INFO LINK
AUTUMN 2009

ISSUE TWENTY TWO

INSIDE THIS ISSUE

• Trace Elements in Health and Disease
• PaLMS Toxicology

Your Future, Your Pathology
Delivering quality comprehensive clinical and laboratory services for all your pathology needs

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Welcome to the Autumn 2009 edition of Infolink.

This edition of Infolink discusses Trace Element testing. PaLMS has invested in expanding the range and sensitivity of trace elements measured through the acquisition of an inductively coupled plasma - mass spectrometer. Some trace elements have clinical relevance in terms of disorders of deficiency or excess and may also be toxic to humans. Lead, mercury, selenium and arsenic are some of the elements most commonly encountered in toxic levels by the PaLMS trace elements laboratory.

The PaLMS Toxicology Laboratory is introduced with the feature article in the following addition of Infolink.

The formation of the northern pathology cluster into the single entity known as Pathology North occurred on 1st January 2009, creating the largest public pathology organisation in NSW.

Pathology North was formed from the amalgamation of the pathology services of:

- Pacific Laboratory Medicine Services (PaLMS)
- Hunter Area Pathology Services (HAPS)
- Mid-North Coast Pathology Service
- Northern Rivers Pathology Service
- Pathology New England

We now offer pathology services “from the Bridge to the Border” between Sydney and Brisbane. Pathology North serves some 2.5 million population and operates across twenty-four University teaching hospital campuses with a geographic span of 1,000 km and an area of 167,000 km². The organisation employs some 1,000 staff, including 80 Senior Medical Staff, 20 Pathology Registrars, 8 Principal Hospital Scientists and 700 Scientific and Technical Staff.

In keeping with the mission of public pathology, Pathology North fosters close relationships and association between academic life and clinical service provision. Our staff includes some 50 Academics, Conjoint Academics, Clinical Academics and Adjunct Academics, of whom 15 are of Professorial status.

Dr. Campbell Tiley
Acting Director PaLMS Pathology
PaLMS Toxicology operates out of Laboratories located at the Macquarie Hospital North Ryde. The laboratory analyses about 500 urine samples per day. We can detect and measure the concentration of a wide range of drugs but our focus is on drugs that have a potential for addiction or non-therapeutic use.

The origins of the laboratory stem back to 1900 when Dr. Eric Sinclair, the then Inspector-General of the Insane, established a Neuropathology Laboratory. Dr. Oliver Latham joined the laboratory in 1905 and was appointed Director in 1917 and retired in 1941. Oliver Latham was an international expert in diseases of the cerebellum.

The laboratory was variously located at Sydney University, Broughton Hall and Callan Park. A laboratory was established at Macquarie Hospital in 1959 and was named ‘The Oliver Latham Laboratories’. This laboratory developed to provide a range of pathology services for all of the State owned Psychiatric hospitals.

Screening for drugs of abuse in commenced in 1972. The service was established to help in the treatments of patients being treated for drug addiction.

Over a period of time routine Pathology testing was transferred to the Public Hospital system. The Oliver Latham Laboratories developed into State Reference laboratories for specialised disciplines such Biochemical Genetics and Toxicology.

In 1992 The Oliver Latham Laboratories were dispersed and integrated into the Public Hospital system. Toxicology became part of the Northern Sydney Pathology Service (now called PaLMS). Toxicology is the only laboratory service still operating on the Macquarie Hospital campus. The laboratory employs about 22 staff.

The samples analysed at Toxicology can be broadly classified as ‘Employment Testing’, ‘Evidentiary Testing’ and Drug Clinic Testing.

Further details of testing undertaken by the toxicology laboratory will be included in a later edition of InfoLink.
Ross Wenzel, Senior Hospital Scientist

Ross Wenzel has worked in pathology 18 years, including 10 years experience in trace elements. He graduated from the University of Technology Sydney with a Bachelor of Applied Science in Biomedical Science in 1994, followed by a Graduate Diploma in Clinical Biochemistry 1996 and a Master of Science by Thesis in 2001 (Arsenic Speciation in Urine by Solvent Extraction / Graphite Furnace Atomic Absorption Spectrometry and Capillary Electrophoresis / Inductively Coupled Plasma Mass Spectrometry). Ross has extensive experience in the determination of trace elements and has worked for PaLMS for many years commencing in 1994. He worked in Biochemistry (1995), Trace Elements (1996-2003), Genetics (2004-2006), and in 2007 commenced working exclusively in the PaLMS Trace Elements Laboratory. Previous roles include working for Astra Pharmaceuticals, CSIRO Division of Exploration Geoscience, Auburn Hospital Pathology, Quinn Pathology Services haematology, SWAHS (Division of Analytical Laboratories Trace Inorganics).

Phone: 9926 7682
Email: rwenzel@nsccahs.health.nsw.gov.au

Dr Christopher Farrell

Dr Christopher Farrell graduated from University of New South Wales with MBBS in 2004. He then worked at Bankstown-Lidcombe Hospital as an Intern and Resident Medical Officer (2005 & 2006), completing terms in cardiology, aged care, emergency and intensive care. Dr Farrell commenced working for PaLMS in 2007 as a Clinical Biochemistry Registrar, spending time in a number of the laboratories including express biochemistry, endocrinology, renal and trace elements. He has found the benefit of working for PaLMS is having access to the latest technology in clinical chemistry.

Phone: 9926 5530
Email: cjfarrell@nsccahs.health.nsw.gov.au
The term ‘trace element’ refers to those elements present in human tissue at concentrations of μg/kg or less. Recently, PaLMS has invested in technology to expand the range and sensitivity of trace elements measured through the acquisition of an inductively coupled plasma - mass spectrometer (ICPMS). Therefore, it is timely to provide a brief overview of the more frequently encountered presentations of trace element deficiency and toxicity. For the more technically minded, we also provide a brief description of the new ICPMS.

While almost any element, or indeed isotope, can be quantified by mass spectrometry, only a limited number of elements have clinical relevance in terms of disorders of deficiency or excess. Those elements for which a specific deficiency syndrome exists are known as the ‘essential’ trace elements. Essential trace elements include iron, zinc, copper, manganese, selenium, iodine, molybdenum, chromium, and cobalt. Deficiencies may result from inadequate intake, disorders of absorption and/or excessive loss.

Conversely, trace elements may also be toxic to humans. This applies both to the essential trace elements, if present in high enough concentration, as well as to a range of ‘non-essential’ trace elements that may be toxic at relatively low concentrations. Lead, mercury, selenium and arsenic are some of the elements most commonly encountered in toxic levels by the PaLMS trace elements laboratory.

TRACE ELEMENT DEFICIENCY

While deficiency states have been reported for a number of trace elements, deficiencies of chromium, cobalt, manganese, molybdenum have not been documented in humans eating natural diets. Iron deficiency is the most common deficiency of trace elements but, iodine, zinc, copper, and selenium also occur. Iodine and selenium status are highly dependent on the availability of these minerals in soil, and in Australia we have relatively low levels of these elements in our environment, increasing the likelihood of deficiency.

Table 1 provides a summary of the more common clinical features of deficiency and the initial investigations that may be considered.

TRACE ELEMENT AND HEAVY METAL TOXICITY

Trace elements have long been recognised as toxic agents. Trace element toxicity may be suspected in an individual on the basis of risk of exposure or symptomatology. Industrial exposure is the most frequent setting for trace element toxicity. However, non-industrial exposure may also occur from environmental contamination, excessive consumption of supplements, even contamination of some complementary medications. Certain clinical features implicate trace element toxicity as a potential differential diagnosis. Respiratory, gastrointestinal, neurological, renal and dermatological manifestations are common to many trace element toxicities.

Not surprisingly, the route of administration frequently informs the clinical presentation. Exposure to fumes or dust containing trace elements may result in respiratory symptoms. ‘Metal fume fever’ caused by exposure to zinc-containing fumes is well recognised. Patients may present with fever, chills, cough, dyspnoea, nausea, headache, myalgia and a metallic taste in the mouth. Similar clinical presentations may occur due to exposure of the respiratory tract to cadmium, chromium, cobalt, copper, manganese, or mercury. In addition, rhinitis, laryngitis and bronchitis may arise from pulmonary exposure to arsenic, beryllium and antimony. Ongoing inflammation from beryllium or antimony exposure may lead to chronic pulmonary disease.

Toxic exposure to trace elements, particularly following oral ingestion, commonly results in gastrointestinal complaints. Abdominal pain, nausea, vomiting and alterations in bowel habit are manifestations common to aluminium, arsenic, cobalt, copper, lead and mercury. Hepatotoxicity may represent arsenic, copper, chromium, or selenium toxicity. Importantly, the copper toxicity in Wilson’s disease may lead to hepatic injury in the absence of other symptoms.

Furthermore, the neurological system is a frequent casualty of trace element toxicity. The central nervous system is a target of numerous elements including aluminium, arsenic, cobalt, copper, lead, manganese, mercury, and selenium. Manifestations may range from subtle neuropsychiatric changes to seizures and coma. Peripheral neuropathy may indicate significant exposure to arsenic, lead or mercury.
The kidneys, and particularly the renal tubules, are sensitive to toxicity from cadmium, copper, mercury, lead, and nickel. The tubular injury may produce proteinuria, Fanconi’s syndrome or frank acute tubular necrosis.

Antimony, arsenic, beryllium, and mercury exposure may cause unusual skin rashes or lesions. Cobalt, chromium and nickel may be responsible for contact dermatitis.

SAMPLE TYPE, COLLECTION AND STORAGE

The very low concentrations of trace elements measured necessitate attention to detail when collecting and processing samples for trace element determinations. This is particularly pertinent to the selection of tubes used for collection. Common contaminants of collection tubes include aluminium, manganese and zinc. Other elements are less likely to be affected by contamination, however, for simplicity PaLMS recommends collecting blood in tubes specifically prepared for trace element analysis. These tubes have a Royal Blue closure and contain sodium heparin as an anticoagulant. They are available on request from PaLMS collecting rooms. Requests for trace element testing must state the individual elements of interest as ‘trace elements’ or ‘heavy metal screen’ are not valid requests.

The most appropriate sample type must also be considered when investigating trace elements. For example, on exposure to organic mercury, as may occur from fish consumption, there is a rise in whole blood mercury because the element concentrates in red cells while plasma and urine mercury show little change. However, exposure to inorganic mercury is best demonstrated from a urine sample. The PaLMS collection manual provides information on sample collection and the trace elements laboratory is happy to be consulted regarding the optimal specimens to collect in a given patient.

REFERENCE INTERVALS


HOW WE MEASURE TRACE ELEMENTS

ICPMS is a rapid, specific and highly sensitive means of measuring trace elements. The technology has been adapted from its traditional role in geological, water quality and environmental laboratories for elemental analysis in biological specimens. ICPMS is capable of measuring some elements below concentrations of one part per billion, analogous to a single drop of water in an Olympic-sized swimming pool.

To achieve this all elements in the relevant sample are first ionised in an argon plasma that reaches temperatures up to 10,000°C, the temperature of the sun’s surface. The ionised elements are then passed through a quadrupole mass filter that separates elements by atomic weight using oscillating electric fields of specific characteristics. ICPMS is capable of accurate quantitation of more than 20 elements from 100μL of blood in less than 3 minutes.

ICPMS may also be used as a sensitive, element specific detector when coupled to a chromatography column to enable the measurement of specific chemical forms of elements present in samples. This is of relevance when different chemical forms differ significantly in toxic properties. For instance, chromium (III) is an essential trace element, while chromium (VI) is toxic and carcinogenic. Utilisation of chromatographic separation prior to ICPMS analysis allows the measurement of individual chemical forms of chromium, arsenic and mercury.

Additionally, ICPMS is able to measure individual isotopes of elements. This capability makes ICPMS a very useful research tool in the study of the biological pathways that various elements participate in. It may also allow sources of lead exposure be traced. A further area of research interest is the use of isotopes as labels for immunoassays in the clinical laboratory. This may lead to significant improvement in the analytical sensitivity of immunoassays. It also makes possible the quantitation of multiple antigens in a single reaction mixture, simply by labelling antibodies specific for various antigens with different isotopes.

References and Further Reading

<table>
<thead>
<tr>
<th>Element</th>
<th>Causes of Deficiency</th>
<th>Presenting Features</th>
<th>Laboratory Tests</th>
<th>Additional Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chromium (Cr)</td>
<td>TPN without Cr supplementation&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Impaired glucose tolerance.</td>
<td></td>
<td>Plasma and serum chromium are not considered useful indicators of chromium deficiency. Deficiency has not been documented in humans eating natural diets.</td>
</tr>
<tr>
<td>Copper (Cu)</td>
<td>Chronic diarrhoea, malabsorption syndromes, foreshortened surgery, high dose zinc supplementation, peritoneal dialysis, TPN with inadequate supplementation, Menkes' disease (rare x-linked condition).</td>
<td>Haematological – anaemia, leucopenia.  Neurological – neuropathy, ataxia, cognitive deficits.</td>
<td>Plasma Cu. Plasma caeruloplasmin.</td>
<td>Elevated plasma copper may be seen in pregnancy, patients taking exogenous oestrogen and in liver disease.</td>
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<tr>
<td>Iodine (I)</td>
<td>Inadequate diet. Prevalence of deficiency is increasing in Australia&lt;sup&gt;3,4&lt;/sup&gt;</td>
<td>Goitre, hypothyroidism and, in the young, impaired central nervous system development.</td>
<td>Serum TSH.</td>
<td>Urine iodine reflects recent intake, and is poor indicator of long-term status. Main use is in epidemiological studies.</td>
</tr>
<tr>
<td>Iron (Fe)</td>
<td>TPN without Mn supplementation.</td>
<td>Impaired growth, skeletal abnormalities, impaired glucose tolerance.</td>
<td></td>
<td>Plasma, blood, and urine manganese are not considered useful indicators of manganese deficiency. Deficiency has not been documented in humans eating natural diets.</td>
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<tr>
<td>Manganese (Mn)</td>
<td>TPN without Mn supplementation, inadequate dietary intake&lt;sup&gt;5,6,7&lt;/sup&gt;</td>
<td>Cardiomyopathy. Putative role in immune function, inflammatory conditions and cardiovascular disease.</td>
<td></td>
<td></td>
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<tr>
<td>Selenium (Se)</td>
<td>TPN without Se supplementation, inadequate dietary intake&lt;sup&gt;5,6,7&lt;/sup&gt;</td>
<td>Anorexia, diarrhoea, immune dysfunction, impaired wound healing, skin lesions, impaired growth, hypogonadism, depression.</td>
<td>Whole blood Se (long-term status). Plasma Se (short-term status). Urine Se (recent dietary intake).</td>
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</tr>
<tr>
<td>Zinc (Zn)</td>
<td>Inadequate diet, malabsorption syndromes, catabolic states, pancreatic disease, TPN with inadequate supplementation.</td>
<td>Anorexia, diarrhoea, immune dysfunction, impaired wound healing, skin lesions, impaired growth, hypogonadism, depression.</td>
<td>Plasma Zn.</td>
<td>Plasma Zn is the most commonly used marker of zinc status. It may be influenced by factors other than zinc status such as hypoalbuminaemia, the acute-phase response, steroid therapy (including oral contraceptive pill use) and pregnancy will lower plasma zinc in subjects without deficiency. However, concentrations below 7μmol/L indicate marked deficiency.</td>
</tr>
<tr>
<td>Element</td>
<td>Source of Exposure</td>
<td>Clinical Effects</td>
<td>Laboratory Tests</td>
<td>Additional Notes</td>
</tr>
<tr>
<td>---------</td>
<td>-------------------</td>
<td>-----------------</td>
<td>-----------------</td>
<td>-----------------</td>
</tr>
<tr>
<td><strong>Aluminium (Al)</strong></td>
<td>Renal failure patients given Al-based phosphate binders or antacids, or with contaminated dialysis fluid. Industrial (as Al dust).</td>
<td>Osteomalacia. Encephalopathy (myoclonus, mental changes, speech disturbances, hallucinations and seizures). Anaemia (microcytic). Cardiomyopathy.</td>
<td>Plasma aluminium (usually ~1.5µmol/L). PTH (~16pmol/L) (inappropriately low in the setting of advanced renal failure).</td>
<td>Whole blood Al. Plasma Al. Urea, creatinine and Cr. Elevated months to years after exposure.</td>
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<tr>
<td><strong>Antimony (Sb)</strong></td>
<td>Industrial (used as alloy and in dyes).</td>
<td>Acute exposure: metallic taste, headache, nausea, dizziness, vomiting, diarrhoea, upper respiratory tract and ocular irritation. Chronic exposure: cardiac arrhythmias, spontaneous abortion, premature birth, and dermatitis. Chronic exposure to dusts can cause pneumoconiosis.</td>
<td>Whole blood Sb. Urine Sb (24-hour or spot Sb: creatinine ratio).</td>
<td>A single meal of seafood can greatly increase total urine arsenic levels. Fish should be eliminated from the diet for five days prior to testing. Elevated urine As levels are confirmed in the laboratory to ensure As is not from seafood consumption by extracting As species of marine origin from those associated with toxicity.</td>
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<tr>
<td><strong>Arsenic (As)</strong></td>
<td>Industrial (multiple applications) or domestic (used as insecticide, herbicide and wood preservative).</td>
<td>Acute: GI – nausea, vomiting, diarrhoea, garlic odour to breath. Cardiovascular – arrhythmias, hypertension. Neurological – encephalopathy (delirium, coma, seizures). Respiratory – laryngitis, bronchitis, rhinitis. Renal – proteinuria, haematuria, acute tubular necrosis. Chronic: Skin lesions, symmetrical sensorimotor polyneuropathy, or any of above features.</td>
<td>Plasma As (24 hour or spot As: creatinine ratio): is the favoured test. Whole blood As: elevated for only ~4 hours after exposure.</td>
<td>Chronic arsenic disease is an immune mediated inflammatory disorder and urine arsenic measurements play no role in its diagnosis or management.</td>
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<tr>
<td><strong>Beryllium (Be)</strong></td>
<td>Industrial (used as alloy and in ceramics) as Be dusts.</td>
<td>Acute: eye, skin and respiratory tract irritation. Chronic: granulomatous disease of the lung, liver, and/or skin.</td>
<td>Plasma Be (24-hour or spot). Whole blood Be (24-hour or spot).</td>
<td>Chronic beryllium disease is an immune mediated inflammatory disorder and urine beryllium measurements play no role in its diagnosis or management.</td>
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<tr>
<td><strong>Cadmium (Cd)</strong></td>
<td>Industrial (used in electropolishing, zinc and lead smelting and as pigment).</td>
<td>Acute: fever, nasopharyngeal irritation, pneumonitis, headaches, malaise, abdominal pain, nausea and diarrhoea. Chronic: proteinuria, renal dysfunction, osteomalacia, anaemia.</td>
<td>Whole blood Cd Urine Cd (24-hour or spot). Plasma Cd.</td>
<td>Whole blood cadmium is the best indicator for chronic exposure, but less reliable for recent exposure (remains elevated long after exposure ceased).</td>
</tr>
<tr>
<td><strong>Chromium (Cr)</strong></td>
<td>Industrial (used in stainless steel, chrome plating, tanning leather, as a dye, a cleaning solution and anti-corrosive).</td>
<td>Respiratory – dyspnoea, cough, wheezing, rhinitis, increased risk lung cancer. GI – abdominal pain, vomiting, GI bleeding. Renal – proteinuria, haematuria, acute renal injury. Contact dermatitis.</td>
<td>Whole blood Pb Urine Pb (24-hour or spot Pb: creatinine ratio). Plasma Pb.</td>
<td>Urine chromium reflects intake over the previous ~2 years.</td>
</tr>
<tr>
<td><strong>Cobalt (Co)</strong></td>
<td>Industrial (used as an alloy).</td>
<td>Acute exposure: pulmonary oedema, allergy, nausea, vomiting, haemorrhage and renal failure. Chronic toxicity may include interstitial lung disease, skin irritation, allergy, intestinal irritations, nausea, cardiomyopathy, memory loss, nerve deafness, impaired vision, haematological disorders.</td>
<td>Whole blood Mn. Plasma Mn. Urine Mn.</td>
<td>Chronic cobalt exposure may cause anaemia.</td>
</tr>
<tr>
<td><strong>Lead (Pb)</strong></td>
<td>Industrial (used in batteries, solder, muntions, car radiators) or domestic (paint produced prior to 1970, contaminated soil) and contaminated Ayurvedic preparations.</td>
<td>Acute: GI – abdominal pain, constipation, anorexia. Neurological – memory deficits, impaired concentration, peripheral neuropathy. Haematological – anaemia. Chronic: myalgia, fatigue, insomnia, anorexia, impaired memory, poor concentration, renal impairment, hypertension.</td>
<td>Whole blood Pb. Urine Pb (24-hour or spot Pb: creatinine ratio).</td>
<td>Whole blood is the preferred sample type for investigating most cases of Pb exposure. Urine Pb is used to assess exposure to infrequently encountered organic lead. For further information on lead exposure from seafood, including consumption advice during pregnancy, refer to <a href="http://www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf">www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf</a>.</td>
</tr>
<tr>
<td><strong>Manganese (Mn)</strong></td>
<td>Industry [as dust or fumes]: during mining of various ores, used as alloy, binding agent in red brick, pigment in some paints. Prolonged cholestasis (impaired excretion).</td>
<td>Acute: fever, pneumonitis. Chronic: parkinsonism, psychiatric disturbances.</td>
<td>Plasma Mn. Whole blood Mn. Urine Mn.</td>
<td>Chronic manganese exposure may cause anaemia.</td>
</tr>
<tr>
<td><strong>Mercury (Hg)</strong></td>
<td>Inorganic mercury: Industrial (production of mirrors, thermometers, plastics, paper, incandescent lights) Organic mercury: Contaminated fish.</td>
<td>Acute: Inhalation of fumes – fever, dyspnoea, chest pain. Oral ingestions – abdominal pain, vomiting, PR bleeding, metallic taste, discoloration of mucous membranes, renal impairment. Chronic: intention tremor, ataxia, paraesthesia, visual/auditory/speech impairment, renal toxicity.</td>
<td>Whole blood Hg. Plasma Hg. Urine Hg (24 hour or spot Hg: creatinine ratio). Whole blood Hg.</td>
<td>Whole blood Hg is best for assessing exposure to inorganic mercury. Whole blood Hg is best for assessing exposure to organic mercury, including that from contaminated fish. For further information on mercury exposure from seafood, including consumption advice during pregnancy, refer to <a href="http://www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf">www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf</a>.</td>
</tr>
<tr>
<td><strong>Selenium (Se)</strong></td>
<td>Consumption of Se-containing supplements in excess of recommended dose.</td>
<td>Acute: abdominal pain, vomiting, garlicky breath, haemolysis, hepatic necrosis, cerebral and pulmonary oedema. Chronic: hair and nail brittleness, skin rash, garlicky breath, fatigue, irritability, nausea and vomiting.</td>
<td>Whole blood Se (long-term status). Plasma Se (short-term status). Urine Se (recent dietary intake).</td>
<td>Whole blood Se is used to assess exposure to Se. Urine Se is used to assess exposure to inorganic Se. For further information on Se exposure from seafood, including consumption advice during pregnancy, refer to <a href="http://www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf">www.foodstandards.gov.au/_srcfiles/brochure_mercury_in_fish_2004v2.pdf</a>.</td>
</tr>
</tbody>
</table>
### Past Issues

<table>
<thead>
<tr>
<th>Issue</th>
<th>Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twenty One</td>
<td>Cytogenetic advances in the detection of prognostic factors in B cell malignancies, Trace Elements in Health and Disease</td>
</tr>
<tr>
<td>Twenty</td>
<td>Human Papilloma Virus, Expanding Molecular Biology</td>
</tr>
<tr>
<td>Nineteen</td>
<td>B-type Natriuretic Peptide (BNP)</td>
</tr>
<tr>
<td>Eighteen</td>
<td>Down Syndrome - Population Screening</td>
</tr>
<tr>
<td>Seventeen,</td>
<td>Anti-Neutrophil Cytoplasmic Antibodies - ANCA</td>
</tr>
<tr>
<td>Sixteen,</td>
<td>Changes in the use of RH D immunoglobulin products in Australia: Introduction of Antenatal Prophylaxis</td>
</tr>
<tr>
<td>Fifteen,</td>
<td>Laboratory Diagnosis for Infection with Human Immunodeficiency Virus (HIV)</td>
</tr>
<tr>
<td>Fourteen,</td>
<td>Hereditary Haemochromatosis</td>
</tr>
<tr>
<td>Thirteen,</td>
<td>Infectious Mononucleosis</td>
</tr>
<tr>
<td>Twelve,</td>
<td>Thrombophilia</td>
</tr>
<tr>
<td></td>
<td>Faecal Occult Blood Testing</td>
</tr>
<tr>
<td>Eleven,</td>
<td>Acute Phase Response</td>
</tr>
<tr>
<td></td>
<td>New Targeted Treatment for CML</td>
</tr>
<tr>
<td>Ten,</td>
<td>Laboratory Markers of Bone Metabolism</td>
</tr>
<tr>
<td>Nine,</td>
<td>Thyroid Function Tests - Dead Easy or Impossible</td>
</tr>
<tr>
<td>Eight,</td>
<td>Synovial Fluid Analysis in the Investigation of Arthritis</td>
</tr>
<tr>
<td>Seven,</td>
<td>The Biopsy Diagnosis of Gastritis</td>
</tr>
<tr>
<td>Six,</td>
<td>Diabetes Mellitus</td>
</tr>
<tr>
<td>Five,</td>
<td>New Markers for Coronary Artery Diseases</td>
</tr>
<tr>
<td>Four,</td>
<td>Auto-Antibodies in Connective Tissue Diseases</td>
</tr>
<tr>
<td>Three,</td>
<td>Laboratory Tests to Detect Allergiv Reactions to Drugs</td>
</tr>
<tr>
<td>Two,</td>
<td>Investigation of Hypertension</td>
</tr>
<tr>
<td>One,</td>
<td>Important Concepts in Laboratory Utilisation</td>
</tr>
</tbody>
</table>

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Please photocopy this page and fax to Vicki Samson, PaLMS Marketing and Communication Manager (02) 9437 1477
Or send an email: vsamson@nsccahs.health.nsw.gov.au
## PalMS COLLECTION FACILITIES

### Northern Sydney
**Enquiries** 9926 6066 or 1300 30 PaLMS

**Chatswood**  
Katherine Street Medical Centre  
Unit 235, 1 Katherine Street  
Chatswood NSW 2067  
Ph: (02) 9411 8197  
Monday to Friday: 8.00am to 4.30pm

**Dee Why**  
Unit 1 "Seascape"  
22-26 Fisher Road  
Dee Why NSW 2099  
Ph: (02) 9982 9433  
Monday to Friday: 8.30am to 5.00pm

**Eastwood/Ryde Hospital**  
Denistone Road, Eastwood  
Ryde NSW 2122  
Ph: (02) 9858 7542  
Monday to Friday: 8.00am to 4.30pm  
Saturday: 9.00am to 12.00 midday

**Hornsby Ku-ring-gai Hospital**  
Adj. Main Entrance Palmerston Rd  
Hornsby NSW 2077  
Ph: (02) 9477 9527  
Monday to Friday: 8.30am to 5.00pm  
Saturday: 8.30am to 12.00 midday

**Manly Hospital**  
West Wing, Darley Road  
Manly NSW 2095  
Ph: (02) 9976 9686  
Monday to Friday: 8.00am to 4.30pm  
Saturday: 9.00am to 12.00 midday

**Mona Vale Hospital**  
Level 2, Coronation Street  
Mona Vale NSW 2103  
Ph: (02) 9998 0278  
Monday to Friday: 8.00am to 4.30pm  
Saturday: 9.00am to 12.00 midday

**Neutral Bay**  
Suite 3, 5 Waters Road  
Neutral Bay NSW 2089  
Ph: (02) 8969 6157  
Monday to Friday: 8.30am to 5.00pm

**St Leonards**  
North Shore Private Hospital  
PaLMS Collection Suite,  
St Leonards NSW 2065  
Ground Floor, Westbourne St  
Ph: (02) 8425 3066  
Monday to Friday: 8.00am to 6.00pm  
Saturday: 9.00 to 1.00pm

**St Leonards**  
Royal North Shore Hospital  
Clinic 13, Outpatient Department,  
Level 3, Pacific Highway  
St Leonards NSW 2065  
Ph: (02) 9926 7557  
Monday to Friday: 8.30am to 5.00pm

### Central Coast
**Enquiries** (02) 4230 3375

**Gosford Hospital**  
Central Coast Specialist Centre  
Holden Street  
Gosford NSW 2250  
Ph: (02) 4320 5607  
Monday to Thursday: 9.00am to 3.00pm

**Outpatient Department**  
Level 3, Holden Street  
Gosford Hospital  
Ph: (02) 4320 3767  
Monday to Friday: 8.00am to 4.00pm

**Wyong Hospital**  
Pathology Collection Centre  
(near Outpatients Clinic)  
Pacific Highway  
Wyong NSW 2259  
Ph: (02) 4394 7554  
Monday to Friday: 8.00am to 2.00pm

### Mid North Coast
**Enquiries: Please contact your local hospital collection centre telephone number**

**Coffs Harbour Hospital**  
345 Pacific Highway  
Coffs Harbour South NSW 2450  
Ph: (02) 66 567500  
Monday to Friday: 8.00am to 5.00pm

**Forster**  
10-12 South Street  
Forster NSW 2428  
Ph: (02) 65 547569  
Monday to Friday: 8.00am to 4.30pm

**Kempsey**  
3/24 Clyde Street  
Kempsey NSW 2440  
Ph: (02) 65 628951  
Monday to Friday: 8.00am to 4.30pm

**Kempsey Hospital**  
119 River Street  
Kempsey NSW 2440  
Ph: (02) 65 620281  
Monday to Friday: 8.00am to 5.00pm

**Macksville Hospital**  
Boundary Street  
Macksville NSW 2447  
Ph: (02) 65 680624  
Monday to Friday: 8.00am to 4.30pm

### Manning Base Hospital
**30-35 York Street**  
Taree NSW 2430  
Ph: (02) 65 929343  
Monday to Friday: 8.00am to 4.00pm

### Taree
**17 York Street**  
Taree NSW 2430  
Ph: (02) 65 524377  
Monday to Friday: 8.00am to 5.00pm

### Wauchope Hospital
**69 High Street**  
Wauchope NSW 2446  
Ph: (02) 65 851300  
Monday to Friday: 8.30am to 11.30pm

### Northern Rivers
**Enquiries: Please contact your local hospital collection centre telephone number**

**Ballina District Hospital**  
Fox Street  
Ballina NSW 2478  
Ph: (02) 6620 6270  
Monday to Friday: 9.00am - 12.00pm

**Grafton Base Hospital**  
Arthur Street  
Grafton NSW 2460  
Ph: (02) 6640 2234  
Monday to Friday: 8.30am to 5.00pm

**Lismore Base Hospital**  
76 Uralba Street  
Lismore NSW 2480  
Ph: (02) 6620 2900  
Monday to Friday: 8.30am to 5.00pm

**Maclean Hospital**  
21 Union Street  
Maclean NSW 2463  
Ph: (02) 6640 0163  
Monday to Friday: 9.00am - 3.00pm

**Murwillumbah District Hospital**  
Ewing Street  
Murwillumbah NSW 2484  
Ph: (02) 6672 0263  
Monday to Friday 7.30am - 5.00pm

**The Tweed Hospital**  
Florence Street  
Tweed Heads NSW 2485  
Ph: (07) 5506 7425  
Monday to Friday: 8.00am to 5.00pm

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For further information visit www.palms.com.au