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Contents:

Editor: Elizabeth Baranyai

"The HGV aims to provide a dynamic continuing education program in which all persons with an interest in Histology and Histotechnology are freely invited to participate."

Committee Page:

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Please feel free to contact any of the committee members listed above with any comments or suggestions. Contributions are always welcome.

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Blurb from the Bush

We have hit winter! Here in Ballarat we have had days during May with only a 7C maximum. Embedding suddenly becomes a favoured task, wrapping your hands around a warm wax bath.

During May we had what is becoming a traditional meeting with the ASC, presenting a series of short presentations on a variety of topics. Unfortunately we had some technical hitches with the venue, but despite this, we were able to provide some well presented and interesting topics.

With winter also come trivia. Now is the time to start thinking about your teams and booking tables. We are locked into the same venue, which has limited availability of tables. This edition has information on how to book a table, so don't leave it too long.

The 5th National Histology Meeting in Sydney is on November 4th – 6th 2011. Information and registration form is in this edition, but can also be obtained from http://www.histonsw.org.au/conference/ It would be great to get a good contingency from Victoria to invade the harbour city, so start approaching your boss to get them to fork out some educational dosh for the trip.

All things going as planned Victoria will host the next National meeting in 2013. These events are a lot of planning so anyone interested in assisting in event organising please nominate for the committee.

Adrian Warmington
HGV President

Review of a Weekend Workshop in Diagnostic Immunohistochemistry-Part 2

After lunch on Sunday, Mogens Vyberg took the floor again, to discuss **the Immunohistochemical classification of endocrine tumours.** The endocrine organs and tissues include the diffuse endocrine system within the gastrointestinal tract, respiratory tract, other mucosa and skin, islets of Langerhans in the pancreas, C-cells of the thyroid, the parathyroid glands, and pituitary gland ,the liver, adrenal cortex, the follicular thyroid, thecal cells of the ovary, Leydig cells of the testis and paraganglionic cells. General markers include synaptophysin and chromogranin A, special markers include hormones, TTF-1,CDX-2 and PAX8. CD 56 and NSE are too non-specific to be of great use. Synaptophysin is an integral-membrane glycoprotein of pre-synaptic vesicles and is present in neurons, neuroendocrine cells and adrenal cortical cells. Some clones of this antibody are better than others. Chromogranin is an acid-calcium binding glycoprotein closely associated with the matrix of dense –core neurosecretory granules, probably involved in packaging and processing of neuropeptides and peptide hormones. It is present in neurones and neuroendocrine cells.

Are we there yet? No, but more than half-way! Next: **Immunohistochemical classification of sex cord and germ cell tumours.** Germ cell and sex cord stromal tumours in females account for 20-25% of all ovarian tumours. The majority are mature teratomas. Mainly young patients are affected by germ-cell tumours, while sex-cord stromal tumours occur in all age groups. 95% of ovarian tumours are germ-cell tumours including: Dysgerminoma, yolk-sac tumour, teratoma.5% are sex-cord stromal tumours including: granulosa cell tumours, theco-fibromas, Sertoli-stromal cell tumours. Germ cell and sex cord tumours in males account for all testicular tumours, the majority of which occur in the 3rd and 4th decade of life, but can occur in small children. The prognosis is good to very good. 95% of testicular tumours are germ-cell tumours and include: seminoma, embryonal carcinoma, polyembryoma, yolk sac, choriocarcinoma, teratoma. 5% of testicular tumours are sex-cord stromal tumours including: Leydig cell tumour, Sertoli cell tumour, granulosa cell tumour. The clinical relevance of IHC enhances differentiation between germ-cell/sex cord tumours vs non GC/SC tumours, seminomas vs non-seminomas and GCT vs SCT. Usually >60% are successfully treated. Antibodies include: placental alkaline phosphatase (PLAP), OCT-4, CD30,CD 117,CK8/18,podoplanin, EMA, glypican 3, human chorionic gonadotrophin(HCG), α-foetoprotein, α-inhibin, vimentin. The take home message: all germ-cell and sex-cord stromal tumours are negative for CA 125 and EMA, but positive for vimentin, with the exception of choriocarcinoma. Inhibin is not specific for sex-cord stromal tumours and should be used in a panel with other antibodies.

Immunohistochemistry of epithelioid, pleomorphic and small cell tumours. Soft tissue tumours are rare, comprising only 1% of all tumours, growth patterns overlap, and a great deal of experience is required to accurately diagnose them, IHC is often inconclusive, rarely EM will give a definitive diagnosis, sometimes molecular biology is helpful. Antibodies discussed included: CD 31, (endothelial cells), virtually all vascular and haematolymphoid tumours are positive. CD117 (mast cells, melanocytes, interstitial cells of Cajal, and various epithelia) ,Gastro-intestinal stromal tumours(GIST), mast cell neoplasms, seminoma, malignant melanoma and malignant lymphomas are positive. Desmin (intermediate filaments) striated and smooth muscle cells, except in most blood vessels, some myofibroblasts. Sarcomas are usually positive and mesothelioma is usually negative. Myogenin (nuclear phosphoprotein transcription factor which determines the differentiation of foetal cells into rhabdomyoblasts) rhabdomyosarcoma is positive for myogenin. Small cell tumours

include rhabdomyosarcoma, Ewing's sarcoma and neuroblastoma. CD99 is a cell adhesion molecule present in T-cells, myeloid progenitor cells, ependymal cells among others and shows strongly positive membrane staining in Ewing's sarcoma.

First up Sunday morning (no respite after the dinner I might add, we were at it again at 830am!) was **Immunohistochemical classification of spindle and clear cell tumours** presented by a bright-eyed and bushy-tailed Jan Klos. This was quite a can of worms! There are several reasons why tumours consist of clear cells: it could just be artefact caused by tissue processing and poor preservation, hydropic cell degeneration, the cells could contain an abundance of glycogen, mucin, lipid, mucopolysaccharides or phagocytosed foreign material. Cancers falling into this category include: hepatocellular carcinoma, renal cell carcinoma, clear cell carcinoma (of soft tissue). This resembles melanoma, and in fact their IHC profiles are identical. Others mentioned include adrenal cortical carcinoma and chordoma. Differential diagnostic problems arise when clear cell and spindle cell tumours metastasise to organs where primary clear cell or spindle cell tumours may also arise, eg. renal cell carcinoma metastases in ovary or lung. The list of spindle cell tumours is extensive, and includes carcinoma, smooth muscle neoplasms, endometrial stromal sarcoma, myoepithelial. neoplasms and malignant melanoma to name but a few. Clear cell and spindle cell tumours are heterogeneous groups with quite characteristic but not always specific morphology of tumour cells or growth pattern. The same histological type of tumour can show either clear or spindle cell morphology, or both, making these tumour groups particularly challenging. Alfred Lam described the use of **Immunohistochemistry and molecular pathology as research tools,** particularly in colorectal and thyroid cancer. Genetic changes in cancers are detected by molecular pathology, and these in turn are confirmed by immunohistochemistry. This has implications in the diagnosis and management of treatment for certain cancer types, and emphasises the need for reliable methodology and expertise in interpretation of results.

Chris Philippa presented his findings on the effect of prolonged formalin fixation on antigen retrieval methods. The aim of fixation is to immobilise and stabilise tissue and components in as like-like a state as possible, while preserving cell shape and volume (morphology) during subsequent processing and treatments. This achieved by using formalin, which firstly diffuses into the tissue, and covalently binds to protein molecules. This occurs most efficiently at pH 7.4-7.6. It is these bonds which are broken during antigen retrieval, exposing the antigenic sites, essentially reversing fixation. This can be achieved several ways, using enzymes, heat and chemical retrieval. Some antigen sites are more susceptible to prolonged fixation, such as TTF-1, ER and Cd10. However, with optimal buffer and pH it is possible to retrieve most antigens after prolonged formalin fixation.

The major role of **Immunohistochemistry in Respiratory Medicine** is in neoplastic lung conditions, differentiating between small cell (SCLC) and non-small cell (NSCLC); adenocarcinoma vs squamous cell carcinoma; a neuroendocrine malignancy, undifferentiated (pleomorphic) and spindle cell malignancy, and clear cell tumours. The initial differential in all malignant lung tumours lies between SCLC and NSCLC. SCLC gets chemotherapy and not surgery. The diagnosis can be based on H & E and/or cytology alone. IHC is occasionally useful on small crushed biopsies, but large cell neuroendocrine tumours have essentially the same IHC profile. Lung cancer is the most common cause of cancer death. NSCLC accounts for 80% of incidence, and 20% of all cancer deaths. Most cases are advanced, so surgery is not an option. The biopsy or cytology sample is often the only tissue available for investigation. The classification is based on standard histology/cytology by the current WHO classification system 25% of bronchoscopic biopsies cannot be sub-typed by histology alone. On small biopsies, accuracy and reproducibility are low, correlation between biopsy and

resection specimen is poor, due to sampling issues. Emerging therapies having greater efficacy and toxicity between sub-types is increasing the need to further categorise all cases of NSCLC, typically adenocarcinoma vs squamous cell carcinoma. IHC has extended the depth of interpretation to a diagnosis of poorly differentiated adenocarcinoma as opposed to NSCLC. The suggested approach is to initially try to rely on H & E and a mucin stain, followed by TTF-1 and CK5/6, if there's still no answer, try CEA and p63. There will be a significant number that will not fit neatly into either! It has not yet been determined whether further classification of tumour sub-type translates into different responses to treatment or indeed survival rates, although the clinicians widely assume that it does! A common challenge is to determine whether the tumour is a lung primary, or a metastases. The most common type of lung malignancy is metastatic disease. Making the distinction on morphology alone is difficult bordering on impossible, however IHC can be very useful here. A multidisciplinary approach is useful. Most metastases are multiple, bilateral, sharply circumscribed and peripheral(this is where the blood vessels supplying the lungs terminate) and originate from breast, GIT, melanoma, kidney or sarcomas. Adenocarcinomas that usually go to the lung include bowel, breast, prostate, upper GIT (stomach, biliary and pancreas), uterus and kidney. Useful antibodies are: TTF-1 (1° lung), PSA (metastatic prostate), CK7, & CK20,(metastatic colorectal ca) ER/PR(12% lung ca is ER +)CEA. Other tumours that turn up in the lungs include poorly differentiated malignancy (always consider malignant melanoma, particularly in Queensland!), spindle cell and clear cell malignancies, pleural tumours such as mesothelioma, and thymoma. Future directions include: Can IHC be used to predict the response to therapy? Can IHC be used to stream lung cancer patients into different treatment arms?

Mogens Vyberg discussed **Tissue Controls and the Design of Multi Tissue blocks.**
Immunohistochemistry is just one stop along the pathway a tissue sample travels from removal from the patient until the point where a satisfactory diagnosis has been reached. There are at least 5 variables at each phase, starting with the tissue type, dimension and method of sampling, followed by specimen preparation (does it require decalcification?), fixation time, type of fixative and volume used (are these adequate?), processing protocols, sectioning (thickness, drying protocol and time, section storage prior to IHC), with IHC, pre-treatment, choice of antibody clone and dilution, buffer choice, time and temperature, staining method manual or automated platform (choice), visualisation, sensitivity and specificity, localisation. On the home stretch, validation and interpretation complete the journey. With roughly 3 choices for each of the 5 variables at each phase of the process there are potentially 4 million protocols! However, for the optimal performance of IHC the following are crucial: appropriate tissue fixation and processing; appropriate and efficient epitope retrieval, appropriate choice of antibody and/or clone; a robust and sensitive detection system and appropriate choice of control material. Reagent and tissue controls are necessary for the validation of immunohistochemical staining results. Without them, interpretation of staining results would be haphazard, and the results of doubtful value. Controls determine if the staining protocols were followed correctly, whether day-to –day and worker-to-worker variations have occurred and that reagents remain in good working order. The Aalborg method for designing control blocks for approximately 180-200 antibodies is as follows: MB1: Appendix, liver, tonsil and pancreas. MB2: Brain, striated muscle, skin, malignant melanoma. MB3: lung, prostate, placenta, thyroid. MB4: Thymus, Hodgkins lymphoma, bone marrow, tonsil. Special controls include ALK-1, hormones and Her-2. The control slides are for the most part pre-cut, each test section has its own control, which frees up stations in the autostainers, and quality control by the technologists is facilitated by using standardised normal tissue. The main disadvantage is the increased amount of work in sourcing the control material and preparing the tissue blocks.

Jan Klos in his presentation on **Quantitation in Immunohistochemistry** started by demonstrating how easily the human brain can be deceived by what it thinks the eye has seen. Most diagnostic immunohistochemistry is based on qualitative interpretation: is it positive or negative? Is the arbitrary cut-off of 10% for positive staining correct? Many publications use cut-off values that are statistically significant (eg the percentage of BCL-2 + cells in DLBCL). Quantitation is essential in lesions where there is a continuous spectrum of positivity: how much? How many? Measurements must be reproducible, and stable results in IHC require standardised procedures such as fixation, process. The conditions for reproducibility that must be met are: stable results of immunohistochemical staining, adequate method of evaluation (estimation, scoring, counting, interactive point counting using a grid, automatic evaluation by digital image analysis) and measurements must occur in a biologically relevant area with random selection of fields of measurement. This type of measurement is expected to be more and more precise, and variation needs to be reduced to the absolute minimum. If done properly, consensus scoring can be a cheap alternative to digital image analysis.

Lunch on Sunday was followed by Mogens Vyberg's presentation on **Immunohistochemistry and therapeutic markers.** Unfortunately, I had a plane to catch, and wasn't able to stay for the duration of this session. My report from this is drawn from the CD. Prognostic markers indicate an increased or decreased risk of recurrence of death with a given type of cancer. Predictive markers indicate an increased or decreased response to a specific therapy for a given type of cancer. Therapeutic markers represent a target to a specific therapy for a given type of cancer. Oestrogen and progesterone receptors and Her-2 were discussed. Oestrogen receptor status is a weak prognostic cancer marker, but a strongly predictive and therapeutic cancer marker and the effect of anti-oestrogen treatment is strongly correlated with IHC ER expression. Progesterone receptor status is a strongly predictive, prognostic and therapeutic cancer marker. ER+/PR- carcinomas are less sensitive to antihormone therapy, ER-/PR+ tumours may be sensitive to antihormone therapy. The Her-2 gene is multiplied 50-100-fold via mutation in ~15% of ductal breast carcinoma, 10-15% of gastric adenocarcinoma,5% of ovarian carcinomas,10-15% of endometrioid carcinoma and 5% of lung carcinoma. The corresponding over-expression of the protein is 10-100 –fold. Her-2 activation causes malignant transformation and increases the malignant potential. Monoclonal antibody trastuzumab (Herceptin) inhibits tumour growth and increases survival. It is therefore imperative to get it right when reporting the status of these receptors as the results directly influence the subsequent treatment made available to the patient.

In conclusion, the breadth and depth of this series of workshops was amazing and the information shared invaluable. As described in the introduction, this was the inaugural workshop in Diagnostic Immunohistochemistry, and I thoroughly recommend attendance at the next, whenever and wherever it may be.

Judy Brincat

Dorevitch Pathology

Meeting Reports:

A combined Histology and Cytology Student Presentation was held on May 5th.

In combination with three cytology case studies, three histology case studies were also presented:

Case Study- Ovarian Teratoma with Malignant Transformation - *Ebony Forrest*

(Monash Medical Centre)

Ebony presented a well illustrated case of an ovarian teratoma with malignant transformation.

What is an ovarian teratoma? AKA a dermoid cyst, a teratoma is a benign, germ cell tumour that contains a diversity of tissues from all three germ layers. The tumour mainly produces bone, hair and teeth. It develops from a totipotential germ cell retained with in the ovary. It is most common in females under the age of 20.

Clinical Notes: A 19 year old female presented with a 3 month history of backache and increasing poor health. On investigation, bilateral ovarian masses, ascites, a mediastinal mass, pleural effusion, enlarged lymph nodes and liver cysts were discovered. The initial evaluation made by doctors was of some sort of metastatic germ cell tumour.

Cytological findings: 1.5L of heavily blood-stained peritoneal fluid was received. 2 air-dried, 2 wet-fixed and 4 cell block slides were prepared. In all the slides, abundant malignant cells were seen presenting singly and in groups. Immunohistochemical stains were performed on the cell block which showed that the ovarian tissue was positive for malignant melanoma.

Histological findings: The patient's right and left ovaries were then removed, as well as the omentum and para-aortic lymph nodes. The right ovary measured 130x95x75mm and the left ovary measured 75x65x45mm. Both exhibited a smooth, pale-grey surface with haemorrhagic and cystic areas with fine hairs. No large discrete lesions were identified in omentum, and the lymph nodes appeared congested. Microscopically, the ovarian masses showed characteristic benign teratoma features: keratinizing squamous epithelium (skin), empty areas where hair had existed, a cyst lining with columnar epithelium, and hyaline cartilage. Malignant features were also identified which were seen to be infiltrating within the teratoma; these showed similar features to the tumour cells seen in cytology. Melanoma markers Melan-A, S100 and HMB45 were positive.

Differential Diagnosis: Other ovarian neoplasms had to be excluded. Ovarian tumour markers CA125 and BetaHCG were negative. Cytokeratin markers were also negative.

Diagnosis: The IHC stains were in keeping with a malignant melanoma with evidence of a pre-existing teratoma. The source of malignant melanoma was not identified; however, it was likely the tumour has arisen from the stem cells in the teratoma, possibly in areas of neural tissue. The patient sadly passed away a couple of weeks after being diagnosed due to the extensive spread of the disease.

Teratoma statistics: Teratoma's account for 10-20% of all ovarian neoplasms.

1-2% undergo malignant transformation, of which 85% are squamous cell carcinomas, 6% are sarcomas and 5% are adenocarcinomas. The median age for malignant transformations is 54-61 years. Malignant melanoma developing from a teratoma is extremely rare!

Colon: The Only Black Hole Known to Man - *Solito Rabaja (St. Vincent's Hospital)*

Solito presented a detailed case study using a panel of special stains to help diagnose a histological section of colon.

Clinical Notes: 85 year old female who presented with severe stomach pains and constipation.

Colon: A normal functioning colon is important to the digestive system. It has three combined functions: 1. Absorbs all remaining water, electrolytes and nutrients from ingested food; 2. Stores nutrients and eliminates waste; 3. Microbacterial flora which inhabit the colon are responsible for maintaining the colonic lining by feeding on indigestible matter.

Histology Section: The H&E section revealed normal crypts with normal goblet and glandular formation, a normal muscularis mucosae lined with columnar epithelial cells, unremarkable lamina propria and smooth muscle, and a normal serosa. A brown pigment between the crypts of the muscularis mucosae was also identified. At a closer look, the section also exposed glandular structures which were not consistent with normal colonic tissue showing a loss of glandular structure and nuclear stratification. Neoplastic pleomorphic cells were seen beginning to infiltrate the muscularis propria. Ulceration was also identified.

Initial Diagnosis → Adenocarcinoma. Differential diagnosis: ulcerative colitis. What was that pigment? Melanin versus lipofuscin.

Special stains chosen to support preliminary diagnosis:

Alcian Blue Periodic Acid Schiff's ± Diastase - used to demonstrate acidic and neutral mucins, as well as glycogen: No positive staining was seen in the neoplastic cells.

Masson's Trichrome (Halls Modification) - used to demonstrate fibrosis/desmoplasia within the tissue: stain didn't work well and so was unhelpful.

Schmorl's Ferric Ferricyanide – used to demonstrate if the brown pigment was either melanin or lipofuscin. The pigment stained black and thus demonstrated that it had been derived from a melanin basis. This result reveals that this patient has a condition known as melanosis coli. This condition is seen in people who consume high amounts of laxatives → the patient in this case was chronically constipated and so was probably taking a laxative.

Final Diagnosis: Moderately differentiated primary adenocarcinoma. TNM classification = stage 1. The mesenteric lymph nodes would need to be removed and assessed to see if the tumour has metastasised.

Additional tests: Immunohistochemistry would be needed to confirm the origin of the tumour. CK7 and CK20 are usually diagnostically run together. CK7 stains positive for transitional epithelium of breast, lung, ovary and uterus, whereas CK20 stains positive for gastric and intestinal mucosa. Mismatch repair genes (MLH1, MSH2, MSH6, and PMS2) can also be performed to identify whether there are any DNA sequencing errors in the tumour.

Colorectal Cancer: The most common form of cancer in the GI tract. 95% of cases are adenocarcinoma. It is mostly prevalent in the 60-70 age bracket. There are 3 types of known colorectal cancers: *1. Familial Adenomatous Polyposis (FAP)* – rare autosomal trait on Chromosome 5; *2. Hereditary Non-Polyposis Colorectal Carcinoma (HNPCC)* - autosomal trait; *3. Sporadic form* – caused by ulcerative colitis/colonic polyps/diet habits (indigestible fibre, high fat).

Treatment: Surgical excision, chemotherapy, radiotherapy.

The Silent Disease - *Bandar M Alshehri (currently completing his Masters in Laboratory Medicine and working in the Histopathology Lab at RMIT)*

Bandar presented a well structured case study using a panel of special stains to help diagnose a histological section of bone.

Clinical Notes: 75 year old female who presented with general malaise, chronic pain and fatigue, and lesions in her bone and other different organs.

Histological section: The H&E section was assessed to identify whether any pigments, pathological changes or signs of infection were present. No signs of infection or pigment were seen, however, an amorphous, extracellular material was identified in some areas of the tissue, and some variation in nuclear size was noted in some of the cells.

Initial/Differential Diagnosis: Osteosarcoma, Ewing's sarcoma, Amyloidosis.

Special stains chosen to reach a diagnosis:

Reticulin stain – used to highlight the complex reticulin networks surrounding tumour groups as would be seen in Osteosarcoma. *Result: normal reticulin network*

PAS Stain – used to demonstrate the cytoplasmic glycogen that would be present in Ewing's sarcoma. *Result: negative*

Congo Red – to demonstrate amyloid that would be present in Amyloidosis. *Result: positive*

Final Diagnosis: Amyloidosis

Amyloidosis: Is a disease characterized by the abnormal deposit of an extracellular protein, called amyloid. This deposit can be localized or systemic. Systemic amyloidosis can be further classified into four subsets: 1. Primary amyloidosis (related to an abnormal production of antibodies by plasma cells); 2. Secondary amyloidosis (develops along with a chronic infectious or inflammatory disease); 3. Hereditary (familial) amyloidosis; 4. Beta-2 microglobulin amyloidosis. Organs affected by this disease include: brain, heart, liver, spleen, pancreas, kidney and bone. Symptoms include: fatigue, weakness, fever and dysfunction of involved organs.

Further Investigations: Urine and serum electrophoresis, immunohistochemistry, genetic studies, anti-nuclear antibodies (in secondary amyloidosis), radioimmunoimaging and X-ray.

Treatment: In primary amyloidosis, the affected individual may be treated with bone marrow transplantation. The causes that lead to secondary amyloidosis need to be treated to cure this form of the disease. And organ transplantation may be required in familial amyloidosis.

Reported by Michelle Zammit

The Alfred Hospital

Under the Microscope

Reported by Maria Chavez

1. **What was your first job?**
I began nursing at the RAH in 1973 and only stayed for about 6 months, as it interfered with my social life too much!

2. **What attracted you to Histology?**
Well, only the fact that it was the only job going at the IMVS when I decided to leave my nurse training and find something on the RAH campus. In fact, even after I was taken around the laboratory during the interview, I still didn't really understand what Histopathology entailed!

3. **What is the worst decision you have ever made?**
Probably doing this interview!

4. **What is the best decision you have ever made?**
Having a child before it was too late. However, now that he is 19, and I look at his bedroom and study, I do wonder what on earth I was thinking!

5. **Who would you most like to have dinner with and why?**
George Clooney, for obvious reasons…!

6. **What music do you enjoy listening to?**
All kinds, but Van Morrison is probably one of my favourites.

7. **What is your favourite stain?**
Probably the ATPase reaction (technically it's not a stain). It demonstrates the skeletal muscle fibre types.

8. **What is your favourite food/Restaurant?**
Chocolate (is it considered a food?) or pasta of some kind.

9. **What are you reading at the moment?**
Robert Goddard's "Play to the end".

10. **What is the best conference you have ever attended?**
12th International Congress on Neuromuscular Diseases in Naples, Italy in July 2010

11. **Are there any current projects you are working on at the moment?**

The only "project" at the moment, is trying to keep my sanity, with the amount of diagnostic work coming in.

Next Scientific Meeting:

Histology Group of Victoria Inc.
Org. No. A0035235F

The Victorian Cancer Biobank
A Consortium of Tissue Banks supporting Cancer Research & Clinical Trials

Speakers:	**Katie-Lee Alexander** **Matthew Chapman**
Date:	Thursday 30th June, 2011
Time:	6:00 – 6:45 Refreshments 6:45 – 7:30 Presentation
Venue:	Peter MacCallum Cancer Institute 7 St. Andrews Place East Melbourne
Presentation:	Brockhoff Lecture Theatre Level 3, Smorgan Family Building

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Future Events:

2011

~~**12th - 14th March**~~
~~Coonawarra Joint Meeting HGV/HGSA~~
~~Venue – Coonawarra, South Australia~~

~~**5th May**~~
~~HGV/ASC Scientific Meeting – Student Presentations~~
~~Venue – PeterMac~~

30th June
Scientific Meeting – The Victoria Cancer Biobank
A Consortium of Tissue Banks supporting Cancer Research & Clinical Trials
Speakers: Katie-Lee Alexander (St. John of God Pathology/Pathcare)
Matthew Chapman (Royal Melbourne Hospital)
Venue – PeterMac

22nd July
Social Event – Trivia Night
Venue – Mount Erica Hotel

15th September
Scientific Meeting – Further Education Possibilities for Histologists
Venue – PeterMac

4th – 6th November
National Histology Conference
Sydney

17th November
Scientific Meeting/AGM – Molecular Techniques (KRAS/BRAF)Venue – PeterMac

Trivia Night

Friday 22nd July 2011

7pm – 11pm

Histology Group of Victoria Inc.

The Mount Erica Hotel

420 High St, Prahran

Sponsored by TekMed MORTUARY

$25 per person or $250 per table.

(inc finger food, Drinks extra)

Tables of less accepted.

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Trivia

Spelling bee

Musical challenge & More.

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Prizes

Full payment must accompany this form. Payment will not be accepted on the night. No exceptions!!!Seats are limited, so book early to avoid disappointment. RSVP & payment due Friday 15th July. Contact Maria Chavez 9594 3493.Please make cheque or money order to HGV Inc. Return this section with payment and send to: 39 Viewgrand Drive, Berwick 3806.

Contact Name .....Contact number.....

Institution/s.....Number of seats.....

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Horses for Courses

FIFTH NATIONAL HISTOTECHNOLOGY MEETING

4-6 NOVEMBER 2011

Hosted by

HISTOTECHNOLOGY GROUP of NSW

Celebrating 30 years of Histotechnology

Postal address:

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GUILDFORD NSW 2161

31 March 2011

Dear Member,

The Histotechnology Group of NSW invites you to attend the next National Conference which is to be held at Rosehill Gardens, Sydney, 4-6 November 2011. Rosehill Gardens is one of Sydney's premier horse racing centres. This weekend is an excellent opportunity to meet your fellow Histotechs from around Australia and from further afield. We are expecting over 300 delegates.

There will be two **Gross Dissection** workshops on Friday 4 November run by Anne Prins and Penny Whippy at nearby Granville TAFE. Numbers for the Gross Dissection workshops will be limited. There will be two separate on site workshops - **Histo Hypotheticals (morning)**: an interactive workshop which will present problems that affect the quality of our results and participants will be encouraged to advise suitable courses of action; and **Histochemistry (afternoon):** a 'wet' workshop exploring alternative staining procedures (Microwave technology, detergent de-waxing.....). The workshops will be led by Tony Henwood and Linda Prasad from The Children's Hospital at

Westmead. These will be held once only, so that if you want to attend a cut up workshop, you will need to choose which of these you would like to attend.

The preliminary program for Saturday and Sunday includes an international speaker as well as a speaker from the Brain and Mind Research Institute and speakers on Lymphomas, Haematoxylin, Molecular Pathology, Moh's surgery, Skin Cancer, Case studies in bone tumours, Breast pathology, Colorectal pathology.

There will be a significant trade display with a large number of companies being represented and prizes for posters and abstracts.

Also a gala dinner has been arranged for Saturday night at Rosehill Gardens costing $74. You might like to dress for the occasion: 'Horses for Courses' is our theme, just a few days after the Melbourne Cup. There will be a prize for the best race call.

Accommodation is not included in your registration fee but rooms have been reserved at Rydges Hotel at Parramatta (across the road from the venue). Please contact Rydges via our Website linkage (www.rydges.com/cwp/histotechnologyconf) or direct on (02) 8863 7600 to book and pay for your own accommodation.

Your registration includes all meals **except** breakfasts and Saturday dinner.

Saturday and Sunday registration:	early bird, before 31 August 2011	$310
	after 31 August, before 4 October	$340
Single day registration:	early bird, before 31 August 2100	$210
	after 31 August, before 4 October	$230

Each workshop will cost an **additional** $50 and includes morning or afternoon tea plus lunch for Friday.

Payment can be made by cheque to "Histotechnology Group NSW Conference" or via internet, but you **MUST** complete all details on the internet so we know who is paying and **return your completed registration form** for early bird registration by 31 August 2011 or by 4 October 2011:

mail to PO Box 496, Guildford.NSW. 2161 or

email to kdrummond@dhm.com.au or

fax to (02) 9855 5169

BSB:**802 084**; Account number: **94099;** Account name: **Histotechnology Group of NSW**.

Prices **include** GST.

Further conference information is available on our Website (www.histonsw.org.au) or by contacting:

Kathy Drummond	Phone:	(02) 9855 5059
	E-mail:	kdrummond@dhm.com.au
	or	
Trevor Hinwood	Mobile:	0427 249 794
	E-mail:	trevor.hinwood@hdscientific.com.au

We look forward to seeing you at Rosehill in November.

Yours sincerely,

Kathy Drummond (Hon Secretary)

Histotechnology Group of NSW

National Histology Conference 2011

Name:.....		
Address:..... ..... Is this address work☐ or home☐?		
Phone no.: work ..... mobile:		
Place of work:		
Email (please print legibly):		
Dietary requirements:		
Workshops: Surgical dissection (morning) ..... **OR** Surgical dissection (afternoon) ..... Histo Hypotheticals ..... **OR** Histochemistry	☐ ☐ ☐ ☐	$..... $..... $.....
Early bird registration: **Saturday and Sunday** ..... **Saturday only** ..... **Sunday only**	 ☐ ☐ ☐	 $..... $ ..... $.....
Saturday registration ..... **Sunday registration**	☐ ☐	$..... $.....
Saturday night dinner: delegate ..... guest/s	☐ ☐	$ ..... $ .....
Payment method: cheque to Histotechnology Group NSW (National Conference) internet banking ..... (your name)	 ☐ ☐	$ ..... $ .....
Submission of **abstract**:	☐	
Submission of **poster:**	☐	
TOTAL		$.....