-term effect from exposure to lead is 'unlikely'

Although it is not directly related to the above extract, it is necessary to refer here to the apparently ignored part of one paper that has been used by LSG in its analysis of the implications of exposure to lead. In their paper on lead contamination extracted by the acid water passing through steel mains pipes, Powell at al (1995) noted that the acid would also have extracted uranium from the pipes, and that this represented a potential toxicological hazard to people drinking the water. It is unclear why the authors' recommendation that the presence of this uranium merited further investigation has not been noted by the LSG in the Draft Report. There is clearly a need to review the toxicological analysis of the health hazards of this entire incident using a much wider view than has been adopted so far.

***"1.18** It is not anticipated that concentrations of manganese after the incident would have caused, or would be expected to cause, delayed or persistent harm to health in those who were adults at the time of the incident, nor is it considered that there would be any substantial increased risk to health to those who were toddlers at the time. It is unlikely that there would have been any delayed or persistent harm to health in those who were bottle-fed infants but recommendations have been made for further monitoring of this age group."*

Comment Again, the suggestion is that only bottle-fed infants were at particular risk. Until it is certain that manganese is not excreted by lactation, it is unsafe to

assume that those infants that were breast-fed were at less risk than those that were bottle-fed.

There was recently a case of a rapidly fatal Parkinson-like neurodegenerative disease in Tintagel (the person concerned was not present during the 1988 incident) where it has been reported that manganese levels have been persistently extremely high recently. Manganese is reported to affect the same brain cuproprotein that is affected by aluminium and copper ions. If there is a link between any of these metals and Parkinson-like or other neurodegenerative conditions, it implies that older people in the population exposed in 1988 may also be at risk. Although evidence of this possible link between these metals, sensitive neuroproteins and neurodegenerative or neurotoxic conditions was presented to the LSG, no mention of this appears in the Draft.

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*"**1.19** It is not anticipated that the concentrations of iron in drinking water after the incident would have caused or would be expected to cause, delayed or persistent harm to health."*

Comment This is based on evidence from the literature sources consulted. However, some characteristics of iron metabolism are increasingly suspected of being a contributory factor in the pathology of Parkinson-like conditions, and this view needs to be subject to expert appraisal.

***"1.20** The sporadic high concentrations of sulphate in drinking water after the incident may have caused acute, adverse gastrointestinal symptoms. It is not anticipated that they would have caused, or would be expected to cause, delayed or persistent harm to health."*

Comment The toxicological implications of drinking what was effectively dilute sulphuric have not been adequately examined by the LSG. Sulphuric acid is a Schedule 2 Poison under the 1972 Poisons Act. Since pH of itself does not appear to be responsible for damage to the mouth mucosa (Cross's measurement of the pH of common consumable products -section 7.40, Fig.33 - is relevant here) there is clearly some unidentified mechanism, possibly but not inevitably associated with the sulphate ions) that the LSG has failed to identify to explain this very common symptom of drinking the highly acidic water (see below also)

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*"**1.21** There may have been an additive effect of those contaminants with the potential to cause adverse gastrointestinal effects and this may have led to an unpleasant, acute gastrointestinal response among those who drank the water, even when the concentration of individual contaminants alone was not high enough to cause such a response. The recorded pH values of the water after the incident were not low enough to cause the cases of sore throat and skin irritation which are reported. It may be that high concentrations of sulphate and metal salts rendered the water more irritant than would be anticipated from its pH alone."*

Comment Despite the apparently far higher acidity of many common foodstuffs, as demonstrated in the Draft, the mouth and throat ulceration reported by some of the people who drank the acidic water cannot be explained by any mechanism so far proposed. This apparently simple observation is very important. If the pH was not low enough to cause the widely reported mucosal damage (the existence and severity of which is supported by pathological evidence in animals that drank the water, and obtained from creditable veterinary sources) and nothing in the toxicological literature explains it, then the LSG has clearly failed to identify the cause of this damage All toxicological data on the absorption of metals from the gut relies on the assumption that the gut lining is intact, so this is clearly a serious obstruction to the diagnosis of risks and possible consequential effects.

Whilst most of the literature dealing with the toxicology of sulphuric acid deals with the corrosive effects of exposure to more concentrated solutions, the potential synergistic effects of consuming a combination of dilute sulphuric acid and the relevant metals in combination appears not to have been explored. The pH of water is an important ecotoxicological factor in metal pollution, as metals such as aluminium, copper, zinc and lead become much more toxic in acidic solution, and show a greatly enhanced toxicity to fish in waters with a pH value below 7. Although the human stomach may contain considerable amounts of hydrochloric acid, the combined effects of metals and sulphuric acid in the stomach and intestine cannot be assumed to be identical to that of the same metals when present in the naturally acidic human stomach.

***"1.22** On the basis of the available data, it is not anticipated that the combination of metals which occurred as a result of the pollution incident would have caused or would be expected to cause delayed or persistent additive or synergistic effects."*

Comment The literature does not provide adequate data on the possible synergistic effects of consuming two or more of the metals prominent in this study to draw such a conclusion, and is even less forthcoming when dealing with them in conjunction with the extraordinarily improbable presence of the registered poison, sulphuric acid. Nor is it safe to accept the general assumption that the low apparent toxicity of one of the metals present would have indicated relative immunity from toxic effects on all ages and conditions of people exposed to them. This is example of the failure of the compilers of the Executive Summary to recognise that lack of adequate understanding is an unsuitable basis for assuming a consequent lack of risk.

"Are the symptoms or illnesses reported by individuals or identified from epidemiological studies considered to have been caused by delayed or persistent effects of the contaminants?"

***"1.23** The symptoms reported as being health effects of the incident were identified using a number of sources. The types of chronic symptoms and diseases which were most commonly reported to the Subgroup in interviews with, and written submissions from, individuals fell into the categories of neuropsychological effects, joint pains and/or swelling, nail problems, cancer and thyroid disease. These were similar to those reported by 70 people in the report of a homeopathic project in 1992; this also reported malaise, tiredness and exhaustion, a dry thirst, and a sensitivity to tapwater.*

The Subgroup recognised that the incident was unique and that there was a recognizable pattern of symptoms and diagnoses among the individuals who provided personal evidence. It also recognised, through its contact with the local population, that many individuals were concerned and distressed about the possible health consequences of the incident in relation both to themselves and to the community as a whole."

Comment The clinical evidence of brain damage in a number of people who complained of severe mental effects, including memory loss, within a short period of exposure is well documented, and is not a matter of opinion. However, information on medical consultation rates is difficult to assess. It is important to bear in mind that many people have reported that they were very actively dissuaded from reporting later symptoms to their medical advisers – indeed, in some cases they claim to have been asked to leave surgeries when they attempted to consult on related issues. Some local GPs are reported to have been reluctant to examine or treat anyone presenting with conditions that they believed to have been caused by the incident. This may be attributed to a letter circulated from Mr. Michael Waring (DoH) in August 1988, in which he assured the local medical sector that adverse health effects were unlikely to occur.

Unlike the detailed critique of published medical papers provided in this Draft, there is no corresponding review of the apparent reliability of verbal evidence about the responses of the medical services following this incident, yet this is relevant to the evaluation of the reliability of data from all sources. Readers without relevant professional qualifications should not be left to infer any inconsistencies in recorded evidence, and thereby decide for themselves on the reliability of the evidence from their own reading of this complex and long document.

We also note with concern that the detailed epidemiological data (referred to above) of the North Cornwall Homoeopathic Project was omitted from the Draft Report, and appeared on the COT website only after its exclusion was drawn to the attention of the Secretariat.

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*"**1.24** In Chapter 8, each of the symptoms, or symptom groups and disease is considered in the context of the evidence relating to the potential exposures to the contaminants, their known toxic effects, and the results of studies on the exposed population. An assessment is made of the likelihood that the reported health effects were caused by the contaminants."*

Comment This is, of course, a fundamental point that must be made clear to the public. The toxic effects of the contaminants are 'known' only as far as past experimentation or study relates to the unique conditions of this incident, and as they are accordingly reported in the literature. Without an appropriate precedent, there can be no truly relevant literature!

As for the epidemiological studies described and assessed in the text, some that were carried out by medical specialists for lawyers acting for claimants have been wrongly denigrated as having been somehow tainted by association with vulgar financial interests. Other studies have been so obstructed by the inaction of the health sector that vital evidence has been irretrievably lost. And a few, as detailed below, have been

of such dubious quality that their value as scientific studies must be rejected. It is improper to draw benign conclusions from such sources – where a potentially lethal or debilitating outcome may be possible, adopting the 'Precautionary Principle' is an absolute requirement. When the balance of probability indicates a possible adverse outcome with a low safety factor, then that should be sufficient to trigger a 'fail safe' response. Adopting scientific aloofness and applying the test of 'beyond reasonable doubt' is a dangerous and unacceptable approach when the health, and even lives, of many may be at risk.

***"1.25** The estimated exposures to the contaminants are not considered to have been sufficient to cause neurotoxic effects in adults nor in those who were children at the time of the incident. However, the Subgroup was advised that the overall pattern of results in one of the neuropsychological studies indicated subtle effects in the individuals tested but that it was not possible to determine whether these effects were due to the contaminated water because of deficiencies in the design of these studies. Further work is recommended on this endpoint."*

Comment These statements are mutually exclusive. The advice indicated that such effects were possibly the result of exposure. There has been abundant evidence, both from the public and from some medical specialists, that in their opinion such a direct causative link was present, even if the mechanism was unclear. This is always the case when a new medical condition appears.

The scope of the neuropsychological effects that appear to have resulted from exposure therefore needs to be clearly defined. The issue is not what the literature does or does not suggest, but what actually appears to have occurred amongst some individuals following their exposure, especially amongst those that were entirely free of such problems immediately before the incident. Any initial monitoring needs to cover the widest possible field. It must attempt to identify those effects that are now amenable to further examination and relevant to the study. Then a targeted series of studies is required to quantify as far as possible the effects on specific conditions.

And also note that a neurodegenerative condition is not necessarily in itself an 'endpoint'. The real endpoint is death.

***"1.26** There is no indication <u>from the toxicological data</u> that the estimated exposures to the contaminants which occurred after the incident can cause effects on joints and <u>it is not possible to conclude that</u> there is a causal relationship between the joint pains and/or swelling reported and exposure to the contaminants. It should be borne in mind that arthritis and related problems occur commonly in the population. However, the Subgroup recognised that many individuals with whom they spoke were concerned about joint problems. Therefore, further work is recommended on this endpoint."*

Comment Joint pain, especially amongst males, was a prominent finding of the Cross and Newman study in 1988. In many cases people were virtually crippled for weeks or months afterwards. This paragraph suggests that any causal relationship is unlikely – the facts suggest otherwise. Whilst it may not be possible to conclude that there was a direct relationship <u>on the basis of evidence from the literature</u>, or to

discover if such an effect has previously been recorded, this does not mean that it is impossible – only that it may result from a novel form of exposure that has triggered a response that is more familiar as the result of other causes.

However, since such conditions naturally do develop and progress as people age, the possibility that any instances that have developed in people exposed can now be recognised as in some way distinct from those that arise naturally is likely now to be remote. Unless exposure caused some new and unique form of joint damage, it is unclear what the recommended study might be capable of revealing at such a late stage.

***"1.27** A consultant dermatologist who, two years after the incident, examined individuals suffering from nail and skin problems reported that the types of nail problems seen were common and that further metabolic investigation of the patients' nails was not required. There is no relevant information in the epidemiological studies nor from the toxicological data on possible effects of the contaminants on nails which can add to this opinion."*

Comment On the contrary, several specialist who also saw some of these patients stated that they could not identify the cause, but in some cases at least the cause was emphatically not fungal. Precisely what 'types of nail problems' did this specialist refer to? How typical were the instances from Camelford people, and how prevalent is non-fungal damage that arises spontaneously and persists for years after its appearance? Why did other specialists say that at least some of these examples were not typical of the usual nail loss conditions?

It is crucial to understand that people reporting nail loss also consistently presented with a number of other conditions that also appeared at the same time – in other words **they exhibited a syndrome**. It is completely unacceptable to discount one of the symptoms within such linked conditions – or indeed, several of them – by dividing them from the entire spectrum of medical conditions shared by the group. This dismissal of any significance in the shedding of finger and toenails is unjustified. At the time local doctors were baffled by the repeated loss of nails. The statement in section 1.27 indicates that this decision is based on the opinion of a single specialist, irrespective of the opinion of others, and is therefore unacceptable. Metals of Group 3A in the periodic table (including aluminium, gallium and thallium) are all known to concentrate in nails after exposure, and skin rashes and nail losses that were commonly reported within this group have been improperly dismissed in isolation.

***"1.28** The results of a study of cancer incidence and mortality between 1988 and 1998 in the population living in the area which received contaminated water provide no evidence of an increased overall cancer risk arising from the incident."*

Comment This dismissal of the significance of cancer rates is based on a single flawed study by Dr. Miles and his colleagues – why is this unreliable study used to support this claim? This study diluted the 'test' group with the entire remaining population of North Cornwall, a technique guaranteed to conceal all but the most overwhelming incidence of abnormal incidence of a pathological condition.

The significance of cancer in the community as a whole is in fact open to interpretation. We have heard a number of local people observe that people from the exposed area seem to have a much shorter life expectancy following initial diagnosis than people elsewhere, but no data have been made available on the survival time of cancer patients that might discredit such a claim. Purely recording mortality rates would not identify any other factor, such as more rapid lethality, that might be relevant.

Unfortunately, the issue of cancer is impossible to evaluate at present, not least because of the uncertainty of the accuracy of either reporting, record keeping or the effectiveness of 'flagging' patients' medical records. The incidence of lost medical records, or the reported alteration of some long after they have been written, has become an issue of concern for some patients. Such records should never be subject to revision, only addition, since this inevitably raises the question of possible attempts to conceal past medical mistakes. Although this Report makes no recommendation regarding the management of medical and other official records, it is our view that all relevant records should be sealed and never subjected to any form of retrospective editing. This would include both medical records, school attendance records, and any other relevant quantitative records that might be of value for statistical analysis after an incident such as this. Their relevance to future investigations needs to be preserved and they should be properly protected from disposal.

Where a large population is exposed to a potentially dangerous emergency, all previous and subsequent medical records should be accessible to official investigation; there is no reason that medical confidentiality should be used to frustrate the identification of population responses that could prove of vital importance in managing the consequential effects. Access to some information is also provided under the new Freedom of Information Act, as well as the Freedom of Access to Environmental Information Act. All relevant records must be preserved and made available to investigators, and medical records should not be exempt from such protection and accessibility.

***"1.29** The results of an investigation of a cluster of three cases of acute leukaemia in children attending a secondary school in the area which had received contaminated water were consistent with the hypothesis that the incidence of leukaemia could be affected by prior exposure to infectious agents. However, the study found that the pollution incident did not cause an increased incidence of infection."*

Comment The LSG did not find that the incident did not cause an increase in leukaemia cases. It has accepted the proposal that some cases of leukaemia may be caused by infection, but has then assumed that this provides sufficient evidence of causation in these cases. It has not established that infection was the only possible such explanation.

In fact, the three leukaemia cases occurred in a singe group of 22 children who were, apparently uniquely, given orange juice with their morning drink in the nursery class at Camelford on 7th July 1988. This forms a valid sub-group against which other matched groups should be compared. Diluting data from such a sub-group with the entire population of North Cornwall is not a scientifically or statistically valid way of

examining this cluster. A new analysis of the health of that sub-group of children must be made immediately.

The recommendation that more investigations of properly selected sub-groups should be undertaken to assess possible delayed or persistent neuropsychological effects of exposure should certainly include this sub-group, but should also identify the causes of chronic illness (including morbid psychological conditions) or death in any members of all identifiable sub-groups. During its investigations and case-taking, the North Cornwall Homoeopathic Project recorded many instances in which subjects needed four, five or six prescriptions of antibiotics to clear infections that would normally have been expected to responded to one or two prescriptions. This suggests lowered immunity and, by implication, an impaired ability to hold back the rate of growth of cancers.

***"1.30** There was no indication from the toxicological data on the contaminants of an adverse effect on the thyroid gland. Thyroid disease is common in the population and the cases reported are considered unlikely to be caused by exposure to the contaminants resulting from the incident."*

Comment This is again linear thinking in a situation that demands a lateral approach. Why should there be relevant literature upon which to draw when this incident was unique? The factual evidence of unusual thyroid conditions (Hashimoto's Disease, for example) is available; conclusions should be drawn from it, not 'reverse engineered' to discount the significance of the evidential data. The thyroid gland is sensitive to many forms of infection and toxic assault, and may provide an early warning of developing conditions.

***"1.31** The homeopathic report cited a sensitivity to tap water as a common finding after the incident but, from the symptoms described, this does not appear to be the immune condition termed "sensitisation". It has been proposed that it may be a manifestation of the non-immune condition termed "chemical sensitivity". It is difficult to assess the potential significance of this process in the context of the Lowermoor incident in view of the lack of firm mechanistic evidence and of robust means of diagnosis. Therefore, at this stage, it is not possible to draw conclusions or make recommendations in relation to these symptoms."*

Comment There is abundant research and case-study literature dealing with chemical sensitivity, yet the LSG has failed to treat this topic with the rigour that it deserves. The argument that there is no immunological basis for such 'sensitivity' has been proposed to dismiss conditions such as ME, yet that is now known to be a real and debilitating condition. Sensitisation to local tapwater has been shown to occur under 'double blind' (but non-clinical trial) conditions; its significance should be investigated, not simply dismissed as inexplicable. The 'lack of evidence and of a robust means of diagnosis' blamed for the LSG's inability to reach a conclusion is attributable to the failure of the health sector to consider the claim of sensitivity as a serious issue, despite the ease with which the claims of those reporting it could be tested.

***"1.32** The Subgroup was informed that there was a higher proportion of children with a statement of Special Educational Needs (SEN) ("Statements") in North Cornwall than in the rest of Cornwall and concern was expressed that this might be related to the pollution incident. The Subgroup received expert advice that the determination of children with SEN is influenced by many different factors and that no conclusions could be drawn from SEN figures about the long-term impact of the incident on health. In addition, a detailed investigation did not find there to be any consistent difference between the rates of children with Statements in the secondary school likely to have had the highest proportion of children from the affected area and those in other schools in Cornwall."*

Comment This statement indicates an unwillingness to examine the data critically. The Richmond Test methodology from the USA used as the measurement criterion has been widely dismissed by teachers throughout Britain as inappropriate for British children. Available SEN and Statement data for the area and schools are indeed useless, but this statement fails to highlight the fundamental flaw in relying on any formal method of classifying children that is based upon the economic provisions within the Education Sector for the support of children with special needs.

To put it bluntly, if an Education Authority does not provide adequate funds according to the actual demands of a community, then the rate of Statementing, and any other formal classification of less serious educational problems, will not reflect the needs of the children, but simply the depth of the purse of the School that they attend.

We have received authoritative reports that after the incident the proportion of children in some groups recognised by their teachers (but not by the funding provided) as having special educational needs was over 30%. This is not merely a 'higher proportion' but one far above the national upper average limit of around 16%. This needs to taken at face value and investigated in detail. If there was an increase in children having difficulties, then the possibility that this may be linked to the incident, even if only in a proportion of cases, is of extreme concern and requires immediate investigation.

We have seen from the modelling, and learned from members of the public giving evidence to the LSG, that exposure to extreme levels of contamination could differ wildly, even between adjacent dwellings. Consequently, the validity of attempting to use residential postal code groupings as the basis for broad-based epidemiological studies is highly questionable. In contrast, children attending the schools (particularly in Camelford where the model shows that exposure levels would have been at their highest) can be identified as members of specific sub-groups, all of whom were subject to similar exposure risks. It is our contention that the achievements and behaviour of sub-groups of children who attended the schools should form the basis of detailed surveys into the possible long-term and delayed effects of the incident.

"Recommendations for further research"

A. Population Studies

Neuropsychological investigations

***"1.33** Further studies should be carried out to explore the neuropsychological status of those individuals who consumed the contaminated water. Expert advice will be required on both the design and conduct of a suitable study or studies. It is suggested that the following groups are investigated:*
• individuals who drank the water and have symptoms
• a matched sample of individuals who drank the water and are without symptoms
• a matched control group from another community where exposure did not occur."

Comment The school classes provide valid sub-groups, since their members were, in general, all in the same large premises at the relevant time of highest contamination within the same area of the water distribution network. If the attendance records still exist, then the actual presence of individuals could be established, and if possible these sub-groups should be identified from class attendance records. The use in the health authority studies of statistics based on subjects' residential post-codes is clearly invalid. This is further exacerbated by the model's indication that the worst affected area was around Camelford itself; many children travelled to the school from outside the worst affected area, but are more likely to have been exposed to similar levels of contamination through the drinking water fountains at the school, even if not at home.

"Investigations of the cognitive, behavioural and educational development of children"

***"1.34** Investigations should be carried out into the cognitive and educational development of individuals who were under 1 year of age at the time of the incident. Expert advice will be required on both the design and conduct of suitable studies."*

Comment We do not accept this; the medical history of ALL children present in the worst affected area must be investigated. There were and are alarming reports concerning some children's behavioral problems that cannot simply be ignored because they were not members of this highly selective age cohort. Extra non-teaching staff had to be recruited to help the teachers to control certain classes.

"Joint pains and/or swelling"

***"1.35** Routine health statistics cannot be used to monitor the prevalence of joint problems. It is recommended that, if feasible, a study should be carried out to assess whether the prevalence of joint pains and/or swelling in the population receiving contaminated water is higher than normal."*

Comment It is almost certainly now too late to carry out any such study. If initial pathological changes did occur after the incident, they may well now be obscured by the subsequent development of more conventional arthrosis at the damaged sites. We know that even the unique aluminium-rich surface layer shown in the bone biopsies disappeared after a year or so. Any persistent physical changes remaining now are likely to be indistinguishable from subsequent consequential or incidental changes.

"Monitoring of routine health statistics"

"1.36 *The monitoring of routine health statistics for the population potentially exposed to contaminated water after the Lowermoor pollution incident, recommended by the Lowermoor Incident Health Advisory Group (1991), should continue. The monitoring should include analysis of overall cancer incidence and mortality rates, and analysis of cancer subgroups. If possible, the assessment of the exposed population should be refined to take account of the fact that some areas experienced a higher level of contamination than others. If such a refinement is possible, it could also be applied retrospectively. It is suggested that monitoring is continued until 2008, twenty years after the incident, and that the burden of this work is removed from the local primary care trust and is, in future, carried out by an academic department familiar with the analysis of routine health statistics.*

Comment The LIHAG monitoring proposal was far too restricted, and credit for the original proposal should not be permitted. Had adequate professional monitoring been carried out, then the LSG might well have had far better evidence on which to base its conclusions now.

Given the history of public sector pressure on scientists and others wishing to investigate this incident, that an independent research body should undertake such studies should not be a recommendation but an absolute requirement. It must have no dependence on research funding from the health sector, and the termination date should not be set merely at 2008. The relevance of the effects of aluminium and copper on neuroproteins, for instance, may cause damage that becomes fatal only as the affected person ages. It is possible that some individuals who were young adults or even middle aged may eventually develop dangerous or lethal conditions in the next twenty five years, whilst those who were infants at the time may eventually develop fatal conditions only in their mid-fifties, as far ahead as 2040.

Since improper medical advice was issued to the public by both the Water Authority (" the water is bacteriologically pure!", and, "If you don't like the taste, mix it with orange juice!") and the health sector at the time of the incident, the possibility that the public prosecutor's office might wish to consider bringing charges of corporate manslaughter and/or medical negligence cannot be ruled out. There is no limitation on the time for bringing such a serious charge against a defendant, and in such eventuality the requirement for transparently independent forensic analysis of all data and relevant materials is central to the concept of justice.

"Toxicological studies"

"1.37 *The toxicological data on aluminium, although extensive, is insufficient to make a definitive hazard assessment. There is a need for further work on the toxicity of aluminium, including:*

• studies to identify No Observed Adverse Effect Levels for aluminum salts using both acute and chronic exposure and a range of salts of different bioavailabilities

• *mechanistic data on the neurotoxicity of aluminium and of its potential role in neurological disease and other disorders such as macrophagic myofasciitis*

• *further investigations of the bioavailability of aluminium in humans, including of the reasons for the reported interindividual variation."*

Comments This recommendation refers only to the toxicology of aluminium, and not to the possible synergistic effects of a cocktail of contaminants. We still do not (and probably never shall) know what substances might have been leached from the sludge layer within the contact tank at the time of the incident, or the implications of the possible mobilization of uranium proposed by Powell et al.

The refusal of the medical establishment to recognise the potential significance of animal data collected from the affected area as an 'early warning' tool is unacceptable.

Animal data provided new insights into unexpected mechanisms of toxicity that would have alerted health authorities to possible adverse effects in the human population. Examples include the previously unreported excretion pathway of aluminium in lactating cows; the altered metal status of pig tissues; changes in reproductive performance, reduced fetal and neonatal survival of pigs; and the death of livestock forced to drink the worst of the contaminated water. All of these indicated at least a potential risk to human consumers, yet were entirely rejected by both the veterinary specialist employed by SWWA and by those (both lay and medically trained) dealing with requests for advice on the possible hazards and medical consequences of exposure.

No other pathological materials were either available or could ethically have been secured, yet the potential significance of this animal evidence has been repeatedly dismissed.

We suggest that further work on the response of common livestock and their suitability as early warning models for possible effects in humans is merited; they represent important sources of reference data and tissue preparations in investigation future chemical accidents.

The failure of the Health sector to carry out post mortem examinations on people from the exposed area who have subsequently died, with the specific aim of determining whether or not their death was related to the incident, is astonishing. Until exposure can be conclusively proven not to have been responsible for human fatalities, all such incidents should be subjected to detailed investigations to discover the scope of any pathological or biochemical changes that may have occurred in those subjected to the incident.

The relationship between the metals – specifically aluminium, copper and manganese - involved in this incident and possible induced changes in neurological tissues and biochemical components is an emerging issue as a result of this incident. Research should be directed at more reliable identification of rare conditions that may result in unusual sensitivity to exposure to metals, as a marker to the epidemiological implications of chemical accidents in which large populations are exposed.

In particular, the incidence of Alzheimer's Disease, and of Parkinson-like neurodegenerative and neurotoxic conditions over long periods following such incidents may be of relevance to the Lowermoor Incident. Special attention needs to be paid to identifying appropriate methods of assessing the neurotoxicology of these metals, and the long-term implications of the response of critical brain components to

them. The possibility that rare genetic conditions may be implicated in the expression of neurological conditions, even if only in a small minority within a population, should be discussed.

"Future handling of similar incidents"

***"1.38** There have been considerable improvements in contingency arrangements for and the management of any future chemical incidents since 1988. However, it is noted that the following areas may require particular consideration in the management of a future incident of the type which occurred in Cornwall:*
• the early identification of populations which may need to be monitored in any later epidemiological studies
• rapid, widespread dissemination of clear and accurate advice. Individuals should be informed about what has happened, the likely consequences and any action they may need to take as promptly as possible. An information point, such as an enquiry line or drop-in centre, should be set up and should continue to operate for some time after the incident so that individuals can seek advice on new concerns if and when they arise
• if the exposed population includes a large number of transient individuals e.g. holiday makers who are in the area temporarily at the time of the incident, consideration must be given as to how to identify this population for inclusion in any future monitoring programme
• consideration of the effect of contamination upon the intake of chemical species from food when there are either direct or indirect routes for the contamination of food."

Comment What improvements have been made? An internationally important effect of the initial analyses of the 1988 incident was that the Clayton Reports were adopted by the Food and Agricultural Organization of the United Nations as an example of how to manage a chemical spillage – which is simply bizarre! Unless the LSG is prepared to spell out these improvements, this section is worthless as a discussion of the lessons that should be learned about handling such incidents

Nor do these recommendations relate to the need for a more precautionary approach to the response to chemical spillages of aluminium sulphate. In a recent incident, approximately 1.3 tonnes of concentrated aluminium sulphate solution was spilled at South West Water plc's Pynes Water Treatment Works in Exeter, Devonshire. The spilled chemical, along with an unspecified amount of water from an underground channel below the works, was 'recovered and subjected to a cleaning process' – apparently the dead fish were strained out of the water into which it leaked – and then recycled through the water treatment process. There appears to have been no consideration of the possible chemical effects of the extremely acidic water on unspecified detritus lying in that channel before the dirty solution was recovered and recycled into the food chain. This illustrates the extent of the industry's failure to appreciate the potential hazards of spillages of this chemical, and brings into question whether in fact lessons have been learned.

Early identification of vulnerable populations depends on who decides what the threat is and on what basis the risk is assessed. This paragraph provides no proposal on how the manifest defects in the management of this incident could be replaced by an effective non-political response system. The Guy's Hospital Poisons Unit is reported to have been ordered not to send the emergency team promised immediately after the incident. The lack of authority of the Director of the Unit to over-riding any such political instructions in the face of Britain's worst ever water poisoning incident is alarming.

It suggests that political expediency may still play a controlling role in obstructing rapid and effective emergency responses, even when the situation presents an overwhelming need for coordinating public responses and essential data salvage in politically sensitive circumstances.

The role of the public in organizing community responses to such incidents is repeatedly ignored by Government. Even quite recently, the public sector's priority in any chemical emergency was still stated to be to get the public out of an incident area, by military force if necessary, and not to allow local people any autonomy or authority in dealing with local social issues. The Camelford Scientific Advisory Panel is now internationally cited as an example of how such incidents can draw on the skills of local people to deal with their problems. Communities do not always panic if provided with full information, and it is patronizing and insulting to try to exclude them and let 'authority' take over.

A crucial question is, who will pay for running the 'drop-in centre' proposed above? The reluctance of Government to ensure that emergency funds allocated for even overwhelmingly severe disasters such as the recent Asian tsunami actually get to the target locations does not provide confidence that adequate – or indeed, any – funding would be available in a purely local incident such as this.

It is now quite apparent that the Department of Health does not wish to incur the costs implicit in a full assessment of the health of the people of North Cornwall affected by this incident. Expecting it to pay for a 'drop-in centre' for an indefinite period is probably equally naïve. After the Lowermoor Incident, the need for this essential social function was recognised and carried out without funding or support by local people in North Cornwall from the start of the incident until the present day.

Indeed, the demand for this service has actually increased as time has passed. The social dimension of community support has expanded to include many aspects of personal and group interaction and advice that are entirely absent from any official recognition or acknowledgement. The cost of this, if supplied from the public purse, would have been substantial. Instead it has fallen on local people already financially compromised by the failure of the public sector to provide even the most basic of support services to the traumatized population.

Assessment of the hydraulic model of the Lowermoor Works.

As part of its technical investigations, the LSG commissioned a detailed hydraulic model of the Lowermoor Works and the main distribution pipelines and service reservoirs from Black and Veatch Consultants. The purpose was to provide estimates of the time and concentrations of contamination within the Works and as the polluted water travelled through the distribution network to consumers around North Cornwall.

It was hoped that the output from such a model would help to extrapolate from the information provided by the delayed and sporadic water sampling programme carried out by South West Water Authority (SWWA) following the incident. This would give the LSG a clearer over-all picture of the physical characteristics of the incident, and assist it in estimating the limits of exposure of the people in different parts of the area served by the distribution network.

What the model does well.

Previously, the only predictive model was a physical one constructed and tested by Philip Allen of SWWA in 1988-9. On running an aluminium sulphate solution through a wooden mock-up of the contact tank, he found that the outflow could have contained between 600 and 1200 mg Al/l. But no indication of the time scale, or of the concentrations of aluminium that might have appeared in the water distribution system to North Cornwall was possible.

An important benefit of the new model is that it is now possible for the first time to understand the time-scale and approximate levels of contamination that could have been associated with the water distribution system. It describes the rate of mixing of the aluminium sulphate within the treatment works, and identifies the time when it entered the main distribution system. It also predicts the contamination levels over a period of four days, both within the Lowermoor Treatment Works itself and in the water distribution system in at least the more proximal part of the area served by the Lowermoor Works.

The sampling operation initiated by SWWA immediately following the incident was hopelessly inadequate. It collected only ten water samples on the 7th July and twelve on the 8th July, yet the results of the analysis of samples taken on the 8th appear to form the basis of all subsequent official estimates of the health implications of the incident.

The new hydraulic model reveals that in fact the peak concentration of contaminated water passed through the system in the Camelford area many hours before any attempt was made by SWWA to collect samples from the distribution system. It establishes that virtually all of the data obtained by analyzing those water samples that were taken on the 7th and 8th July have very little practical value in helping to assess the level of contamination in the critical few hours immediately after the incident. The value of such delayed sampling in assessing the possible medical implications of the incident is low.

We know that shortly after the incident two SWWA chemists were sent to the Lowermoor Treatment Works on the evening of 6th July. They spent a large part of the night attempting to find out where the contamination came from, and "they were getting such high aluminium readings that they did not believe their instruments". Unfortunately, no trace of any records that they might have made has emerged, and no reference to such information was made in the Lawrence Report on the incident in 1988. Consequently, data that could have been used to calibrate the new model have been lost.

In the Lawrence Report, the claim was made that concentrations of aluminium of '4 mg/l, peaking at 40 mg/l' were experienced briefly, then fell rapidly. This was misleading, because it was applied only to data collected on the 8th of July and later – at least one and a half to two days after the incident, and long after the actual peak contamination levels had passed through the sections of the system close to Camelford. The very sparse SWWA water quality data relating to conditions on the 7th July indicated the existence of much higher levels of aluminium contamination, including one of 109 mg Al/l, but although briefly acknowledged by Lawrence,

remarkably these were not treated as particularly important, and were relegated to a more or less coincidental status.

Yet this assessment formed the foundation of the reassuring reports issued by Waring (DoH) and the Clayton Committee a short time after the incident, and repeated interminably whenever any discussion of the Lowermoor Incident takes place. They also provided the basis on which highly dismissive views on the potential adverse health effects were issued repeatedly by the health sector, right up to the present.

What the model fails to describe adequately

Even in the present study the validity of the new model has been compromised. There is a curious absence of data on the times that SWWA water samples were taken; none of the sample times are recorded in the material provided to the LSG. Yet one of us has direct practical experience in this work with SWWA's predecessor; we can state categorically that recording sampling times has always been a fundamental and routine requirement when collecting water samples for the SWWA.

As a result of this unexplained deletion, the new model has had to assume that all samples from this source were collected at mid-day on the date indicated; this is reflected by plotting of all sample data at the mid-day point for the relevant date in the plots shown in the Draft. The model itself concludes that

'99.9% of the aluminium would have been discharged from the (contact) tank after 10 hours',

'After 24 hours, 92% (by mass) of the aluminium sulphate . . . was predicted to have exited the clear water tank' (paragraphs 3.67 and 3.68).

Introducing an uncertainty factor of up to 12 hours in the times at which these downstream samples were taken seriously compromises capacity to calibrate the model using real-time field data derived from SWWA's own analytical data.

The peak contamination spread to relatively distant sections of the system within 48 hours, yet the SWWA sampling regime still failed to locate the peaks of contamination that were critical to understanding the incident, because their sampling programme started far too late.

Yet one crucial privately-collected water sample for which the exact time of sampling is available is treated by the modellers as if the time at which it was taken is also uncertain. If the information is correctly re-plotted in Fig 19 (page 68), it reveals that the May Rose Farm sample actually provides the only reliable indication of the concentration of aluminium present in those parts of the distribution system immediately following the incident. Yet the Draft contains the following extraordinary statement,

"*Given that this (sample containing 620 mg Al/l) is the only major anomaly with the modelling results, it raises serious doubt about the validity of the sample."* (para 3.70)

In other words, if the evidence does not fit the theory, then it is the evidence (and not the model) that must be wrong!

It is a fundamental principle of scientific methodology that the test of any theory is whether or not it reflects reality. When evidence indicates a theory is wrong, then it is the theory that should be modified, and not the evidence. Discarding inconvenient evidence is a serious scientific mischief, to be deplored in any research study. Yet at present this new model remains purely a theoretical construct; remarkably, it has not been calibrated.

It is wrongly assumed that since the predicted decay curves of the contamination in the system correspond well with almost all of SWWA analytical results from the 7th and 8th July onwards, they must therefore reflect reasonably well what happened during the crucial early hours after the incident. This is unacceptable – the data that it claims to reflect accurately were all obtained from samples that were taken after the initial slug of highly contaminated water had passed through much of the system. There are also questions regarding the accuracy of some of the analyses.

Evidence of a deep layer of solids in the contact tank.

In fact, data from the May Rose Farm sample could and should be used to calibrate the model. Two witnesses have provided written depositions to the LSG recording the evidence of sludge in the contact tank at the time of the incident. This was given by a former SWWA staff member to a formal meeting of the Lowermoor Incident Liaison Group in Camelford, a few months after the incident. He stated that the contact tank into which the aluminium sulphate was discharged did not in fact contain only treated water under chlorination at the time. Instead, there was

> **"about a metre of sludge on the bed of the contact tank, firm enough for a man to walk on it, and reaching up to the level of the outlet."**

As the modellers themselves state, the contact tank outlet was approximately one metre above the floor of the tank. Unfortunately, the schematic showing the components of the Lowermoor Treatment Works (Fig 2, page 30) shows every component except the contact tank in profile – the tank is in plan. So the vertical relationship between the outlet pipe and the floor of the tank is not evident in the plan of the treatment works.

The configuration of this 'high level outlet' has led to a remarkable dispute that has distorted the output of the model. This has resulted in an unduly optimistic (i.e., low) estimate of the concentration of aluminium sulphate that existed in the distributions system near to Camelford immediately after the incident. It also suggests that the estimate of the time when the peak concentration left the works and passed through the distribution system may require revision.

In his original analysis of the incident, Cross (1990) noted that the bottom of the contact tank, below the level of the outlet pipe, effectively formed a 'sump' into which the dense solution of aluminium sulphate would have flowed. This would have slowed the rate at which the solution mixed with the water flowing through the tank, and would have provided more time for SWWA to have realized that there was a serious water quality problem, and to have responded more effectively.

The existence of this 'sump' has been disputed, yet the original evidence came from a SWWA staff member who had inspected the contact tank shortly after the incident. A cross-sectional view at the location of the outlet of the contact tank shows that the

outlet was and remains, as the modellers state, a 'high-level outlet', approximately half way up the side wall of the tank. The lower half of the contact tank would therefore have acted as a partial trap for the extremely dense aluminium sulphate solution as it was poured into the contact tank. The claim of the eye-witness that there was a layer of sludge to a depth of one metre is therefore credible, especially in view of the mismanagement at the works for many years before this incident.

In fact the model, as it has been run so far, effectively recognizes the existence of Cross's 'sump effect'. He commented that the effect of sludge in this 'sump' would have been to accelerate the rate at which the pollutant could have passed out of the system. The model output therefore mirrors the potentially delayed release of the aluminium sulphate into the distribution system had the 'sump' been clear of sludge, as postulated by Cross fifteen years ago.

In the account of the modelling the level of the water recorded in the contact tank at the time of the incident indicated an apparent depth of 2.2m, (Appendix 10. Page 267), and the calculated flushing time and concentrations of contaminants entering and leaving the treated water tank downstream were based on the assumption that the true depth of water in the contact tank was indeed 2.2m.

But if the sludge was present as the witness records, then the actual volume of the tank would have been only around half of that assumed in the model. This would have accelerated the rate at which the contamination passed through the works, and increased the peak concentration of the contamination entering the distribution system. Indeed, without the 1 metre high differential between the bottom of the tank and the outlet, the aluminium sulphate solution would have begun to emerge from the contact tank within, at most, a few minutes of the start of the delivery. The model therefore does not provide a reliable description of the time and concentration of pollutant in the outflow in the presence of the sludge deposit filling half of the contact tank.

The so-called 'anomalous' May Rose Farm water sample contained 620 mg Al/l, and is the sole evidence of the actual concentration from the distribution mains. It would have been drawn into the long feeder pipe to the Farm during the late evening of the 6th July, exactly when the model indicates that the highest concentration of pollutant would have been in the region of Helstone. It was stored overnight in the pipes and tanks of the cottages on the Farm, and used to make morning drinks at 053o hrs on the 7th July.

The concentration of aluminium in this sample is probably close to the concentration that the model would have predicted had the modellers accepted that the sludge was indeed present as stated. Remarkably, they did not re-run the model to assess this alternative scenario, which would in effect have calibrated the model against the actual field evidence.

Instead they sought the views of a specialist in water treatment who was not present at the time, and indeed may never have visited the Lowermoor Works. They accepted his view that it was extremely improbable that there had been any such sludge in the tank, or that it could support the weight of a man. Instead he apparently proposed that those making the observation must have mistaken the sludge for some hypothetical and non-existent 'benching' in the contact tank.

Yet again, evidence has been rejected to support theory!

Conflicting evidence from 'blue baths' downstream of service reservoirs.

Had the model been adjusted and calibrated as described above, the result would almost certainly have been more consistent with other observations that also raise doubts about the validity of its predictions. For instance, Cross demonstrated to the LSDG that when water containing the secondary contaminant copper comes into contact with some soaps and detergents, it only develops the strong blue coloration reported by many local people if the copper concentration is in excess of 1000mg/l. The aluminium concentration in the polluted water wherever a 'blue bath' was reported would have had to be in the region of 285 mg/l or greater to release sufficient sulphuric acid to dissolve sufficient copper in the acidic water.

The model predicts that the maximum concentrations of aluminium in the outflow of the service reservoirs at Delabole and St Endellion would have been around 125 mg Al/l, yet 'blue baths' were reported downstream of these reservoirs. The concentrations predicted by the model for these reservoir outflows are too low to account for the development of this indicative phenomenon. Had the model accepted the presence of the sludge layer, the resultant output predictions for these reservoirs would have been consistent with the development of the 'blue bath' effects downstream.

Additional toxicological implications of the contamination of the water supply by sludge in the contact tank.

One important potential health implication of the presence of sludge in the contact tank is obscured by the modellers' decision to reject of the evidence of its presence. The extremely acidic water above this layer would have dissolved out some components of the sludge, and possibly more would have been physically mobilized and carried out into the distribution mains. But since there is no information about the composition of that sludge, this is likely to remain an unresolved additional factor in the toxicology of the incident.

In conclusion

- **The Local Representative Members of the LSG Study of the 1988 Lowermoor Incident disassociate themselves from the conclusions expressed in the Executive Summary of the Draft Report. The view that this study was a 'toxicological risk assessment' is inconsistent with the requirements of the Terms of Reference and inappropriate for the analysis of a historic event.**

- **The study has failed to investigate all of the available evidence, notably the past and present medical condition of those exposed to the contamination, and particularly of those who continue to exhibit residual medical conditions that they attribute to their exposure to it.**

- **We note with concern the re-emergence of the reassurance first issued by the DoH immediately after the incident, and repeated interminably for the past sixteen years, that the Lowermoor Incident is not expected to pose any substantial threat to human health. In the Executive Summary this outdated official mantra is resurrected, and implies discredit to the members of the Lowermoor Sub-Group who have worked hard to compile an invaluable summary of evidence on the actual effects of the incident.**

1. Methodology

Because the incident was unprecedented, the Draft's assessment of its medical risks relies far too heavily on inadequate literature sources

- It is not with the collection and recording of data in the Draft that we are concerned, but with the interpretations placed upon that evidence expressed in the Executive Summary. The statements are dependent on analyses based far too heavily on literature sources and previous experience for an understanding of an incident in which the health risks to a large and disparate population were in fact unprecedented and unpredictable.

- Failure examine personal medical records means that the conclusions presented in the Draft are not supported by any clinically robust assessment of the present conditions of those still claiming to be experiencing severe and long-lasting symptoms. Instead, reliance is placed upon a random and extremely small collection of often-controversial studies, most carried out over a decade before this present study.

2. The Lowermoor syndrome

Within a small group of severely affected people there is a distinct syndrome that should be investigated as such; the attempt by the Draft to deal with, and even dismiss, individual symptoms as if they were unrelated is inappropriate.

- In analyzing the individual symptoms, the Draft fails to consider the common set of symptoms exhibited by a small but clearly defined group of complainants as possibly constituting a novel clinical syndrome. We hold that the totality of the symptoms should be regarded as indicative of a toxic overload resulting from

exposure to an undefined mixture of contaminants; individual symptoms should be re-assessed within that framework, and not as if they are unrelated**.**

- The assistance of experts in aluminium (and other metal) toxicology, in relevant associated fields of neurotoxicology, and in the field of chemical sensitivity, should be sought to provide a more comprehensive view of the implications of this incident. Those people exhibiting this condition should receive immediately the detailed medical examination to which they are entitled.

3. Political obstruction

The study has been hamstrung by political obstruction and the failure of the health sector to collect reliable and comprehensive data on the medical effects of the incident. The origins of this obstruction and those responsible for implementing it should be identified and replaced by sound and accountable strategies for dealing with such chemical accidents and their health impacts in the future.

- Political obstruction to providing an effective medical response to the incident has been evident from July 1988, and remains an issue right through to the present day. At the time of the Incident the health sector accepted without question medically unjustified dismissals of the health risks, and has stifled all subsequent dissenting concern. Access to medical services and justice for those affected has been blocked repeatedly, resulting in an absence of critical data on the medical effects of exposure. This has seriously obstructed the work of the LSG in assessing the implications of the evidence now available to it.

- The death of a wide range of livestock forced to drink the contaminated water provided important early warnings of the potential severity of the toxicological risks to people. The persistent dismissal of the relevance of such data when applied to a large and diverse population implies a serious failing in professional standards. Those persons and policies responsible for obstructing the victims of this incident from having such assistance for the past sixteen years should be identified and removed, to ensure that such injustice is not repeated.

- The Terms of Reference for the LSG study were amended at the last moment by the DoH to prohibit the Sub-Group from identifying the reasons and persons responsible for the resultant absence of reliable medical data. The failure of the health sector to advise the population of health risks at the time, without political bias, or to engage subsequently in an adequate monitoring and support programme for those worst affected, must be investigated. Suitable safeguards should be put in place to ensure that such bias and deficiencies in responding to chemical emergencies do not occur again.

- The study's second Term of Reference requires the LSG to make recommendations on future management of the medical monitoring of, and research on, the incident. This is impossible without a full understanding of those aspects of its administrative and political history that are responsible for the present difficulties in collecting and interpreting data. The scope of the all such studies should be widened to allow them to identify and comment on the defects in the medical management of the incident and their consequences.

4. Public policy in chemical emergencies

The Draft recommends that drop-in centres should be funded in similar circumstances. But it fails to recognise that providing financial and administrative support to local self-help groups is the most socially- and cost-effective method of promoting strong liaisons between public sector authorities, the emergency services and the public exposed to such incidents. The Draft should contain more targeted proposals, which can be used as a model for responding to future chemical emergencies.

- In chemical emergencies of this type, provided that the immediate threats to public safety are abated, public involvement is the only reliable means whereby any resultant social problems may be managed and all possible relevant data salvaged without political interference.

- Public funds should be rapidly available for local self-help groups to establish community contact centres and support services, and liaise with public authorities and experts in the management and recording of such incidents and their impacts.

- The policies and procedures of local and national emergency response services engaged in dealing with such incidents should be reviewed, and robust structures developed to ensure that interference in the name of political expediency does not occur.

5. The hydraulic model of the Lowermoor Works and distribution system.

The hydraulic model used in the study should be revised to take into account evidence that has been improperly rejected in the initial computations. The strengths and limitations of the model to predict the full range of chemical implications in this incident should be identified and explained.

- Whilst providing valuable provisional insights into the timing of the peak contamination levels in and close to Camelford, and in some of the nearby communities, the refusal to recognise evidence on the presence of the sludge layer in the contact tank is unjustified. The model should be re-run to include this additional factor, and the output calibrated against the water quality data from the May Rose Farm water sample, to reflect recorded field conditions. The revised output data should then be re-interpreted.

- The output from the model reveals that the Water Authority failed to act quickly enough to secure crucial water quality data during the critical periods when peak contamination concentrations existed in the distribution mains, but the Draft fails to emphasize this important lesson.

- Contingency planning in industries in which chemical accidents could affect large areas should include provisions to identify critical nodes in the distribution system or potential contamination areas at which recording pollution monitors can be installed to provide adequate monitoring data on the timing and intensity of public exposure or risk. Such records should be securely sealed and in the possession of local public health authorities to ensure their availability id they become relevant to future incidents or emergent health issues.

- Contemporary analytical data show that secondary chemical reactions were taking place between the primary pollutants and materials deposited over a period within the distribution system itself. The model does not provide information on the potential extent of contamination of secondary pollutants such as copper, lead and manganese.

- There is a clear chemical relationship between the potential of primary contaminants (such as the sulphuric acid released in this incident) to engage in complex secondary chemical reactions. Within broad limits, the theoretical maxima of the resultant secondary contaminants resulting from acid attack of domestic plumbing systems are predictable. The model can be used to estimate some 'worst case' scenarios that could have developed in different parts of the distribution system. The Draft has failed to discuss the use of the calibrated model's predictions to expand understanding of potential 'downstream' effects in incidents of this type.

6. The importance of social factors in community perceptions of the incident.

The social disruption caused by the incident, and the social damage resulting from the defective response of the medical sector, are important but neglected aspects of this study. The Draft should provide a clearer analysis of how socio-economic factors regulated the individual risks of exposure to the contamination. It should also provide a section explaining the relevance of the sociological factors that subsequently affected community relations within the population after this incident.

- Severe social divisions have developed within the local community as a direct result of the public sector's refusal to provide full medical and social support to those needing them. The variability of individual exposure to the contaminated water, of individual medical responses to it, and of the failure of the health sector to be seen to be providing the best possible response and assistance to those in need of support have promoted conflicting entrenched beliefs about the incident. These could have been avoided with full and open admission by the medical establishment of its uncertainty of the implications of the incident.

- Socio-economic factors were highly relevant in affecting the exposure risks experienced in different locations. There were very significant discrepancies between the actual exposure to contaminants of individuals living in different properties, even if they were adjacent. Different houses had widely differing plumbing systems; some had direct cold water supply to their taps, others had header tanks in their lofts; domestic water pipes were of copper, cast iron, polyethylene, or even in some cases of lead. In some holiday accommodation properties, high occupancy rates resulted in large numbers of people using the toilet facilities before retiring on the evening of the 6th July.

- Consequently, some properties were more likely to draw off highly contaminated water passing through the nearby water mains, and become exposed to the worst of the contaminated water during the following morning. The salvage of the most critical water quality data of the entire incident from such a location emphasizes the importance of recognizing such factors in planning emergency and monitoring responses to such incidents.

- The public needs to be helped to understand how variables within the system, the characteristics of different properties, and the physiological differences between individuals can result in highly variable exposures and medical responses in homes and those living in them. This is necessary to reconcile disparate and by now entrenched views of the physical and chemical effects of the contamination in homes and on the health of the population.

- The Draft should provide a clearer explanation of the reasons for the variability in exposure risks, and provide more discussion on the sociological factors that led to the present climate of social division and public distrust of the health sector.

Doug Cross and Peter Smith
4th April 2005

Subgroup Report on the Lowermoor Water Pollution Incident

COMMENTS ON **THE DRAFT FOR CONSULTATION**

General Point

I am extremely disappointed about the general tone of the report. In particular it does not come across as being independent, rather it gives the impression of always looking for ways to dismiss any possibility that individuals were harmed or will be harmed by the poisoning of their drinking water with aluminium sulphate. In doing so the subgroup have chosen to ignore, or have not consulted, large swathes of the scientific literature and have chosen to cite a limited literature which supports what appears to be a prejudiced view of the event. I have no intention of directly addressing these inadequacies in this submission. The subgroup had more than enough time and opportunity to undertake a thorough review of the relevant literature and they have decided that such would not be necessary. Whilst ignorance cannot change the facts it can help to keep them under wraps and this seems to have been the motive of the subgroup. Why the Department of Health should prefer to protect the interests of South West Water Ltd and the Aluminium Federation and not the health of the residents of this area of the United Kingdom is an open question.

Specific Points

1. The subgroup has no expertise in any aspect of the environmental toxicology of aluminium. The subgroup made almost no attempt to remedy this situation. Review data pertaining to aluminium were provided by the Department of Health Toxicology Unit at Imperial College, London, and all documents were written by postgraduate and postdoctoral staff with no direct experience of any aspect of the environmental toxicology of aluminium. The subgroup took oral evidence on aluminium from only **four** individuals with recognised experience in the environmental toxicology of aluminium. I was one of these individuals (the others were, Dr P Altmann, Dr N Ward and Dr N Roberts) though I was not invited to give evidence I, independently, offered to give evidence. In addition to my oral evidence I also provided the subgroup with a written critique of their summary document LSG/02/29. I am not aware that any of the information which I supplied to the subgroup in either oral or written form has formed any part of the consultation document. In addition after I had given my oral evidence to the subgroup the chairman, Professor HF Woods, thanked me and asked if I would be prepared to assist the subgroup in preparing the sections of the final report which pertained to the environmental toxicology of aluminium. In spite of my agreement to this effect I have never had any further communication with Professor Woods. The latter was in spite of the fact that I was in regular contact with members of the secretariat, Mr George Kowalczyk and Mr Khandu Mistry for the entire duration of the enquiry.

2. The subgroup took oral evidence from the Aluminium Federation though they omitted to point out in the report (Appendix 3) that Professor J Edwardson and Professor N Priest gave their evidence on behalf of the Aluminium Federation. Thus the subgroup took oral evidence from **four** representatives of the Aluminium Industry and only **four** representatives of independent research on the environmental toxicology of aluminium. Why did the subgroup take evidence from an umbrella organisation the members of which are the worldwide aluminium industry which is an industry which does not fund any open research on the environmental toxicology of

aluminium ? I have been told that it is only a coincidence that the Aluminium Federation gave evidence at the meeting which immediately followed the meeting at which I gave evidence and that they wrote to the subgroup and requested to give evidence without any prior knowledge that I had given evidence. Documentary evidence to support this chain of events should be made forthcoming as an alternative scenario is that the subgroup or someone associated with the subgroup invited the Aluminium Federation to give evidence at this time. Is it a coincidence that the Department of Health are supporting financially at least one of the individuals, (Priest) who gave evidence on behalf of the Aluminium Federation? Professor N Priest was awarded a contract by the Department of Health to commence on the 1st of January 1999 valued at £95,600 to undertake studies on; 'The development of assays for the determination of aluminium body burden in man' (D.H. Reference No: PRIEST/CHEM/98/1). The individual in the Department of Health whom acted as signatory to this contract was **Miss FD Pollitt**, who also happens to be the **Scientific Secretary to the Lowermoor subgroup**. Questions should be asked as to why the Department of Health funded an individual (who they new to be a representative of the aluminium industry) who, to use Priest's own words as written in the contract, **'is a consultant to the International Aluminium Industry'** !? The award of this contract is even more of a scandal if one considers that even though the contract was due to expire on the 1st of November 2001 the Department of Health have, over three years later, still **not received a single published outcome from the research**. Indeed, upon my latest enquiry I was told that they were still awaiting the interim report on this project! It is also a scandal that this award to a consultant of the aluminium industry represents the **only grant of any kind awarded by government**, including all of the research councils, during the past **ten years** in the subject area of aluminium and human health.

3. The fact that the subgroup took evidence from the Aluminium Federation would not be so important if it was not that the evidence of Priest and Edwardson was the most heavily used and cited in the report. The scandalous misuse of the published literature is one of the subjects of a Letter to the Editor recently published in the British Medical Journal (http://bmj.bmjjournals.com/cgi/eletters/330/7486/275-a?ck=nck). A particular example of the subgroup's misuse of the published literature is their extensive reference to Priest (2004). Not only is this a review article written by a known representative of the Aluminium Federation it is also currently the subject of a Letter to the Editor of the Journal of Environmental Monitoring (the RSC journal which published the paper) concerning the author's **failure to disclose 'conflicts of interest'** relating to his connections with the international aluminium industry. This letter will appear in the May Issue of the journal. Clearly the connections between the Department of Health and the International Aluminium Industry run deep and the Lowermoor subgroup would have been better advised to have steered clear of such complications.

4. The terms of reference of the COT Lowermoor subgroup are outlined in section 2.9 on page 23 of their report;
"To advise on whether the exposure to chemicals resulting from the 1988 Lowermoor water pollution incident has caused, or is expected to cause, delayed or persistent harm to human health; and

To advise whether the existing programme of monitoring and research into the human health effects of the incident should be augmented and, if so, to make recommendations."

These terms of reference have since been amended to;
"The committee took at face value the information which members of the public told them about their health. Members did not consider that there was a need to confirm what they were told by looking at medical notes or by commissioning medical assessments, neither was this the purpose of the investigation. The assessment made by the committee was a toxicological risk assessment, in which the key questions were:

1. what levels of exposure were individuals likely to have had to the contaminants and,
2. given what is known about the toxicity of the contaminants, were they likely to have caused harm to health at these exposures.

Medical notes and clinical investigations of individuals claiming persistent ill-health would not have assisted in this risk assessment."

(Frances Pollitt, DoH Secretariat, 17th March 2005)

The above is a quotation which was made in response to a question concerning whether or not any assessment was made by the subgroup of the 'medical' evidence presented to the subgroup. Clearly, and perhaps in spite of the original terms of reference, **the subgroup were not concerned with the health of individuals who might have been affected by the incident**. They have interpreted the terms of reference such that their remit is limited to a risk assessment of an hypothetical exposure to the poisoned drinking water supply. In this respect the subgroup have employed a number of strategies to try to ascertain the concentration of aluminium to which individuals were exposed and how this concentration would have changed in the days which followed the incident. Much of the available information is contained in Chapter 3. One does not have to read far into this chapter to have the ignorance of the subgroup's knowledge of anything to do with aluminium confirmed. The equation in section 3.14 and its corresponding footnote are non-sensical and yet the information that the subgroup was trying to deliver by using them was absolutely critical to their understanding of this aspect of the poisoning event. This is not a good start! The majority of the water quality data were provided by the polluters, South West Water Ltd. However, in a similar approach to the medical evidence, the subgroup **have not appraised this data in any way**. Neither the haphazard manner in which water samples were collected for analysis (the sampling) nor the methods by which the samples were analysed have been critically appraised. These water quality data will form the major part of the subgroup's risk assessment analysis and yet the risks associated with using these potentially flawed data were not determined. From only a preliminary look at the data presented in Table 4 it is immediately obvious that there are some significant discrepancies between the concentrations recorded for aluminium and those recorded for sulphate. (Remember that the aluminium was 'dumped' into the water supply as a slurry of aluminium sulphate.) Conveniently, perhaps, there were not any measurements for sulphate on the days immediately following the incident. However, the measurements for sulphate and aluminium on the 9th of July (three days after the incident) revealed that the measurements given for aluminium are a **significant underestimate** of what would have been expected from the corresponding sulphate concentration. The aluminium concentrations offered by South West Water Ltd are between 2 and 5 times lower than would be predicted by

the sulphate concentration. Interestingly the trend of underestimating the aluminium concentration is only continued for water samples collected up to about the 18th of July at which time the measured aluminium concentrations (which are now all at or below 1 mg/L) are exactly as would be predicted by the corresponding sulphate concentrations. The significant discrepancies between the measured concentrations of aluminium and sulphate are mentioned in section 3.65 on page 63 of the report but they do not prevent the subgroup from continuing to use the South West Water Ltd data in their subsequent risk assessments. The data concerning aluminium concentrations in tapwater in the days following the poisoning of the potable supply are clearly flawed in such a way as to underestimate the degree to which individuals were exposed to aluminium. These data, though pretty horrendous in themselves, are clearly what South West Water Ltd. are willing to accept in the terms of their liability though they have little if any scientific credibility and this should have been clear to anyone with any relevant experience on the subgroup. In many ways the subgroup allowed themselves, conveniently, to be confused by the modelling exercise that they commissioned to try to determine how the aluminium which had been dumped into the treatment tank at Lowermoor would subsequently have been distributed throughout the potable water network. Like many models this can only be a very crude approximation of events and as such may be useful to present ideas concerning proportional differences in aluminium concentration throughout the network but it cannot be definitive in terms of the absolute concentrations of aluminium. Indeed, even the modellers have themselves questioned the validity of their model beyond twenty four hours after the event.
The subgroup would have been better served by asking the question as to whether the water quality analyses which were at their disposal were sufficiently reliable to be used in their subsequent risk assessments. Clearly, if they had asked this question they would have concluded that such reliable data were not available to them. No one could have argued with such a conclusion.

5. The subgroup then proceeded to use the flawed water quality data to calculate estimates of human exposure to aluminium and other contaminants in the poisoned tap waters. The futility of the textbook approach taken by the subgroup should be evident to anyone interested in the scientific method. The test of the validity of their approach should be that it would survive peer review and could be published in a quality scientific journal. Irrespective of the fact that the water quality data used in the calculations of exposure were flawed it is the simplistic manner in which these data were used that negates their scientific credibility. I am making my assessment as someone who reviews more than thirty scientific manuscripts each year and as someone who has reviewed for more than fifty different scientific journals. There are members of the subgroup who should have a similar experience in the scientific method and yet they have not questioned the approach taken in the draft report. Why ? For whatever reason, though it cannot have a scientific basis, it is clear that the subgroup believe that their estimates of 'worst-case exposures' for aluminium (and other contaminants) are of considerable value and consequently all of their recommendations concerning the likely impact of the pollution incident on human health have been based upon them. In my original submission to the subgroup I pointed out that it would be **impossible to determine human exposure without looking at the individuals that were exposed**. The subgroup ignored this advice as being outside of their remit, almost certainly their understanding and most probably their budget. Later on in these comments I shall include some brief recommendations on how we might, even today, be able to determine the likely exposure of the

Camelford residents to aluminium without relying upon unsatisfactory water quality analyses supplied by the polluters.

6. Chapter 5 of the draft report is at least useful in that it confirms that many individuals were exposed to the contaminated drinking water and suffered ill effects commensurate with aluminium poisoning. All of the ill effects reported in this chapter are documented in the scientific literature in respect of exposure to environmental aluminium. However, what is truly intriguing about the evidence presented in this chapter is not what was reported but indeed what was not reported. Human exposure to aluminium has been linked with a number of classes of disease, namely, (i) **neurodegenerative diseases** such as Alzheimer's disease, Parkinson's disease, motor neurone disease, multiple sclerosis, epilepsy..etc. (ii) **diseases of the bone and connective tissues** including osteomalacia, adynamic bone disease, arthritic conditions..etc. and (iii) **haematological conditions** including an anaemia and an insensitivity to erythropoetin. There is an exhaustive scientific and medical literature covering the role or putative role of aluminium in these diseases (including effects relating to the thyroid !!) and yet the Department of Health, as part of their ongoing assessment of the possible health effects of the incident funded studies looking at, pregnancy outcomes, child growth, mortality, cancer incidence including leukaemia, and educational achievements of schoolchildren. Why were these 'health outcomes' chosen whilst those which might have identified likely exposure to aluminium largely ignored. The subgroup were often dismissive of the accounts of the health of many individuals in that they were criticised for being 'self-reported'. It is quite clear that whoever was responsible for organising the so-called monitoring of the health of the local population following the poisoning was extremely careful in selecting health criteria which were least likely to indicate any influence of the known exposure to aluminium. Why were these criteria chosen ?

7. As someone who lives and breathes the subject of aluminium, whether through its chemistry with silicic acid to form hydroxyaluminosilicates and so to keep aluminium out of biota or its interaction with beta-amyloid in the aetiology of Alzheimer's disease, I am confident when I say that even with the best will in the world and all of the relevant scientific literature immediately available because of the lack of reliable data concerning individual exposure to aluminium during and following the poisoning incident it would not be possible to provide unequivocal answers relating to the terms of reference of the Lowermoor enquiry. We have two unassailable facts; (i) twenty tonnes of aluminium sulphate were dumped into the potable water supply of Camelford and the surrounding areas and (ii) many individuals were exposed to the poisoned water and many of those experienced illnesses following exposure. We now need to know how many people; (i) continued to suffer ill-effects; (ii) have developed ill-effects as a consequence of their exposure and (iii) are still to suffer ill-effects as a consequence of their exposure. This cannot be achieved in any other way than by looking at the affected population. A first approach would be to determine if the population had a higher than normal body burden of aluminium. We are all aware that our bodies have no requirement for aluminium and so the storage or retention of aluminium in our bodies can only put unneccessary stress upon our physiology. How this may manifest itself will be entirely dependent upon the individual and individual susceptibility to disease. Larger body burdens of aluminium will increase our individual susceptibility to an aluminium-related disease. Thus estimates of the body burden of aluminium will enable a better understanding of the likelihood that an individual has suffered, is suffering or will suffer in the future from an aluminium-

related illness. The determination of an estimate of the body burden of aluminium in an individual is not a trivial matter but there are a number of almost completely non-invasive techniques which could be used to achieve this. For example, it was announced at the **Sixth Keele Meeting on Aluminium** (26^{th} Feb – 2^{nd} March, 2005) that the aluminium content of bone, which is an important indicator of a prior exposure to aluminium, can now be measured without the requirement of a biopsy using in vivo neutron activation. The 'patient' will simply be asked to place their hand in small tube for a short period of time during which the hand will be bombarded with neutrons and data corresponding to the aluminium content of the bone will be collected. This is only one of a number of ways that we should be able to make useful estimates of the body burden of aluminium in individuals. These body burdens will be a first step in establishing that people absorbed and retained aluminium in their bodies following the pollution incident. Those individuals showing the highest burdens might then have their past, present and future health scrutinised more thoroughly to establish whether or not their health had been or was being impacted by their exposure to the poisoned drinking water. Only by such a human-based approach will any subgroup be able to make strong conclusions concerning the possible health effects of what happened at Lowermoor treatment works on the 6^{th} of July 1988.
The conclusions drawn by the present subgroup and contained within their draft report have neither scientific foundation nor credibility and can do nothing to allay the very real fears of the individuals who believe that they were poisoned by the polluted drinking water.

Dr Christopher Exley

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TOX/2012/38

From The Registrar
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Ms Khandu Mistry
Lowermoor Report Consultation
Room 692D
Department of Health
Skipton House
80 London Road
London SE1 6LH

21 April 2005

Dear Ms Mistry

Re: Subgroup report on the Lowermoor water pollution incident

The Royal College of Physicians welcomes the Report from the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment on whether the Lowermoor pollution incident in July 1988 has caused delayed or persistent harm to human health.

On the basis of the available data, the Royal College of Physicians concurs with the conclusion of the Report that the combination of metals which occurred as a result of the pollution incident would not have caused, or would not be expected to cause, delayed or persistent harm to health.

The Royal College of Physicians supports the recommendations that:

(i) Further studies should be carried out to explore the neuropsychological status of those individuals who consumed the contaminated water;

(ii) Investigations should be carried out into the cognitive, behavioural and educational development of individuals who were under one year of age at the time of the incident;

(iii) A study should be performed to assess whether the prevalence of joint pains and/or swelling in the population receiving the contaminated water is higher than normal;

(iv) The monitoring of routine health statistics for the population potentially exposed to contaminated water after the Lowermoor pollution incident should continue;

(v) There is a need for further work on the toxicity of aluminium, specifically studies to identify the NOAEL (no-observed-adverse-effect-level) for aluminium salts, for both acute and chronic exposure and a range of salts at different bioavailabilities;

(vi) Mechanistic data on the neurotoxicity of aluminium and its potential role in neurological disease and other disorders should be generated;

(vii) Further studies on the bioavailability of aluminium in humans should be performed.

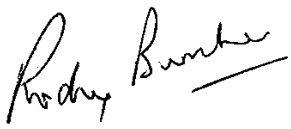
DRAFT December 2012

The Royal College of Physicians also endorses the recommendation that there should be rapid and widespread dissemination of accurate advice, particularly to medical practitioners, if further chemical incidents of this kind were to take place.

In conclusion the College is reassured by the thoroughness of the evaluations contained in the report and the fact that no neurotoxic effects have been identified almost seven years since the exposure.

I trust these comments are of use.

Yours sincerely



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TOX/2012/38

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Re: Lowermoor Water Pollution Incident:
Comments on the DRAFT FOR CONSULTATION

22 April, 2005

Dear Mr Mistry

I herewith would like to submit comments on the above indicate report.

I trust you shall pass this on to the committee.

Sincerely

Dr Bettina Platt

Comments on the subgroup report on the

Lowermoor Water Pollution Incident

As a neuroscientists working in a medical research institute, and with an interest and an expertise in metal toxicity and mental health, I have to express my concern about the enquiry in the above incident. My main concern lies with the lack of medical and epidemiological data sought by the committee from the individuals exposed. It is entirely unacceptable to conduct an enquiry of such an incident without essential medical data and related continuous monitoring of the affected population. Furthermore, the assessment of parameters *relevant* in cases of aluminium exposure should be the focus of such an assessment, such as repeated cognitive and neurological assessments, particularly in the elderly and in people with pre-existing neurological conditions. Monitoring of the current aluminium load and correlations to relevant neurological parameters are also still possible, and should be investigated in connection with the individuals' medical history.
Other points of concern are:

1) I could not find any evidence that the committee has sought appropriate expert advice on various issues of aluminium toxicology. Aluminium is unique in many aspects related to its chemical properties, interactions with biological system, and analytical problems related to its determination in the environment and in biological samples, and this is not considered appropriately in the your report.
2) The involvement of the Aluminium Federation in the proceedings, and scientists associated with them, is rather questionable and of major concern.
3) Clarification is required with regards to why the company that caused the pollution (South West Water Ltd.) was involved in the water analyses following the incident. The resulting data cannot stand up to scientific and indeed ethical scrutiny.
4) As an expert in the filed of aluminium neurotoxicity, I would like to stress that the scientific data and information considered by the committee appears to be more than inadequate and incomplete.

I sincerely urge the committee to take action to rectify this situation. This incident requires more rigorous scrutiny, firstly to help the affected population, but also to gain insights into the effects of high Al exposure to the human body, and learn necessary lessons on how to deal with other pollution incidents in the future.

Re: Comments on COT Lowermoor Subgroup Report on Water Pollution Incident

Dear Sirs,

We have reviewed the Lowermoor Subgroup report on the 20 ton aluminum sulphate accidental dumping into the water supply at Camelford in July 1988.

We have to totally agree with the comments of Dr. Chris Exley. We have seen so many of these types of reports in the United States, where many pages are produced to generate the illusion that the environmental and public health matter at hand has been considered. We are so regretful to conclude that the report appears to be aimed at whitewashing the issue.

It is well known that aluminum causes cognitive impairment in humans, and the Camelford spill involved a very large dosage. Profound effects are seen in very young children, in elderly persons. For example, there are now 22 drinking water epidemiology studies statistically linking aluminum to either elderly cognitive impairment or Alzheiemr's disease.

Cognitive effects are also seen in high dosage situations like welders for middle-aged persons. (1)

It is documented by many sources that individuals differ in their absorption of aluminum. For example, P.Brian Moore et al found the absorption of aluminum-26 to average higher in Alzheimer's disease patients. But there was a considerable individual scatter of absorption in both the control group and the AD cases. (2) And so, in Camelford one would expect some individuals to be more significantly and aversely affected than others, just based on differing absorption rates. Undoubtedly, there are also metabolic or dietary variables that would make some individuals more prone to injury than others from the massive exposure.

We are most impressed with the study of Paul Altmann et al, of the Camelford incident. Significant differences in cognitive function was found in exposed persons, compared to sibling controls using a range of tests, but most importantly the very sensitive flash and pattern visual evoked potentials. (3) This type of test is not only very sensitive, but it is also non-subjective.

We conclude that Lowermoor Water Pollution Incident Subgroup Report needs to be reworked by experts who have actual experience with aluminum in biology. The public deserves safe drinking water, and alternative safer purification systems such as iron based coagulants are used widely in Europe, in a number of cities in the United States, and undoubtedly in locations in the UK. Many utilities, such as that in Philadelphia, find that they can reduce costs by using iron coagulants.

For all these reasons, we think that you should adopt the recommendations of Dr. Chris Exley on this situation. We are a US-Canadian group, and have worked on the aluminum in health issue since 1989.

Best regards,

Erik Jansson, Pres.

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Department of the Planet Earth, Inc.
701 E Street, SE, Ste. 200
Washington, DC 20003

(1) H. Hanninen et al, Internal load of aluminum and the central nervous functioning of aluminum welders, Scand J Work Environ Health 20 (1994) 279-85
(2) P. Brian Moore et al, Absorption of aluminum-26 in Alzheimer's disease, measured using accelerator mass spectrometry, Dement Geriatr Cogn Disord 11 (2000) 66-9
(3) Paul Altmann et al, Disturbance of cerebral function in people exposed to drinking water contaminated with aluminum sulphate: retrospective study of the Camelford water incident, BMJ 319 (1999) 807-11

Khandu, here are my comments on the January 2005 draft subgroup report of the Lowermoor water pollution incident. These are not comments on behalf of the Health Protection Agency.

Para 1.37 The highlighting of macrophagic myofasciitis (here and at para 9.6) seems odd, given that the impression from para 6.55 and page 388 (appx 16, section 6.3.1) is that the basis is speculation in one paper, and little is made of it in para 8.29. It would be helpful if the group could explain its apparent interest in the hypothesis, in the context of the incident. Were any soundings taken from vaccination experts in DH or MHRA or HPA about the status of this hypothesis?

Para 1.38 The order of the bullet points is different from (and less logical than) that in para 9.7. These recommendations are not supported by any discussion or explanation in the body of the report or its appendices. Despite the careful wording of the stem, this paragraph may give the presumably incorrect impression that LSG has identified these points as deficiencies in present-day management of chemical incidents. This impression could be avoided by rewording the paragraph along the lines of "There have been considerable improvements.....The Lowermoor incident highlighted in particular the importance of the following aspects of management…"

Para 2.2. The quote from the first LIHAG report should insert dots after the end of the second sentence, to indicate a deletion.

In the last sentence, insert "the very real current health complaints to" after "attribute".

Para 2.5. The last word in the first paragraph of the quote from the second LIHAG report should be "categorically", not "completely".

Table 3 (p 40). Number of samples for pH should presumably be 130 (since the 50 exceeding the GV make up 39%).

Table 8 (p 58), and Figures 10-14, and Table 9. If the units really are mg rather than micrograms, the maximum concentrations are extremely high if they are samples at the potable water tap.

Figures 11-13 Incorrect metal on y-axis.

Table 10. heading in table should be "number of samples" not "concentration…"

Table 11. If the units really are mg rather than micrograms, the maximum concentration of copper is extremely high.

Table 12. If the units really are mg rather than micrograms, the maximum concentration of copper is extremely high.

Table 11 maxima for Al, Mn and Fe are not reflected in Table 12.

Para 3.70. second sentence "consistent"

Fig 25 the key to the trend lines is the wrong way round.

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Para 4.31 1991 not 1999, at end.

Para 6.36 At end, 6.28 not 4.28

Figure 32 the key to the trend lines is the wrong way round.

Para 7.29 The RHS of the equation is "upside-down" (as is the description in the second sentence of point 3 on page 196).

Figure 33 The heading should refer to "concentration of hydrogen ions", not "amount of hydrogen ions present".

Abbreviations In "JECFA", also need to expand "FAO" somewhere?
In "SWWA", Authority not Association.
In "TDS", Study not Survey (or has it changed?)
Re "WRc-NSF", I think that the acronym *per se* is actually the name of the company, although its derivation is as described.

I should be included in Appendix 1 in the Secretariat as:

Mr Michael Waring MA MB BChir BA FRCS LRCP Medical Secretary (until 31 October 2001)

In Appendix 3, maybe "Those who provided written information to the Subgroup" should include "Officials from Department of Health and Department for the Environment Food and Rural Affairs" – or maybe this aspect could be included in the bullet points in para 2.12 of the body of the report.

Michael Waring
Medical Toxicologist
Health Protection Agency
Chemical Hazards and Poisons Division HQ
Chilton.

Appendix 6: Reply to the Consultation Responses

Introduction

1. A draft version of the Subgroup's report was published in January 2005 and a consultation exercise to consider the draft report was run from 26 January to 20 May 2005. Twenty-six consultation responses were received, four of which were from one correspondent. Two submissions were received from one of the lay representatives on the subgroup; one of these reported information provided by an individual who had previously given oral evidence to us. Five submissions were received from individuals who had previously provided personal evidence about the incident to the Subgroup, and one from an individual who provided new personal evidence. This was reviewed but the evidence was not added to Tables 31 and 32 of the report, because it had not been provided in oral form (see Chapter 5, paragraph 5.16). Four responses were received from individuals who had previously provided technical information. Several responses provided new technical information, raised new issues or pointed out minor errors in the report.

2. A public consultation meeting was held in Camelford on 17 February 2005. Thirty-three individuals attended and a number of helpful points were raised. These were considered by the Subgroup with the written consultation responses.

3. The responses were helpful to us and we are grateful to the correspondents for their contribution to the development of this report. The exercise raised issues which we had not previously addressed, such as the question of what other trace contaminants might be present in the contaminated water (see Chapter 3, paragraphs 3.90 to 3.96). It has allowed us to identify issues which require further explanation, such as why we did not review individual medical records (see below) or why it has been difficult for us to make use of medical samples provided as personal evidence (see Chapter 5, paragraph 5.180). We have also been alerted to further scientific references on aluminium, which has enabled us to extend our review of this contaminant (see Chapter 6, paras 6.7 to 6.84). As a result of the consultation exercise, we have extended the recommendation for investigations into the cognitive, behavioural and educational development of children who were under 1 year of age at this time of the incident to include children who were *in utero* at the time of the incident (see Chapter 9, paragraph 9.4).
We also recommend that the monitoring of routine cancer incidence and mortality statistics for the previously established cohort which was exposed to contaminated water after the pollution incident should continue.

4. Many of the points raised by the consultation exercise have been dealt with by amendments or further text in the body of the report. However, the responses raised a number of generic issues which it became clear to us had either not been addressed or had not been adequately explained in the draft report. We discuss these below.

<u>Individual medical records</u>

5. Three responses expressed disappointment that we had not asked to see the medical records of those who provided personal evidence to us and who were worried that the incident had adversely affected their health.

6. We discussed the question of individual medical records during the investigation and again in detail after receiving these responses. We considered whether obtaining and reviewing the records of individuals who had been exposed to the contaminated water would provide us with any new information or would be useful *within the remit of our study.* Our terms of reference is "to advise on whether the exposure to chemicals resulting from the 1988 Lowermoor water pollution incident has caused, or is expected to cause, delayed or persistent harm to human health." It is not to discover the reason(s) why individuals are experiencing health complaints nor to develop a treatment protocol. Within the context of our terms of reference, medical records are of limited value. The record of the consultation between the doctor and patient made in a medical record reports the symptoms the patient is experiencing and discusses what is wrong with the patient from the point of view of the doctor and patient. The consultation, and the record of it, is not made from the point of view of whether the patient's symptoms are caused by the contaminants released in the Lowermoor incident. There is no prior hypothesis which we could test in an examination of medical records, as there would be if a scientific study was carried out, such as those we have recommended in Chapter 9.

7. We wish to emphasise that we accepted the information which individuals provided to us about their health at face value and, therefore, it is not clear what further information would be gained from seeing the medical records of these individuals. However, we also wish to emphasise that we recognise, from both the oral evidence and written evidence we received, that some individuals have continuing ill health for which they are concerned to find a cause.

Adequacy of scientific data reviewed by the Subgroup

8. Three respondents were critical of the extent to which we had reviewed the scientific literature on the contaminants whose water concentrations were increased after the incident. Another respondent commented that there were 22 drinking water epidemiology studies statistically linking aluminium to either elderly cognitive impairment or Alzheimer's disease. All four respondents who addressed this issue were asked for further details of missing references which they regarded as important and two replied with these details. Some of the references cited by these respondents were already referenced in the report or in the reviews prepared by the Department of Health Toxicology Unit at Imperial College (see Appendices 20, 21 and 22). Abstracts of any which had not been considered previously were reviewed and relevant papers obtained. Chapters 6 and 7 of the report were updated accordingly.

8. We do not agree that we conducted an inadequate review of the scientific literature on the contaminants. We describe in Chapter 6 the way we went about our review and the data which was used.

9. Another respondent provided a list of 548 hyperlinks to references on aluminium. We obtained and reviewed the abstracts of the papers and determined that some had already been reviewed and some were not relevant to the exposure of individuals from the contaminants in water, for reasons which are described in Chapter 6. Nine references were relevant and we are grateful that these have been brought to our attention. We have referred to these in the revised text on the human and animal toxicity of aluminium.

Adequacy of other information available to the Subgroup

10. Some correspondents considered that we had cited inappropriate information and studies. For example, we were criticised for including a published study of Richmond tests of educational attainment in schoolchildren (see paragraphs 5.116 to 5.122) and data on Special Educational Needs (SEN) (paragraphs 5.146 to 5.155). The data on Richmond tests was included because it was a published study on children in the affected area and, in our report, we discuss all relevant published studies. We discussed the (SEN) data because we were asked to consider these data by a local journalist. We expressed reservations about the suitability of using SEN data to assess effects on health in the draft report (paragraph 5.155).

11. Other correspondents expressed reservations that the report made use of water quality data from SWW plc. These were the only water quality data that were available for the greater part of the period of contamination. It should be noted that these data were not used exclusively and, in our assessment of the implications for health of exposure to the contaminants (Chapter 7), we have also included estimated exposures based on contaminant concentrations in the water samples taken by private individuals.

12. In our investigation, we have attempted to use all the data which is available and we are aware of the limitations of some of these data. We wrote to a number of individuals who responded in the consultation exercise to obtain further information. Where we consider that further studies would be beneficial, we have recommended them in Chapter 9 of the report.

Appendix 7: Drinking water quality – the legislative framework for public drinking water supplies

The situation before 1989

1. Under the Water Act 1945, water undertakers - the Water Authorities, created in 1974, and the statutory water supply companies - were required to "... provide in their mains and communication pipes a supply of wholesome water sufficient for the domestic purposes of all owners and occupiers within the limits of supply...". UK law did not define a quantitative measure of what made water wholesome and there was no requirement to monitor the quality of supplies.

2. General guidance on safe levels of substances that might be permitted in drinking water supplies, and safeguards and best practice to be adopted to ensure production and delivery of a wholesome supply of water were available when WHO published its 1st edition of "International Standards for Drinking Water" in 1958. This was updated in 1964 when the 2nd edition of "International Standards for Drinking Water" were published and the 1st edition of "European Standards for Drinking Water" were also published. Both these were updated in 1970 and 1971 respectively. In 1984 the two editions were combined and published as the WHO "Guidelines for Drinking Water Quality".

3. The 1980 EC Directive on 'Water Intended for Human Consumption' (80/778/EEC) set out standards for drinking water quality. However, this was not enshrined in national law until 1989 (see below). There was no regulatory regime to oversee that water undertakers were carrying out their duties in respect of supplying wholesome water. There was no offence of supplying water unfit for human consumption. There was a formal requirement to notify the appropriate department (usually the then Department of the Environment) of major incidents affecting water supplies. Usually, but not always, the local Medical Officer of Health would be informed if there was a health risk.

4. In the case of the Lowermoor water pollution incident, this lack of suitable water/environment legislation under which investigation and prosecution could take place resulted in the police investigating the incident. A prosecution was initiated by the Director of Public Prosecutions for Public Nuisance by supplying contaminated water. South West Water Authority was fined £1,000 and required to pay costs of £25,000.

The situation since 1989

5. EC Directive 80/778 on 'the Quality of Water Intended for Human Consumption' came into effect on 17 July 1980. It set out Maximum Admissible Concentrations (MACs) (often and usually based on WHO Guideline Values) for various chemical, aesthetic and microbiological quality parameters which tap water was required to meet.

6. In 1989 the water industry was privatised under the water Act 1989 and the Water Authorities became the ten Water and Sewerage Companies of today. The

Statutory Water Companies became private water supply only companies. All the water companies were appointed water undertakers under section 6 of the Act.

7. The Water Act 1989, which was consolidated into the Water Industry Act 1991 ("the Act"), made it a legal duty for water undertakers to "... supply only water which is wholesome at the time of supply" (Section 68). The Water Supply (Water Quality) Regulations 1989, made under section 69 of the Act, came fully into force on 1 January 1990. These Regulations for preserving the quality of water reflected the requirements of Directive 80/778 in terms of standards to be met, and also set some nationally derived standards. The 1989 Regulations remained in force until the end of 2003.

8. A new EC 'Drinking Water Directive' came into force on 25 December 1998 and its requirements are incorporated into the new Water Supply (Water Quality) Regulations 2000 (the new Regulations). These new Regulations apply to water companies whose area of supply is wholly or mainly in England. The National Assembly for Wales adopted similar new Regulations at the end of 2001 and these apply to water companies whose area of supply is wholly or mainly in Wales. Whilst some of the requirements of these new Regulations came into force as early as 2001, the majority came into force at the end of December 2003. The new Regulations set some new standards, tightened some existing standards, relaxed some standards, and dispensed with others. There are also changes in the monitoring requirements of supplies.

9. During 2006/7 revisions of the 2000 Regulations were proposed and consulted upon. It is likely that the 2000 Regulations will be amended at the end of 2007 to take into account changes in EU legislation contained in the Surface Water Abstraction Directive and Water Framework Directive. The opportunity has also been taken to incorporate the use of Drinking Water Safety Plans, recommended by WHO, as the most effective means of consistently ensuring the safety of a drinking water supply through the use of a comprehensive risk assessment and risk management approach that encompasses all steps in water supply from catchment to consumer.

10. The standards provide a numerical definition of wholesomeness. Water supplied which contravenes one or more of the standards listed in the Regulations is, by definition, unwholesome.

The old and new Regulations (and the proposed 2007 amendment) set out requirements on:

- monitoring drinking water quality
- water treatment
- the provision of information
- the use of water treatment chemicals
- drinking water system construction products.

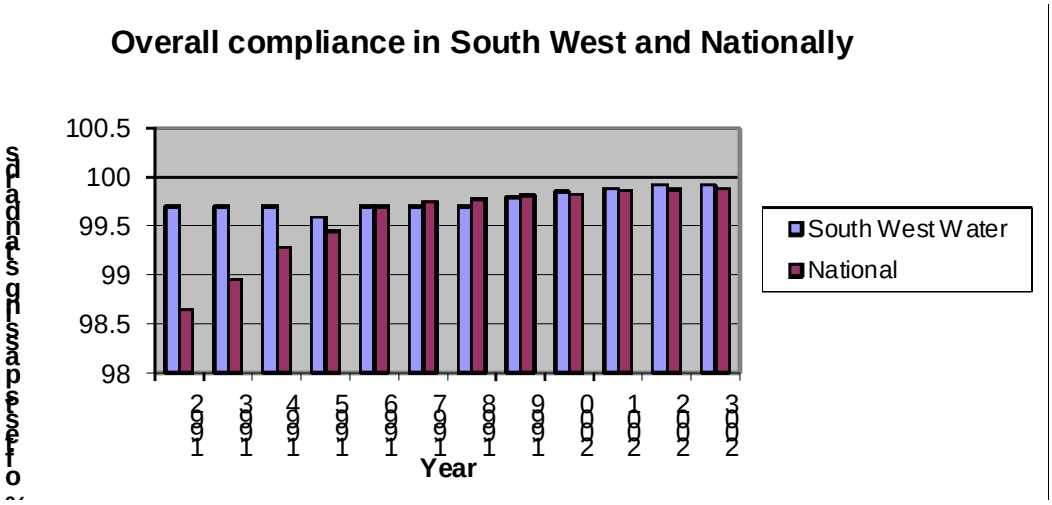
11. Figure 1 shows the number of tests carried out between 1992 and 2003 in the South West and Nationally which met the required standards. From 2004 onwards, when the new Regulations and revised standards came fully into force, the method of reporting on compliance also changed. It is not possible therefore to compare data from 2004 - 2006 with those from previous years. It was recognised that simply reporting on the number of tests meeting the standards, which indicated that overall

compliance was high, could mask localised problems experienced by consumers. The Chief Drinking Water Inspector's Annual Reports on drinking water quality for these years have therefore been issued on a regional basis and more detailed reporting provided on performance by water companies. These reports can be found at www.dwi.gov.uk. For information on water quality in the South West Water area, the Western Regional Reports should be consulted.

12. Section 18 of the 1991 Act requires enforcement action to be taken for any breach of wholesomeness standards, monitoring and treatment, and/or records and information requirements of the Regulations. However, enforcement action is not taken forward if the breach is

- deemed to be trivial or
- unlikely to recur or
- if the water company has taken immediate remedial action to prevent a recurrence or
- if the water company has submitted a legally-binding programme of work to achieve compliance within an acceptable timescale.

Figure 1:



The bars show the proportion of tests which met the required standards.

13. Section 70 of the 1991 Act made it a criminal offence to supply water which is unfit for human consumption. It also provides a defence for the company if it can show that it had no reasonable grounds for suspecting that the water would be used for human consumption; or took all reasonable steps and exercised all due diligence for securing that the water was fit for human consumption on leaving its pipes, or was not used for human consumption. It is for the courts to decide whether water is unfit but, in general, water that causes illness on consumption, or where its appearance, taste or smell is such that people cannot reasonably be expected to drink it, is likely to be deemed unfit. It is important to understand, however, that whilst water which is unfit is likely to be unwholesome, unwholesome water is not necessarily water unfit for human consumption.

Deterioration of water beyond the point of supply

14. Water companies are not responsible for any deterioration in the quality of the water within a consumer's premises, except in the case of concentrations of copper or lead (the 1989 regulations also included zinc but there is now no longer a standard for this parameter under the new regulations). If the standards for these metals are likely to be exceeded in the water supplied to the cold tap in the kitchen, then the water company must consider further treatment of the water to reduce the risk of the water becoming unwholesome. This measure is intended primarily to reduce exposure of consumers to lead.

The role of the Drinking Water Inspectorate

15. Under the 1991 (and 1989) Act, responsibility for regulating the quality of public drinking water supplies lay with the Secretary of State for the then Department of the Environment. Technical Assessors were appointed under section 86 of the Act to act on their behalf in these matters. Following devolution, this duty also applied to the National Assembly for Wales.

16. The Water Act 2003 amends section 86 of the Act to reflect that such assessors are more generally known as the Drinking Water Inspectorate and to enable the Chief Inspector of Drinking Water to initiate prosecutions in relation to drinking water quality in his/her own name.

17. Water companies are responsible for monitoring the quality of their supplies. This 'self-monitoring' role is subject to checks by local authorities and the Drinking Water Inspectorate. One of the main tasks of the Inspectorate is a rolling programme of continuous technical audit to ensure that water companies are meeting all their regulatory obligations. Water companies must make all results of regulatory sampling available to the general public via their public record.

Drinking Water Quality Incidents

18. The Water Industry (Suppliers' Information) Direction 2009 (and earlier versions) require water companies to notify the Secretary of State or the National Assembly for Wales (in practice, the Inspectorate) of any event, which by reason of its effect on the quality or sufficiency of drinking water, may give rise to a significant risk to consumers' health. The Regulations require that similar notifications are made to health authorities (normally the relevant Consultant in Communicable Disease Control in the Health Protection Agency and Director of Public Health in the relevant Primary Care Trust) and local authorities (normally the relevant Environmental Health Officer). The Inspectorate investigates all such notifications and, in some cases, the investigation could result in the water company being prosecuted for supplying water unfit for human consumption. The results and recommendations arising from the Inspectorate's investigations are made public.

Drinking Water Inspectorate
September 2007 and 2012

Appendices 8 to 11

The following Appendices:

Appendix 8: Water quality data for the parishes of Camelford, Davidstow, Advent, St Minver Lowlands and St Minver Highlands;

Appendix 9: Water quality data for the parishes of Camelford and Davidstow;

Appendix 10: Water quality data for the parishes of St Teath, Tintagel and Trevalga; and

Appendix 11: Water quality data for the parishes of St Endellion, Forrabury & Minster and St Juliot

Please see http://cot.food.gov.uk/pdfs/lowermoorappendices65.pdf where these are found as Appendix 6 to 9 of the 2005 Consultation Report

Appendix 12: Lowermoor water quality modelling report.

Black & Veatch Ltd, August 2004

Please see http://cot.food.gov.uk/pdfs/lsgreportapp10.pdf

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Appendix 13: Lowermoor water quality modelling report (Phase 2)

Black & Veatch Ltd, August 2006.

Attached as separate document

Appendix 14: Other water pollution incidents involving aluminium sulphate

A number of other reported incidents in which the water supply has been contaminated with aluminium sulphate are listed below. Little information is available on most of these.

October 1988: The water supply to properties in Hatfield, Hertfordshire was contaminated with aluminium sulphate (Cross, 1990)

March – April 1989: Penwhirn water supply district of Dumfries and Galloway Regional Council: because of a change in raw water quality and subsequent plant failure, water entering the supply contained 'flocculated material' including raised concentrations of aluminium. The maximum concentration of aluminium entering the supply was 3.5 mg/l (Water Research Centre, 1989).

June 1989: The water supply to residents around Newry, Ulster was contaminated with concentrated aluminium sulphate solution (amount unclear, may be up to 30 tonnes) (Cross, 1990).

November 1989: Amlaird, Kilmarnock: a breakdown in treatment led to raised aluminium concentrations in the water supply for at least 4 months (Cross, 1990).

March 2011: Control of the treatment process at Burncrooks water treatment works, Glasgow, was lost for approximately four hours. Concentrations of aluminium exceeded the regulatory standard for a period of 24 hours, with concentrations exceeding 4 mg/l for approximately 6 hours (Drinking Water Quality Regulator for Scotland, 2011).

References

Cross D. Something in the water. Green Magazine, July 1990.

Drinking Water Quality Regulator for Scotland, September 2011. DWQR Investigation into the Burncrooks Incident, North-west Glasgow 17 – 19 March 2011. Ver 2: 26 September 2011. Available at: http://www.dwqr.org.uk/technical/water-quality-incidents/2011-water-quality-incidents

Water Research Centre. Penwhirn district water treatment and distribution: An independent investigation by WRc. Unpublished report, November 1990.

Appendix 15: Report on the estimated consumption of aluminum, sulphate, copper, zinc, lead and pH following the contaimination incident on 6th July 1988. Crowther Clayton Associates. Report no. 91/2737.

Note: during the printing of the Consultation report, it became apparent that the quality of the above report was too poor to be reproduced in a published document. A photocopy of the Crowther Clayton report can be obtained by contacting the Secretariat , whose details can be found at the front of the Consultation report, or it can be viewed as a scanned document on the Subgroup's website (home page: http://cot.food.gov.uk/cotwg/lowermoorsub/)

See http://cot.food.gov.uk/pdfs/crowtherclaytonassociates.pdf

Appendix 16: Extract from 'The Health of the Population', Department of Public Health Medicine, Cornwall and Isles of Scilly of Health Authority, 1988.

See http://cot.food.gov.uk/pdfs/healthofthepopulation88.pdf

Appendix 17: Letter from Department of Health and Social Security to Dr CR Grainger, 'Lowermoor Incident', 24 August 1988

See http://cot.food.gov.uk/pdfs/lsgreportapp14.pdf

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Appendix 18: Summary and critique of epidemiological studies of the North Cornwall population

See http://cot.food.gov.uk/pdfs/lsgreportapp15.pdf

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Appendix 19: Report of the North Cornwall Homeopathic Project

See http://cot.food.gov.uk/cotwg/lowermoorsub/draftlowermoorreport/

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Appendix 20: Summing-up by the West Somerset Coroner

Attached as separate document

Appendix 21: Review of the scientific literature on aluminium (1994 to April 2002) prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London

Note: this was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

See http://cot.food.gov.uk/pdfs/lsgreportapp16.pdf

Appendix 22: Review of the scientific literature on aluminium (January 2002 to October 2003) prepared for the Lowermoor Subgroup by the Department of Health Toxicology Unit, Imperial College, London

Note: this was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

AN UPDATE OF PUBLICATIONS RELATING TO THE TOXICITY OF ALUMINIUM, 2002-October 2003

A report prepared for the Department of Health Committee on Toxicity, Lowermoor Subgroup by the Department of Health Toxicology Unit at Imperial College London.

1 Introduction

Aluminium (Al) toxicity was reviewed in detail in an update of the 1997 WHO (IPCS) Environmental Health Criteria 194 report on Aluminium, prepared for the COT-Lowermoor Subgroup in Spring 2002 (LSG/02/7). This report is a further update, describing relevant publications from the period Jan 2002-October 2003 (by publication date).

The data included have been restricted to toxic and/or other biological effects of Al in humans and in animal models. In vitro/mechanistic studies have not been described, but a bibliography of these recent publications is provided in Appendix 1.

2 Aluminium toxicity publications 2002-2003

2.1 General literature

The US Food and Drug Administration (FDA) published a final monograph on antiperspirant drug products for over-the-counter human use (Food and Drug Administration, 2003). Section II. F. of this ruling details comments on the safety of aluminium ingredients, including critical discussion of some publications regarding the potential toxicity of aluminium by various routes of intake, in particular, neurotoxicity and the possible involvement of aluminium in Alzheimer's and Parkinson's diseases and amyotrophic lateral sclerosis (ALS).

The FDA concluded that the literature showed that high doses and long-term industrial exposures to aluminium can be associated with recognisable specific neurological effects, but that the evidence to date was insufficient to link aluminium to Alzheimer's disease, Parkinson's disease or ALS. It was noted that people with renal dysfunction should be alerted to consult a doctor before using or continuing to use Al-containing antiperspirant products. It was also recommended that a general warning be included to keep antiperspirant drug products away from infants, who may be at higher risk from Al exposure due to immature renal function. In support of this, the agency stated that it "… acknowledges that small amounts of aluminum can be absorbed from the GI tract and through the skin. Assuming a person has normal renal function, accumulation of aluminum resulting from usual exposures to antiperspirant drug products (application to the underarms once or twice daily) and subsequent absorption is considered minimal. However, people with renal dysfunction have an impairment in normal renal excretion of aluminum… The agency considers it prudent to alert these people to consult a doctor before using or continuing to use these products on a regular basis and is including a warning in the final monograph: 'Ask a doctor before use if you have kidney disease'."

2.2 Human data

2.2.1 Acute/sub-acute exposure

(Owen *et al*., 2002) carried out a retrospective study to evaluate mortality rates (from July 1988 to December 1997) in the population of a region of Cornwall supplied by water from the Lowermoor treatment works at the time of the 1988 aluminium sulphate contamination incident. The ratio of standardised mortality ratios (SMRs) for subjects living in an area supplied by the Lowermoor works (n = 11 114), as compared with those in an adjacent area with a different water supply (n = 5359) was 1.08 (95% CI, 0.97-1.21). The SMR for the Lowermoor-supplied region was lower than that for the county of Cornwall as a whole (81.6; 95% CI, 77.2-86.2) and lower than that for the England and Wales standard population (77.7; 95% CI, 73.5-82.0).

2.2.2 Chronic exposure

Renal failure patients

Encephalopathy due to aluminium overload in renal failure patients is a well-documented syndrome (see LSG/02/7) and further publications will generally not be described here. One recent case report described autopsy findings in a 59-year-old female encephalopathy patient, who had chronic renal failure and took 3.0 g hydroxy-aluminium gel per day during a 15-year period. Aluminium deposition and neuropathological changes in the brain were noted, but there were no signs of Alzheimer's disease (AD), supporting the hypothesis that aluminium alone is not causal for AD (Shirabe *et al*., 2002).

Occupational exposure

(Polizzi *et al*., 2002) reported that foundry workers who had previously (≥ 10 years before) been exposed to aluminium dust (low-level occupational exposure for several years) had significantly higher serum aluminium concentrations and blood iron concentrations than a control group without occupational exposure. A positive relationship was observed between serum aluminium concentration and some tests of cognitive function.

2.3 Animal data

Studies described have been restricted to those in which aluminium administration was by the oral route.

2.3.1 Biodistribution

(Ogasawara *et al*., 2002) reported that oral administration of 270 mg/l aluminium (as hydroxide or chloride, in tap water) and citric acid (molar ratio aluminium : citric acid = 1:2) for 7 weeks did not increase brain aluminium levels in rats.

2.3.2 Acute exposure

(Micic *et al*., 2003) reported that oral application of a single, high dose of aluminium chloride (3.7 g/kg bw $AlCl_3.6H_2O$, ~ 414 mg/kg bw aluminium) resulted in a biphasic pattern of increased superoxide dismutase (SOD) activity in the brains of Mongolian gerbils during the subsequent 4 days (up to 200% and 171% increase as compared

with control animals at 24 hours and 96 hours, respectively, after treatment; no significant difference between treated and control animals at 48 hours). Twenty of the 52 treated animals died within 24 hours of aluminium treatment, the other 32 animals survived until the end of the experiment, but showed signs of sickness such as slow gait, splaying of extremities and loss of appetite.

2.3.3 Reproductive/developmental effects

A reproductive toxicity study in which high dose (probably 1000 mg/kg diet, ~ 150 mg/kg bw/day, although this is unclear[36]) was fed to female Swiss Webster mice from conception to weaning showed reduced weaning weight associated with aluminium treatment. Pregnancy weight gain was not affected. Maternal food intake was not reported (Golub *et al*., 2003).

2.3.4 Sub-chronic/chronic exposure

Neurobehavioural effects

Treatment for 6 months with 0.1% aluminium, as sulphate in drinking water (~ 200 mg/kg bw/day aluminium) did not affect tests of spatial working memory in rats (von Linstow *et al*., 2002).

Treatment of young and old male rats for 100 days with 100 mg/kg bw/day Al[37] (as nitrate nonahydrate, in drinking water, + citric acid) did not affect performance in behavioural tests. The total number of synapses in the left CA1 fields of hippocampal formation decreased with both age (~ 22% lower in control aged vv. control young rats) and aluminium exposure (~ 32% lower in aluminium -loaded young vv. control young rats; ~ 8% lower in aluminium-loaded aged vv. control aged rats) (Colomina *et al*., 2002).

Gavage treatment of rats with 50 or 200 mg/kg bw/day aluminium chloride (~ 10 or 40 mg/kg bw/day aluminium, described by the authors as one twentieth and one fifth of the oral LD_{50}, respectively) for 8 weeks (5 days/week) had no significant effect on central electrophysiological or behavioural parameters evaluated. Brain aluminium levels were increased by ~ 34% and 153% in the low and high-dose aluminium groups, respectively, as compared with a control group (Baydar *et al*., 2003).

Neuropathology

Chronic (24 month) exposure of mice to a diet containing very high levels of aluminium (15600 mg/kg di*et al*uminium hydroxide, ~ 810 mg/kg bw/day aluminium), with or without low levels of Ca and Mg, resulted in a significant increase in tau-positive neurons in the brains of these animals (Kihira *et al*., 2002).

(El Rahman, 2003) reported that gavage treatment of rats with aluminium (43, 86 or 172 mg/kg bw/day, as sulphate) for 35 days was associated with pathological changes in brain tissue. These changes included congestion of cerebral blood vessels (all

[36] The dose given is unclear - described variously throughout the report as 1000 μg/g, 1000 mg/g and 1000 μg/kg diet.

[37] It is not entirely clear from the report whether the dose was 100 mg/kg bw/day Al or 100 mg/kg bw/day Al nitrate.

doses) and haemorrhage (2 higher doses); meningeal damage (highest dose); neuronal degeneration of the cerebral cortex (all groups, dose-dependent), subcortical region, base of the brain and hippocampus (2 higher doses). Dose-dependent increases in brain glutamate and glutamine, decreases in GABA, and increases in brain aluminium levels were observed.

Neurophysiology

(Chen *et al*., 2002) reported that neonatal exposure of rats to aluminium from birth to weaning (day 21) affected electrophysiological indicators of pre- and post-synaptic mechanisms of (central) synaptic transmission which were evaluated at 90-120 days. (Exposure was *via* breast milk, dams were given water contain 0.3% aluminium chloride [~ 120 mg/kg bw/day Al]).

Neurochemistry

Cholinesterase activity

Brain acetylcholinesterase (AChE) activity was increased (around 1.5-fold) in mice given 10 mg/day aluminium (~ 500 mg/kg bw/day, in water, as chloride or lactate) for 1-3 months (Zatta *et al*., 2002).

(Dave *et al*., 2002) reported that dietary supplementation with aluminium (100 mg/kg bw/day aluminium chloride, ~ 20 mg/kg bw/day aluminium) for 100-115 days was associated with inhibition of rat brain AChE activity (V_{max} of soluble fraction component I of soluble form decreased by 34%; V_{max} of components I and II of membrane-bound form decreased by 20% and 19%, respectively), whilst butyrylcholinesterase (BChE) activities in heart and liver were increased (V_{max} soluble fraction components I and II heart increased 2.3-fold; V_{max} components I and II membrane bound heart increased 74% and 160%, respectively; V_{max} components I and II soluble form liver increased by 58% and 83%, respectively; V_{max} components I and II membrane-bound liver increased by 91% and 168%, respectively).

5-hydroxytryptamine (5-HT)

(Kumar, 2002) reported that oral administration of aluminium to rats as aluminium chloride (320 mg/kg bw, ~ 36 mg/kg bw/day Al, by gavage) for periods of 4 to 60 days had varying effects on brain 5-HT levels depending on the brain region and duration of exposure. The authors suggested that these changes may be related to the cholinergic toxicity of aluminium.

Neuronal nitric oxide synthase (nNOS)

Rats were exposed to 0, 5 and 10 mM aluminium chloride (in drinking water) beginning 3 weeks after birth and continuing through mating and gestation, and suckling (pups exposed for 3 weeks gestation, 3 weeks suckling). Pups were then analysed for nNOS-immunoreactive neurons in regions of the cortex; levels were increased (10%) in the 5mM group and decreased (17%) in the 10 mM group. The authors suggested that impaired expression of nNOS induced by aluminium treatment may be neurotoxic because it disturbs the link between glutamatergic and monoaminergic neurons (Kim, 2003).

Oxidative stress

Chronic treatment (8 months) with drinking water containing 0.2% aluminium nitrate affected indices of oxidative stress in rat brain regions (catalase activity increased 49% cortex, 11% midbrain; GST activity decreased 49% cortex, 46% cerebellum, 26% pons, 23% mid-brain; GPx levels increased 18% cerebellum; TBARs increased ~ 100%; GSSG increased ~ 30%; GSH no significant change). Blood δ-ALAD were decreased by ~ 25% and ZPP increased by ~ 40%. Aluminium levels were significantly increased by the treatment (~ 5-fold increase in blood; ~ 2-fold increase in brain) (Flora *et al*., 2003).

(Pratico *et al*., 2002) reported that feeding an aluminium-enriched diet (2 mg/kg diet, ~ 0.3 mg/kg bw/day aluminium) for 9 months to transgenic mice which over-express human amyloid precursor protein led to an increase in markers of oxidative stress and increased amyloid β peptide formation and deposition in the brain. These effects were ameliorated by co-inclusion of vitamin E in the diet.

2.4 In vitro/mechanistic studies

Several recent papers have described studies of the effects of aluminium in vitro. Many of these studies have used neural cell cultures to investigate the possible effects/mechanisms of aluminium involvement in neurodegenerative syndromes such as Parkinson's and Alzheimer's disease and ALS. These studies are not described here.

3 Summary

Very few new studies have been published regarding potential adverse effects of aluminium in healthy (i.e. non-renal failure) human subjects. One study (Owen *et al*., 2002) compared mortality rates in subjects likely to have been exposed to aluminium sulphate-contaminated water following the 1988 Lowermoor incident with those in a neighbouring area (not Lowermoor-supplied). The rate for the "supplied" population was slightly higher (1.08), but the difference was not significant, and rates in both areas were lower than national rates and those for the county of Cornwall. (This paper has previously been discussed by the LSG).

Several recently-published studies have evaluated effects of oral aluminium dosing in animal models. The majority of these studies have focussed on neurological effects. Studies in rats showed no effects of chronic aluminium supplementation (10-200 mg/kg bw/day[38] for several weeks or months) on behavioural measures. Some adverse effects (neuropathological and neurophysiological) were observed in cases where chronic treatment with very high levels of aluminium was given. Gavage treatment of rats with high levels of aluminium sulphate (43-172 mg/kg bw/day aluminium) for 5 weeks was also associated with neuropathological and

[38] For comparison, the maximum theoretical Al concentration in the water supply following the Lowermoor incident was estimated as 21 mg/kg bw/day for a 60 kg adult (based on daily intake of 2l water containing 620 mg/l Al [the maximum estimated concentration in the cold water supply – see LSG/03/07]. The time period of exposure is unclear, but perhaps in the region of several hours or days.

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neurochemical changes (El-Rahman, 2002). One study showed that high-dose aluminium supplementation (0.3% aluminium chloride in drinking water, ~ 120 mg/kg bw/day aluminium) to rat dams from birth to weaning affected indicators of neurotransmission in offspring several months later (Chen *et al*., 2002). Gavage treatment of rats with aluminium chloride (320 mg/kg bw/day $AlCl_3.6H_2O$, ~ 36 mg/kg bw/day aluminium) for periods of 4 to 60 days was associated with increased, decreased or unaltered brain 5-HT levels, depending on the specific region of the brain and the duration of treatment (Kumar, 2002).

October 2003

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Appendix 23: Review of the scientific literature on aluminium (October 2003 to April 2005) prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London

Note: this was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

AN UPDATE OF PUBLICATIONS RELATING TO ALUMINIUM TOXICITY October 2003-April 2005.

A report prepared for the Department of Health Committee on Toxicity, Lowermoor Subgroup by the Department of Health Toxicology Unit at Imperial College London.

1. Aluminium toxicity was reviewed in detail in an update of the 1997 WHO (IPCS) Environmental Health Criteria 194 report on aluminium, prepared for the COT Lowermoor Subgroup in Spring 2002 and updated in 2003. This following report is an update of studies from 2003 to date, relating to biological and toxic effects of aluminium in humans and in animal models. The report is divided into two main sections: A] neurotoxicity and effects on the brain, and B] other toxic and biological effects. Reports of experimental studies in animals were only included if aluminium treatment was given by the oral route.

A. Neurotoxicity and effects on the brain

Human data

Epidemiological studies of cognitive impairment, dementia and Alzheimer's disease

2. An association was observed between serum aluminium concentrations and Alzheimer's disease in a group of 35 elderly patients evaluated for potential correlations between serum trace element concentrations and presence of cognitive impairment and/or dementia. Patients were divided into 4 groups: control [n = 11], cognitive impairment non-dementia (CIND) [n = 8], Alzheimer's disease (AD) [n = 8], vascular dementia (VaD) [n = 8]). The paper reports that trace element serum concentrations were in a normality range in all subjects. Serum aluminium concentrations (mean ± SD, in mg/ml) were 0.215 ± 0.106, 0.353 ± 0.145, 0.735 ± 0.158, 0.303 ± 0.183, respectively[39]. A significant negative correlation was also noted between MODA scores ('Milan Overall Dementia Assessment' - a test for the presence of dementia) and serum aluminium concentration in this group of 35 subjects (ie, higher aluminium correlated with lower cognitive function; $r = -0.628$, $p<0.0001$). MODA scores were also negatively correlated with copper serum concentrations, and positively correlated with selenium, cobalt, chromium and iron levels (Smorgon *et al*., 2004).

3. (Gillette-Guyonnet *et al*., 2005) reported an evaluation of potential associations between drinking water composition (aluminium, silica and calcium content) and cognitive impairment in a group of 7598 women aged ≥ 75 in France (the EPIDOS study cohort). Daily intakes of aluminium, silica and calcium supplied by drinking water were 0.0231 ± 0.025, 10.17 ± 10.01 and 134 ± 154.1 mg, respectively (mean ± SD). Cognitive performance was positively correlated with daily silica intake, but not with aluminium or calcium intakes. The authors concluded that the study did not show any evidence for aluminium as a risk factor for Alzheimer's disease.

Inhalation

[39] There is a question regarding the units cited by the authors here, as most reports cite standard serum Al concentrations in a range around ~ 10 μg/l.

4. (Buchta *et al*., 2003) described evaluations made in 1999 and 2001 as part of a longitudinal study of 98 welders with occupational exposure to aluminium welding fumes, as compared to a control group of 50 car-production workers at the same plant in Germany. Median plasma aluminium concentrations were approximately 10 (range ~ 2-40) μg/l in 1999 and 4 (range ~ 1-11) μg/l in 2001, whilst median urinary aluminium concentrations were around 40-70 (range ~ 2-250) μg/l [or 30-40 (range ~ 5-230) μg /g creatinine] (data for control subjects were not reported). There were no significant differences between test and control subjects in psychomotor performance and other neurobehavioural tasks, except that test subjects showed slower reaction times. The difference in reactions times between the groups did not change during the period of evaluation, and the authors suggested that it may be due to pre-exposure differences between the groups. Further evaluations were scheduled to be carried out in 2003.

5. A study in China showed differences in neurobehavioural parameters between a group of 32 men with occupational (14.91 ± 6.31 years, mean ± SD) aluminium exposure, as compared with a control group (workers at a flour plant). The aluminium workers had significantly higher scores for confusion and tension/anxiety, lower scores for standard reaction times, and lower scores for DSY (described as "digital symbol") and PA (pursuit aiming) tests. Other parameters tested were not significantly different between the two groups[40]. Mean urinary aluminium concentrations were 40.08 ± 9.36 and 26.84 ± 8.93 μg/mg creatine, in test and control groups, respectively (He *et al*., 2003).

Renal failure patients

6. Calcium intoxication was suspected in a group of 27 end-stage renal disease patients in Curacao, Netherlands Antilles, who presented with symptoms including nausea, vomiting and hypercalcaemia. Despite subsequently changing to a low-calcium dialysate, a number of the patients developed microcytic anaemia and neurological symptoms. Ten patients died of convulsions, sepsis and coma. Analysis showed mean ante mortem serum aluminium concentrations of 808 (359-1275) μg/l and 255 (113-490) μg/l in deceased patients and survivors, respectively (normal aluminium concentration < 10 μg/l, or < 50 μg/l in non-symptomatic dialysis patients). Investigations revealed high calcium and aluminium levels in the dialysis water supply due to leaching from a replacement supply pipe (de Wolff *et al*., 2002).

7. A patient with chronic renal failure, but not on dialysis, developed fatal aluminium-related encephalopathy due to self-dosing with large doses of antacids (total cited as at least 3 kg) for approximately 3 years (Zatta *et al*., 2004).

Reviews

8. (Gupta *et al*., 2005) published a review of the literature regarding potential associations of aluminium and Alzheimer's disease (AD). They concluded that aluminium is undoubtedly neurotoxic, that the involvement of aluminium as a factor

[40] The scoring systems are not explained in the report and it is not clear to the non-specialist what increases or decreases in scores indicate, except that, in the discussion, the authors state that Al workers performed better in neurobehavioural tests than controls, with quicker reaction times.

in AD cannot be discarded. However, whether aluminium is a sole factor in AD and whether it is a factor in all AD cases still needs to be understood.

Animal studies

Rats

9. (Jing *et al*., 2004) reported that treatment of adult male rats for 3 months with 500 mg/kg bw/day aluminium (in water solution, by perfusion through the stomach) led to increased brain aluminium content, changes in synaptic ultrastructure in the hippocampus and frontal cortex, and adverse effects on measures of memory function.

10. Wistar rats treated intragastrically with 500 mg/kg bw/day aluminium chloride (~ 100 mg/kg bw/day aluminium) for one month, followed by continuous exposure *via* drinking water containing 1600 ppm aluminium chloride for up to 5 months, showed impaired ability in tests of learning and memory function (Morris water maze). Subsequent treatment for 2 months with *Ginkgo biloba* extract was reported to ameliorate these effects (Gong *et al*., 2005).

11. Zhang *et al*. (2003) reported that they carried out a study to assess the potential of a herbal medicine (*Dipsacus asper*) to protect against cognitive impairment and overexpression of hippocampal β-amyloid protein induced by chronic aluminium exposure in rats (salt not specified). In this study groups of male, Sprague-Dawley rats (total aluminium -treated n = 84) were treated for 90 days with drinking water containing 0.3% aluminium chloride. Treated animals showed decreased performance in the one measure of cognitive function evaluated (passive avoidance task/step through latency), as compared with a group of 15 control animals treated with distilled water (mean latency of aluminium-exposed rats reported as only 19% that for control group). Treated animals also showed increased staining for β-amyloid protein in the brain (123 % more positive Aβ cells in aluminium-treated compared with control rats). Subsequent treatment with *Dipsacus asper* was reported to ameliorate some of these effects (Zhang *et al*., 2003).

12. Brain myelin phospholipid profiles were altered in male, albino rats treated with 100 mg/kg bw/day aluminium chloride (estimated to be ~ 20.3 mg/kg bw/day aluminium) in the diet, for 90 to 100 days. The authors noted that many of the changes observed were similar to those seen in the brains of subjects with Alzheimer's disease (Pandya *et al*., 2004).

13. Dietary supplementation for 4 months with 0.03 g/day aluminium chloride (estimated to be ~ 75 mg/kg bw/day aluminium chloride or ~ 15 mg/kg bw/day aluminium) was reported to alter the kinetic behaviour of brain Na^+/K^+ ATPase in adult, male rats (Silva and Goncalves, 2003).

14. (Fattoretti *et al*., 2003) measured copper, zinc and manganese concentrations in three brain regions (prosencephalon + mesencephalon, PME; cerebellum; pons-medulla, PMD) of aged, male Wistar rats treated with drinking water containing 2 g/l aluminium chloride ($AlCl_3.6H_2O$) (estimated to be ~ 11 mg/kg bw/day aluminium) for 6 months. Aluminium content increased in all three regions. The only other significant changes observed were increased PMD copper content and cerebellum zinc content. Histological examination showed an increase in the hippocampal area

occupied by mossy fibres. Treated animals were reported to show aggressive behaviour. A subsequent report by the same authors, apparently describing the same experiments and data, determined that all the changes (increases) measured in copper, zinc and manganese levels in PME and PMD regions were significant, whilst no significant changes occurred in the cerebellum (Fattoretti *et al*., 2004).

15. Treatment of male albino rats for 90 days with 2% aluminium chloride in drinking water (described by the authors as equivalent to 50 mg/kg bw/day aluminium chloride, or ~ 10 mg/kg bw/day aluminium) was reported to enhance lead deposition when co-treatment with 2.5% lead acetate was given. Some effects of leas and/or aluminium treatment were also noted on brain AChE and lipid peroxidation levels and on motor neurological functions (Shakoor *et al*., 2003).

16. (Kaur and Gill, 2005) reported that intragastric application of 10 mg/kg bw/day aluminium (as lactate) to male, albino rats for 12 weeks altered brain intrasynaptosomal calcium homeostasis.

17. Treatment of male and female HSd:W1 rats with 91.8 mg/kg bw/day aluminium lactate (8.42 mg/kg bw/day aluminium[41]) +/- 3.0 g/kg bw/day ethanol for 90 days by gavage was associated with decreased brain synaptosomal ATPase and AChE activities. The difference was detected two weeks, but not immediately, after discontinuation of treatment (Kohila *et al*., 2004).

18. Indicators of lipid peroxidation and lactate dehydrogenase (LDH) activity were increased, whilst AChE activity was decreased, in the brains of male Sprague-Dawley rats treated orally with 34 mg/kg bw aluminium chloride, on alternate days, for a period of 30 days (El Demerdash, 2004).

Mice

19. Aluminium accumulated in the brains and other organs of male ddy mice given drinking water supplemented with 0.1 mg/ml (~ 16.7 mg/kg bw/day) aluminium, as chloride (ionic) or maltolate (complex), for up to 120 days. In aluminium maltolate-treated rats, brain aluminium accumulation peaked at 60 days, then fell, which the authors interpreted as suggesting that aluminium accumulation in the brain is a reversible process. Brain tissue from aluminium maltolate-treated, but not aluminium chloride-treated animals showed indicators of oxidative stress (TBARS and NOx levels), and clusters of neurofilament cells upon immunostaining (Kaneko *et al*., 2004).

20. Increased levels of some indicators of inflammation were observed in the brains of male B/6C3F1 mice treated with drinking water containing 0.01, 0.01 or 1 mM aluminium, as lactate (0.26, 2.6, 26 mg/l aluminium; ~ 0.043, 0.43, 4.3 mg/kg bw/day aluminium) for 10 wks, but there was no clear pattern of dose-response and no increase in brain aluminium levels (Campbell *et al*., 2004).

Rabbits

[41] It is not entirely clear whether the dose was 91.8 mg/kg bw/day Al lactate or Al.

21. Groups of 6 male New Zealand white rabbits were treated every other day, by gavage, with 40 mg/kg bw L-ascorbic acid (AA) and/or 34 mg/kg bw aluminium[42] (cited by the authors as 1/25 LD_{50}), for 16 weeks. Aluminium treatment was associated with indicators of increased oxidative damage in plasma, liver, brain, testes and kidney, decreases in liver and testes AST, ALT, ALP and AcP enzyme activities, whilst plasma, liver, testes and brain LDH activities were increased. The activities of acetylcholinesterase (AChE) were decreased in brain and plasma. Some haematological parameters were also affected. Co-administration of ascorbic acid provided some protection against these effects (Yousef, 2004).

B. Other biological and toxic effects

Human data

Inhalation and effects on the respiratory system

22. (Fishwick *et al*., 2004) reported that workplace exposure to aluminium fume was associated with reduced respiratory function (FEV_1) (at least 5% reduction after 15 min exposure) in welders in New Zealand.

23. Studies have described evaluations of asthmatic manifestations in workers at aluminium smelting plants ("potroom asthma"). It is not currently clear what is the specific cause of these effects (workers are exposed to a mixture of particulates and gases including aluminium oxide), although the major candidate is suggested to be fluoride compounds (Barnard *et al*., 2004; Sjaheim *et al*., 2004).

Dermal absorption and effects

24. A 43 year old woman who presented with bone pain and fatigue showed normal values for biochemical/haematological analyses, but an elevated plasma aluminium concentration of ~ 3.9 μM (~ 10.4 μg/dl, or ~ 100 μg/l) (normal values ~ 10 μg/l or less). Neuropsychologic and electroencephalographic tests were normal. The patient had no history of aluminium antacid use or occupational exposure to aluminium, and raised levels were attributed to use for the preceding 4 years of ~ 1 g/day aluminium-containing antiperspirant cream. Bone pain symptoms disappeared within a few months of discontinuation of antiperspirant use (Guillard *et al*., 2004).

25. (Akyol *et al*., 2004) described the case of a 9 year old boy who exhibited contact sensitivity to aluminium. This was apparent as an accidental finding when positive reactions at all test sites were observed in allergen patch-test evaluations (presumably due to the use of aluminium test chambers). The authors attributed this aluminium sensitivity to prior exposure to aluminium-absorbed vaccines (although they noted that the patient had received his childhood vaccinations without any adverse effects).

26. Some reports have described the development of persistent itching nodules at the site of injection of aluminium-containing vaccines in children (Bergfors *et al*.,

[42] It is not entirely clear from the report whether the dose was 34 mg/kg bw Al or $AlCl_3$.

2003; Netterlid *et al*., 2004; Thierry-Carstensen and Stellfeld, 2004; Frederiksen and Tofte, 2004).

Others

27. (Cimma *et al*., 2004) reported that consuming foods cooked in Al pots was not associated with adverse effects on parameters of calcium metabolism or increased serum aluminium concentrations in young Bangladeshi children with calcium-deficient rickets (*note: data taken from the abstract: the full text of this paper was not available during the preparation of this report)*.

Animal studies

Developmental/reproductive effects

28. (Wiles *et al*., 2003) evaluated the bioavailability and toxicological effects of montmorillonite clays (which are frequently added to animal feeds, and of which aluminium is a major component) by supplementing clay minerals to pregnant Sprague-Dawley rats throughout pregnancy at a level of 2% (w/w). Aluminium was not detected above background levels in any tissues evaluated and no effects were seen on fetal or maternal toxicity *(note: these data taken from the abstract: the full text of this paper was not available during the preparation of this report)*.

Haematological effects

29. Groups of adult female Wistar rats were exposed for 18 months to tap water, 35 mM sodium citrate solution, or a solution of 35 mM sodium citrate + 30 mM aluminium sulphate (~ 810 mg/l aluminium, or ~ 46 mg/kg bw/day aluminium). Aluminium treatment was associated with significant decreases in red blood cell count, haematocrit, serum iron concentration, and an increase in bone marrow δ-ALA-D activity (Farina *et al*., 2005)

Absorption/bioavailability

30. (Arnich *et al*., 2004) reported a comparative study of the intestinal absorption of aluminium, manganese, nickel and lead in rats using the *in situ* intestinal perfusion technique. Perfused metal solutions at concentrations likely to occur during oral intoxication were used. The authors reported that aluminium (48 and 64 mM), even as citrate complex, crossed the brush border with difficulty (0.4% of the perfused amount). Of this, ~ 60 % was retained in the intestine and the remainder was found in target tissues (*note: data taken from the abstract, the full text of this paper was not available during the preparation of this report)*.

31. (Yumoto *et al*., 2003) used aluminium^{26} chloride as a tracer to measure aluminium^{26} incorporation into the brain of suckling rats by accelerator mass spectrometry. Lactating rats were subcutaneously injected with aluminium^{26} chloride from day 1 to day 20 postpartum. Suckling rats were weaned from day 21 postpartum. From day 5 to day 20 postpartum, the amounts of aluminium^{26} measured in the cerebrum, cerebellum, spinal cord, liver, and kidneys of suckling rats increased significantly. After weaning, the amounts of aluminium^{26} in the liver and kidneys decreased remarkably. Alternatively, in the cerebrum, cerebellum, and spinal cord, as

much as 12 to 20% of the aluminium[26] amounts present on day 20 postpartum remained in the tissues on day 730 postpartum. The authors concluded that considerable amounts of the aluminium[26] taken up into the brain of suckling rats through maternal milk remain in their brain throughout their lifetime (*note: these data taken from the abstract: the full text of this paper was not available during the preparation of this report).*

May 2005

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Appendix 24: Review of the scientific literature on aluminium (May 2005 to July 2006) prepared for the Lowermoor subgroup by the secretariat

Note: this was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

Appendix 25: Review of the scientific literature on aluminium (August 2006 to December 2006) prepared for the Lowermoor subgroup by the secretariat

Note: this was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

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Appendix 26: Review of the scientific literature on aluminium (January 2007 to September 2011) prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London

Note: This was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

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Appendix 27: Review of the scientific literature on aluminium (October 2011 to May 2012) prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London

Note: This was a paper prepared for discussion by the Lowermoor subgroup. It does not necessarily represent the views of the subgroup

Appendix 28: Review paper on metal-metal interactions prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London (January 1970 to February 2003)

Note: this was a paper prepared for discussion by the Lowermoor Subgroup. It does not necessarily represent the views of the subgroup.

See http://cot.food.gov.uk/pdfs/lsgreportapp17.pdf

Appendix 29: Second review paper on metal-metal interactions prepared for the Lowermoor subgroup by the Department of Health Toxicology Unit, Imperial College, London (March 2003 to April 2012)

Note: this was a paper prepared for discussion by the Lowermoor Subgroup. It does not necessarily represent the views of the subgroup.

Appendix 30: Potential study designs to address recommendations for neuropsychological and neurological investigations

Neuropsychological studies

1. Adult study

The purpose of this study would be to assess whether exposure to the contaminated water in the 1988 contamination incident is associated with an increased risk of abnormal neuropsychological status.

This study would examine two groups of individuals:

i. Randomly selected adults of 42 years or over[44] who were living in 1988 in an area supplied with contaminated water and who drank the water.
ii. A reference group of matched individuals who were living in 1988 in an area not supplied with contaminated water and who did not drink the contaminated water.

The subjects would undergo an appropriate assessment of pre-morbid IQ and a battery of neuropsychological tests to assess neuropsychological status. These should address factors such as attention, memory, learning and information processing speed, and the potential moderating influences of depression and anxiety.

2. Developmental study

The purpose of this study would be to assess whether exposure to the contaminated water in the 1988 contamination incident has affected the cognitive development of children who were under 1 year of age at the time of the incident and children who were *in utero* at the time of the incident.

This study would examine the following groups of individuals:

i. Randomly selected individuals who were *in utero* during the period 7 to 10 July 1988, inclusive, and whose mothers were living in an area supplied with contaminated water and drank the water.
ii. A reference group of matched individuals whose mothers were living during the period 7 to 10 July 1988 in an area not supplied with contaminated water and who did not drink the contaminated water.
iii. Randomly selected individuals who were children under one year of age at the time of the contamination incident, were living in an area supplied with contaminated water and who drank the water.
iv. A reference group of matched individuals who were living in an area not supplied with contaminated water and who did not drink the water.

Sufficient individuals should be recruited to inclusion of subjects potentially exposed to the contaminated water during the first, second, and third trimesters and throughout to first postnatal year.

[44] This is to ensure that the individuals were adults (≥ 18 years) at the time of exposure.

The subjects would be tested 1) with a broad neuropsychological test battery which addresses factors such as attention, memory, learning and information processing speed, and the potential moderating influences of depression and anxiety; 2) with an appropriate estimate of pre-morbid IQ, and 3) with questionnaires which assess implications in daily life, such as a quality of life questionnaire.

In both the adult study and the developmental study, applicants should provide the following:

i. Evidence that they are experienced in the administration and interpretation of the neuropsychological tests described in the tender.
ii. Details of how the tests will be analysed and interpreted.
iii. Justification of the group sizes to be used with power calculations.
iv. A consideration of whether it is possible to incorporate a dose-response assessment into the study design.
v. Information on how results will be fed back to individual subjects.
vi. Full costings.

Neuropathological studies

1. Long-term study

This study would use an enhanced protocol from the Medical Research Council (MRC) study on Cognitive Function and Aging (CFAS) and would recruit a random sample from GP practices in Cornwall, including the area which received contaminated water following the 1988 incident. Participants would be invited to donate their brains when they die.

If the study was to determine and compare the emerging incidence of dementia in different regions of Cornwall, a large sample size would be needed to account for attrition. Follow-up would be required every 2 years. To assess whether any higher incidence in the contaminated area was due to aluminium exposure, the study would need to include a detailed lifestyle questionnaire with relevant questions to determine total exposure to aluminium and, if possible, an assessment of body burden of aluminium.

Donated brains would undergo neuropathological examination and aluminium estimation on frozen brain samples. In order to diagnose CAA, the whole brain would be required.

This study was considered to be most likely to produce valuable results. It could produce incidence rates within 4 years to compare with the rest of the country but any work relying on accrued brain donation could take up to 20 years.

The questions which could be answered by such a study are:

- Does the incidence of different types of dementias in individuals living in the area which received contaminated water differ from those in other parts of Cornwall and England and Wales?

- Is any higher incidence in individuals in the contaminated area associated with either a higher past or current intake of aluminium, or with a higher body burden of aluminium?

2. Shorter term study

This would only be appropriate for individuals developing dementia before the age of 65 as health service records are poor for assessing the prevalence of dementia cases in older patients.

The study would use existing data from clinics, GP practices and other sources to draw up a register of early-onset dementia cases. Where possible, cases with CAA should also be included, although these may not present as dementia but with other symptoms. Those whose next-of-kin have symptoms would be invited to donate the brains of the individuals on the register at the time of death for full neuropathological examination and aluminium estimation on frozen brain samples.

The register could then be used to compare early-onset dementia prevalence in the area receiving the contaminated water with that in other parts of Cornwall and, if sufficient brains samples were donated, to compare the incidence of neuropathological diagnoses in different areas and the aluminium concentrations in individuals with the same neuropathology from different areas.

This study would indicate whether the prevalence of early-onset dementia in individuals living in the area which received contaminated water differed from those in other parts of Cornwall.

Appendix 31: Current procedures for the management of chemical incidents

1. At the time of the Lowermoor incident, there were essentially no structures in place to deal with chemical incidents such as pollution of the water supply. Following the incident, a number of new procedures and organisations were established by the Department of Health to improve the arrangements within the NHS for the investigation and public health management of chemical incidents. Together with other major chemical incidents, such as the Bhopal disaster of 1985 and the deliberate release of the organo-phosphate nerve agent sarin on the Tokyo underground in 1995, the Lowermoor incident provided a major impetus to the development of local, regional and national structures for the management of the public health consequences of chemical incidents in the UK.

3. In the mid 1990s, regional service provider units (RSPUs) were established in England, Wales and Scotland, providing advice and support in the event of a chemical incident across the whole of the UK, as well as Eire. Health authorities were required to contract with one of the four RSPUs, which were in Birmingham, Cardiff, London and Newcastle. As a consequence of the "internal market" and the "purchaser-provider" split of the time, the provision of advice to health authorities by RSPUs was geographically diverse, with neighbouring areas frequently receiving support from different RSPUs. The Scottish Centre for Infection and Health (SCIEH) served as the RSPU in Scotland.

4. The establishment of RSPUs was further supplemented by the formation of the National Focus for Chemical Incidents in 1997. Funded by the Department of Health and devolved administrations, the National Focus for Chemical Incidents served to co-ordinate the activities of the RSPUs and collected, integrated and collated information from all agencies and organisations involved in management of incidents, providing timely advice to the DH and/or devolved administrations. All these organisations were consolidated in April 2003 when the Health Protection Agency (HPA) was established.

5. In the event of a chemical contamination of the water supply, there is now a statutory requirement for water companies to inform the Director of Public Health in the primary care trust[45] covering the affected area. The primary care trust would seek support from the local Health Protection Unit (HPU). HPUs are part of the Local and Regional Services Division of the HPA (LARS) and there are about 100 serving the 301 primary care trusts in England, giving support in the event of chemical, biological or radiological incidents.

6. In Wales, the infrastructure is slightly different, with the formation of 23 local health boards instead of primary care trusts. These are supported by the National Public Health Service for Wales, which is composed of consultants in communicable disease control, support staff and the former Public Health Laboratory Services in Wales. Although not a part of the HPA *per se*, the National Public Health Service for Wales has functions analogous to LARS.

[45] Primary Care Trusts have taken over most of the duties of district health authorities. They cover a smaller area and population size than the old health authorities

7. The model, therefore, is for local expertise, whether in LARS or the National Public Health Service for Wales, to provide the initial management of chemical, biological and radiation events.

8. To support the HPUs and National Public Health Service for Wales in the management of chemical incidents, the HPA has 4 regional centres of chemical expertise in its Radiation, Chemicals and Environmental Hazards Directorate (CRCE), which has a headquarters in Harwell, Oxon. CRCE provides 24 hour, 365-day/year support and advice to first line responders, the NHS, local authorities, the HPA and other agencies, and government departments on the likely public health consequences of exposure to environmental chemicals. Advice provided encompasses the principal areas of environmental risk assessment and decontamination, modelling and sampling, clinical management and biological sampling, public health consequences, risk communication and epidemiological follow up.

9. The local structures, whether primary care trusts and HPU or local health boards and National Public Health Service for Wales, would decide, in discussion with other relevant bodies, what action was necessary to ensure protection of public health, and whether any follow-up action should be taken and, if so, what the action should be.

The Water Industry

10. Changes in the organisation and regulation of the water industry were already planned at the time of the incident. The legal framework before and after 1989 is outlined in Appendix 7 of this report. The incident itself led to at least three changes: immediate new procedures by water companies to prevent such an incident happening again (eg stringent procedures for checking and supervision of chemical deliveries, improved monitoring of the water treatment process and of the finished water); a new criminal offence of supplying water unfit for human consumption; and, as mentioned above, a statutory requirement for water companies to inform the NHS in the event of an incident.

11. Also, with the privatisation of the water industry, the Drinking Water Inspectorate (DWI) was established. This body acts as a technical auditor. It has three main functions: assessment of compliance data against statutory standards; inspection of sites, procedures and policies in relation to the supply and treatment of drinking water; and assessment of water quality incidents. In the event of an incident, it can take enforcement action and initiate prosecutions under Section 70 of the Water Industry Act 1991. DWI is notified of all events and, where appropriate, notifies the HPA and other stakeholders based on an assessment of the individual circumstances. In the case of major incidents (not just pollution incidents but, for example, plane crashes which might affect the water supply) the water company must also notify the Department of the Environment, Food and Rural Affairs (Defra) which oversees water-related major emergencies. Water companies must have contingency plans in place in the event of an emergency eg provision of alternative supplies to the public.

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Appendix 32: Declaration of LSG Members' Interests

To be completed