Issue 2

The Cutting Edge

Word from the Chair

June is upon us and the next education session will be at the RAH. Flyers for the evening will be posted shortly.

The reviews of the first session in April should make interesting reading material. Roche sponsored the evening and there was a good turnout to listen to and be amused by Dr Rebecca Morrow and Dr Nicole Sladden.

Progress on the National Histology Conference is moving at a brisker pace with all working towards a National Logo for the conference. Our young and enthusiastic local crew are full of verve and energy, contemplating the theme of the conference amongst other things like workshop topics.

Expressions of interest for submissions of abstracts will be posted via State newsletters with final dates to be confirmed. Early-bird dates for registrations will probably commence August or September this year, giving all states and their members time to flag their intention to attend with their lab managers.

Alex Szabo – HGSA, SA Pathology

Contents

Dr Koszyca treated the Histology Group with a fascinating and entertaining talk on Dementia at the Royal Adelaide Hospital last October.

Dementia is a complex process involving interplay between specific molecular pathways affecting cellular functions, leading to loss of synaptic connections, cell death, gliosis, inflammation and disruption of functional networks. This affects personality behaviour, and sensorimotor functions and eventually attacks an individual's autonomy. It was originally described as a collection of symptoms that are caused by disorders affecting the brain and not related to one specific disease.

There are two classifications:

1. Vascular - this can be a result of
   - small vessel disease
   - large vessel disease
   - hypoperfusion lesions
   - rare local vascular disorders

2. Non-vascular – caused by abnormal protein aggregates in neurons and / or glia as well as in the extracellular component.
   - amyloid
   - tau
   - α synuclein
   - PrP
   - TDP 43
   - FUS

Amyloid

-β A4 Alzheimer's disease
-A Bri British familial dementia
-PrP Creutzfeldt-Jakob disease

Tau
- Alzheimer's disease
- Progressive supranuclear palsy
- Corticobasal degeneration
- Argyrophil grain disease
- Pick's disease
- Tangle predominant dementia
- Guam parkinsonian-dementia complex
- Chronic traumatic encephalopathy

Synuclein
-Parkinson's disease
-Dementia with Lewy bodies
-Multiple System Atrophy

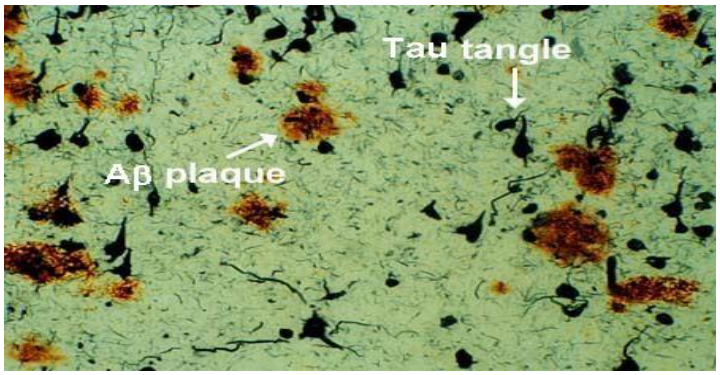


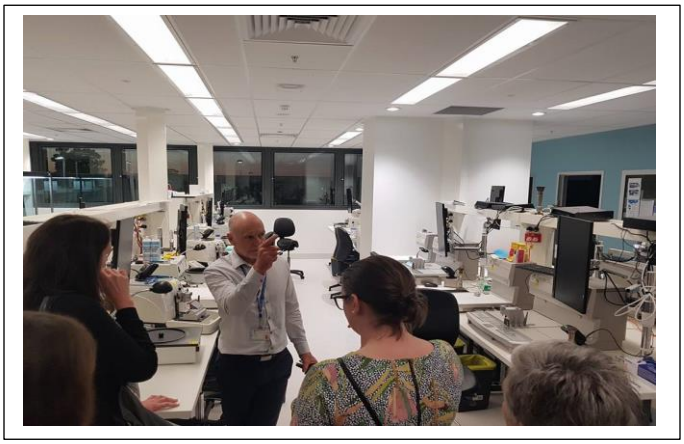
Figure 1. Postmortem tissue sample from an AD patient brain reveals AD pathology including amyloid-beta plaques and Tau tangles. (Photo credit Dr. Dale Bredesen).

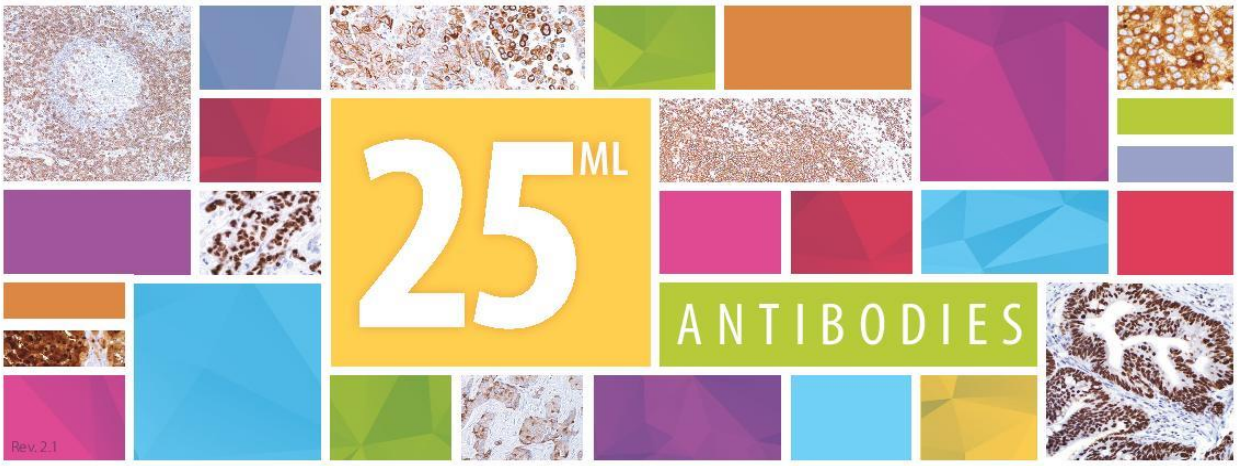
Alzheimer's Disease
Familial AD has 3 causative genes.
-APP chr21 (10%familial AD)
-PS1 chr14 (50% of early onset familial AD)
-PS2 chr1 late onset FAD
Epidemiologic studies have shown an increased risk of AD with severe head injuries. The mechanisms are not clear. Not all studies have shown an increased AD risk in adults sustaining a severe head injury.

Chronic Traumatic Encephalopathy
90% of cases have been in athletes with one third symptomatic at time of retirement from sport and one half within 4 years of retirement. Gridiron players have a shorter duration of symptoms (3-10 years) compared with boxers (5-46 years). Those diagnosed had mood disorders and all died in middle age.

Rod Coombe – HGSA, SA Pathology RAH

Tour of the new Royal Adelaide Hospital Histology Laboratory and Mortuary, October 2017





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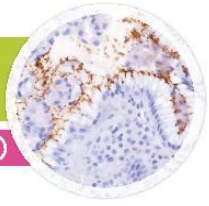
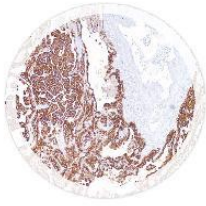
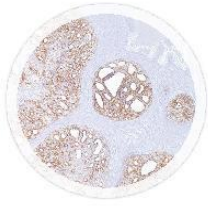


Helicobacter pylori
(polyclonal)

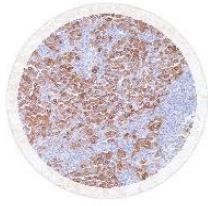
Cytokeratin Cocktail
(AE1 & AE3)



Cytokeratin 7
(OV-TL 12/30)



P504s
(13H4)



MART-1 (Melan A)
(M2-7C10)

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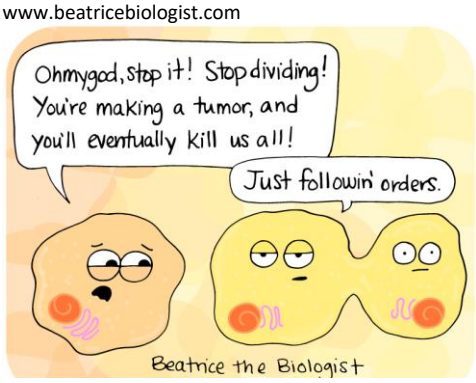

"Wherever the art of medicine is loved, there is also a love of humanity"

-Hippocrates-

Cryptic Corner

GP's are not producing sepsis: it is confused with State

Answer page 21

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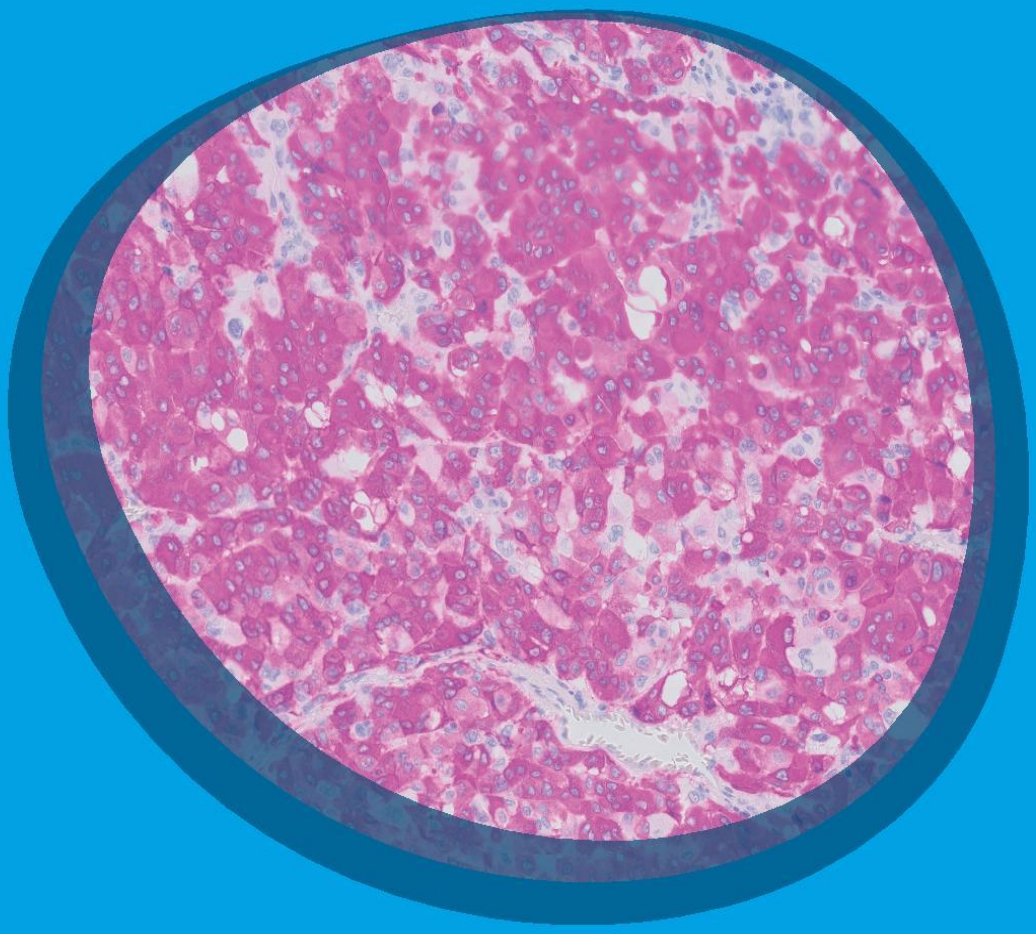

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Dr. Nicole Sladden made a wonderful lecture on direct immunofluorescence (DIF) for diagnosing auto-immune skin diseases. The event was hosted by Clinpath (Kent Town).

The DIF along with other routine histological techniques are employed to clarify dermatological diseases such as blistering diseases, lupus erythematous and vasculitis. The types of antigens, sites of deposition and strength of deposition are hints to distinguish diseases from quite similar clinical appearance thereafter determines the personal treatment protocols.

Applications on blistering diseases are good examples: according to the various antigens (*figure 4*) been attacked, pemphigoid vulgaris, bullous pemphigoid, mucous membranes pemphigoid and epidermolysis bullosa acquisita are clearly classified. These targeted antigens are distributed from epithelial to basal membrane. Systemic Lupus Erythematous (SLE) (*figure 3*) has wide spread multiple auto-antibodies targeting nuclear or/and cytoplasmic proteins. Linear IgA (*figure 2*) disease features with deposition of IgA at epidermal side, close to lamina densa however, it has heterogenous group of antigens. Dermatitis herpetiformis (DH) needs to distinguish from Linear IgA disease as DH shows patchy granular IgA on basement membrane.

Dr Sladden's emphasis on the bullous pemphigoid, pemphigus vulgaris, Linear IgA disease, lupus erythematous and Henoch-Schönlein purpura. The variants of clinical appearance, the features of HE and DIF morphology and the pathogenesis are discussed in detail. Much appreciation is given to Dr Sladden who made such an educative lesson.

Shuming Tang – Clinpath Laboratories

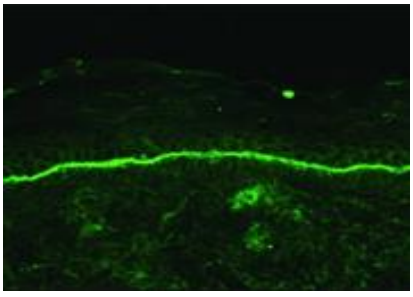


Figure 2. Linear deposition of IgA at the dermo-epidermal junction.

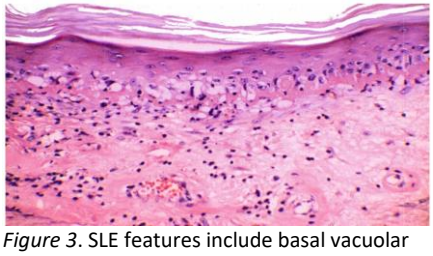


Figure 3. SLE features include basal vacuolar damage, perivascular lymphocytic infiltrate, basement membrane thickening and fibrinoid material around vessels, collagen and interstitium.

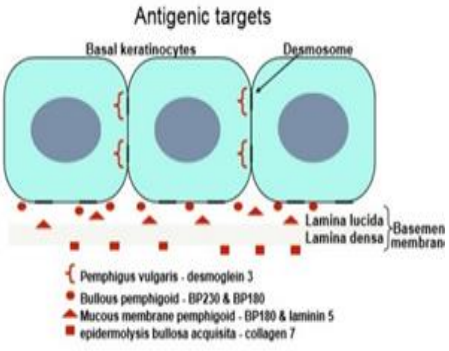
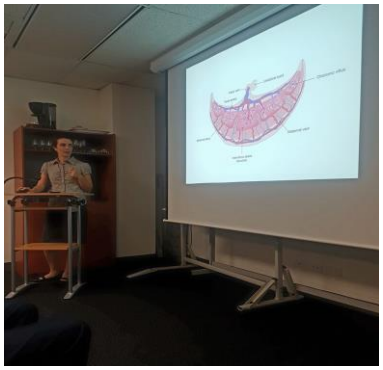


Figure 4. Antigen targets.

Dr. Rebecca Morrow (Clinpath) has extensive placenta pathology experience and presented us with some grossing essentials. With 6.5 million stillbirths and neonatal deaths worldwide, 11-65% of the answers are found in the placenta. Placentas are examined due to maternal, obstetric or paediatric difficulties and abnormalities throughout the pregnancy or at the time of delivery. Some of these indications for histological examination include (but are not limited too); autoimmune disease, uncontrolled diabetes, physical abnormalities of the placenta (e.g. colour, size, lesions), preterm birth (<37 weeks), pre-eclampsia, infections during pregnancy (e.g. rubella, toxoplasmosis), Amniotic Fluid Index (AFI) abnormalities, antepartum/intrapartum haemorrhage and any other maternal diseases affecting pregnancy. It is recommended that placentas are received fresh (i.e. not fixed in formalin) due to possible microbiological, cytogenetics and metabolic studies that may be required.



In 2014, a group of 26 pathologists standardised the sampling criteria, placental gross descriptions, pathological terminology and diagnostic criteria.

The current macroscopic grossing template, for both single and multiple gestation placentas, is from the Royal College of Pathologists Australasia (RCPA) 'Macroscopic Cut-up Manual' (*figure 5*).

Placenta –single gestation — Macroscopic reporting dictation template

	Data element	Response
	Fresh tissue received	No / Yes / *If yes, describe any additional tests/ microbiology/cytogenetics performed*
	Membrane description	
	Membrane completeness	Complete with single point of rupture / Closest distance to edge of placenta __mm
		Incomplete (stripped or ragged)
	Membrane appearance	Opacity / Colour / Texture
	Membrane insertion	Marginal / Circummarginate / Circumvallate / ____% of circumference involved
	Other findings in membranes	Plaques / Nodules / Any vessels
	Umbilical cord description	
	Umbilical cord dimensions	Length__ mm / Diameter , minimum__ mm and maximum __ mm
	Number of umbilical cord vessels at both ends	__
	Umbilical cord insertion point	Eccentric / Central / Marginal / Velamentous / Distance from placental edge __mm
	If velamentous	Maximum length of vessel in membrane __ mm / Describe _____
	Umbilical cord colour	Normal / Abnormal / Describe colour and area involved_____
	Umbilical cord coiling index	Count of coils__ per total length of cord __mm / Localised areas of abnormal coiling.
	Umbilical cord other abnormalities	No / Yes / *Knot, stricture, thrombosis, haematoma or oedema*
		Describe appearance _____ / Size ___mm / Location _____
		If cord is tethered to fetal surface, length of tethering __mm
	Placental disc description	
	Placental disc shape	Oval / Round / Irregular / Bilobed / Accessory lobe __x__x__mm / Fragmented
	Iatrogenic procedures	No / Yes / Describe _____
	Fetal surface	Normal / Abnormal, describe focal lesions inc. abnormalities of chorionic vessels _____
		Overall involvement of placental disc__ %
	Maternal surface	Complete / Incomplete / Ragged and unable to be assessed
	Blood clot	No / Yes / Size ___% involvement / Weight __g
		Location / Central 2/3 / Peripheral 1/3
	Placental disc trimmed weight	__g / *Placental weight trimmed of cord and membranes*
	Placental disc dimensions	__x__x__mm
	Parenchyma	Normal / Abnormal, *describe focal lesions*
		Number / Appearance _____
		Location / Central 2/3 / Peripheral 1/3
		Overall involvement of placental disc__ % of volume
	Block identification key	Text / *Describe nature and site of blocks*

Placenta single dictation template — Date: 22 October 2015 — Version: 1.1

Figure 5. RCPA 'Single placenta dictation template' https://www.rcpa.edu.au/Library/Practising-Pathology/Macroscopic-Cut-Up/Specimen/Gynaecology-and-perinatal/Placenta/Placenta-single

The placenta consists of the fetal side (chorionic villi) with the umbilical cord and the maternal side (decidua basalis) (*figure 6*). A macroscopically normal placental membrane should be shiny, smooth and translucent. A yellow-grey colour may indicate chorioamnionitis (caused by several bacterial, fungal and parasitic organisms) and green-brown may indicate meconium.

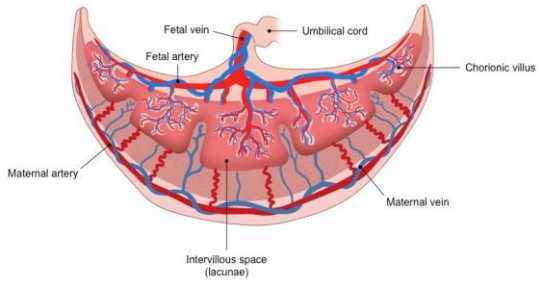


Figure 6. Placenta anatomy

Description of membrane insertion is important, in particular if it is circumvallate (*figure 7*). Where marginal is normal, in a circumvallate situation the amnion and chorion fetal membranes 'double-back' around the edge of the placenta. There can be associated tethering and risks include placental abruption, haemorrhage and fibrin deposition. Circummarginate is similar to circumvallate without the thickened, rolled up edges.

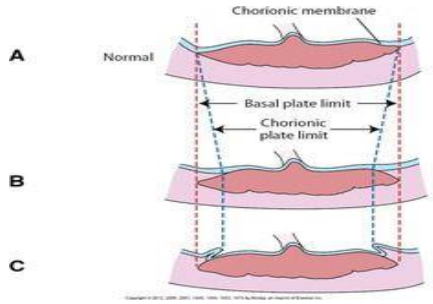


Figure 7. Normal membrane insertion (A), circummarginate (B) and circumvallate (C).

The umbilical cord has one vein and two arteries (i.e. there should be three vessels at 'both' ends). One less artery could indicate congenital heart and kidney problems. Cord insertion includes marginal (<1cm from the edge), peripheral (<3cm from the edge) and velamentous (where the cord inserts into the membranes). These vessels are exposed and not protected by the umbilical cord's gelatinous substance 'Wharton's Jelly). This increases risk of rupture and haemorrhage. It is important to count the coils of the umbilical cord per 10cm. A hypo-coiled cord has <1 coil/10cm and a hyper-coiled cord has >3 coils/10cm. Abnormal cord coiling can cause thrombosis and stenosis. In hyper-coiled cords, blood flow can decrease thus affecting fetal growth. Knots in the cord can also prevent passage of blood flow.

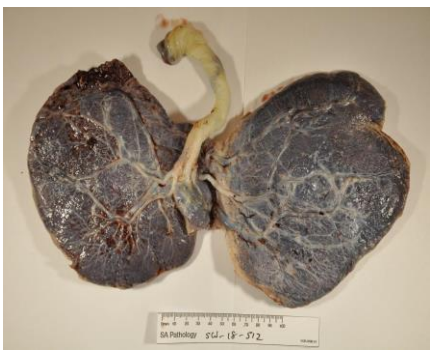


Figure 8. Bi-lobate placenta (fetal side)

Abnormal shapes have been associated with a decrease in placental efficiency (e.g. reduced vascular supply). The typical placental shape is round or oval, as they generally grow uniformly out from the umbilical cord insertion, thus having a centrally inserted cord. Placentas can be bi- or multi-lobate or otherwise irregular (*figure 8*). The succenturiate placenta in particular, has one or multiple accessory lobes connected to the main part of the placenta by blood vessels. These accessory lobes vary greatly in size unlike a bi-lobe placenta where both segments are almost equal in size. Succenturiate implications include placenta retention and post-partum haemorrhage.

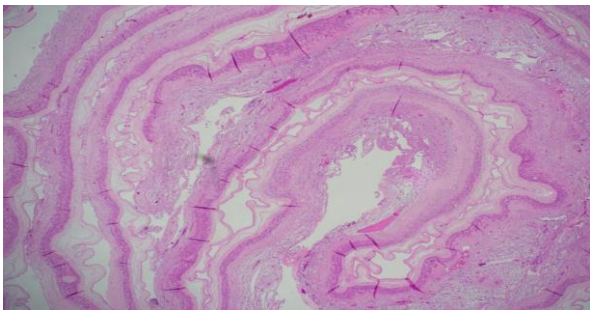


Figure 9. Membrane roll H&E stain

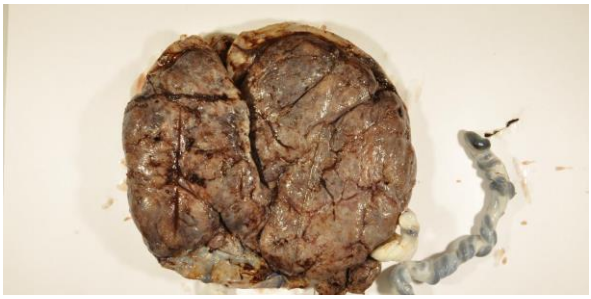


Figure 10. Maternal surface / parenchyma

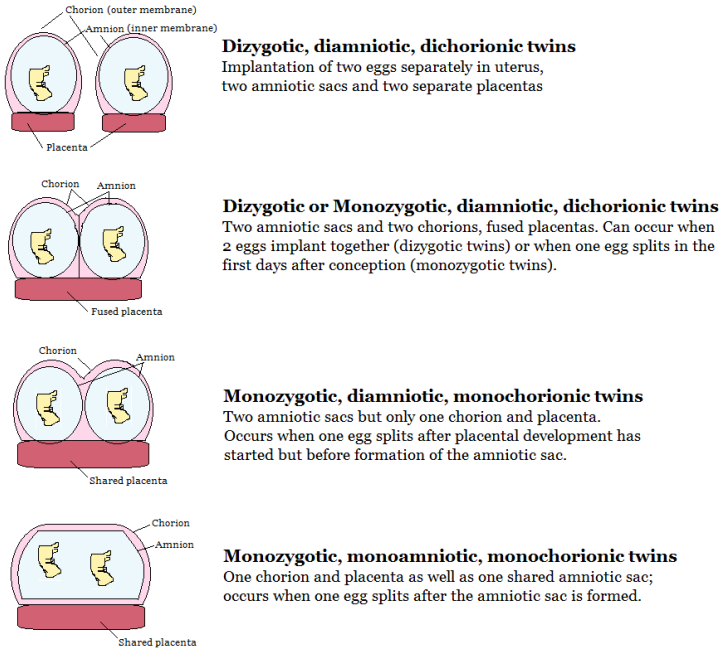


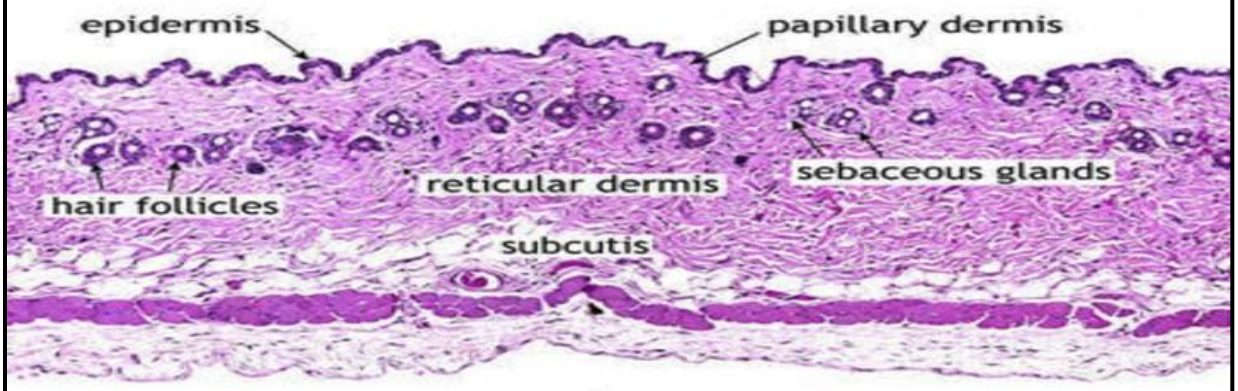
Figure 11. Twin placenta types

Placental shape can be influenced by where they implant within the uterus. Other implantation complications include placenta accreta where the placenta attaches too deeply into the uterine wall; placenta increta where attachment is deeper, penetrating the muscle and placenta percreta where the placenta penetrates through the uterine serosa and can adhere to other organs such as the bladder. These conditions may require surgery to remove the placenta and the mother is at risk of severe haemhorrage. Placental previa involves the placenta overlying the cervical os thus covering the birth canal and placental abruption is a condition where the placenta separates from the uterus early (i.e. prior to delivery) and can cause haemhorrage and fetal death (due to lack of oxygen and nutrients).

The average weight of a placenta is 537g (normal range = 442-632g). This is the trimmed weight i.e. minus the cord and membranes. Decreased placental weight can indicate chronic hypertension or pre-eclampsia and increased placental weight can indicate anaemia or gestational diabetes.

Five blocks should be sufficient with a macroscopically normal placenta. This includes 2 sections of the cord (one from each end) in one block, a membrane roll (*figure 9*) and three full thickness sections of parenchyma (from maternal to fetal end). The maternal surface/parenchyma should have a uniform, wrinkled, dark red-brown, vascular cut surface (*figure 10*). Any paleness, lesions, infarcts and blood clots should be described and sampled. For twin placentas, only one extra section is required of the dividing membrane to be able to distinguish the type of twins (*figure 11*).

Karen Bampton – HGSA, Clinpath Laboratories



"Skin: common specimens, problems and how to approach" - *Dr. Harry Kasmeridis, SA Pathology*

HGSA
HISTOLOGY GROUP OF SOUTH AUSTRALIA

Educational Session

Monday 25th June 6:30pm
6pm Light Refreshments
Room 2F 492-494
Level 2 Meeting Rooms
Royal Adelaide Hospital
1 Port Road, Adelaide

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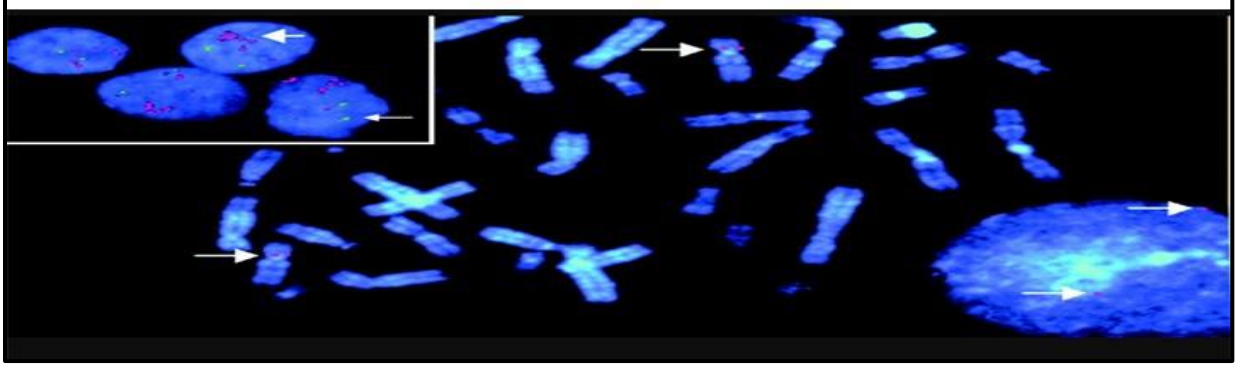
"FISHing in Anatomical Pathology" – *Sarah Moore, SA Pathology*



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Upcoming Events

Check out our website www.histosa.org.au and Facebook page https://www.facebook.com/HistoSA/info for further details of the following events.

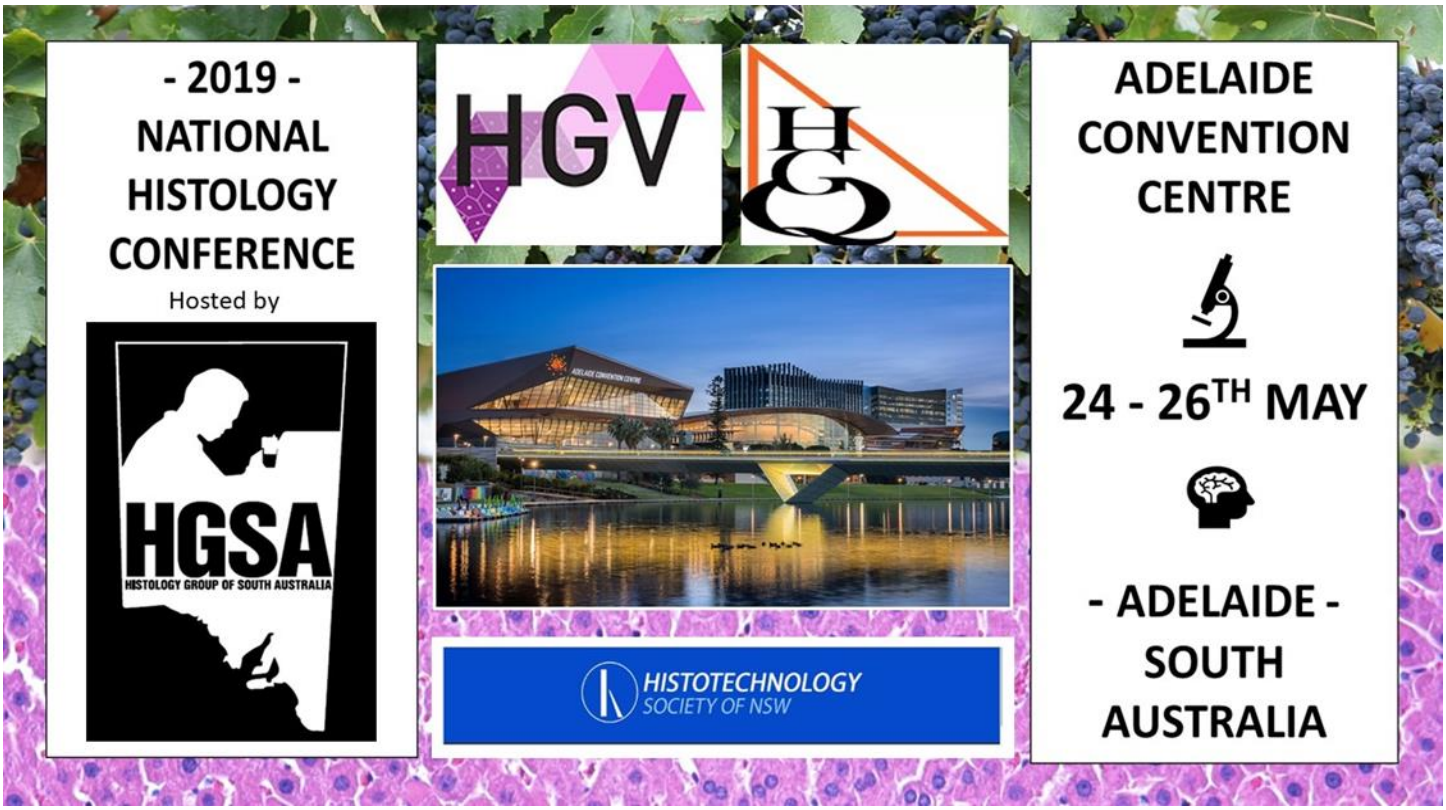
Don't worry, you will also get sent an email closer to each event, so make sure you are on our mailing list!

Educational Session #2	**Monday 25th June: RAH – Adelaide**
The Cutting Edge Newsletter #3	**Monday 6th August**
Educational Session #3	**Monday 20th August: Clinpath – Kent Town**
The Cutting Edge Newsletter #4	**Monday 1st October**
Educational Session #4	**Monday 24th October: RAH – Adelaide**
HGSA Christmas Dinner	**Monday 3rd December**

The Histology Group of South Australia will host the next National Histology Conference here in Adelaide in 2019. We will be working with the Histology Group of Victoria, Histotechnology Group of Queensland and the Histotechnology Society of New South Wales to provide a range of workshops and plenary sessions, aimed to provide continuing education and professional development to those within the medical science, clinical and research fields. Modern equipment and consumables will also be showcased by trade sponsors.

Like and follow the National Histology Conference – Australia facebook page https://www.facebook.com/National-Histology-Conference-Australia-179877572580038/

Watch this space for more details…..



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A/Prof Lisa Butler's research program at the University of Adelaide and SAHMRI investigates new therapies and diagnostic tests for prostate cancer. Most research relies on cell line or animal models of prostate cancer, which do not always accurately reflect how the cancer behaves in the human body. This is a major reason for the failure of many developmental drugs in clinical trials.

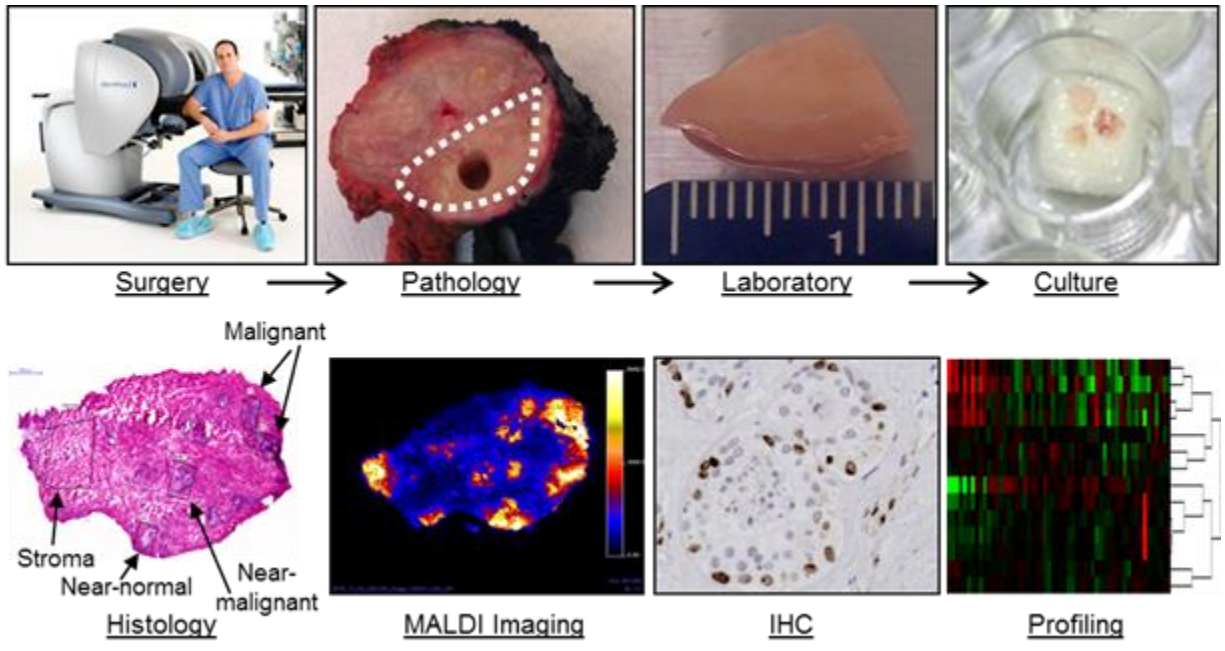
A/Prof Butler's group has developed a unique model where tissues collected from consenting patients undergoing prostate cancer surgery are cultured in the laboratory in a way that retains their 3D structure. She then assesses changes in prostate cancer cell growth and death as well as changes in genes, proteins, lipids, and cellular pathways that occur when the tissues are exposed to drug treatments.

This approach can uncover important information about how prostate cancers behave and greatly increases the likelihood that the findings will quickly translate to clinical practice. Understanding the pathology of each tissue sample is essential to successfully working with human prostate samples in this way.

Interaction between researchers and pathologists provides a detailed analysis of the different morphologies within each tissue sample and helps to relate our research findings to the aggressiveness of each cancer. We are working towards developing accurate and non-invasive diagnostic tests to monitor the aggressiveness of a patient's cancer and how it responds to treatments, as well as analysing efficacy and mechanism of action of potential new drugs.

Clinpath – Kent Town Histology lab are currently assisting this exciting research program. Histology scientists sample the tissue from core biopsy positive sites of the fresh prostates that are brought into the lab. These samples are then taken away by Kayla Bremert, from the University of Adelaide's School of Medicine, for their team to work their magic. The prostates are then processed routinely for histology at Clinpath and the University of Adelaide and SAHMRI research team utilise their samples for various tests.

Watch this space for updates on the team's research!



Legend: Prostate tissues are collected from surgery and assessed for pathology then a core is taken to the laboratory for culture on gelatine sponges. Some of each tissue is used for H&E stain to visualise tissue pathology. Other tissue pieces are used for experiments such as MALDI Mass Spectrometry Imaging, immunohistochemistry or RNA, protein or lipid profiling.

HURST SCIENTIFIC

New -Microtome Blades-

Histo Cutter Plasma Microtome Blades

We are pleased to launch our new Microtome Blades.

Histo Cutter Plasma, with Plasma technologies that improve sharpness and durability of the blades considerably.

Code #	Description	model	Quantity	Price
LH35	**PLASMA** (LOW PROFILE)	LH35	50 pcs/Pack 10 Packs/Box	POA
LS35		LS35		
HS35	**PLASMA** (High PROFILE)	HS35		

LH35: Suitable for Hard specimens

LS35: Suitable for Soft specimens

HS35: Suitable for Cryo Sectioning

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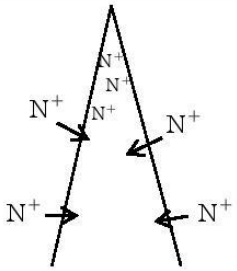
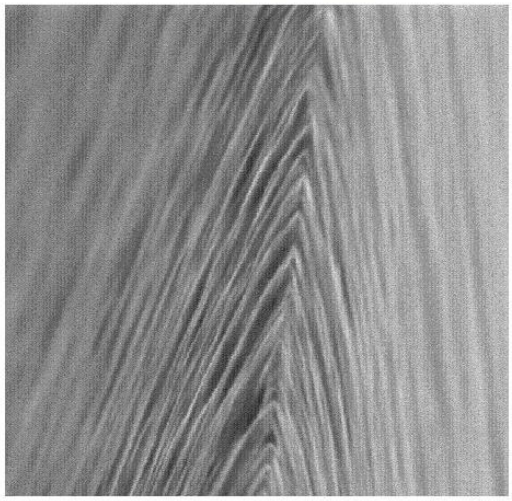
Plasma technology

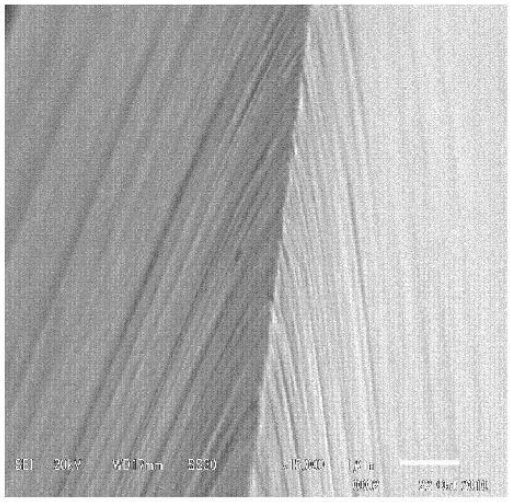


- In the plasma state let N^+ ion get into the blade edge
- The blade edge gets hardened by 1100 Hv(Vikers)
- Thus increased sharpness and durability more than conventional blades

After Plasma Process



Conventional Process



How to set Plasma blades onto microtomes

"Triangles" etched onto blade surface indicate optimal cutting side.
Place blade into holder with triangles facing the user.

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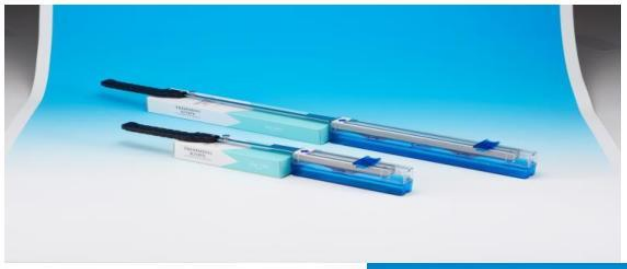
New -Dispenser Type- Trimming Blades

Code #	Description	model	Quantity	Price
F130	Trimming Knife 130mm in Dispenser	#130	Knives 50 pcs/box	POA
F130H	Handle for Trimming Knife 130mm	#130H		
F260	Trimming Knife 260mm in Dispenser	#260	Handles 1 pc/box	
F260H	Handle for Trimming Knife 260mm	#260H		



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Competition: Word Search

R	B	P	N	A	P	P	L	S	T	V	E	M	F	D	E	C	D	M	I
P	I	T	E	L	Y	A	R	M	S	S	R	U	N	L	F	N	D	D	Q
X	I	Y	J	K	N	Z	V	T	I	S	J	S	E	R	F	R	Z	V	A
U	E	X	I	I	M	I	X	S	G	G	H	C	U	M	A	E	V	G	X
I	E	X	P	X	Q	K	O	Y	O	W	T	L	R	V	F	U	U	R	Z
T	G	S	Q	F	B	R	Q	R	L	R	X	E	O	T	P	T	J	U	K
E	V	D	V	Q	E	O	Y	U	O	W	E	S	D	N	A	U	O	X	Z
R	V	O	G	L	H	E	G	M	R	R	T	N	E	D	T	I	L	Z	G
S	V	I	C	D	P	U	Y	K	U	B	R	E	G	F	V	W	Q	J	Z
D	E	S	S	D	M	O	H	A	E	K	O	N	E	U	R	O	N	E	S
Q	Z	V	W	S	G	R	T	F	N	K	C	K	N	U	B	P	S	I	F
E	J	S	R	R	E	R	D	C	N	P	X	Y	E	B	H	H	S	A	Q
M	P	I	A	E	O	R	C	C	I	H	P	O	R	T	O	Y	M	A	F
R	N	P	K	P	N	N	G	E	D	D	E	S	A	E	S	I	D	R	Q
O	H	B	H	Q	X	L	F	O	R	R	H	A	T	S	Y	L	A	Q	L
Y	I	Y	H	I	M	G	X	V	R	E	F	P	I	G	O	R	M	Z	I
U	E	D	M	V	X	U	J	A	A	P	B	T	V	B	M	F	Q	H	I
F	C	R	C	O	J	C	I	R	Z	N	L	R	E	F	U	X	A	Q	I
I	B	Z	E	M	R	A	V	N	X	O	R	T	A	Z	A	H	C	R	G
Y	H	W	Q	I	V	M	J	P	T	R	C	R	Y	L	E	S	S	M	X

Which of the 15 words from the list below is **missing** from the word search?

AMYOTROPHIC	ELECTROMYOGRAPHY	NEURONS
ATROPHY	MUSCLES	PALSY
CEREBRAL	NERVES	SCLEROSIS
CORTEX	NEURODEGENERATIVE	SPINAL
DISEASE	NEUROLOGIST	PROGRESSIVE

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Cryptic Corner Answer

SPECIALISTS

Did you get it right…??!!!

Congratulations to newly married HGSA Committee member **Rebecca Dyer**!

Congratulations to **Deb Dyer** (Clinpath, Kent Town) on winning the April 'Word Search' competition!

Are you looking for a good histotech handbook to improve your quality of work in the lab? Try "Histology Hacks" written by North Carolina's LabCorp histotechnician Mike Backhus. There are over 75 demonstrated histo hacks for embedding, microtomy, frozen sections and grossing. Have you ever used dryer sheets, soap or alfoil? No...well for more secrets and tips check it out https://www.facebook.com/michaelpathology/
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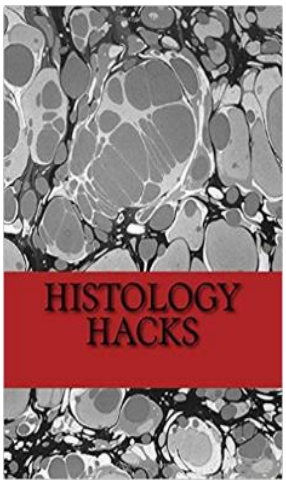

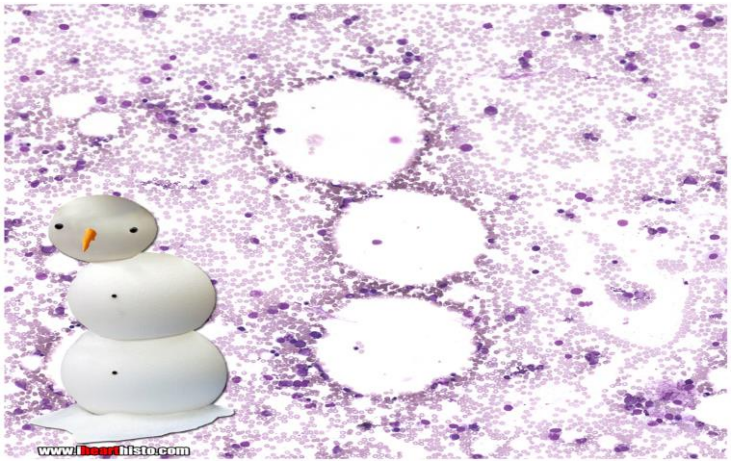

Stay warm this winter!!

A smear made from the red marrow extracted from the iliac crest of the donor's pelvis prior to transplantation.https:// http://www.ihearthisto.com/post/135581514897/