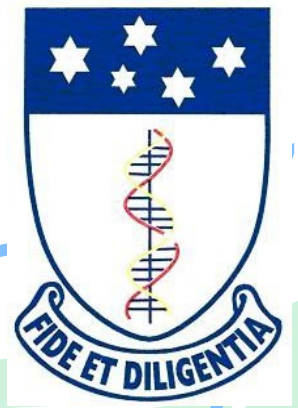
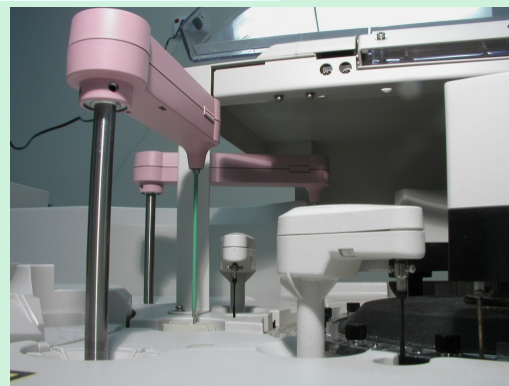
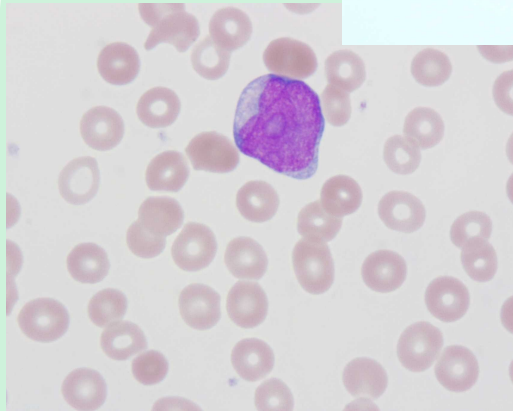
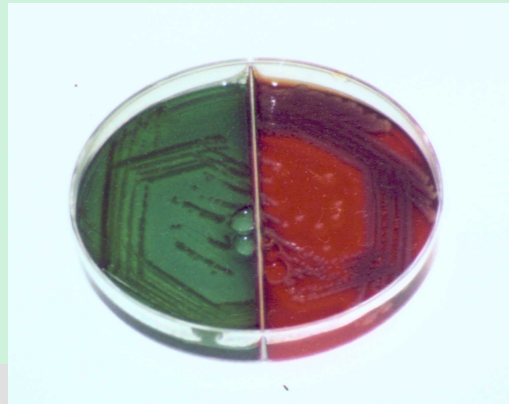


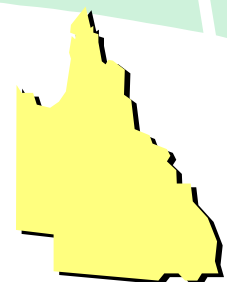
AIMS



Australian Institute of Medical Scientists



AIMS Qld Branch Newsletter
February 2007



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Disclaimer:

Opinions expressed in this newsletter are not necessarily the views of AIMS or the editor, or the branch committee.

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CHAIR REPORT

Welcome to another wonderful year for the Queensland Branch. Your committee has been hard at work planning the various continuing education events for the year, and we are looking forward to seeing you at our meetings.

Our first meeting will be the AIMS AACB Clinical Review, to be held during Pathology Week. Speakers from Chemical Pathology, Haematology and Microbiology will take you on a journey through the fascinating world of sepsis. The event will be held at the Royal Brisbane Hospital Patient Education Centre on Tuesday 27 February 2007, commencing at 6.00pm.

Pathology Week, from 24 February to 4 March, is a celebration of the outstanding contribution medical technologists make to patient outcomes. Please support your local branch by promoting our industry and celebrating our professional achievements.

The Queensland Branch is actively preparing for our State Scientific Meeting to be held in late July. At this time, we anticipate a two-day program combining workshops and case presentations. We are also planning workshops for November, which will focus upon Haematology.

Warm Regards
Lara

CONTINUING EDUCATION CALENDAR 2007**AIMS QLD BRANCH**

Clinical Review, RBWH Tuesday 27th February

CHIGM (Haematology and Coagulation Interest Group)

Tuesday March 6th

Thursday May 10th

Wednesday August 1st

Monday November 12th

These meetings are held at the ESA Leukaemia Village, 64 Raymond Tce, South Brisbane commencing at 6pm.

Contact Robyn Wells, QHPS-Central ph 3636 8466

Australian Society of Microbiology (ASM)

March 7th PAH

March 28th PAH

April 18th S&N

May 16th QML

June 20th PAH

August 22nd Mater

Sept 19th QHPS-Central

October 17th PAH

Nov 28th TPCH

These meetings are held as far as possible on the third Wednesday of the month, commencing at 6pm for refreshments and talks at 6.30pm.

For further information, contact Jacqueline Schooneveldt

Jacqueline_Schooneveldt@health.qld.gov.au

AIMS Tropical Division

June long weekend meeting 9-11th June, Mackay

Contact: Jeffrey.Warner@jcu.edu.au

MEDICAL TERMINOLOGY Part 8

Here are some more clinical notes that have been seen recently:

Lumpoproliferative disorder (lymphoproliferative)

Cold eggs (cold aggs)

Gem cell tumour (germ cell tumour)

?Thai minor (Thal minor)

?Sopsis (sepsis)

Viral malaria (vivax malaria)

If you come across any that make you chuckle, please send them in to me Robyn_Wells@health.qld.gov.au (and thanks to those who did)

NEWSLETTER DEADLINE

Articles/notices/reports/letters for the next issue.

Please submit articles etc. as word or RTF files either on disc or as email attachments (preferred). Items for publication should be received by 31st March 2007

Email: Robyn_Wells@health.qld.gov.au

Post: Editor

AIMS Qld Branch Newsletter

PO Box 8027

APACE NEWS

The Washington University CPD scheme (accessed through the members lounge) attracts APACE credits. The committee have allocated 1 credit for each hour of reading for the modules and 5 credits for passing the test at the completion of the modules. This is a great way to get those extra credits for your application, as well as refreshing your knowledge for your special discipline, or increasing your knowledge in another area.

Congratulations go to the following Qld members who have attained their APACE accreditation in the last 2 months:

Robert Flatman, Suzette Gordon, Robyn Wells, Jeff Warner, Kayleen Geyer and Daniela Arbuckle.



Topical Science for the Tropics


Call for Abstracts now open for AIMS Tropical Division June Conference in Mackay. See link below.

<http://www.jcu.edu.au/school/bms/units/aims/index.html>

PATHOLOGY WEEK 2007 26 February to 4 March


Pathology Week 2007, a combined event of all organisations in pathology (AIMS, AACB, ASM, RCPA, HGSA, ASC, ANZSBT) will be held 26 February to 4 March. All AIMS Branches are organising events and displays and information will be placed on the AIMS web site as it comes to hand.






Any enquiries aimsnat@aims.org.au





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ascia  **ESA** **HSANZ**  **RCPA** **NCOPP**

% * # Brain Teaser Page @ % *

1. Sudoku (Su-difficult)

	6	5	2	9	7	1		
				3				2
7	2				4		5	
2	8							
9			3		1			5
							9	4
	5		6				1	8
6				1				
		2	4	7	8	5	3	

Sudoku puzzles do not require any skill with arithmetic. It is all based on logic! The numbers 1-9 can only appear once in each horizontal line, vertical line and 3x3 block within the sudoku square.

2. Which of the following dishes is the odd one out?

- Moussaka
- Risotto
- Tiramisu
- Lasagne
- Chow Mein
- Coq au vin
- Vindaloo

3. The computer term "pixel" is derived from which two words?

4. What is the term for a period of 7 years?

5. In which hand does the Statue of Liberty hold her torch?

Can't work them out - Answers are in the back!

CONTINUING EDUCATION FILM SET JANUARY 2007

The suggested responses to the set of films distributed in January as part of the AIMS continuing Education programme are listed.

1. Waldenström's macroglobulinaemia

Monoclonal Kappa IgM paraprotein present (56 g/L), lymphocytes have increased pale blue cytoplasm and there is marked rouleaux and protein background on the film.

2. Hairy cell leukaemia

Classic hairy cells in the peripheral blood, with 64% in BM. Cell markers: CD11c (strong), CD 19 (strong), CD 20 (strong), CD23 (weak), CD 25, CD103, FMC-7, and strong lambda light chains.

3. Large granular lymphocytic leukaemia

The majority of the lymphocytes are LGLs, with cell markers, CD2, CD3, CD5 (weak), CD8, CD16 (weak) and variable CD57 confirming this. The molecular testing demonstrated monoclonal DNA with TCRB and TCRG rearrangements present.

4. Multiple myeloma

Circulating plasma cells, but not 20% or $2.0 \times 10^9/L$ to meet the criteria for plasma cell leukaemia.

The BM had 58% plasma cells with CD38 and variable CD138 expression.

5. B-ALL

Large immature, lymphoid looking cells in peripheral blood. Immunophenotype consistent with B-ALL, although aberrant expression of variable CD11b and equivocal CD13. BM: 97% lymphoblasts of intermediate size with high N:C ratio and fine chromatin. Cytogenetics was very unusual with hyperdiploidy as he had 54 chromosomes (extra copies of X, 4, 5, 6, 14, 15, and 17) as well as the Ph' t(9;22) and a derived 22. The extra copies is a good prognostic indicator, but the Ph' is not.

6. Acute sepsis.

Marked left shift and toxic changes (although quite neutropenic), very vacuolated monocytes. This patient died 24 hours later.

ADVERTISING RATES FOR 2007**AIMS QUEENSLAND BRANCH NEWSLETTER**

Single page A5

\$66 (includes GST)

Four consecutive issues single A5 page

\$185 (includes GST)

The deadline for copy and advertising for the next

issue is 31st March 2007

Contact: Robyn_Wells@health.qld.gov.au

ROBYN'S RECIPE

Pears are coming into season now, and this cake is very rich and luscious – and also very big! You can quite easily halve the recipe

Pear and ginger cake

Serves 10

2 pears, quartered, cored and sliced thinly

300g (2 cups) plain flour

1 ½ teasp bicarb soda

3 teasp ground ginger

165g unsalted butter, chopped

225 g brown sugar

4 eggs

1 ½ cups golden syrup

285g sour cream

Caramel

60g unsalted butter

175g brown sugar

For caramel, place butter and sugar in small saucepan and stir over medium heat til melted and well combined. Pour into a greased and baking paper lined 28cm springform pan. Place pears in slightly overlapping layers over base.

Sift flour, bicarb soda and ginger into a bowl. Beat sugar and butter with electric mixer, til light and fluffy, then add eggs, beating well after each addition. Add golden syrup and beat well, then fold in sour cream and flour mixture alternately until just combined.

Pour mixture into pan and bake at 180°C for 1 ½ hours or until skewer withdraws clean, covering cake with baking paper for last ½ hour if over-browning. Stand cake in pan for 5 minutes before turning out onto wire rack to cool. Serve warm or at room temperature. Will keep up to 3 days in airtight container.

For half mixture, use 20cm pan and bake for approx 1 hour.

FAST FACTS- CHRONIC REJECTION

Chronic allograft rejection is a gradual reduction in graft function. Intravascular ultrasound imaging is an important technique to support the diagnosis in heart transplants. Needle biopsy is a valuable tool in the diagnosis of chronic allograft rejection in the liver, pancreas and kidney.

Characteristic histological features:

- Inflammation. Patchy distribution of lymphocyte and macrophage infiltrates
- Fibrosis. Initially patchy but increases as chronic rejection intensifies
- Vasculopathy. Occurs in all solid organ transplants particularly prominent in cardiac transplantation. Histologically distinguishable from atherosclerosis although both conditions may co-exist.
- Destruction of epithelial conduits. This effect is most visible with the bronchiolar destruction and in the liver it is prominent with the loss of bile ducts.

RISK FACTORS

There are a range of factors that increase the possibility of chronic allograft rejection, these include

- the occurrence of acute rejection - a transplant recipient is 2.7 times more likely to develop chronic rejection with the onset of an acute rejection
- timing
- number and severity of acute rejection episodes, eg. A single late episode of acute kidney graft rejection increases the risk 31 fold, greater than one single episode increases to 78 fold compared with those patients who have had absence of acute rejection. Effectively graft survival time is directly affected by episodes.

Cellular and humoral assays are used to predict risk of developing chronic rejection although any of these assays alone would not suffice as a single clinical indicator.

- T-cell assays identify donor-Ag specific hyporeactivity, effective for long-term graft survival, however this assay is of low specificity.
- ELISA-based humoral assay has increased sensitivity than the traditional complement-mediated cytotoxicity assay; this assay shows good correlation with graft survival.

LUNG TRANSPLANTS

- 50-60% lung transplant patients develop chronic rejection
- Immune-mediated inflammation and destruction of airways, causing scarring and a fibroproliferative response that results in obliteration of bronchioles. Obliterative bronchiolitis involves the migration of fibroblasts to the bronchioles causing progressive airflow obstruction and ultimately graft failure.
- Healthy lung function cannot be restored although this destructive process can be halted by effective therapy
- Symptoms show shortness of breath and progressive decline of Forced Expiratory Volume
- Risk factors in the development of chronic rejection in lung transplant patients arise from acute rejection, CMV, bacterial pneumonia, pneumocystis pneumonia, airway ischaemia injury all. The role of cytokines in the Obliterative bronchiolitis is still under investigation.

HEART TRANSPLANTS

- Allograft vasculopathy and coronary artery disease are both risk factors in chronic rejection
- Risk assessment involves the evaluation of intimal thickening, hyperlipidemia, and donor transmitted atherosclerosis
- Vascular damage is recorded at the time of transplant and is monitored for progression after transplant

- Intravascular ultrasound is a visual assessment of vessel lumen, not only is this method provide a 360° view of the heart but is highly reproducible and is more sensitive than an angiography.
- Coronary flow is an effective assessment in predicting graft function. Endothelial response to vasodilators is monitored to predict vascular disease, eg. Acetylcholine causes vasoconstriction instead of vasodilation in endothelial dysfunction

CLINICAL MANIFESTATIONS

A complex immune and non-immune interactive process results in chronic rejection. T cells hold a significant role in acute and chronic allograft rejection. Immune and non-immune factors stimulate humoral immune response subsequently affecting intimal smooth muscle proliferation and vascular thickening.

Grafts undergoing chronic rejection may have

- Increased matrix protein,
- Increased elastase activity in coronary arteries and myocardium. collagen production is promoted and fibrosis in the myocardium is stimulated,
- Induced TGF- β production, stimulating fibronectin expression and promoting T-cell and smooth muscle cell migration.
- An increase in almost all serum cytokines

Laminin, proteoglycans and tenascin are other factors linked to chronic rejection as they have an involvement in increasing smooth muscle proliferation.

Mismatched or poorly matched HLA (human leukocyte antigen) is a certain risk factor for chronic rejection. Other Immunological risk factors include

- Inadequate immunosuppression,
- High immune responsiveness to graft: detected by cellular reactivity to donor antigen and monitored by panel reactive antibody (PRA) status
- Early rejection episodes
- Late acute rejection
- Genetic (eg Afro-American, Afro-Caribbean)

Non-immune risk factors can be split in to two categories: Donor organ and Recipient organ. Listed below are associated risks.

Donor organ:

- Brain death,
- Cold ischaemia,
- Donor-recipient size mismatch (known as Nephron mass),
- Primary non function,
- Gender

Recipient organ:

- Hypertension
- Hyperlipidemia
- Obesity
- Infections
- Proteinuria
- Drug nephrotoxicity
- Age (poor compliance in adolescent patients – thought to be caused by patients forgetting to take tablets, or side effects)

Rebecca Klee

Reference Article: *Clinical Opinion in Transplantation*
Symposium of the American Society of Transplant Physicians,
January 1998

SNIPPITS FROM THE NEWS

All these facts appeared in a recent copy of Time Magazine (13/11/2006)

Bottle nose dolphins (thought to be the smartest mammal) have been observed by a group of scientists to use sea sponges when fishing.

Elephants do not only have a great memory, they are self aware. This was noted during a study using mirrors.

Rooks, Britain's version of a crow, have discovered how to get food out of a trap without getting caught.

Researchers have observed gorillas using tools to open nuts and gauge water depth

While not the brightest animal, the humble chicken has been noted to leave a piece of food in case a better piece arrived a little later.

Scientists in America have discovered mice that have been fed on a high calorie diet that contains large doses of a natural substance found in grape skins (resveratrol) lived longer than the controls whose diet lacked resveratrol. While the mice still got fat, they did not show liver damage or diabetes. For a human to get the same proportion of resveratrol in their diet, 100 glasses of red wine would have to be drunk daily.

Haematology Morphology Workshop 2007

School of Life Sciences

QUT George St Brisbane
16th to 19th July 2007

Registration fee \$495 (includes GST)

For all details including registration
please visit

→ <http://omsp.net/wksp/>

% * # Brain Teaser Answer Page @ % *

1. Sudoku

8	6	5	2	9	7	1	4	3
4	1	9	8	3	5	7	6	2
7	2	3	1	6	4	8	5	9
2	8	4	9	5	6	3	7	1
9	7	6	3	4	1	2	8	5
5	3	1	7	8	2	6	9	4
3	5	7	6	2	9	4	1	8
6	4	8	5	1	3	9	2	7
1	9	2	4	7	8	5	3	6

Sudoku puzzles do not require any skill with arithmetic. It is all based on logic! The numbers 1-9 can only appear once in each horizontal line, vertical line and 3x3 block within the sudoku square.

2. Which of the following dishes is the odd one out?

- Moussaka
- Risotto
- Tiramisu
- Lasagne
- Chow Mein
- Coq au vin
- Vindaloo

Answer: Tiramisu

This is a dessert. The others are all main courses.

3. Picture Element

4. Septenary

5. Right Hand

AIMS QLD BRANCH COMMITTEE 2006 – 2007

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AIMS AACB Clinical Review

***Please join your AIMS and AACB colleagues for
the first 2007 clinical review meeting,
to be held during Pathology Week***

***Our multi-disciplinary topic will be
SEPSIS***

***Date:
Tuesday 27 February 2007***

***Venue:
RBWH Education Centre***

***Time:
Refreshments from 6.00pm
Presentations from 6.30pm***

