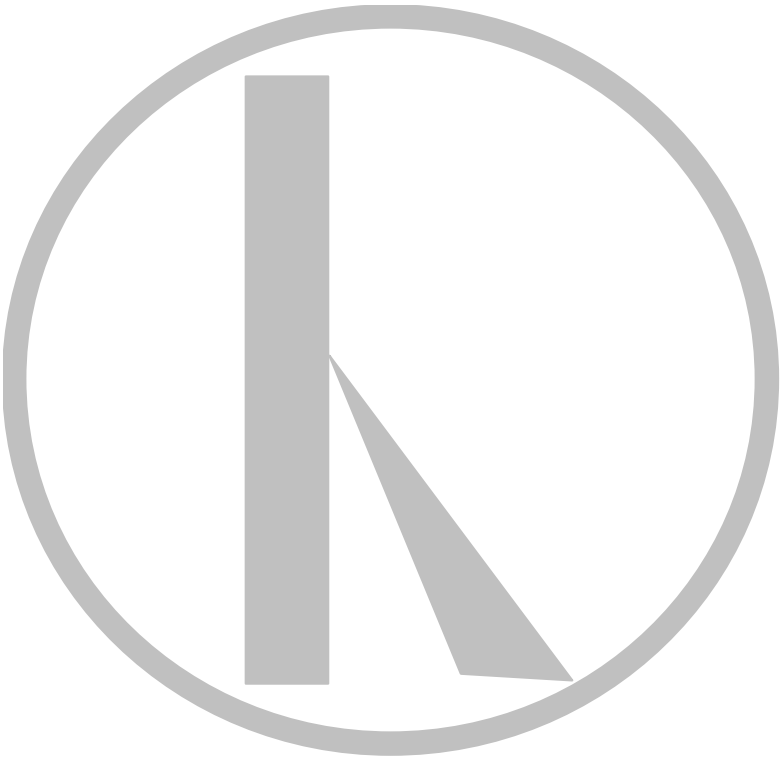
Histograph

ISSUE 3
November 2008



Newsletter of the Histotechnology Group of NSW

Contents

Editorial

Hi all,
I am currently getting over a bout of PND (Post NATA Depression). We have just had two days of NATA accreditation, both the laboratory and mortuary. At least we will have three years to get over it.

This issue contains details of the upcoming Christmas Dinner and National Meeting in South Australia. Ozone or leave your account in your patient? Or what about a review on lipofuscins or de-waxing sections. I hope you find this issue of interest. If not, then sack the editor! Or ply him with alcohol at the Christmas dinner!!

Tony Henwood,
Editor
anthonyh@chw.edu.au

Chairman's Report

Welcome to our new committee members elected to our committee in July. We trust you will enjoy your involvement in our Histotechnology Group and we welcome your contribution. This is a team effort and we have a number of projects and meetings planned for this year. Welcome back to the committee members who have been re elected for another term.

Since our Annual General Meeting in July we have held three successful meetings:

26th of August. I.C.P.M.R. Westmead. Neisha Jefferies. A very interesting presentation on PCR. What is PCR and how it correlates to Histology.

15th of September. Garvin Research Institute. Dr David Chang. Excellent presentation on Pancreatic research. Where this research is currently and future directions.

There are lots of interesting projects happening and we would like to revisit the Garvin next year.

1st November. Newcastle Scientific Meeting hosted by the Histology members in Newcastle. Some great topics and speakers:

- Dr Peter O'Brien. Plastic and reconstruction surgeon. Reviewed facial skin cancers and associated reconstruction work.
- Dr Ravinder Singh, Opthalmic and Optical surgeon. Reviewed eye diseases and abnormalities and associated reconstruction work.
- Dr Kasinathan Nadesan, Senior Staff Specialist, Forensic Pathologist. Review of physical violence against children.
- Prof. Ron Newland, specialist pathologist, retired. Life after pathology.
- Kathy Drummond/ Grant Taggart.Douglas Hanley Moir Pathology. Skins cut-up and pathology.
- Dr Phil Woodford, Senior Staff Specialist, Anatomical Pathologist,Hunter Area Pathology Service. Foetal and placental pathology.

Our next meeting will be our Christmas function on the 5th of December at North Ryde RSL. Our guest speaker is Dr Richard Jaworski who has a keen interest in Astronomy. Please note this in your diary; it will be a great night.

Our Website is now nearly sorted. We are looking forward to having this fully operational in the near future.

As this will be our last Histograph before Christmas, the committee wishes our members and families an enjoyable and safe Christmas break. We look forward to seeing you again in the New Year.

Trevor Hinwood,
Chairperson,
Histotechnology Group of NSW.

Committee Members

Chairman	Trevor Hinwood	trevor.hinwood@hdscientific.com.au	Product Specialist H D Scientific
Secretary	Kathy Drummond	kdrummond@dhm.com.au	Histology Lab supervisor DHM
Assistant Secretary	Dianne Reader	dreader@nsccahs.health.nsw.gov.au	Senior TO Anatomical Pathology RNSH
Treasurer	Lin Parkes	eyepoint2@bigpond.com	Eyepoint Instruments
Membership Secretary	Margaret James	megajam@optusnet.com.au	Dutta Path
Trade Rep	David Jones	d.jones@zeiss.com.au	Zeiss
Country Rep	Margaret McLauchlan	mclauchlans@bigpond.com	
Country Rep	George Youssef	george.youssef@gsahs.health.nsw.gov.au	
Editor	Tony Henwood	anthonyh@chw.edu.au	Histopathology Lab Dept Head CHW
Committee	Bill Sinai	sinai_family2@bigpond.com	Life Member
	Grant Taggart	gtaggart@dhm.com.au	Lab Manager DHM
	Julie Bilkey	juliebilkey@bigpond.com	Sydney Skin Pathology
	Liam O'Donnell	liam@sydneyskinpathology.com.au	Sydney Skin Pathology
	Bharathi Cheerala	bcheerala@dhm.com.au	
	Shaun Kay	skay@dhm.com.au	DHM

Can Melan-A Replace S-100 and HMB-45 in the Evaluation of Sentinel Lymph Nodes From Patients With Malignant Melanoma?

Kucher, et al (2006) Applied Immunohistochemistry & Molecular Morphology. 14(3):324-327

The sentinel lymph node (SLN) biopsy has become an increasingly important procedure used in the primary staging of malignant melanoma. However, micrometastases in a lymph node can be easily missed on routine H&E-stained sections. Therefore, S-100 and HMB-45 IHC stains are standardly performed on grossly negative SLNs for detection of metastatic melanoma. Each of these IHC markers, however, is not ideal. The authors investigated whether the newer IHC marker Melan-A would improve the detection of metastatic melanoma in SLN biopsies. Forty lymph nodes previously diagnosed with metastatic melanoma were retrospectively evaluated for S-100, HMB-45, and Melan-A expression. In addition, 42 SLN biopsies for metastatic melanoma detection were prospectively collected and evaluated for S-100, HMB-45, and Melan-A expression. All lymph nodes with metastatic melanoma from the retrospective study demonstrated S-100 reactivity. Five of the lymph nodes with metastatic melanoma from the retrospective study failed to express either HMB-45 or Melan-A, all of which displayed a desmoplastic morphology. One of the metastases positive for S-100 and HMB-45 failed to show reactivity with Melan-A (3%). The prospective study found 10 lymph nodes from 42 cases to be positive for metastatic melanoma, which were positive for S-100 (100%). Nine of the involved lymph nodes were positive for HMB-45(90%), and nine were positive for Melan-A (90%). Melan-A, although very specific, cannot replace the use of S-100 and HMB-45 for the detection of metastatic melanoma in SLNs. It can, however, substitute for HMB-45 with equally good results.

Safety Zone

Many air purifiers use ozone generators to disinfect and/or purify air. Ozone may indeed be of some concern. Some research notes:

"High levels of atmospheric ozone in Florence air correlated with DNA damage, and to the prevalence of inflammatory pathologies of the upper respiratory tract, although the ozone concentrations were below the Italian recommended attention level. Furthermore, higher levels of DNA damage were correlated with a dysfunction in the ability to maintain a normal epithelial cell structure. These data suggest an association between ozone air levels and damage in the upper respiratory tract. It remains unclear whether ozone itself or other associated pollutants are responsible for the observed alterations." (Environ. Mol. Mutagen. 42:127-135, 2003).

"Beagle dogs exposed for 8 hours a day to 3 ppm O_3 for 18 months showed cytologic changes that indicate metabolic alterations. The endoplasmic reticulum of the type 2 alveolar epithelial cells in the proximal alveoli were frequently dilated and contained a moderately electron-dense substance having a periodicity of approximately 754 A. This condition was accompanied by a substantial reduction in the lamellar membranes in the characteristic lamellar bodies of the type 2 cells, suggesting a sequestering of protein in the endoplasmic reticulum. In addition, endothelial cells contained paracrystalline arrays of cytoplasmic membranes not observed in control animals" (Am J Pathol. 1973 December; 73(3): 711–726).

"The interaction of ozone with some molecules results in an increased production of free radicals. The objective of this study was to identify whether acute ozone exposure to 1–1.5ppm for 4hr, produced cytological and ultrastructural modifications in the olfactory bulb cells. The results showed that in rats exposed to ozone there was a significant loss of dendritic spines on primary and secondary dendrites of granule cells, whereas the control rats did not present such changes. Besides these exposed cells showed vacuolation of neuronal cytoplasm, swelling of Golgi apparatus and mitochondrion, dilation cisterns of the rough endoplasmic reticulum. These findings suggest that oxidative stress produced by ozone induces alterations in the granule layer of the olfactory bulb, which may be related to functional modifications" (Neuroscience Letters Volume 274, Issue 1, 15 October 1999, Pages 1-4).

"Inhalation of the pulmonary irritant ozone is associated with an accumulation of macrophages in the lung. These cells, along with type II epithelial cells, are activated to release increased quantities of hydrogen peroxide and nitric oxide, two reactive mediators that have been implicated in tissue injury" (Am J Respir Cell Mol Biol. 1996 Jun ;14 (6):516-25).

Ozone-induced high-protein alveolar edema is pathogenetically linked to pulmonary hyperemia, deficiency of surfactant tubular myelin, and associated lung dysfunctions" (Toxicol Appl Pharmacol Vol. 117 Issue 1 Pg. 37-45 Nov 1992).

Histology Group of South Australia

Is Proud to Present The

REGISTRATION FORMS

NOW ONLINE

www.nhc.org.au

Workshop 1

Dr. Craig James

Surgical Grossing Of Skin Specimens

Workshop 2

Dr. John K C Chan

Immunohistochemistry - Technical And Interpretation Pitfalls

Keynote Speakers

Dr J K C Chan – Immunogenetics Of Tumours, Achieving New Heights By Immunohistochemistry

Dr J Robin Warren – How A Lifetime's Work With Helicobacter Pylori Led To A Nobel Prize In Medicine

Friday 8th May	10:00	Workshop 1
	13:00	Workshop 2
	18:30	Trade Opening with Cocktail Party
Saturday 9th May	09:00 - 17:00	Plenary Sessions
	18:30	Pre-Dinner Drinks
	19:30	Conference Dinner
Sunday 10th May	09:30	Plenary Sessions
	14:00	Finish With Late Lunch

Gossypiboma What the?

Any ideas what a Gossypiboma is? Is it a benign rumour about a tumour? Well it is also known as a textiloma. Maybe a tumour of our SMS'ing fingers?

OK The name gossypiboma is derived from the Latin gossypium (cotton) and the Swahili boma (place of concealment). Any Takers?

Gossypiboma denotes a mass of cotton or sponge that is retained in the body after surgery. Retained surgical sponge (gossypiboma) is an uncommon surgical complication, with an incidence of 1:1500 for intra-abdominal operations. Patients may be asymptomatic, or it may lead to serious complications such as bowel or visceral perforation, obstruction, fistula formation, abscess, pseudotumour syndrome or foreign body granuloma. Pathologically, two types of foreign body reaction can occur. Most commonly, there is aseptic fibrinous inflammatory reaction, and adhesions encapsulate the sponge in the omentum and near-by organ. The other type is exudative inflammatory reaction, with an abscess or chronic internal/external fistula formation[1].

Gossypiboma often mimic tumour masses radiologically and in a patient with a history of prior malignancy, a recurrence is often considered. Surgically a gossypiboma may look like a dermoid cyst. On slicing, surgical gauze or sponge is usually found[3]. Retained sponges are more common than retained instruments because of their small size, frequent use, and ability to mimic intraabdominal contents when saturated in blood[4].

There have been reports where a retained surgical sponge has eroded from the intra-abdominal space into the intestinal lumen, migrated distally, and spontaneously passed with defecation[2].

Szentmariay et al[4] have described a male patient with a 23-year-old history of right lower lung lobectomy for primary pulmonary adenocarcinoma who presented with recurrent bronchopneumonia and purulent sputum. Pleural callus, lung abscess, bronchopleural fistula, and stitch granulomas were confirmed by chest x-ray, computed tomography scan, and bronchoscopy in the background of his complaints.

An attempt to remove the bronchial purulent discharge and tissue sampling was made by using a flexible bronchoscope. The area of the lower trachea suddenly became clogged during bronchoscopic removal of the suspected piece of tissue (which later turned out to be organizing surgical gauze). The resuscitation following ventricular fibrillation failed to save the patient's life. The postmortem examination confirmed the position of the foreign body extending from the abscess cavity, crossing the midline at carina and obstructing the lower trachea. This foreign body was a remnant of the surgical gauze left behind during a thoracic surgery 23 years ago.

References

1. Mahalik, Puneet, Gupta, Khanna (2008) ANZ J Surg 78(5):417-8
2. Zantvoord, van der Weiden & van Hooff (2008) Obstetrical & Gynecological Survey 63(7): 465-471
3. Possover (2008) NEJM 359(8):e9
4. Szentmariay et al (2004) Am J Forensic Med Pathol 25(4): 324-326.

.

From Histonet – Decolourising a PAS

"We have a section that was incorrectly stained with PAS and no tissue left to recut. We need to do an H. Pylori stain on the slide. Any thoughts on how to destain the PAS?"

- You can destain PAS with a very very very diluted solution of Clorox (Household bleach) (René J Buesa)
- Overnight immersion in concentrated (i.e., 28%) ammonium hydroxide removes the Schiff reagent and restores the original aldehydes in the tissue (Isaac Glickfield).

Mild Increase in Advertising Charges

After several years we have decided that a modest increase in advertising charges is warranted. The committee decided that the new prices be:

A4 PAGE - PRINTED COPIES SUPPLIED BY ADVERTISER
1 or 2 sides..... $75.00

A4 B&W PAGE - PRINTED IN THE JOURNAL
1 A4 page $100.00

A4 COLOUR - PRINTED IN THE JOURNAL
1 A4 page $300.00

Histograph is expertly printed by:

I'm Getting Old – My Pigments are Up

A Review of Lipofuscins

Lipofuscin is the name given to brown pigment granules composed of lipid-containing residues of lysosomal digestion. It is considered one of the aging or "wear and tear" pigments; found in the liver, kidney, heart muscle, adrenals, nerve cells, and ganglion cells. "Liver spots" commonly associated with aging are superficial dermal lipofuscin deposits (1).

Lipofuscin accumulates in post-mitotic cells, located in small granules in secondary lysosomes, and consists mostly of crosslinked lipid and protein residues formed during lipid peroxidation. Antioxidants inhibit lipofuscin formation. Although oxidative damage to organelles seems responsible for depositing lipofuscin in lysosomes of senescent mammals, it is not clear whether it is a biomarker or a mechanism of aging. Two explanations for the increase of lipofuscin with age have been suggested. The first theory is based on the fact that lipofuscin is not totally eliminated and accumulates in post-mitotic cells over time. The second suggests that lipofuscin accumulation reflects a derangement of autophagocytosis associated with decline in intra-lysosomal degradation, as more lysosomal volume is occupied by indigestible material (2).

Lipofuscin has been defined as a pigment that exhibited yellow autofluorescence under specified conditions, i.e. light from a mercury arc lamp passed through a filter that transmitted violet and blue light, and the resulting yellow lipofuscin autofluorescence was examined after passage through a barrier filter which transmitted wavelenghts above 515 nm. The presence of yellow (i.e. yellow-green/orange/red) autofluorescence under these conditions indicated the presence of lipofuscin (3). Even though lipofuscin granules are often autofluorescent; however, when they contain iron or copper the fluorescence is often quenched (4).

Lipofuscin is acid-fast, PAS-positive after diastase digestion, slightly argyophilic and sudanophilic (so called alcohol-insoluble lipids), and markedly Schmorl's- and peroxidase positive in paraffin sections. It is often difficult to see in sections stained with hematoxylin and eosin; the pigment can be detected in unstained sections (4).

Another interesting point is the distinction between lipofuscin and neuromelanin. Lipofuscin has been found in carotid body chief cells, as well as in neoplastic chief cells of the carotid body and jugular paragangliomas. Histochemically, lipofuscin may give misleading staining patterns such as argentaffin positivity, but it is usually considered positive with periodic acid–Schiff and Ziehl-Neelsen stains. Both pigments are similar when ultrastructurally viewed (5).

Neuromelanin function has yet to be determined. This substance is considered a waste

product of catecholamine metabolism, derived from the oxidation of dopamine, norepinephrine, and compounds related to quinones. One of its functions is to protect the cell against toxic quinines produced from catecholamines. It is believable that, in particular conditions such as the neoplastic process, an excessive production of catecholamines may cause defects in their cytoplasmatic storage, transport, or degradation and favor accumulation of neuromelanin by an auto-oxidative pathway. Therefore, because of some different histochemical properties with untreated lipofuscin such as absence of autofluorescence under fluorescence microscopy, it has been suggested that neuromelanin may be a melanised lipofuscin. This aspect could be confirmed by findings of neuromelanin-like lipofuscin in pigmented adrenal cortical nodules, primary pigmented nodular adrenocortical disease, and black thyroid syndrome (5).

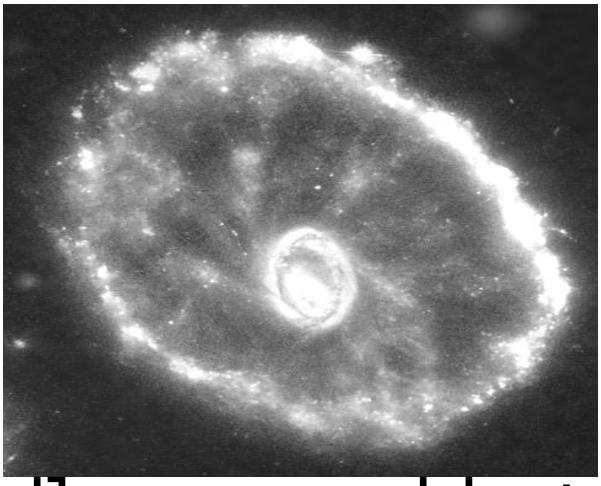
In the prostate lipofuscin is seen in cells undergoing regressive changes, the seminal vesicles and the ejaculatory ducts. It is also present in prostatic adenocarcinoma.
Mahmoodi et al (6) studied the prognostic significance of lipofuscin in prostatic adenocarcinoma.
Lipofuscin pigment was found in 17 (31%) of 60 prostatic adenocarcinomas as random, sparse, fine, yellow-brown intracytoplasmic granules staining positive for cathepsin D and negative for S-100 protein. Using logistic regression to exclude age as a confounding factor, lower Gleason scores and pathologic stages were demonstrated in the lipofuscin-positive group. There was also a significant difference between the 2 groups in tumor volume, degree of capsular invasion, and positive margins. Lipofuscin in prostatic adenocarcinoma correlates with both lower Gleason score and pathologic stage. Mahmoodi et al (6) suggest that lipofuscin probably indicates slow cellular turnover as suggested by the low proliferation rate and p53 expression. The value of lipofuscin in biopsy as a predictor separating aggressive from indolent disease needs further investigation.

The prospect of removing cellular deposits of lipofuscin is of considerable interest because they may contribute to age related functional decline and disease. Fonseca et al (7) have shown that lipofuscin, accrued through normal ageing, can be lost from neural tissue. The mechanism of loss probably involves exocytosis and possibly blood transport. If non-disruptive ways to accelerate lipofuscin removal can be found, their results suggest that therapeutic reversal of this most universal manifestation of cellular ageing may be possible.

References:

1. Wikipedia Encyclopedia http://en.wikipedia.org/wiki/Lipofuscin
2. Melk & Halloran (2001) J Am Soc Nephrol 12: 385–393
3. Mountjoy et al (2005) Acta Neuropathologica
4. Goldfischer & Bernstein (1969) J Cell Biology 42:251-263.
5. Bellezza et al (2004) Arch Pathol Lab Med. 128:e125–e128.
6. Mahmoodi et al (2006) Ann Diag Pathol 10(5): 257-262
7. Fonseca et al (2005) Neurobiology of Aging 26(1): 69-76

Join the stars for Christmas



Proudly sponsored by Leica

Dr Richard Jaworski is an amateur astronomer in his other life, when he's not a pathologist

Come with him on a Sky Tour – both virtual and real (weather permitting) + **Trivia quiz and prizes to be won**

Date: Friday 5 December, 2008

Where: North Ryde RSL, Cnr Pittwater and Magdala Rds, North Ryde

Time: 6.30 pm for 7.00 pm start

Cost: $30 per person includes 3 course dinner and bar tab.

RSVP: For catering purposes by 1st December 2008 with payment to
HISTOTECHNOLOGY GROUP of N.S.W.
P.O. BOX 496, GUILDFORD NSW 2161

Please make cheques payable to the Histotechnology Group of NSW
Or: I**nternet Banking**: BSB: **802 084**; Account number: **94099;**

Account name: **Histotechnology Group of NSW**.

Contact : kdrummond@dhm.com.au or fax attn: Kathy Drummond 9855 5169

The Medical Team

A surgeon, pathologist, physician, psychiatrist and radiologist go duck hunting out on a pond.

As the first duck flies over head, the psychiatrist takes aim, ...but doesn't fire.

"What the hell happened?" demands the surgeon (they're known for their retiring manner)

"Well, says the psychiatrist, I know its a duck, and YOU know its a duck - but does the duck know its a duck?"

The second bird flies over head, and the physician takes aim.....but doesn't fire.

"What now???" roars the surgeon.

Well, says the physician, in a recent article I read in the New England Journal of Medicine, 12% of ducks are actually Terns, and they're a protected species....

The third duck flies over, and the radiologist takes aim.....then paddles the boat further into the pond, then takes aim, then paddles back a bit, then takes a further aim....and by this time the duck has gone.

"Jesus wept!" explodes the surgeon, what the f*** were you doing?

Well, says the radiologist - every time I took aim, I realised that I needed another view.

After lunch, they re-group for more hunting.

This time the surgeon gets out his double barrelled, gold plated bazooka, blows his duck whistle - and as a flock of birds swarm over head, he fires randomly and enthusiastically into the air. Objects rain out of the sky. When he had finally finished blasting away to the heavens - he turned to the pathologist and said: "row over to all those bodies, and tell me if any of them was a duck"

Form Craig (Joe) Farish, School of Agricultural and Veterinary Sciences, Charles Sturt University, Wagga Wagga NSW

.

De-waxing Update

Incomplete wax removal can cause several problems including:

- The birefringence of cell nuclei (figure 1)
- So-called Pink Disease – where there is a patchy distribution of stains and loss of distinction of nuclear margins (figure 2)
- As well as weak to false negative immunohistochemical stains.

Though there may be several causes of Pink disease, one common cause is the incomplete removal of wax.

In the early 60s, it was known that cell nuclei were often birefringent. It was commonly accepted that this phenomena was due to incomplete wax removal by xylene. This artifact is often seen in hepatocyte nuclei as seen here. Vlachos (1) also noted that nuclear staining was weaker in areas that showed increased birefringence.

Faolain (2) used Ramon Spectroscopy to show residual wax in de-waxed sections. They compared xylene, histoclear, antigen retrieval with citrate following xylene, hexane and Trilogy.

Ramon Microspectroscopy is a non-destructive technique that provides information about the molecular structure of the sample. The Raman effect arises when the incident light excites molecules in the sample, which subsequently scatter the light. While most of this scattered light is at the same wavelength as the incident light (λ1), some is scattered at a different wavelength (λ2). This inelastically scattered light (λ2) is called Raman scatter and results from the interaction of the incident light with the molecular motions or vibrations. The positions, intensities and line-widths of the Raman lines, corresponding to vibrational energy levels, yield information on the composition, secondary structure and interaction of molecules, including the chemical microenvironment of molecular subgroups. The usefulness of the technique lies in its possible ability in differentiating normal tissues from tumours.

Their results indicate that apart from hexane, all of the de-waxing agents failed to fully dissolve wax from sections. They also found that immunohistochemical results were significantly better after de-waxing in hexane (28% increase in positivity with Hexane over Xylene).

Falkeholm et al (3) have evaluated a xylene-free method that excludes xylene, not only as the intermediate step before the paraffin baths, but also for de-waxing of the cut sections, which also eliminates the need for rehydration and dehydration for the staining and mounting steps. Elimination of xylene from tissue processing cuts costs, saves time, and improves the laboratory environment. They have used this technology since 1995.

The dewaxing procedure involves placing the slides in a heated detergent solution for 30 seconds (x2) followed by rinsing in hot water and then storage in room temperature water prior to staining. After staining, slides were air-dried prior

to routine coverslipping (ie without alcohol or xylene). Overall results were equitable to routine staining with the exception of the van Gieson stain which scored poorly.

A collaborative study is currently underway at the Children's Hospital at Westmead in conjunction with Rene J Buesa (USA), Maxim (Russia) and Dr. Ian Montgomery (University of Glasgow, Scotland) to confirm these findings. At the Children's Hospital conventional dewaxing is being compared with detergent dewaxing in the immunolocalisation of several antigens using the Bond system. To date the results have been comparable and seem to be worth serious consideration.

References

1. Vlachos (1968) "Birefringence and Paraffinophilia of Cell Nuclei" Stain Techn 43(2):89-95
2. O'Faolain E et al. (2005) Raman spectroscopic evaluation of efficacy of current paraffin wax section dewaxing agents. J. Histochem. Cytochem. 53(1): 121-129.
3. Falkeholm et al (2001) "Xylene-Free Method for Histological Preparation: A Multicentre Evaluation" Lab Invest 81:1213–1221.

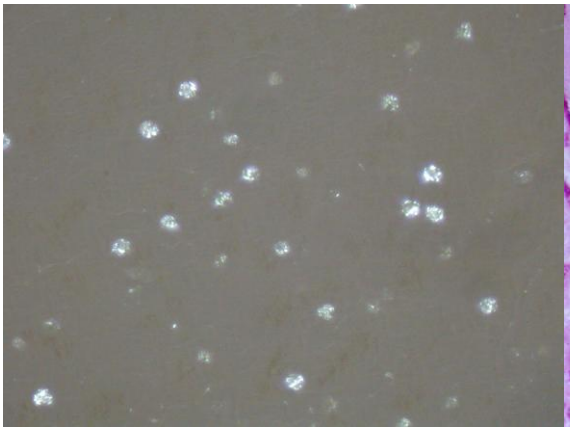


Figure 1 Birefringence of cell nuclei after incomplete wax removal

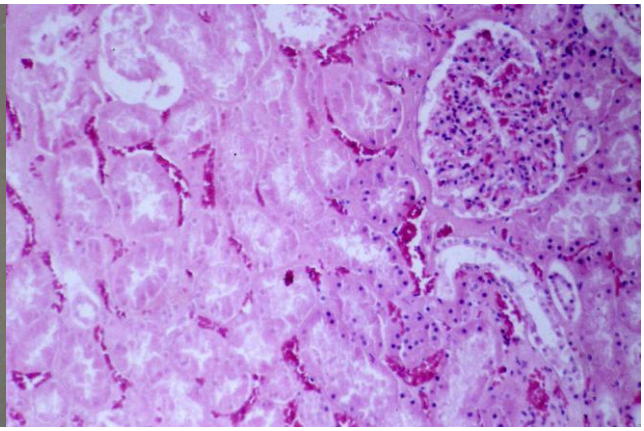


Figure 2 Kidney showing incomplete wax removal H&E stain

The Batman-Black Beauty Virus

Oh the lengths I go to get a catchy title. So what is he talking about, you ask? It is the Hendra Virus. In September 1994, a sudden outbreak of an acute respiratory syndrome occurred among thoroughbred horses in a training complex in Brisbane. Thirteen horses and their trainer died. The causal agent, a previously undescribed member of the family Paramyxoviridae, was initially named equine morbillivirus, but was renamed Hendra virus (after the Brisbane suburb where the outbreak occurred). A second (apparently unrelated) outbreak resulted in the death of two horses and their owner near Mackay, nearly 1000 km north of Brisbane. The outbreak preceded the events at Hendra and was retrospectively identified in 1995. Most recently, a single fatal equine case occurred near Cairns in North Queensland in January 1999[1].

To evaluate the theory that Hendra virus existed in a wildlife reservoir, serologic surveillance of wildlife species was undertaken, and, in April 1996, anti-Hendra virus antibodies were identified in a black flying fox (Pteropus alecto). Within weeks, evidence of infection was found in the other three species of Australian flying foxes; grey-headed flying fox (P. poliocephalus), little red flying fox (P. scapulatus), and spectacled flying fox (P. conspicillatus). In 1996, a Hendra-like virus was isolated from the reproductive tract of a seemingly healthy, pregnant grey-headed flying fox. A range of tests showed the bat isolate to be indistinguishable from the Hendra virus isolated from horses. However, no evidence of illness exists in flying foxes infected naturally or infected experimentally that can be attributed to infection with Hendra virus, supporting epidemiologic evidence that flying foxes are the probable hosts of Hendra virus[1].

Hendra virus does not appear to be very contagious, and there has been no evidence of infection in humans even in those who have had close contact with injured bats. Transmission from flying foxes to horses has not been demonstrated; however, studies done on different species infected experimentally and flying foxes and horses infected naturally have indicated possible modes of transmission. Virus has been isolated from the kidney, urine, and (less so) oral cavity of horses and from the kidney and urine of cats experimentally infected with Hendra virus. Horses have been experimentally infected by the naso-oral route, and cat-to-cat transmission and

suspected cat-to-horse transmission have been reported[1].

Most patients present with a severe acute encephalitic syndrome, but some also have significant pulmonary manifestations. The virus causes cells to clump together in giant multinucleate cells or "syncytia".

Histological findings show severe endothelial damage and vasculitis, mainly in the arterioles, capillaries, and venules. The most severely affected organ was the brain, but other organs including the lung, heart and kidney were also not spared. Vasculitic vessels were characterized by vessel-wall necrosis, thrombosis, and inflammatory-cell infiltration of neutrophils and mononuclear cells. Syncytial-cell formation was seen in the endothelium of affected blood vessels in the brain and lung, and in the Bowman's capsule of the glomerulus. Zones of microinfarction and ischaemia were commonly found around or adjacent to vasculitic blood vessels, affecting both the grey and white matter of the cerebrum, basal ganglion, cerebellum, brainstem and spinal cord[2].

Although there have been no cases of human-to-human transmission, the CDC classified the Hendra Virus as a P4-pathogen. That meant samples can be collected and handled only by researchers clad in space suits and examined only in high-level safety labs. As for the virus's mode of transmission, one theory is that it is present in horse lungs and urine and that humans can get infected by inhaling aerosols. What is particularly worrying is that one of the Australian victims of the Hendra virus died 14 months after he was infected[3].

References

1. Mackenzie et al (2001) Emerging Infectious Diseases Vol. 7, No. 3 Supplement, pp 497-504.
2. Lam (2003) Antiviral Research 57:113–119
3. Science 16 April 1999: Vol. 284. no. 5413, pp. 407 - 410

Histotechnology Group of NSW Membership Application

2008 - 2009

I wish to become a member of the Histotechnology Group of N.S.W. and enclose

PLEASE TICK

- ☐ $38.50 for annual subscription of $35.00 and $3.50 GST.
- ☐ $16.50 for student subscription of $15.00 and $1.50 GST
  (Full-time or working toward first qualification)
- ☐ $82.50 for company subscription of $75.00 and $7.50 GST
  (2 representatives, one of whom must be a NSW representative)
- ☐ RENEWALS ☐ ANY CHANGES TO PREVIOUS DETAILS.

PLEASE PRINT ALL INFORMATION.

SURNAME_____ GIVEN NAME_____

TITLE: MR, MRS, MS, DR, MISS. (Circle one)

OCCUPATION_____POSITION_____

INSTITUTION_____DEPARTMENT_____

ADDRESS for CORRESPONDENCE:

STREET/P.O.BOX._____

CITY,TOWN,SUBURB,_____POSTCODE._____

IS THIS ADDRESS HOME OR BUSINESS ? (Circle One).

PHONE No. WORK_____ EXT_____ HOME_____

E-MAIL ADDRESS: _____

2ND COMPANY CONTACT

SURNAME_____ GIVEN NAME_____

TITLE: MR, MRS, MS, DR, MISS. (Circle one)

POSITION_____ INSTITUTION_____

ADDRESS STREET/P.O.BOX._____

CITY,TOWN,SUBURB,_____POSTCODE._____

PHONE No. WORK_____ HOME_____

E-MAIL ADDRESS: _____

SIGNATURE_____**DATE**_____

RETURN TO:

SECRETARY
HISTOTECHNOLOGY GROUP of N.S.W.
P.O. BOX 496
GUILDFORD NSW 2161

Office use only