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The Histotechnology Group of Queensland Newsletter

TISSUE PAPER

"Bridging histology laboratories since 1982"

2015 HGQ executive committee

President's Report - Jerres Alcober

Welcome to the first edition of the Tissue Paper for this year. As we move forward from a successful 2015, 2016 is set to be another actioned pack year.

Last year's AGM hosted by Pathology QLD - TPCH at Kedron Wavell RSL, brought about a new committee with new faces and fresh ideas. The 2016 HGQ Executive Committee are as follows - President: Jerres Alcober; Secretary: Michael Staunton; Treasurer: Becca Thuell; Editor: Anthony Van Zwieten; Committee Members: Jason Tu; Melissa Hillas; Greg Viggers; Lloyd Blundell; David Gan; Kellie Vukovic; Jessica Hurst & CK Tan.

Thank you to the 2015 committee for all their hard work, efforts & contributions to the 2015 NHC in Brisbane. I'd like to take this opportunity to personally thank Anthony Van Zwieten & Jason Tu for all that they have achieved in their respective roles as President & Treasurer. I am looking forward to working closely with Michael Stuanton & Becca Thuell and the entire committee in my new role as president of the HGQ.

The HGQ have brought and will be bringing members the following events in the upcoming months: Scientific Meetings - RBWH: Thu 25 Feb; Mater Pathology: Thu 26 May; AIMS/HGQ: Wed 3 Aug; ASC/HGQ: TBC (Nov); Conference - HSNSW/HGQ State Histology Conference – 30 Sep – 2 Oct. Make sure you mark the upcoming events in your calendar as we'd love to see you there.

Join 300 plus members that "Keep in the know" with the HGQ by signing up for "Free" membership at www.hgq.org.au the official HGQ website.

Until the next edition, take care, stay safe and enjoy!!! Happy reading ;)

Newsletter Design : Jerres Alcober

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Secretarial Report - Michael Staunton

A big hello to all of our readers and welcome to all our members, old and new. I have recently taken over the reins as secretary from Jerres Alcober (who has moved onto greener pastures as the HGQ president) and hope to have a great time in the HGQ.

Firstly we would like to congratulate the RBWH Central Lab for the great scientific meeting in February and to thank everyone for the wonderful turnout. Special mention to Christiano Samuela for his talk on fetal brain retrieval and the Abacus/ Milestone team for their presentation of the PrestoCHILL.

More events to watch out for include the upcoming social event and the joint HGQ/HistoNSW conference "Tides of Change" in Port Macquarie (Sep 30th – Oct 2nd, see our website for more information).

Make sure you keep up to date with the HGQ by registering your details on our website (it's easy and best of all, free) and keep an eye out for updates to the HGQ app leading into the Port Macquarie conference.

Hope to see you all in upcoming events and that 2016 continues to shine for everyone in the "Sunshine State".

Wishing you all the best.

Publication Guidelines

Newsletter Correspondence

Histotechnology Group of Queensland
Anatomical Pathology Department
Level 2 – Clinical Sciences Building
The Prince Charles Hospital
Rode Road, Chermside, QLD 4032
admin@hgq.org.au
(07) 3139-4379

Tony Reilly - Life Member Inductee

Guidelines to Contributors

Please forward submissions in Microsoft Word or compatible program either via email and/or CD & DVD. For any attached photos, please also include these in a separate file. Include your name and address if required. Submissions can be in the form of a brief note, letter or as a complete article.

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Histotechnology Group of Queensland

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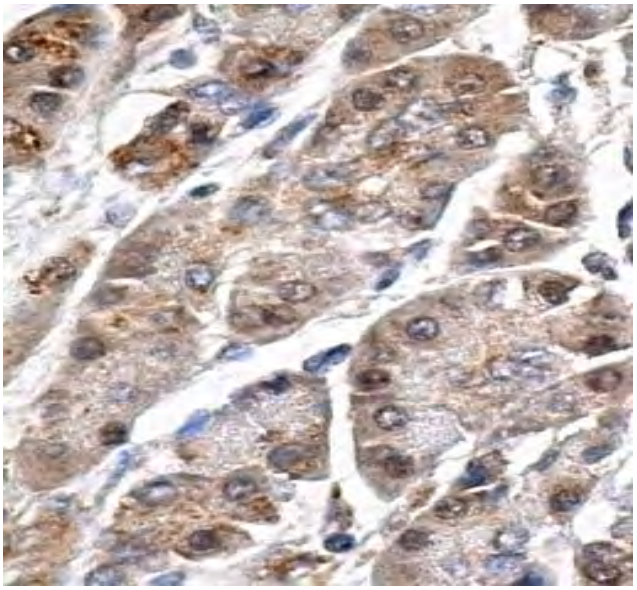
Spotlight

Summer



BAP 1 (C-4) #SC-28383

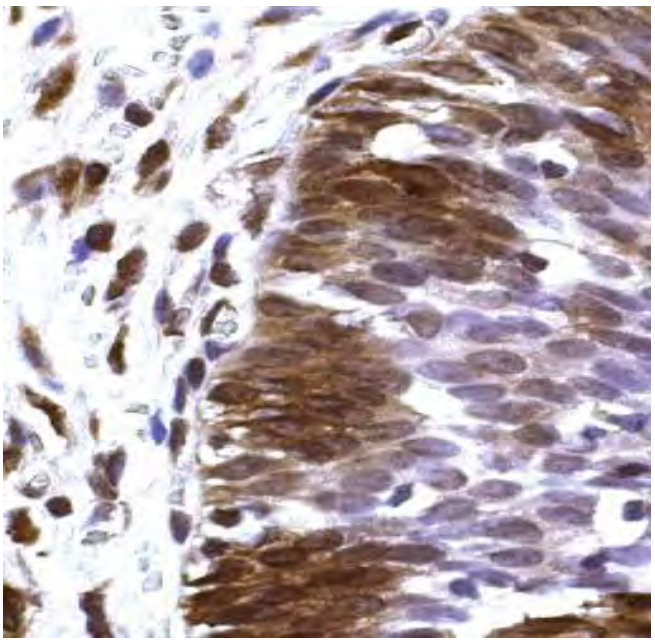
Mouse Monoclonal

Mutations within the BRCA1 gene, localized to chromosome 17q, are believed to account for approximately 45% of families with increased incidence of both early-onset breast cancer and ovarian cancer. The BRCA1 gene is expressed in numerous tissues, including breast and ovary, and encodes a predicted protein of 1863 amino acids. This protein contains a RING domain near the N-terminus and appears to encode a tumor suppressor. BARD1 (BRCA1-associated RING domain protein 1) and BAP1 (BRCA1-associated protein 1) have both been shown to bind to the N-terminus of BRCA1 and are potential mediators of tumor suppression. BARD1 contains an N-terminal RING domain and three tandem ankyrin repeats. The C-terminus of BARD1 contains a region with sequence homology to BRCA1, termed the BRCT domain. BAP1 is a ubiquitin hydrolase and has been shown to enhance BRCA1-mediated cell growth suppression.

STAT 6 (S-20) #SC-621

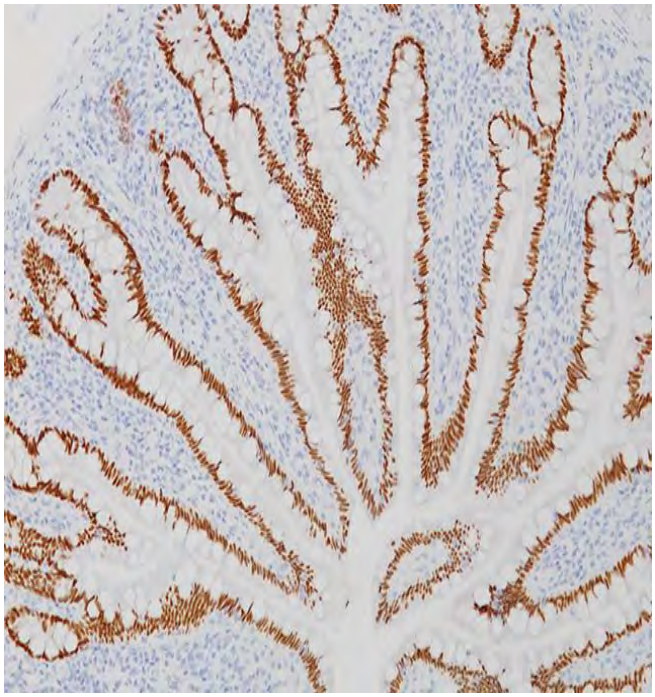
Rabbit Monoclonal

Membrane receptor signalling by various ligands, including interferons and growth hormones such as EGF, induces activation of JAK kinases which then leads to tyrosine phosphorylation of proteins that have been designated Stats (signal transducers and activators of transcription. The first members of this family to be described include Stat1å p91, Stat1ſ p84 (a form of p91 that lacks 38 COOH-terminal amino acids) and Stat2 p113. Stat1 and Stat2 are induced by IFN-a and form a heterodimer which is part of the ISGF3 transcription factor complex. Stat3, which becomes activated in response to epidermal growth factor (EGF) and interleukin-6 (IL-6), but not interferon-© (IFN-©) or Stat4, is an additional member of this family. It has been suggested that the phosphorylated forms of both Stat3 and Stat4 form homodimers as well as heterodimers with the other members of the Stat family, and that differential activation of different Stat proteins in response to different ligands should help to explain specificity in nuclear signalling from the cell surface. Highest expression of Stat4 is seen in testis and myeloid cells. IL-12 has been identified as an activator of Stat4. Other members of the Stat family include Stat5, which has been shown to be activated by prolactin and by IL-3, and Stat6 (also designated IL-4 Stat), which is involved in IL-4-activated signalling pathways.

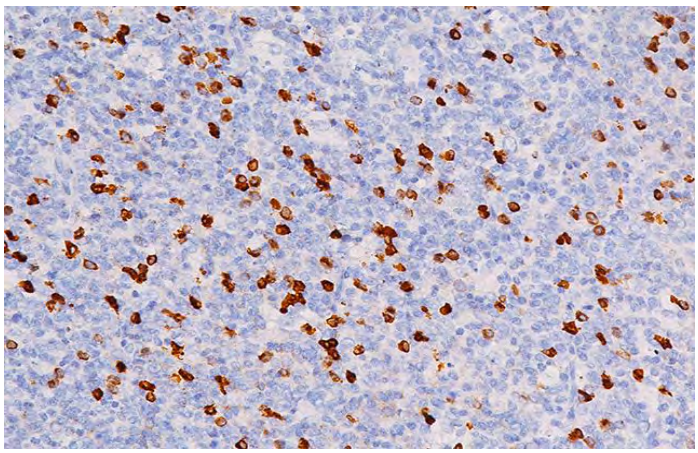


CDX2 #ACI3144

Rabbit Monoclonal



CDX2 is a homeobox gene that encodes an intestine-specific transcription factor (1). CDX2 has been useful to establish gastrointestinal origin of metastatic adenocarcinomas and carcinoids and can be especially useful in distinguishing metastatic colorectal adenocarcinoma from tumors of unknown origin (1-7). CDX2 has been shown to be more specific and more sensitive than villin or CK20 (1,4,6). CDX2 has also been shown to be expressed in mucinous ovarian cancer, bladder adenocarcinoma, cholangiocarcinoma and malignant germ cell tumors of the testes (1,2,6-8). Only very rare examples of carcinomas of the genitourinary and gynaecologic tracts or breast, lung, and head and neck cancers showed elevated levels of CDX2 expression (1). Recently, a new rabbit monoclonal CDX2 has been developed and studies have shown that CDX2 rabbit monoclonal is a more sensitive clone than other CDX2 mouse monoclonal antibodies. Data has also shown that rabbit monoclonal CDX2 had fewer false negatives (9). The specificity was similar when compared to other mouse monoclonal CDX2 antibodies. However, in certain cancers, rabbit monoclonal CDX2 displayed a slightly higher percentage (9). The overall specificity for CDX2 antibodies can be significantly improved in a panel with CK7, TTF-1 and CDH17 (3,4,6,10).



KAPPA LIGHT CHAIN [L1C1] #ACI3149

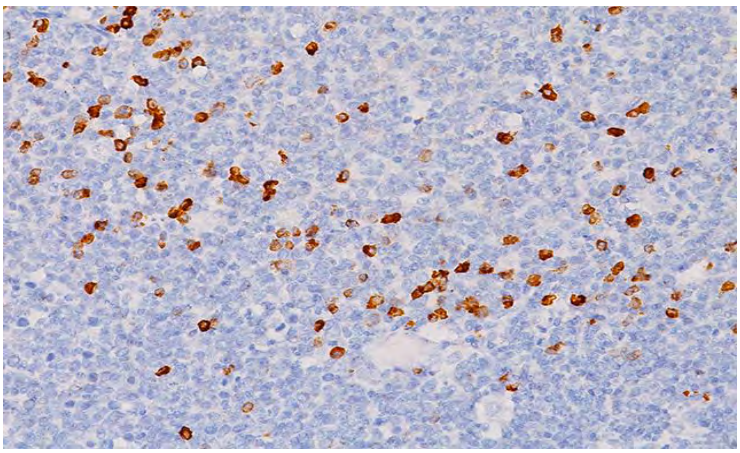
Mouse Monoclonal

This antibody recognizes kappa light chains of human immunoglobulins, which may be useful in the identification of leukaemia, plasmacytomas, and certain non-Hodgkin's lymphomas (1-5). The most common feature of these malignancies is the restricted expression of a single light chain class. The normal human kappa/lambda ratio is approximately 2:1. The presence of clear cut light chain restriction with a kappa/lambda ratio more than 10:1 is consistent with a malignant proliferation (2,6).

LAMDA LIGHT CHAIN [N10/2] #ACI3063

Mouse Monoclonal

This antibody recognizes lambda light chains of human immunoglobulins, which may be useful in the identification of leukaemia, plasmacytomas, and certain non-Hodgkin's lymphomas (1-5). The most common feature of these malignancies is the restricted expression of a single light chain class. The normal human kappa/lambda ratio is approximately 2:1. The presence of clear cut light chain restriction with a kappa/lambda ratio more than 10:1 is consistent with a malignant proliferation (2,6).



Pagets Disease

Beverley Pardon - Anatomical Pathology
Nambour General Hospital

Biography:

Sir James Paget (11 January 1814 – 30 December 1899) was an English surgeon and pathologist who is considered, together with Rudolf Virchow, as one of the founders of scientific medical pathology.

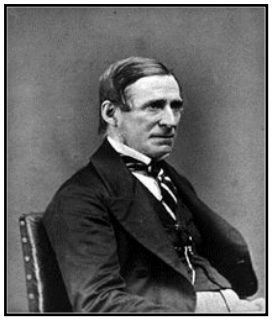
His famous works included Lectures on Tumours (1851) and Lectures on Surgical Pathology (1853).

He was born in Great Yarmouth, England, the son of a brewer and shipowner. At the age of 16 he was apprenticed to a general practitioner for 4 years.

He discovered the pathogen for trichinosis, a parasitic disease caused by Trichina spiralis, a minute roundworm that infests the muscles of the human body which is usually acquired by eating infected pork.

In May 1836, he passed his examination at the Royal College of Surgeons, and became qualified to practice. He was a friend of Charles Darwin. Paget's greatest achievement was that he made pathology dependent, in everything, on the use of the microscope, especially the pathology of tumours.

In 1858 he was appointed surgeon to Queen Victoria, and in 1863 surgeon to Albert Edward, Prince of Wales. He had for many years the largest surgical practice in London. His name is also associated with certain great practical advances such as being the first to urge removal of the tumour, instead of amputation of the limb in cases of myeloid sarcoma. In 1869 he was elected President of the Clinical Society of London.



In 1871, he nearly died from infection at a post mortem examination. In 1875, he was president of the Royal College of Surgeons and the Medical and Chirurgical Society of London. In 1878, he gave up operating, but for eight or 10 years longer, he still had a very heavy consulting practice. In 1887 he was elected President of the Pathological Society of London. He died in London on 30 December 1899, at the age of 85.

While most people know of one or another type of Paget's disease, three diseases were named after him:

1. Paget's disease of bone (*osteitis deformans*) is a chronic disorder that can result in enlarged and misshapen bones. The excessive breakdown and formation of bone tissue causes affected bone to weaken, resulting in pain, misshapen bones, fractures, and arthritis in the joints near the affected bones.

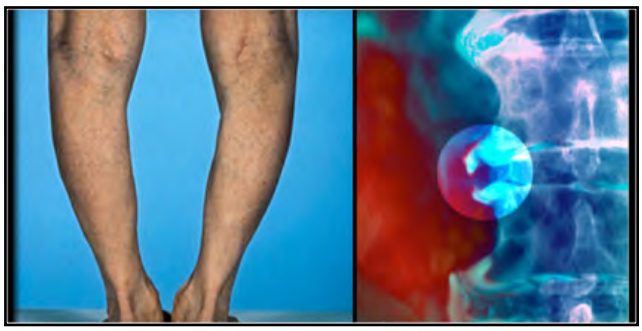
Paget's disease may be diagnosed using one or more of the following tests:

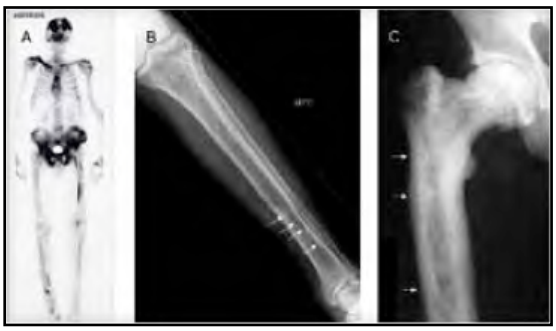
- An elevated level of alkaline phosphatase in the blood in combination with normal calcium, phosphate and aminotransferase levels in an elderly patient.
- Elevated levels of serum and urinary hydroxyproline
- Bone scans to determine the extent and activity of the condition. If suggestive of Paget's disease, the affected bone(s) should be X-rayed to confirm diagnosis

Today's medications, especially when started before complications begin, are often successful in controlling the disorder. Paget's disease is rarely diagnosed in people less than 40 years of age. Men are more commonly affected than women. Because early diagnosis and treatment is important, where there is a family history, after age 40, family members of someone with Paget's disease may wish to have an alkaline phosphatase blood test every 2 or 3 years. If this level is above normal, other tests such as a bone-specific alkaline phosphatase test, bone scan, or X-ray can be performed.

In general, patients with Paget's disease should receive 1000–1500 mg of calcium and at least 400 units of vitamin D daily. This is especially important in patients being treated with bisphosphonates. Exercise is very important in maintaining skeletal health, avoiding weight gain, and maintaining joint mobility, although undue stress on affected bones should be avoided.

Prognosis is generally good, particularly if treatment is given before major changes in the affected bones have occurred. Symptoms progress slowly and the disease does not spread to normal bones. Treatment can control Paget's disease and lessen symptoms, but is not a cure.





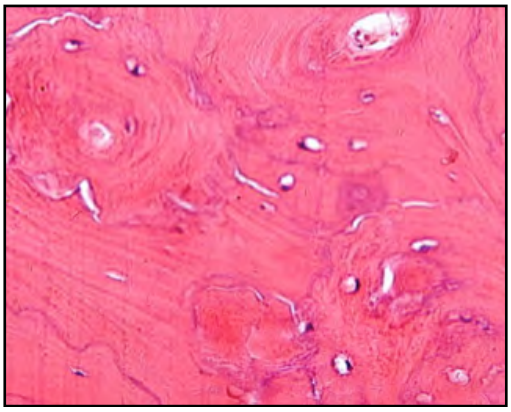
Clinical and Radiological features of Paget's disease of bone

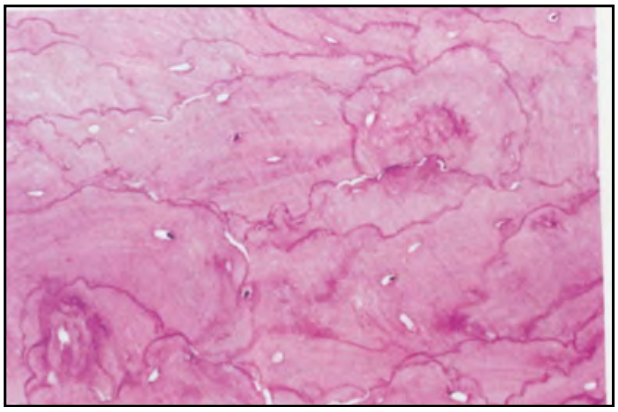
The most conspicuous pathologic changes are seen in the pelvis, skull, the bones of the legs, and in the vertebral column.

The mosaic-like irregular segments of lamellar bone with their scalloped cement lines are uniquely characteristic of this disease. The pathogenesis of Paget's disease is described in 4 stages:

1. ***Osteoclastic activity***
2. ***Mixed osteoclastic - osteoblastic activity***
3. ***Osteoblastic activity***
4. ***Malignant degeneration***

Initially, there is a marked increase in the rate of bone resorption at localized areas caused by large and numerous osteoclasts. The osteolysis is followed by a compensatory increase in bone formation induced by osteoblasts recruited to the area. This is associated with accelerated deposition of lamellar bone in a disorganized fashion. This intense cellular activity produces a chaotic picture of trabecular bone ("mosaic" pattern), rather than the normal linear lamellar pattern. The resorbed bone is replaced and the marrow spaces are filled by an excess of fibrous connective tissue with a marked increase in blood vessels, causing the bone to become hypervascular. The bone hypercellularity may then diminish, leaving a dense "pagetic bone," also known as burned-out Paget's disease.





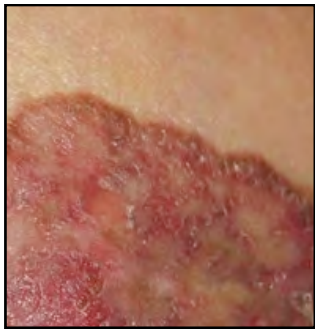
Characteristic jigsaw-like mosaic pattern

2. Paget's disease of the nipple/breast

Rare and accounts for 2-3% of all patients with breast cancer. It typically occurs in postmenopausal women (50-60 years of age). Men can also develop Paget's disease but it is extremely rare. It typically starts as a scaly erythematous patch on the nipple, then spreads to the areola.

It is characterised by infiltration of the nipple epidermis by adenocarcinoma cells, which cause an eczematous eruption on the nipple and areola. Most patients also have one or more tumours inside the same breast. These breast tumours are either ductal carcinoma in situ (DCIS) or invasive breast cancer.

The symptoms of Paget disease of the breast are often mistaken for those of some benign skin conditions, such as dermatitis or eczema. These symptoms may include:



- *Itching, tingling, or redness in the nipple and/or areola*
- *Flaking, crusty, or thickened skin on or around the nipple*
- *A flattened or inverted nipple*
- *Discharge from the nipple that may be yellowish or bloody*

Because the early symptoms of Paget disease of the breast may suggest a benign skin condition, and because the disease is rare, it may be misdiagnosed at first. People with Paget disease of the breast have often had symptoms for several months before being correctly diagnosed.

Diagnostic Tests:

1. A nipple biopsy for histopathology allows doctors to correctly diagnose Paget disease of the breast. In some cases, doctors may remove the entire nipple
2. A clinical breast examination to check for lumps or breast changes
3. Mammogram
4. Ultrasound exam
5. Magnetic resonance imaging scanPaget disease of the nipple shows large cells with atypical prominent nucleoli and pale cyoplasm which are intermingled with normal keratinocytes (arrowed)

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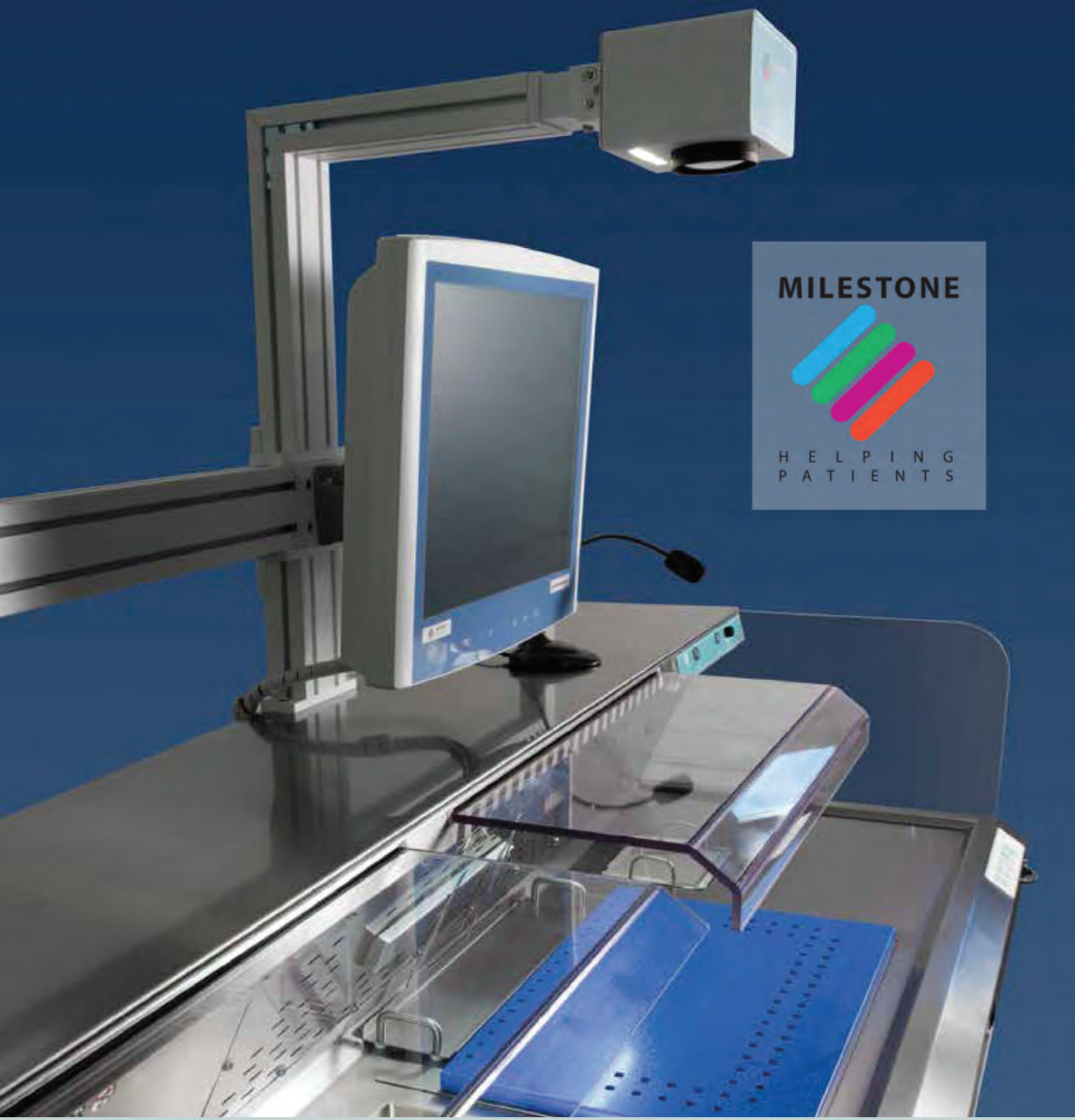

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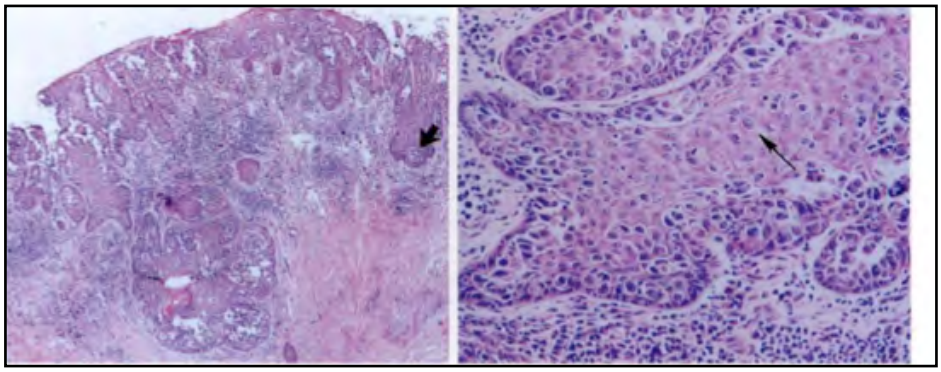


Paget disease of the nipple shows large cells with atypical prominent nucleoli and pale cyoplasm which are intermingled with normal keratinocytes (arrowed)

Primary treatment of Paget's Disease is still surgical. There are typically 3 possible clinical patterns of the disease:

1. Changes in the nipple or areolar complex only
2. Changes in the nipple or areolar complex with an underlying breast mass
3. Clinical presentation of a breast mass with histologic confirmation

Prognosis and treatment are dependent on the presence or absence of palpable masses or axillary nodes and underlying invasive carcinoma. In patients with only nipple or areolar complex involvement,skin excision with radiation therapy. In patients with a palpable mass and negative skin margins, lumpectomy with radiation is recommended. In patients with a palpable mass and involved margins, complete mastectomy is recommended

Depending on the stage and other features of the underlying breast tumor (eg., the presence or absence of lymph node involvement, estrogen and progesterone receptors in the tumor cells, and HER2 protein overexpression in the tumor cells), adjuvant therapy, consisting of chemotherapy and/or hormonal therapy, may also be recommended.

The 5-year prognosis for patients without breast masses is 85% versus 32% with breast masses. The 10-year survival rate for node-negative versus node-positive individuals is 79% versus 28%, respectively. Men have a worse prognosis, with a reported 5-year survival rate of 20% to 30%.

Paget disease of the breast should be strongly considered when any areolar or nipple lesion fails to heal with topical steroid therapy. Most lesions would clear within a month or so. A high index of suspicion is key to diagnosis.

3. Extramammary Paget's disease, which includes Paget disease of the vulva and Paget disease of the penis

Extramammary Paget's disease (EMPD), is a rare, slow-growing, usually non-invasive intraepithelial adenocarcinoma outside of the mammary gland and includes Paget's disease of the vulva and the extremely rare Paget's disease of the penis.

It involves primarily the epidermis, but occasionally extends into the underlying dermis. It tends to occur in apocrine gland-bearing areas, mostly the perineum, vulva, axilla, scrotum, penis and perianal region.

Extramammary Paget's disease is usually seen in isolation and is associated with an underlying invasive malignancy about 12% of the time.

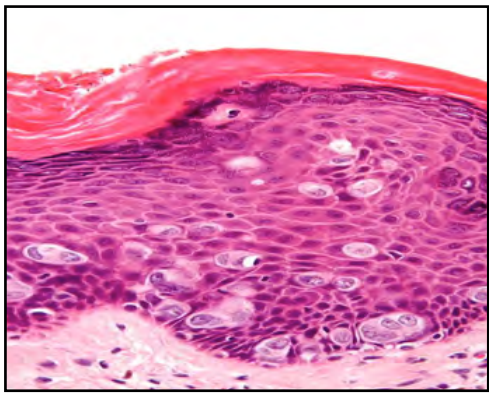
Signs and symptoms are skin lesions, often mistaken as eczema, that may be itchy or painful. A biopsy will establish the diagnosis. The histology of the lesion is similar to Paget's disease of the breast.

Types include:

- Paget's disease of the vulva, a rare disease, may be a primary lesion or associated with a secondary adenocarcinoma originating from local organs such as the Bartholin gland, urethra, endometrial, endocervical, vagina, bladder or the rectum. Patients tend to be postmenopausal.
- Paget's disease of the penis may also be primary or secondary, and is even rarer than genital Paget's disease in women. It is thought to be more frequently associated with an internal malignancy (eg. Urethral, bladder, prostatic, and testicular neoplasms). Isolated Paget's disease of the penis is extremely rare
- Anal Paget's disease occurs in the perianal region but may extend up the anal canal. Clinically, signs and symptoms can range from none to florid eczema. It can arise in perianal apocrine glands or spread from a rectal adenocarcinoma. Mucin-containing malignant cells are characteristic, but histological investigations are needed to exclude ordinary adenocarcinoma. These can include CEA, EMA and Cam 5.2 IHC to indicate the cell of origin (intraepidermal or epidermotropic).

The most appropriate management of primary EMPD disease at any site is local surgical excision, ideally with a 1cm margin of normal skin. In EMPD associated with an underlying neoplasm, treatment and management will depend on that used for that particular associated neoplasm.

Prognosis for primary EMPD confined to the epidermis is excellent. Dermal invasion complicates and worsens the prognosis depending on the depth of infiltration. Where inguinal lymph node metastases is present then 5 year survival is 0%. The prognosis for EMPD secondary to underlying neoplasm is worse and depends on the treatment for the particular carcinoma involved.



Paget disease of the vulva with a cluster of large clear tumour cells within the squamous epithelium

References:

The Royal Australian College of General Practitioners

The New England Journal of Medicine

Journal of Clinical Pathology

Manual of Surgical Pathology

Cancer Australia

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There's more to me than Histology

Kellie Vukovic - Scientist - SNP - Taringa

Thanks to Kellie for answering these questions as one of our newest HGQ committee member. For those unaware she has recently moved to QLD from her role at Peter McCallum Cancer Centre in Victoria.

How long have you worked in histology?

I completed my one year of Professional Practice at Peter MacCallum Cancer Centre in Melbourne in 2010 and have been working in Histology ever since.

When people ask, "So, what do you do?" How do you explain Histology?

I tell them my job is to cut up body parts to diagnose disease in tissue.

What is a skill you'd like to learn and why?

I would love to learn how to scuba dive now that I have moved to the Sunshine State.

If you could witness any event of the past, present or future, what would it be?

I would have loved to be around for man's first step on the moon.

What is the best conference you have ever attended?

The best would have to be the joint Aims/HGV conference which was held in Cape Schanck in Victoria in 2012. Pretty much the whole Peter Mac lab of about 10 people hired a house and all stayed together. My favourite part of the weekend was the included winery tour.

What is your dream holiday destination and why?

The Maldives – Bora Bora is a magical place that may end up under water in the future. I want to stay in a bungalow in the middle of the ocean.

Why have you made the move to QLD?

My boyfriend is a soldier in the Australian Defence Force posted to Brisbane. We have done over 2 years of long distance which included flying up almost every weekend. I decided it was finally time to give my Frequent Flyer points a break, so put my car on a truck and hopped on a plane.

What are the differences between working at PMCC and at SNP?

The major difference between the two labs is workload. Peter Mac is a public hospital with an average of 200 blocks processed per day. SNP is a 24 hour private pathology service that is processing over 2000 blocks per day.

What was your first part-time job?

I worked for 7 years at the camping store 'Ray's Outdoors'.

What is your favourite restaurant?

I can't pick one – There are so many amazing restaurants to choose from in Melbourne. I am still looking for a favourite in Brisbane but Watt Restaurant and Bar near the Powerhouse museum was great for a sunset.

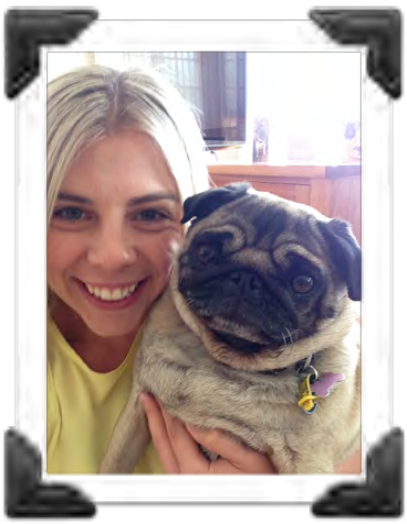
Manual or semi-automatic microtome?

Manual - I have never used a semi-automatic microtome.

Your presentation at the 2015 NHC about your training in complex cut up was well received. How nervous were you before your talk and what did you think of the conference as a whole?

The talk I gave in Brisbane was my very first time presenting at a conference. I was incredibly nervous leading up to that weekend and even though it was my story and experience, I was still afraid I would get it wrong. I practiced for hours and hours and made anyone around me listen.

The conference as a whole was extremely well run and I was very impressed in the calibre of presenters. The venue was excellent and the program was very interesting and informative. I thought the use of the HGQ phone app was a very clever idea to get the audience involved.



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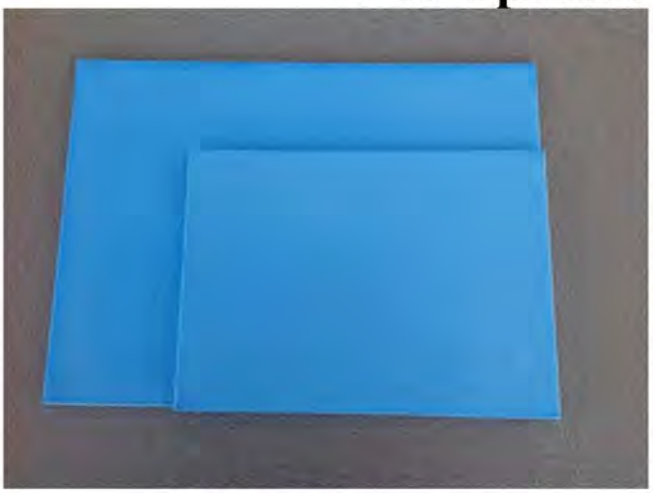

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Destroying Its Harmful Vapors While Dissecting Tissue

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- ***Desirable Blue background for excellent tissue contrast***
- ***Light Weight, High strength, Non-Stick work surface***
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- ***Soft enough to Reduce Wear on Dissecting Blades***
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- ►Specimen Storage Areas
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- ►Autopsy
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Joint State Conference 2016

"Tides of Change"

Panthers Port Macquarie - **Port Macquarie, NSW**

Friday 30 September - Sunday 2nd October 2016

www.histonsw.org.au - Register NOW

Key Note Speaker:

Dr Joe McDermott

Technical Head Anatomical Pathology

LabPlus - New Zealand

Workshops - Friday 30 September 2016

Workshop 1 – *IHC Validation by Tony Henwood*

Workshop 2 – *Presentation Skills by Dr Joe McDermott*

Members: $50 / Non-members: $65

Member Students: $50 / Non-member students: $65

Registration closes on 31st July 2016. Spots limited

Poster Presentations

Abstracts to be sent to:
bharathi.cheerala@sonichealthcare.com.au

Prizes given to eligible & successful recipients

Poster abstracts close on 30th June 2016

Plenary Sessions - Saturday 1st & Sunday 2nd October 2016

Topics include:

Team Development - *Dr Joe McDermott*

Paediatric Tumours – *Dr Susan Arbuckle*

Digital Pathology – *Jessica Unwin*

Biobanking – *Aysen Yuksel*

Interactive IHC 101 – *Anthony Van Zwieten*

Histology in the 70's – *Tony Reilly*

Forensic Pathology – *Dr Allan Cala*

Trivial Pursuit Conference Edition – *David Gan*

21st Century Learning for Laboratory Personnel – *Leah Simmons*

Registration for Conference includes:

Conference Opening Ceremony - Panthers Port Macquarie

Conference Plenary Sessions - Panthers Port Macquarie

Wine Tasting & Conference Dinner - Cassegrain Winery

Early Bird Registration for Conference closes on 31st July 2016:

Members: $300 / Non-members: $350

Registration closes on 14th September 2016:

Members: $350 / Non-members: $400

Member students: $100 / Non-member students: $150

Contacts: Bharathi Cheerala - (02) 9855 5023 - 0411 766 776 - bharathi.cheerala@sonichealthcare.com.au

Kathy Wells - (02) 9855 6271 - 0413 984 751 - kwells@dhm.com.au

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