Hong Kong College of Paediatricians
NEWSLETTER April 2004

Editors: Prof. Leung Nai Kong, Drs Albert Martin Li, Wong Sik Nin, Chris Woo

Published by the Hong Kong College of Paediatricians

Room 808, HKAM Building, 99 Wong Chuk Hang Road, Hong Kong
28718871 27851850 hkcpaed@netvigator.com

Website: http//www.paediatrician.org.hk

Message from the Editors

It has been a year since the global SARS outbreak which posed a great impact on our health care system and society. In the past few months, the avian flu also had significant influence on many Asian countries. Thanks to the Hong Kong health care system that equips with vigilant surveillance on infectious diseases and we have kept ourselves free from avian flu and SARS so far in 2004. We hope we can keep up with our good work and stay alert to cope with all these potential threats to our health care system.

Message from the Council

Coopted Council Members 2004

Congratulations to Dr Chan Hoi-shan Sophelia and Dr Tong Tsz-fun for being nominated as the co-opted Council Members of HKCPaed for one year. Dr Chan works in Child Assessment Service and Dr Tong works in Department of Paediatrics, Kwong Wah Hospital. They will certainly help to bring the views of our young Fellows and Members to the Council.

Congratulations to Our Presidents

Our sincere congratulations go to Professor Fok Tai Fai, our President for being appointed the Dean of the Faculty of Medicine of the Chinese University of Hong Kong. He will assume the office in July 2004.

Congratulations also go to Professor Leung Nai Kong, our Immediate Past President for being elected an Honorary Fellow of the Royal College of Paediatrics and Child Health (RCPCH). Professor Leung received the fellowship certificate from Princess Royal at the Annual Meeting of the RCPCH in York on 1 April 2004.

Congratulations

Congratulations to the following 8 candidates who passed the MRCPCH Pt II Clinical Examination in Feb 2004:

1) Dr Chan Wai Ming
2) Dr Hau Wai Lok
3) Dr Kwok Mei Kwun
4) Dr Lau Wei Sze, Vercia
5) Dr Yuen Lai Kei
6) Dr Yu Wing Sze, Margaret
7) Dr Hui Wai Han
8) Dr Kwok Man Lai

Congratulations also go to the following members who passed the recent Exit Assessment in December 2003 and were admitted as Fellows of our College:

1) Dr Chan Cheong Wai
2) Dr Choi Mui Sum
3) Dr Chow Wing Cheong
4) Dr Fong Kwok Wah
5) Dr Fung Po Gee, Geneveieve
6) Dr Ho Shing Fai
7) Dr Poon Wing Kit, Grace
8) Dr Sit Sou Chi
9) Dr Tay Ming Kut

10) Dr Wong Chi On, Simon
11) Dr Yau Kin Cheong, Eric

Accreditation Committee

The second accreditation exercise for all paediatric units have been completed in late 2003. The Committee has encountered and the Council has discussed about a number of issues. The following resolutions have been made which will supplement the published guideline on accreditation of training units:

1. In training units with age-orientated wards, the rotation through all such wards within the 3-year Basic Training Programme should be even and qualitative-measured.
2. In training units with established subspecialty teams, rotation of trainees through several subspecialty teams may be viewed as 18 months of general paediatrics training in the core programme of Basic Training. Such subspecialty team rotations should preferably be not more than 3 months, but definitely not more than 6 months for each subspecialty. Trainees should not rotate through the same subspecialties again in the one-year flexible programme (except for neonatology in which a trainee can be trained for another 6-month in the flexible programme).
3. A training team is governed by the number of patients looked after per day (10 – 30 beds) and can have one or more trainers. However, a training team can have a maximum of 3 trainees – either 2 Basic and 1 Higher Trainees, or 1 Basic and 2 Higher Trainees.
4. At any one time a trainer/training team cannot supervise more than 3 trainees in total (either one Basic plus two Higher Trainees, or two Basic plus one Higher Trainees). Family Medicine trainees or trainees in other training programmes working in the same training centre will be counted in the trainer: trainee ratio.

In addition, the Council has adopted the following resolution in its Council meeting on 16 March 2004: that a trainer for Basic Training Programme should be a College Fellow, and a trainer for Higher Training Programme should have a minimum of 3 years post-Fellowship experience in an accredited training centre. This new rule will be implemented with immediate effect, i.e. applied to new applicants. All existing Trainers will be Trainers for both Basic and Higher Training.

Examination Committee

Examination Dates - 2004
MRCPCH Part II New Clinical Examination: 8-9 November 2004
DCH Clinical Examination: 10-11 November 2004

Result of MRCPCH Pt IA & IB Examination, 21 Jan 2004
Overall pass rate for Part IA: 76.2%
Overall pass rate for Part IA & IB: 66.7%

Result of MRCPCH Part II Clinical Examination, 17-18 Feb 2004
Pass rate: 44.4%

Invitation for Funding Application to Conduct Reviews of the Development of Paediatric Subspecialities

To support the development of Paediatric Subspecialties in Hong Kong, Hong Kong College of Paediatricians now invites funding application to conduct reviews of the development of one or more paediatric subspecialties in Hong Kong. Each programme should consist of at least the following components:

1. To review the existing services and facilities
2. To advise on the future development of the services
3. To conduct training programmes
4. To assess training needs
5. To advise on research activities

The purpose of the funding is to support the applicants to invite one or more overseas subspecialty experts to come to Hong Kong to conduct the programme.

The reviews are made possible by a generous donation from the Providence Foundation. The upper limit for funding for one programme is HK$300,000.

Information about the applications can be obtained from the College Secretariat. The deadline of application is **31st August, 2004**.

5th Guangdong-Hong Kong Paediatric Exchange Meeting

Guangdong Branch of the Society of Pediatrics of the Chinese Medical Association and the Hong Kong College of Paediatricians will be hosting a joint scientific meeting on 24 July 2004 at Pao Yue Kong Auditorium, Hong Kong Academy of Medicine Jockey Club building. In addition to invited presentations from Guangdong and Hong Kong, there will be poster presentation and members of both organizations are strongly encouraged to submit papers to make this meeting a success.

If you wish to submit a paper for presentation, please complete the attached abstract submission form (**Appendix I**) and email or send the completed form to the college secretariat.

The deadline for postal and email submission is **20 May and 31 May 2004** respectively.

Please mark down the date of this important event.

HKCP Foundation

Cambodia Medical Mission Trip: jointly organized by Hong Kong College of Paediatricians Foundation Ltd. and the Chinese Rhenish Church Hong Kong Synod Mission

Date: Saturday, October 23, 2004 - Thursday, October 28, 2004

Place: Phnom Penh, nearby villages and poor relocation settlements

Objectives:

- to set up a mobile clinic/simple laboratory and at the same time, cooperate with the local professionals to provide free medical consultations to Cambodian citizens
- to deliver talks and organize seminar on "Community Health"
- to arrange field visits to hospitals and local orphanages
- to assign medicines to professionals and give away basic necessities to locals donated by Hong Kong College of Paediatricians Foundation Ltd.

Cost: approximately HK$4,000 (including visa, round trip air ticket, accommodation and meals). This fee is partially subsidized by Hong Kong College of Paediatricians Foundation Ltd. and The Chinese Rhenish Church Hong Kong Synod Mission.

Deadline for application: 10 September, 2004

Enquiries: For further information, please contact Dr. Ko Wai Keung at 23911661 (e-mail: fmko@netvigator.com) or
Dr. Yau Fai To at 26892286 (e-mail: yauft@ha.org.hk) or
Mrs. Christine Leung at 28718871 (e-mail: hkcpaed@netvigator.com)

2004 Annual Subscription

For those who have not yet send in their Year 2004 subscription to the College, please **act immediately**.

As a reminder, the Year 2004 subscription fee structure for Hong Kong College of Paediatricians are as follows:

Fellows:	**HK$1,500**
Overseas Fellows:	**HK$750 (With approved overseas status)**
Members:	**HK$1,000**
Overseas Members:	**HK$500 (With approved overseas status)**
Associates:	**HK$500**

Should you wish to continue your subscription with Hong Kong College of Paediatricians, kindly send in your cheque payable to "Hong Kong College of Paediatricians" to the College Chamber at Room 808, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong **on or before 30th April, 2004**.

Monitoring of Congenital Malformation

The Department of Health (DH) has initiated a project to collect data for monitoring congenital malformation amongst the local births. The success of this endeavour by the DH will very much depend on the cooperation and participation of paediatricians in Hong Kong. I hope that you can help in this worthwhile project. The notification form will be posted onto the DH's website (www.info.gov.hk/dh/forms/index.htm for English version or www.info.gov.hk/dh/forms/index-c.htm for Chinese version) for downloading.

Position Paper on Exposure to Lead and Mercury in Children and Chelation Therapy

There has been a discussion concerning the exposure to lead and mercury in children and the use of chelation therapy in children in the community. A working group was formed under the College and a position paper had been released. Please find the details in **Appendix II**.

****

CME Subcommittee

Synchronization of CME Cycles for Fellows whose Cycle ends on 30th June 2004

Fellows have been informed that from 1st January 2005, CME requirement will become mandatory for all registered practitioners. The annual practising certificate will be replaced by practising certificate renewable once every 3 years subject to compliance of CME requirement of the last CME cycle. To facilitate this change, the CME cycles for ALL Fellows will end on 31st December 2004 and start afresh on 1st January 2005. In order to achieve this end, CME cycles started within 3 years before 1st January 2005 will have to be counted on a pro-rata basis.

The following table shows the number of CME points each Fellow will need to obtain up to 31st December 2004 according to the date his/her CME cycle starts:

Date when current CME cycle starts / next CME cycle will start	Total number of CME points required by 31st Dec 2004	Number of Category A CME points required by 31st Dec 2004
1st January, 2002	90	30
1st July, 2002	75	25
1st January, 2003	60	20
1st July, 2003	45	15
1st January, 2004	30	10
1st July, 2004	15	5

For the Fellows whose CME cycles will end on 30th June 2004 and restart on 1st July 2004, there have been concerns as to whether there will be enough CME meetings for them to attend to get their pro-rota requirements within 6 months. Hence the HKAM Education Committee has resolved at its last meeting

on 9th March 2004, to allow Colleges to combine the new CME cycle (of 6 months or less) with the previous one, while the CME points requirements will be adjusted on a pro-rata basis.

Thus Fellows whose CME cycle originally end on 30th June 2004, will carry over their accumulated CME points till 31st December 2004 (i.e. their CME cycle will become 3 ½ years). Of course the CME points requirement will be adjusted on a pro-rata basis, i.e. they require 105 total CME points of which 35 will be Category A points by 31st December 2004. Similarly all regulations regarding maximum or minimum CME points for the reported year will be counted on a pro-rata basis.

****

CME Category A Activities

Listed below are CME Category A activities organized by the HKCPaed and various paediatric societies and institutions. For the complete list of Category A activities and Category B activities, please refer to the homepage of the HKCPaed.

The accuracy of the information has been checked according to the details submitted by the responsible organizers. *Members and fellows are reminded to enquire the contact person listed below for last-minute alterations*.

April 2004		
7th April (Wed)	Topic:	**1. A boy presenting with severe anaemia and thrombocytopenia** **2. Case discussion**
	Venue:	Seminar Room 9, 2/F, Block G, Princess Margaret Hospital
	Time:	6:00 pm – 7:30 pm
	Speakers:	1. Dr Lai Wai Ming 2. Dr Joannie Hui
CME Cat. A 2 pts	Organizer:	HK Paediatric Nephrology Society
	Enquiry:	Dr Lai Wai Ming, Tel: 29903754

7th April (Wed)	Topic:	**1. Managing two oncology patients with the same name in the same ward: a case study** **2. Constitutional marrow failure presenting in late adolescence**
	Venue:	Room 1, Block M, G/F, Queen Elizabeth Hospital
	Time:	6:00 pm – 7:30 pm
	Speakers:	1. Dr ACW Lee 2. Dr SC Ling
CME Cat. A 2 pts	Organizer:	HK Paediatric Haematology & Oncology Study Group
	Enquiry:	Dr PW Yau, Tel: 29586741

13th April (Tue)	Topic:	**The role of nutrition in health and disease of infants**
	Venue:	Intercontinental Hotel
	Time:	7:00 pm – 9:00 pm
	Speakers:	Prof Hans Buller & Prof Hugo Hyemans
CME Cat. A 2 pts	Organizer:	Nutricia China Fund
	Enquiry:	Mr Eric Chan, Tel: 28619610

13th April (Tue)	Topic:	**New indications in growth hormone therapy and its optimized use in children**
	Venue:	Ching Room , 4/F, Sheraton Hotel, Kowloon
	Time:	6:45 pm – 7:30 pm
	Speaker:	Prof Pinchas Cohen
CME Cat. A	Organizer:	HK Society of Paediatric Endocrinology and Metabolism
1 pt	Enquiry:	Dr Lee Ching Yin, Tel: 34087911

15th April (Thu)	Topic:	**1. Non-alcoholic steatohepatitis** **2. Gastroenterology update**
	Venue:	Lecture Theatre, Block A, G/F, Queen Elizabeth Hospital
	Time:	7:30 pm – 9:30 pm
	Speakers:	1. Dr Ng Chi Hang 2. Dr Y K Leung
	Organizer:	HK Society of Paediatric Gastroenterology, Hepatology &
CME Cat. A		Nutrition
2 pts	Enquiry:	Dr Leung Ying- kit, Tel: 27710698

23rd April (Fri)	Topic:	**Update on the management of asthma in children**
	Venue:	Sheraton Hotel
	Time:	7:30 pm – 8:30 pm
	Speaker:	Dr Colin Robertson
	Organizers:	HK College of Paediatricians & Merck, Sharp and Dohme
CME Cat. A		(Asia) Ltd
1 pt	Enquiry:	Ms Lolita Cheung, Tel: 28359826

28th April (Wed)	Topic:	**Seminar on Community Paediatrics –** **1. Children developmental assessment pearls and perils** **2. Helping children with developmental problems: the responsibility of paediatricians**
	Venue:	Room 02-041, 2/F, Main Block, Pamela Youde Nethersole Eastern Hospital
	Time:	1:30 pm – 3:00 pm
	Speaker:	Dr Rose Mak
	Organizer:	Department of Paediatrics & Adolescent Medicine, Pamela
CME Cat. A		Youde Nethersole Eastern Hospital
2 pts	Enquiry:	Ms Erica Chung, Tel: 25956410

29th April (Thu)	Topic:	**Education programme for management of disabled children**
	Venue:	Room 01, Multicentre Block B, Pamela Youde Nethersole Eastern Hospital
	Time:	4:30 pm – 5:30 pm
CME Cat. A	Organizer:	Comprehensive Paediatric Rehabilitation Centre, PYNEH
1 pt	Enquiry:	Ms Suki Tsang, Tel: 25956860

May 2004		
11th May (Tue)	Topic:	**Medical Teleconference Programme - Topics in Neonatology**
	Venue:	Lecture Theatre, Multicentre Block B, Pamela Youde Nethersole Eastern Hospital
	Time:	7:30 am – 9:30 am
	Organizers:	Department of Paediatrics & Adolescent Medicine, Pamela Youde Nethersole Eastern Hospital & Stanford University School of Medicine
CME Cat. A 2 pts	Enquiry:	Ms Erica Chung, Tel: 25956410

19th May (Wed)	Topic:	**1. Addiction in children and adolescents** **2. Sex and sexual identity problems in adolescents**
	Venue:	Room 02-041, 2/F, Main Block, Pamela Youde Nethersole Eastern Hospital
	Time:	1:30 pm – 3:00 pm
	Speaker:	Dr Tsang Fan Kwong
	Organizer:	Department of Paediatrics & Adolescent Medicine, Pamela Youde Nethersole Eastern Hospital
CME Cat. A 2 pts	Enquiry:	Ms Erica Chung, Tel: 25956410

27th May (Thu)	Topic:	**Meeting of inborn error of metabolism – Kowloon West Cluster**
	Venue:	Room 10, 2/F, Block G, Princess Margaret Hospital
	Time:	3:30 pm – 5:00 pm
	Organizer:	Departments of Paediatrics & Pathology, Princess Margaret Hospital; Clinical Genetics Services, Department of Health
CME Cat. A 2 pts	Enquiry:	Dr Lee Shing Yan, Tel: 29901111

Appendix I

5th Guangdong-Hong Kong Paediatric Exchange Meeting

24 July 2004 Hong Kong

Pao Yue Kong Auditorium, Hong Kong Academy of Medicine

Official Abstract Form

(Please complete this form in BLOCK or TYPE SCRIPT letters. Please give a √ in the appropriate box. Photocopy of this form is acceptable. **Only poster presentation is available at this meeting**)

Presenting author	
Title: ☐ Prof ☐ Dr ☐ Mr. ☐ Mrs. ☐ Ms.	
Last name:	Initials:
Institution:	
City	
Telephone	Fax
Email:	

Co-authors	
To be listed (maximum 2) in the Authors Index in the Final Program	
Last name:	Initials:
Last name:	Initials:

Category

Please check the appropriate box for the category that best describes your abstract.

- ☐ Basic science
- ☐ Cardiology
- ☐ Endocrinology
- ☐ General Paediatrics
- ☐ Gastroenterology
- ☐ Haematology and Oncology
- ☐ Infection and Immunology
- ☐ Intensive care medicine
- ☐ Neonatology
- ☐ Nephrology
- ☐ Neurology and Neuromuscular diseases
- ☐ Respirology and Sleep medicine
- ☐ Others

(About 300 words excluding the title, name(s) of author(s), name of institution and the country of origin)

Abstract Submission Process

Electronic submission instruction

1. Electronic submission is the preferred method.
2. Deadline for submission is **31 May 2004.**

Paper submission instruction

1. Abstracts may also be submitted on paper by mail to the congress
2. Deadline for submission is **20 May 2004.**
3. The entire abstract should be typed or laser-printed within the space provided in the abstract form. .
4. A computer disk (either Macintosh or PC format) containing the text of the abstract must also be included. Label the disk with the name of contact author, the file name and the word processing program and version used.

Submission Regulations

1. More than one abstract could be submitted but a separate submission form is required for each abstract.
2. Once submitted, abstracts will not be returned.
3. The Scientific Programme Committee reserves the right to edit the language content and publish the accepted abstracts in the official publications of the meeting.
4. There is no fee for abstract submission but presenting author selected for presentation must be registered for the meeting as a paying delegate.

Notification

1. Authors who submitted abstract on paper will be acknowledged by mail. Authors who submitted via internet will be notified of receipt online at the end of submission process.
2. Notification of abstract acceptance will be sent to the presenter once accepted by the Scientific Committee via email followed by post not later than **30 June 2004.** .
3. Only the presenting author will be notified of abstract acceptance.

Preparation of Abstracts

1. All abstracts must be submitted and presented in English.
2. Type the abstract using the form overleaf in single-line spacing in Microsoft Word format (6.0 is preferred) using 12-point font of Times New Roman. (maximum 300 words) Any abstract exceeds this limit will be automatically truncated.
3. Please type clearly within the frame, the title of the abstract in BOLD CAPITAL LETTERS, initial and last name of each author (do not include titles e.g. MD, PhD), name of the institution and country of origin on the top of the form, and underline the name of the presenting author. The author's name must be exactly the same in all abstracts when more than one abstract is submitted.
4. The abstract should contain summarized description of the following: a) Objective, b) Method, c) Results and d) Conclusion. Simple tables or graphs (in blank ink) may be included.
5. Standard abbreviations may be used. Special or unusual abbreviation must be placed in parentheses after the full word the first time it appears.
6. Non-proprietary (generic) drug names are preferred and should be written in small letters. Each time the proprietary name of a drug appears, the first letter should be capitalized.

Publication of abstracts

Abstracts will be published in the meeting abstract book.

Congress Secretariat

Address: Hong Kong College of Paediatricians

Room 808

Hong Kong Academy of Medicine Jockey Club Building

99 Wong Chuk Hang Road, Aberdeen

Hong Kong

Tel: 852 28718871

Fax: 852 27851850

E-mail: hkcpaed@netvigator.com

Appendix II

Hong Kong College of Paediatricians

Position paper

on

Exposure to lead and mercury in children and chelation therapy

Working Group Members:
Dr Patricia Ip
Dr Paul Ko
Dr Catherine Lam
Prof Tony Nelson
Prof Virginia Wong

Advisors:
Prof Thomas Chan Yan Keung
Prof C R Kumana

Acknowledgement: Dr Albert Martin Li for assistance in literature search

Endorsed by the Council of the Hong Kong College of Paediatricians 16th March 2004

Summary

As there is relatively little controversy about the need for treatment of acute and chronic lead and mercury poisoning in children, this review focuses on low level exposure to these two metals and the use of chelation therapy.

For lead, although low level exposure may affect children's intellectual development, reduction in the blood lead level does not necessarily correlate with improvement in cognition. Although chelating agents can reduce blood lead levels, this can also be achieved more safely with environmental interventions.

With regard to mercury, major concerns relate to its presence in fish and vaccines, and the hypothesis that it can cause autism. Apart from a few fish high in mercury content identified by the US Food and Drug Administration, common dietary fish in Hong Kong are generally safe. The World Health Organization has recently reaffirmed the safety of thimerosal in vaccines and there is no evidence that autism is related to mercury toxicity. Although newer and safer chelating agents can remove organic mercury from the body, they cannot reverse the damage to the central nervous system.

The use of hair analysis for the screening of lead or mercury toxicity is controversial and is not recommended for routine clinical practice. The use of challenge test as a guide to the necessity for therapy is unreliable and not without danger.

Non-conventional or alternative treatments should be used only in formal research protocols to evaluate their effectiveness. Currently, reduction of environmental pollution and balanced nutrition are considered to be the best strategies to prevent exposure to lead and mercury.

Exposure to lead and chelation therapy

Children can be exposed to lead from many sources including lead in the air from combustion of leaded petrol, licking lead-based paint on furniture and toys, chewing crayons and ingesting contaminated soil particles, especially in children exhibiting pica. Certain populations may be at particular risk e.g. children from fishermen's families in Hong Kong as reported by Yu and Yeung.[1] Over past decades there has been considerable effort to reduce environmental lead exposure with the introduction of legislation related to lead-free petrol and children's products.

Measurement of lead exposure

The Centers for Disease Control and Prevention (CDC)[2] defined in 1991 a blood lead level (BLL) $\geq$10 mcg/dL as an indictor for concern. Lead exposure can be assessed by a number of laboratory means.[3] The standard procedure for determining BLLs is the use of venous blood samples collected properly and analyzed in laboratories with quality assurance programmes. Capillary blood samples from finger prick can be contaminated with environmental lead, and require confirmation with a venous sample when levels are above 10 mcg/dL. The use of hair analysis for assessing lead exposure is not recommended by CDC.[4] The American Academy of Pediatrics (AAP)[5] has stated that the calcium disodium ethylenediaminetetra-acetic acid (EDTA) mobilization (challenge) test is difficult and expensive to perform. The test has the potential to increase lead toxicity when EDTA is used alone, which has made the test "obsolete".

Lead level and development

Although even low levels of lead exposure may affect children’s intellectual development, the threshold at which harmful effects from lead exposure occur is not clearly established. A systemic

review by Pocock et al[6] in 1994 found that doubling the body lead burden (from 10 to 20 mcg/dL blood lead) was associated with a mean deficit in full scale IQ of around 1-2 IQ points. Other explanations for this deficit were also possible e.g. children with a lower IQ might adopt behaviours that could make them more prone to lead uptake. Locally, Chow and Tse[7] reported on the health status of Chinese new immigrant children and found that although 20.7% of the children had BLLs above 0.47 mmol/L (10 mcg/dL), no child had a BLL above 0.96 mmol/L (19 mcg/dL). Some of these children had symptoms, including learning difficulties, that could have been related to lead exposure but these symptoms bear no relationship with BLL.

Chelation therapy and lead levels

O'Connor and Rich[8] in a double-blind placebo-controlled trial found that reduction of elevated BLL could be achieved with environmental remediation as well as chelation therapy with 2,3 dimercaptosuccinic acid (DMSA). However changes in cognitive test results and changes in BLLs with chelation therapy did not always correlate. An earlier observational study in 1993 by Ruff et al[9] reported an improvement of cognitive test scores for children with BLLs between 25 to 55 mcg/dL after chelation with EDTA (with iron therapy when indicated). Tong et al[10] followed a group of children from birth to 11-13 years who lived in the vicinity of a large lead smelter. From 2 years to 11-13 years, there was a fall in the mean BLL but the improvement in cognitive scores did not correlate with the degree of change in BLLs. From 7 years to 11-13 years, cognition was slightly better among children whose BLL declined most but did not reach statistical significance.

In 2001, Rogan et al[11] reported for the Treatment of Lead-exposed Children Trial Group the results of a randomized, placebo-controlled, double blind trial on the effect of succimer (DMSA) in over 700 children with BLLs of 20 to 44 mcg/dL. Although DMSA therapy lowered BLLs, it did not improve scores on tests of cognition, behaviour, or neuropsychological function in the children at the 36 months follow-up. In 2002, Liu et al[12] did another analysis of the results from the above group using change in BLL as the independent variable. By 6 months after randomization, BLLs had fallen by similar amounts in both chelated and placebo groups despite the immediate drops in the chelated group. At the 36 months follow-up, cognitive test scores increased with a fall in BLL in the placebo group only.

CDC has a set of recommendations for action for various BLLs (Table 1).[4] Chelation therapy is only recommended when BLL is $\geq$45 mcg/dL. The AAP[3] has slightly different recommendations in that if BLL is >25 mcg/dL, chelation could be considered after consultation with clinicians experienced in lead toxicity.

Chelation therapy is not without side effects. AAP reviewed the various chelating agents that have been used.[5] As much as 50% of patients experience side effects from dimercaprol (BAL in Oil) which has to be given intramuscularly. Significant haemolysis was reported in patients with glucose-6-phosphate dehydrogenase deficiency. EDTA has to be given parenterally and if used alone in the treatment of patients at risk for encephalopathy, there is a danger of lead redistribution from soft tissues to the central nervous system. In this situation, pre-treatment with BAL has been recommended. Slower infusion rates in patients without the risk of encephalopathy may be safer but loss of zinc could result in zinc deficiency. Careful monitoring of renal and hepatic functions is needed. DMSA can be given orally and only minimally increases the excretion of iron, zinc and calcium. Side effects include mild gastrointestinal upset, malaise, hypersensitivity reactions, transient elevation of liver enzymes and reversible neutropenia. Adverse effects in the longer term are not yet known.

The current situation was summarized by AAP[5] as "Given the lack of data regarding an improvement in outcome associated with any chelation therapy and the lack of sufficient data on safety to exclude

rare but potentially severe side effects, therapy for lower-level exposures should include only environmental and nutritional intervention". If chelation therapy for low-level lead exposure (BLL of 25 to 44 mcg/dL) is considered, it should be undertaken as part of a research protocol.

Table 1

Summary of Recommendations for Children with Confirmed (Venous) Elevated Blood Lead Levels[4]

Blood Lead Level (mcg/dL)				
10-14	**15-19**	**20-44**	**45-69**	**≥70**
Lead education - Dietary - Environmental Follow-up blood lead monitoring	Lead education - Dietary - Environmental Follow-up blood lead monitoring Proceed according to actions for 20-44 mcg/dL if: - a follow-up BLL is in this range at least 3 months after initial venous test or - BLLs increase	Lead education - Dietary - Environmental Follow-up blood lead monitoring Complete history and physical exam Lab work: - Hemoglobin or hematocrit - Iron status Environmental investigation Lead hazard reduction Neurodevelopmental monitoring Abdominal X-ray (if particulate lead ingestion is suspected) with bowel decontamination if indicated	Lead education - Dietary - Environmental Follow-up blood lead monitoring Complete history and physical exam Complete neurological exam Lab work: - Hemoglobin or hematocrit - iron status - FEP or ZPP Environmental investigation Lead hazard reduction Neurodevelopmental monitoring Abdominal X-ray with bowel decontamination if indicated Chelation therapy	Hospitalize and commence chelation therapy Proceed according to actions for 45-69 mcg/dL
The following actions are NOT recommended at any blood lead level:				
- Searching for gingival lead lines - Testing of neurophysiologic function - Evaluation of renal function (except during chelation with EDTA)		- Testing of hair, teeth, or fingernails for lead - Radiographic imaging of long bones - X-ray fluorescence of long bones		

Exposure to mercury and chelation therapy

Children can be exposed to elemental, inorganic and organic mercury. An example of elemental mercury exposure is ingestion of mercury from a broken thermometer. This is generally not a problem to the child as the ingested mercury passes out unchanged.[13] If the mercury is spilt onto the floor, it is

important not to clean up the mercury using a vacuum cleaner as vapourised mercury is rapidly absorbed by the respiratory tract causing acute toxicity. Inorganic mercury in teething powders used to cause acrodynia or 'pink disease' but such teething powders are no longer used. Mercurochrome, once a common household antiseptic, could give rise to extremely high blood mercury levels and acute poisoning after ingestion of 20 ml of 2% of the compound.[14] The major organic mercury compounds of current concern are methyl and ethylmercury.

Mercury in fish

Methylmercury is found in sea sediments and accumulates in predatory fish along the food chain. It was also used as a fungicide. Infants were brain-damaged when mothers ate heavily contaminated fish from industrial release of mercury into Minamata Bay in Japan in the 1950's and bread made from contaminated grain in Iraq in the early 1970's.

A prospective study in the Faroe Islands in the Norwegian Sea[15] found infants of mothers who ate small amounts of cod but had episodic feasts of pilot whale meat with a mean content of methylmercury of 1.9 ppm developed subtle neuropsychological dysfunction. Another prospective study in the Seychelles in the Indian Ocean[16, 17] did not find similar adverse effects in infants followed up to 9 years whose mothers frequently ate fish with relatively low methylmercury content of a mean of < 0.3 ppm. However the mean mercury level in mothers' hair in the Seycelles study (6.8 ppm, range: 0.5-27 ppm) was higher than that in the Faroe Islands (4.3 ppm, range: 0.2-39.1 ppm).

Although the exposure patterns to methylmercury in the mothers of the two studies were different, the US Environmental Protection Agency (EPA) has recommended a limit of mercury exposure of 0.1 mcg/kg/d as a precaution using the results of the Faroe Islands study.[18] The FDA[19] advises pregnant women, and women of childbearing age who may become pregnant, not to eat certain fish with high methylmercury content (> 1 ppm) such as shark, swordfish, king mackerel or tilefish. This advice has also been extended to breast-feeding mothers and young children. Up to 12 ounces a week of other fish can be eaten with smaller portions for children. These recommendations also emphasize the benefit of fish in a balanced diet.

A Hong Kong study on environmental mercury exposure in children by Ip P et al[20] has found that more frequent fish consumption is correlated with a higher blood and hair mercury level. However another study of 29 common dietary fish in Hong Kong, whose mercury content was assessed, has shown that none exceeded the Hong Kong legal limit of 0.5 ppm[21] (Fok TF, personal communication). In June 2003, the Joint Food and Agriculture Organization of the United Nations and World Health Organization Expert Committee on Food Additives (JECFA) revised the provisional tolerable weekly intake (PTWI) for methylmercury from 3.3 mcg to 1.6 mcg per kg body weight per week in order to sufficiently protect the foetus from exposure to methyl mercury through contaminated food eaten by the pregnant mother.[22] As approximately 70% of total mercury in fish is methylmercury, for Hong Kong, around 0.3 kg of mackerel to 5.3 kg of white pomfret could be safely consumed per week (Fok TF personal communication).

Mercury in vaccines

The other organic salt of mercury causing concern is ethylmercury which is metabolized from thiomersal (known as thimerosal in the USA), a preservative in vaccines. Ethylmercury was thought to have similar toxic effects to methylmercury. Ball et al[23] calculated that some infants may be exposed to cumulative levels of mercury during the first 6 months of life that exceeded EPA recommendation. This resulted in the removal of thimerosal from all the vaccines in the US as a precautionary measure. However further studies found that the half-life and toxicity levels of ethyl

and methylmercury are different and the WHO has recently confirmed that it is safe to continue to use vaccines containing thiomersal.[24]

Mercury and autism

Bernard et al[25] in 2001 proposed that autism is a novel form of mercury poisoning. Nelson and Bauman[26] reviewed the evidence for this hypothesis and concluded that mercury poisoning and autism have different clinical and neuropathological features. In Denmark, Madsen et al[27] noted an increase in the incidence of autism despite the discontinuation of thimerosal-containing vaccines. Hviid et al[28] also found in a Danish population-based cohort study that the risk of autism and other autistic spectrum disorder (ASD) did not differ significantly between children vaccinated with vaccines with or without thimerosal. A local study by Ip et al[29] found no significant difference in the hair or blood mercury levels between autistic and normal children. The AAP[30] in their technical report on the diagnosis and management of ASD in children affirmed the lack of any link between mercury exposure and ASD. The report also noted a lack of evidence to support chelation therapy to treat mercury toxicosis in order to improve developmental function and emphasized that chelating agents themselves can have toxic effects and precipitate allergic reactions.

Measurement of mercury exposure

Interpretation of mercury levels need to take into account the type and duration of exposure. Whole blood and urine assays can be used to detect elemental and inorganic mercury exposure. For organic mercury whole blood has to be used, as it is concentrated in the erythrocytes. The reference range from a local laboratory (Prince of Wales Hospital) is less than 10 mcg/L in blood and less than 10 mcg/day in urine. Either a 24-hour urine collection or a spot urine sample adjusted for creatine output should be used. However spot urine mercury alone is very misleading as large variations may occur in the same subject, depending on the hydration state. AAP does not recommend hair analysis for diagnosis of mercury exposure because of the ease of contamination. AAP also discourages the use of provocative chelation tests which have yet to be scientifically validated. [30]

Chelation therapy for mercury

Chelation regimens for mercury were developed for acute mercury poisoning. In theory, a patient can develop subacute or chronic methylmercury poisoning because of excessive intake of fish with a relatively high methylmercury content. In western societies in which all fish sold in the market are closely monitored, there has not been a single report of chronic exposure requiring treatment.

In general a blood mercury level greater than 35 mcg/L and urine concentration over 100 mcg/L requires treatment.[31] As noted, 24-hour urine output or spot urine adjusted for creatine should be used. Dimercaprol and d-penicillamine have been used for chelation but are more toxic. In particular dimercaprol is not recommended for organic mercury toxicity because animal studies have shown an increase in mercury in the brain due to redistribution during treatment. DMSA and sodium dimercaptopropanesulfonate (DMPS) may be used to chelate inorganic, elemental and organic mercury and are safer than the older drugs. They are however not devoid of side effects.[32] Adverse effects of DMSA include gastrointestinal upset, skin rashes, increased serum transaminases, flu-like symptoms, drowsiness and dizziness, and mild to moderate neutropaenia. DMSA should be used with caution in renal impairment and hepatic disease. DMPS can produce skin rashes and increase copper and zinc excretion. The major problem for organic mercury toxicity is that although chelators may remove methyl and ethylmercury from the body, they cannot reverse the damage done to the central nervous system.[13] Hence when balancing the risks and benefits, there is no indication for the use of chelating agents for the treatment of low level exposure to mercury. The most effective and important therapeutic measure for managing excessive exposure to mercury is to identify and remove the source.

Mineral analysis in hair for lead or mercury

Methylmercury can be measured in hair specimens but usually in research settings with rigorous control of contamination.[33] Esteban et al[34] from CDC studied the use of hair lead concentration as a screening method for lead poisoning. The method was considered unacceptable with a sensitivity level of only 57% and with 18% of the children being classified as false negatives. Barret[35] found in 1985 that commercial laboratories in the US gave highly unreliable results of hair analysis for a whole range of minerals and presented potentially frightening reports to clients with various recommendations for the use of food supplements. As laboratory methods may have improved since this time, Seidel et al[36] performed a similar study which was reported in 2001. The study concluded that hair mineral analysis was still unreliable despite being undertaken by "Clinical Laboratory Improvement Act" certified laboratories. Certification of these laboratories was not specifically for hair analysis. The authors recommended that health care practitioners refrain from using such analyses to assess individual nutritional status or suspected environmental exposure. Drasch and Roider[37] assessed hair mineral analysis commercially offered in Germany and came to the same conclusion. This is also the opinion of AAP.[30] Hence the routine use of hair mineral analysis for the screening for lead and mercury toxicity is not recommended.

References:

1. Yu ECL, Yeung CY. Lead poisoning in fishermen's children. HK J of Paediatr 1990;7:62-3.
2. Centers for Disease Control and Prevention. Preventing lead poisoning in young children: a statement by the Centers for Disease Control, October 1991. Atlanta, GA: US Dept of Health and Human Services; 1991.
3. American Academy of Pediatrics, Committee on Environmental Health. Screening for elevated blood lead levels. Pediatrics 1998;101:1072-8.
4. Centers for Disease Control and Prevention. Managing elevated blood lead levels among young children: recommendation from the Advisory Committee on Childhood Lead Poisoning Prevention, March 2002. Atlanta, GA: US Dept of Health and Human Services, Public Health Service; 2002.
5. American Academy of Pediatrics, Committee on Drugs. Treatment guidelines for lead exposure in children. Pediatrics 1995;96:155-60.
6. Pocock SJ, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. Br Med J 1994;309:1189-97.
7. Chow CB, Tse K. Report-survey on the childhealth status of Chinese new immigrants. HK J Paediatr (new series) 2000;5:15-24.
8. O'Connor ME, Rich D. Children with moderately elevated lead levels: is chelation with DMSA helpful? Clin Pediatr 1999;38:325-31.
9. Ruff HA, Bijur PE, Markowitz M, Ma YC, Rosen JF. Declining blood levels and cognitive changes in moderately lead-poisoned children. JAMA 1993;269:1641-6.
10. Tong S, Baghurst PA, Sawyer MG, Burns J, McMichael AJ. Declining blood lead levels and changes in cognitive function during childhood. JAMA 1998;280:1915-9.
11. Rogan WJ, Dietrich KN, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. N Engl J Med 2001;344:1421-6.
12. Liu X, Dietrich KN, Radcliffe J, Ragan NB, Rhoads GG, Rogan WJ. Do children with falling blood lead levels have improved cognition? Pediatrics 2002;110:787-91.
13. Clarkson TW, Magos L, Myers GJ. The toxicology of mercury - current exposures and clinical manifestations. N Engl J Med 2003;349:1731-7.
14. Magarey JA. Absorption of mercurochrome. Lancet 1993;342.1424.
15. Grandjean P, Weihe P, White RF, et al. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol Teratol 1997;19:417-28.

16. Davidson PW, Myers GJ, Cox C, et al. Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: outcomes at 66 months of age in the Seychelles Child Development Study. JAMA 1998;280:701-7.
17. Myers GJ, Davidson PW, Cox C, et al. Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. Lancet 2003;361:1686-92.
18. US Environmental Protection Agency. Mercury Study Report to Congress. Washington, DC: US Environmental Protection Agency; 1997.
19. Center for Food Safety and Applied Nutrition, US Food and Drug Administration. An important message for pregnant women and women of childbearing age who may become pregnant about the risks of mercury in fish. Available at: http://vm.cfsan.fda.gov/~dms/admehg.html Accessed 21 February 2004.
20. Ip P, Wong V, Ho M, Lee J, Wong W. Environmental mercury exposure in children - south China experience. Pediatric International. 2004 in press.
21. Food and Environmental Hygiene Department, HKSAR. Risk of mercury in fish. Risk in brief 2003;13. Available at: http://www.fehd.gov.hk/safefood/report/mercury_fish/report.html Accessed 21 February 2004.
22. Joint Food and Agriculture Organization/WHO Expert Committee on Food Additives. Toxicological recommendations and information on specifications. June 2003. Available at: http://www.who.int/mediacentre/notes/2003/np20/en/ Accessed 21 February 2004.
23. Ball LK, Ball R, Pratt RD. An assessment of thimerosal use in childhood vaccines. Pediatrics 2001;107:1147-54.
24. World health Organization, the Global Advisory Committee on Vaccine Safety. Statement on thiomersal. August 2003. Available at: http://www.who.int/vaccine_safety/topics/thiomersal/statement200308/en/index.html Accessed 21 February, 2004.
25. Bernard S, Enayati A, Redwood L, Roger H, Binstock T. Autism: a novel form of mercury poisoning. Med Hypotheses. 2001;56:462-71.
26. Nelson KB, Bauman, ML. Thimerosal and autism? Pediatrics 2003;111:674-9.
27. Madsen KM, Lauritsen MB, Pedersen CB, et al. Thimerosal and the occurrence of autism: negative ecological evidence from Danish population-based data. Pediatrics 2003;112:604-6.
28. Hviid A, Stellfeld M, Wohlfahrt J, Melbye M. Association between thimerosal-containing vaccine and autism. JAMA 2003;290:1763-6.
29. Ip P, Wong V, Ho M, Lee J, Wong W. Mercury exposure in children with autistic spectrum disorder: a case-control study. J Child Neuro. 2004 in press.
30. American Academy of Pediatrics, Committee on Children with Disabilities. Technical report: The pediatrician's role in the diagnosis and management of autistic spectrum disorder in children. Pediatrics 2001;107(5). URL:
31. http://pediatrics.aappublications.org/cgi/content/full/107/5/e85.
32. Chiang WK. Mercury. In: Ford MD, Delaney KA, Ling LJ, Erickson T, editors. Clinical Toxicology. Philadelphia: WB Saunders Company, 2001:737-43.
33. Sweetman SC. Martindale. Great Britain: The Pharmaceutical Press, 2002.
34. Goldman LR, Shannon MW, the Committee on Environmental Health, American Academy of Pediatrics. Technical report: mercury in the environment: implications for pediatricians. Pediatrics 2001;108:197-205.
35. Esteban E, Rubin C, Jones RL, Noonan G. Arch Environ Health 1999;54:436-40.
36. Barrett S. Commercial hair analysis. Science or scam? JAMA 1985;254:1041-5.
37. Seidel S, Kreutzer R, Smith D, McNeel S, Gilliss D. Assessment of commercial laboratories performing hair mineral analysis. JAMA 2001;285:67-72.
38. Drasch G, Roider G. Assessment of hair mineral analysis commercially offered in Germany. J Trace Elem Med Biol 2002;16:27-31.