Faculty of Medicine

School of Medical Sciences

PATH 3205

Molecular Basis of Inflammation and Infection
MBII
(6 UOC)

SEMESTER I, 2014

Molecular Basis of Inflammation and Infection
Course Manual

2014

Preface

This is the eleventh, revised edition of the manual for Molecular Basis of Inflammation and Infection (previously Molecular Basis of Disease A). This manual contains a large amount of relevant information regarding the course PATH3205 Molecular Basis of Inflammation and Infection. We recognise that the manual may contain some errors. We therefore seek the co-operation of staff and students in pinpointing errors of content or style, so that these may be corrected in subsequent editions.

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Introduction

Welcome back to the Department of Pathology!

PATH3205 Molecular Basis of Inflammation and Infection builds on fundamental principles of human disease taught in PATH2201 Processes in Disease. This is achieved by focusing on the underlying molecular basis or 'molecular mechanisms' of the disease process in humans. The course is suitable for all those who have developed a general grounding in the field of biological sciences and wish to apply this knowledge to human health. For those wishing to pursue a career in research or hospital based laboratory work, it is hoped that the course will not only develop their basic knowledge of molecular processes, but also provide a framework for understanding how these processes link to the modern practice of medicine. Similarly, for those who may wish to pursue a career in the health sciences, the course will provide an understanding of the cellular and molecular processes underlying the clinical manifestations of disease. Furthermore, development of ePortfolios will assist students in understanding career pathways in medical research.

Core topics in Pathology are presented as themed '*Modules*'. The '*Research Lecture and Laboratory Series'* will illustrate 'state-of-the-art' research techniques that address molecular mechanisms of disease, primarily in the context of Inflammation and Infection. Students will have opportunities for interactive learning and engagement in practical and research laboratory settings and upon completing the course should have a better understanding of molecular mechanisms that underlie clinical manifestations of human disease, as well as potential research topics in the areas of Inflammation and Infection.

This course complements PATH3206 *Cancer Pathology*, ANAT3212, BABS3041, PATH3207 *Musculoskeletal Diseases* and PATH3208 *Cancer Sciences: Research Design, Measurement and Evaluation* which also run in the same academic year.

At Level 3, courses are offered for those wishing to undertake the new Major in Pathology, which allows for 'research pathways' in:
Inflammation and Infection (PATH3205, PATH3206, PATH3207, ANAT3212 and/or BABS3041)
and/or
Cancer (PATH3205, PATH3206, PATH3208, ANAT3212 and/or BABS3041)

Level 3

Semester 1

- PATH3205 Molecular Basis of Inflammation and Infection
- PATH3206 Cancer Pathology
- BABS3041 Immunology 1

Semester 2

6 UOC from

- PATH3207 Musculoskeletal Diseases
- PATH3208 Cancer Sciences
- ANAT3212 Research Methods in Microscopy

The Department of Pathology and staff of the Inflammation and Infection Research Centre join me in wishing you and interesting and successful year.

Dr Patsie Polly

Senior Lecturer in Pathology – PATH3205 Convenor

General administrative points

Staff members in theDepartment of Pathology, School of Medical Scienceswith whom you may have contact include the following:

Dr Patsie Polly, Senior Lecturer, Course Convenor for PATH3205, SOMS3001 and Co-Convenor for PATH2201/2202

Dr Cristan Herbert, Co-Convenor for PATH3205;

Dr Christine van Vliet, Lecturer, Course Convenor for PATH3206;

Associate Professor NicodemusTedla, Course Convenor for PATH3207;

Professor Nicholas Hawkins,Head, School of Medical Sciences;

Professor Rakesh K Kumar;

Professor Denis Wakefield, Associate Dean (Strategy & External Relations);

Professor Andrew Lloyd, Head IIRC;

Associate Professor Gary Velan, Head, Department of Pathology;

Associate Professor Nick Di Girolamo;

Dr Fabio Luciani, Senior Lecturer;

Dr Simone Van Es, Lecturer;

Dr Shane Thomas, Senior Lecturer;

DrThuan Thai, Associate Lecturer

Administrative and general problems related to your attendance, or the content and conduct of the course, can in the first instance be addressed by consulting Dr Patsie Polly (patsie.polly@unsw.edu.au) by e-mail. Students wishing to see other members of staff should make an appointment via e-mail. If students have difficulties of a personal nature, or with the course, they should contact the School's Grievance Officer, Dr P Pandey (p.pandey@unsw.edu.au) or Prof N Hawkins, the Head of School.

Should you feel that there are particular circumstances that have affected your performance in the course; you should lodge an application for special consideration via https://my.unsw.edu.au/student/atoz/SpecialConsideration.html.It is intended that supplementary exams for the School of Medical Sciences in Semester 1, 2014 will be held 16th, 17th and 18th July, 2014. Special considerations sought outside the 3 day time period WILL NOT be accepted except in TRULY exceptional circumstances.To have a result reviewed (checking of mark and/or reassessment):
https://my.unsw.edu.au/student/academiclife/assessment/Results.html
To appeal academic standing or ability to progress:
https://my.unsw.edu.au/student/academiclife/assessment/finalisation_results.html
Guidelines on extra-curricular activities affecting attendance:
http://medicalsciences.med.unsw.edu.au/sites/soms.cms.med.unsw.edu.au/files/Extra-curricularActivitiesSOMS.pdf

Information on the different research units in the Department of Pathology and the research interests of each staff member is available at Department of Pathology's home page at

http://medicalsciences.med.unsw.edu.au/staff/departments/pathology

Recommended text

You are expected to acquire the following text:

Robbins. Basic Pathology, 9th Ed. V. Kumar, RA.K. Abbas & J. Aster (2012). Saunders & Co. Philadelphia PA; Elsevier Saunders.

Reference

Students wishing to study the molecular biology or clinical features of diseases in greater depth might consider the purchase of the following text:

Robbins and Cotran Pathologic Basis of Disease. 8th edition. Eds. V. Kumar, A.K. Abbas, N. Fausto and J. Aster. (2009) Elsevier Saunders.

Official communication by email

All students in course PATH3205 are advised that email is the official means with appropriate etiquette by which the School of Medical Sciences at UNSW will communicate with you. All email messages will be sent to your official UNSW email address (e.g.,z1234567@student.unsw.edu.au) and, if you do not wish to use the University email system, you MUST arrange for your official mail to be forwarded to your chosen address. The University recommends that you check your mail at least every other day. Facilities for checking email are available in the School of Medical Sciences and in the University library. Further information and assistance is available from DIS-Connect, Tel 9385 1777. The UNSW Library runs free email courses.

Plagiarism

The Department of Pathology will not tolerate plagiarism in submitted written work. The University regards this as academic misconduct and imposes severe penalties. Evidence of plagiarism in submitted assignments, etc. will be thoroughly investigated and may be penalised by the award of a score of zero for the assessable work. Flagrant plagiarism will be directly referred to the Division of the Registrar for disciplinary action under UNSW rules.

https://student.unsw.edu.au/conduct

Your attention is drawn to the following extract from the above website:

"The basic principles are that you should not attempt to pass off the work of another person as your own, and it should be possible for a reader to check the information and ideas that you have used by going to the original source material. Acknowledgment should be sufficiently accurate to enable the source to be located speedily. If you are unsure whether, or how, to make acknowledgement consult your lecturer.

The following are some examples of breaches of these principles:

a) Quotation without the use of quotation marks. It is a serious breach of these rules to quote another's work without using quotation marks, even if one then refers to the quoted source. The fact that it is quoted must be acknowledged in your work.

b) Significant paraphrasing, e.g. several sentences, or one very important sentence, which in wording are very similar to the source. This applies even if the source is mentioned, unless there is also due acknowledgment of the fact that the source has been paraphrased.

c) Unacknowledged use of information or ideas, unless such information or ideas are commonplace.

d) Citing sources (e.g. texts) which you have not read, without acknowledging the 'secondary' source from which knowledge of them has been obtained.

These principles apply to both text and footnotes of sources. They also apply to sources such as teaching materials, and to any work by any student (including the student submitting the work) which has been or will be otherwise submitted for assessment. You must obtain the prior approval of your lecturer if you wish to submit to that lecturer an essay substantially similar to one which has already been, or will be, submitted to another lecturer.

Using the principles mentioned above about proper acknowledgment, you should also proceed on the general assumption that any work to be submitted for assessment should in fact be your own work. It ought not be the result of collaboration."

Staff contacts in the Department of Pathology

Name	*Title*	*Telephone*	*E-mail*
Dr Patsie Polly	Senior Lecturer, Department of Pathology Convenor	9385 2924	patsie.polly@unsw.edu.au
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Dr Christine van Vliet	Lecturer, Department of Pathology	9385 8434	C.vanVliet@unsw.edu.au

Topics for possible student research

Opportunities exist for all students wishing to undertake undergraduate and postgraduate research programmes within the School of Medical Sciences. Information can be accessed via the Faculty of Medicine directory for the School of Medical Sciences at:

http://medicalsciences.med.unsw.edu.au/research

Administrative Matters

You may also meet the following members of the School support staff during the course of the year:

Ms Soo Han Chup

Position: Administrative Officer

Location: Administrative Wing, 5th Floor Wallace Wurth EAST Building

MsChup is responsible for the distribution of Pathology manuals and Images of Disease CD-ROMs to students, and will assist in arranging interviews with academic staff within the Department.

Ms Susan Dacre

Position: Executive Assistant to the Head of School

Location: Administrative Wing, 5th Floor Wallace Wurth EAST Building

Students wishing to make appointments with Prof Hawkins should do so by first contacting MsDacre.

Mrs Carmen Robinson/ Mr Ryan Ling

Position: Administration Officers/Student Advisors

Location: Room G27, Biosciences Building

Mrs Robinson and Mr Lingare responsible for general administration and student support within the School of Medical Sciences.

Mr Fergus Grieve

Position: Web & TELT LMS Administrator

Location: Administrative Wing, 5th Floor Wallace Wurth EAST Building

Mr Grieve is responsible for uploading resource to the PATH3205 Moodle Module.

Mr Derek Williamson

Position: Museum Manager

Location: Room G04 Ground Floor Samuels Building, Building F25

Mr Williamson provides support for all undergraduate teaching programs. He plays a major role in broadening the use of the Museum of Human Disease by supervising an integrated learning program for senior high school students and community interest groups. Mr Williamson co-ordinates a network of volunteers who assist with the supervision of visitors from outside the University. Contact Mr Williamson immediately if there are any broken or leaking specimens in the Museum.

Ms Julia Kiss

Position: Museum Education Officer

Location: Room G04 Ground Floor Samuels Building, Building F25

Ms Kiss provide support for all undergraduate teaching programs, and assist in delivering an integrated learning program for senior high school students and community interest groups.

The Museum of Human Disease

The Donald Wilhelm Museum of Human Disease is located on the ground floor of the Samuels Building. It was established by Professor Donald Wilhelm, the Foundation Professor of Pathology at this university. Thanks to his foresight, and to the tireless efforts of Dr G. Higgins (the Museum Curator of longstanding), the Museum has been meticulously maintained and updated over the years to reflect the changing patterns of disease in our society. The Museum contains over 2,700 specimens (or "pots"), which display diseased human tissue at the macroscopic level, usually preserved in formalin. Specimens are obtained both from organs removed surgically and from tissue obtained at autopsy, where the natural history of disease is in full view. **Please take note that some specimens of diseases which have become rare, e.g. diphtheria, are over 60 years old, and are irreplaceable.** Each specimen is numbered and is accompanied by a clinical history (when known), a macroscopic description of the abnormalities displayed, and a histopathological description of changes at the microscopic level (where relevant). That information, specific to each of thirty areas (or "bays"), can be found in the Museum catalogues located in a bracket within each bay.

All the specimens in the museum are arranged in one or other of two major groups. One group comprises collections of specimens according to pathological processes such as congenital, inflammation and healing, vascular, neoplasia etc. The second group comprises collections of specimens under organ systems, such as cardiovascular, central nervous, renal etc.

As responsible adults, we expect you to maintain decorum in the Museum, behave with care and respect for the integrity of the specimens, and help to keep the Museum tidy at all times. This means no eating or drinking in the Museum, and always returning specimens and catalogues to their allocated places. **Do not shake the specimens!** This activity conveys no useful information, but often damages the specimens. If you discover that a specimen is leaking or broken, follow the instructions listed in the safety notice below. **Remember that the Museum is a precious learning resource, of which you are encouraged to make full use**.

Security in the museum

It is a crime under the Human Tissue Act to steal or mistreat material preserved in the Museum or practical class laboratories. Anyone who contravenes the Act will be prosecuted.

In order to protect the collection of specimens, access to the Museum is restricted for students in the Department of Pathology during weekdays from 8 a.m. to approximately 5 p.m. The Museum is security locked, and can only be entered by using your student card to enable the doors to be opened. Mr Williamson, Ms Kiss and Mr Cutting play a supervisory role during office hours.

The Museum and practical class laboratories are under constant electronic surveillance.

Safety in the museum

- Always handle museum specimens with care and respect. All specimens consist of generously donated human tissue.
- Specimens are preserved in Perspex and contain a range of preserving chemicals that may be harmful. Chemicals used include **formalin, pyridine, sodium dithionate**. A full list of chemicals and associated MSDS information is available in the H&S Station and on the SoMS website.

Chemical	**Max. Percentage Composition**
Glycerol	17 (v/v)
Pyridine	0.8 (v/v)
Sodium Acetate	7 (w/v)
Formalin	<2 (v/v)
Sodium Dithionate	0.4 (w/v)

- For reasons of hygiene, never take food or drink into the museum.
- Never leave a museum specimen on the floor, or in any precarious position.
- If a specimen is leaking or broken, do not attempt to wipe up the spillage. Clear the area and immediately inform the Museum Manager or a member of academic staff. A spill kit will then be used to absorb the fumes. Remember that the museum is here for your benefit - your cooperation in maintaining neatness and safety at all times is appreciated.

Museum development

In recent years, the role of the Museum of Human Disease has been expanded to include the education of senior high school biology students and community interest groups, with an emphasis on the prevention of common diseases. Visitors are given a supervised two hour multimedia program, including a tour of the Museum, video presentation, microscopic examples of disease and an introduction to the Department's computer assisted learning facilities. Our Museum Manager (Mr Williamson), his Assistants and a full-time technical officer have helped to make these visits possible.

Periodically, special Museum exhibitions will be mounted for viewing by the general public. Recent examples include "Getting on My Nerves" and "Gutsy Stuff".

Health and Safety

UNSW aims to provide a safe, healthy and secure learning and working environment for all students, staff, contractors and visitors. To achieve this goal, everyone has a responsibility to take reasonable care of their own health and safety (H&S), for ensuring that their acts or omissions do not adversely affect the H&S of others and to comply with reasonable H&S instructions. The university has laid out these goals in its Health and Safety Policy**.** The policy applies to all students.

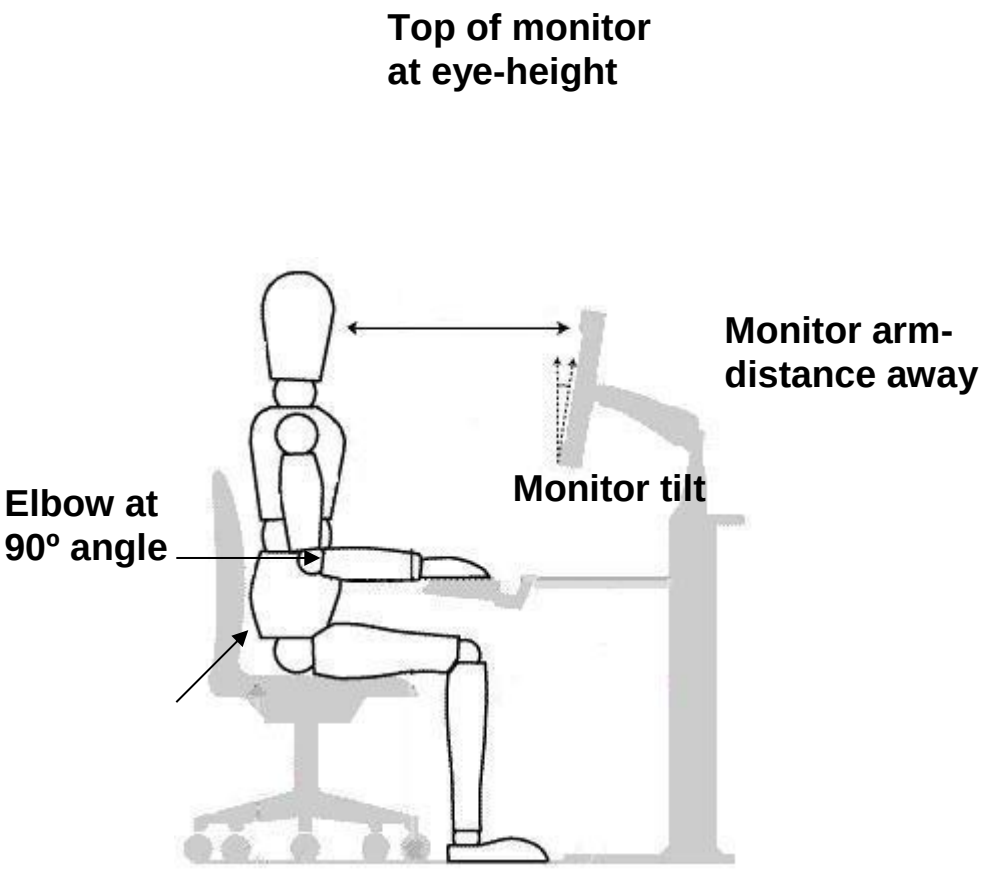
If you identify a hazard at the university, or have an injury or incident then this must be reported. To do this, log in to myUNSW, go to My Student Profile and click on the H_2O link. You must participate in all real and practice emergency situations by following the instructions of your lecturer or the local warden.At the start of the guide to the lecture series and the guide to the practical series there is a student risk assessment for each. Your lecturer will remind you of the hazards at the start of each lecture.

General H&S rules for undergraduate students can be found at the following URL: http://medicalsciences.med.unsw.edu.au/students/health-safety

Practical labs have special and varying H&S risks and rules; these are outlined in the risk assessments below and will be communicated to you by the lecturer before the practical.

Medicine Teaching Laboratory Student Risk Assessment	UNSW THE UNIVERSITY OF NEW SOUTH WALES	Pathology practicals in G6/G7 & G16/G17 in Wallace Wurth for PATH3205 MBII, 2014

Workstation set-up



Personal Protective Equipment Required

All pots contain real human tissue that has been generously donated to medical science and **must be treated with appropriate respect and dignity.**
Specimens are preserved in Perspex and contain a range of preserving chemicals that may be harmful. Chemicals used include **formalin, pyridine, sodium dithionate**. A full list of chemicals and associated MSDS information is available in the H&S Station and on the SoMS website.

MANUAL HANDLING OF POTS
It is recommended that all students wash their hands thoroughly as they leave practical class Chemical residues may be present on pots.
Carry one pot at a time. Use two hands at ALL TIMES and support the base of pot.
Avoid rough handling and/or tilting of pots. This can cause leaking joints or tear tissue in specimen.
Limit the number of pots on a table at any one time.

SPILLS AND LEAKAGES

If a specimen is leaking or broken, do not attempt to wipe up the spillage. Clear the area and immediately inform the Museum Manager or a member of academic staff. A spill kit will then be used to absorb the chemicals.

Emergency Procedures

In the event of an alarm, follow the instructions of the demonstrator. The initial sound is advising you to prepare for evacuation and during this time start packing up your things. The second sound gives instruction to leave. The Wallace Wurth assembly point is in the lawn in front of the Chancellery. In the event of an injury inform the demonstrator. First aiders and contact details are on display by the lifts. There is a first aid kit in the laboratory.

Clean up and waste disposal

Not necessary in these practicals.
No open-toe shoes allowed

Declaration

I have read and understand the safety requirements for this practical class and I will observe these requirements.

Signature:……Date:……
Student Number:……..

Date for review: 13/2/2015

Science Teaching Laboratory Student Risk Assessment	UNSW THE UNIVERSITY OF NEW SOUTH WALES	Pathology practicals in G6/G7 &G16/G17 in Wallace Wurth for PATH3205 MBII, 2014

Hazards	Risks	Controls
Physical Sharp plastic	'Stabbing' wound of hand	• Use disposable gloves
Biological Antibody	Inoculation/Irritant	• Do not eat, drink or smoke in the teaching laboratory • Use disposable gloves
Chemical Acrylamide Azide ...PBS	Corrosive/Flammable Irritant/neurotoxic Irritant Mild Irritant	• Low concentrations of chemicals used • Use disposable gloves

Pipetting ergonomics

Pipetting is another work aspect that can cause aches and pains. Here are some handy hints:

- Adjust your chair or stool so that your elbow is at a 90° angle while pipetting.
- Adjust the height and position of sample holders, solution container, and waste receptacle so that they are all approximately the same.
- Try to work with your hands below shoulder height.
- Let go of the pipette from time to time and give the fingers/hand a break
- Do not twist or rotate your wrist while pipetting
- Use minimal pressure while pipetting
- Try to switch periodically between different types of work.

For more information on preventing repetitive strain while pipetting click onhttp://www.anachem.co.uk/rsi

Personal Protective Equipment required			
	 optional		 optional

Emergency Procedures
In the event of an alarm sounding, stop the practical class and wait for confirmation to evacuate from demonstrators. Then wash your hands and pack up your bags. Follow the instructions of the demonstrators regarding exits and assembly points.

Clean up and waste disposal
• Remove your gloves and dispose in the biowaste bins provided. • Dispose of all pipette tips in the bin provided.

Ethics Approval
This type of practical does not require ethics approval.

Declaration
I have read and understand the safety requirements for this practical class and I will observe these requirements. Signature:……Date:……

Date for review: 13/2/2015

Computer assisted learning

Images of disease

Images of Disease (IOD) is a database of images used for teaching within the Department. The latest version of Images of Disease is now available online, optimised for smart phones and tablet computers, as well as Firefox 4+, Chrome 13+ and Safari browsers on laptop or desktop computers – http://iod.med.unsw.edu.au (zID and zPass required). An interactive Images of Disease app for iPhone and iPad is available to download from that website.

The following information might help you understand more about IOD.

What you get

- Over 3000 images relevant to your study as an undergraduate. Many of these images represent specimens from the Museum of Human Disease, or histopathological images from the student histopathology slide sets. Accompanying x-rays and images of surgical and autopsy specimens are also available.
- A database that links them all together
- A user interface that lets you access the images in a variety of ways
- Interactive "hotspotted" images to assist your understanding of macroscopic Pathology.

What you do not get

- A collection of images that you can send to your friends, put in your magazines, put on the Internet or whatever other scheme seems clever at the time.

  Many of the images used in this program are of a sensitive nature, and are intended for the purpose of private study by pathology students and graduates. You should exercise appropriate standards of professional ethics when using them.

- A high level of technical support

  Unfortunately, it will be impossible for us to answer all your problems immediately, as we have very limited resources. We will of course make every effort to help, and will provide you with a listing of known problems and difficulties on request.

Pathology on the World Wide Web

PATH3205 Moodle Module

Students enrolled in PATH3205 will be able to access the timetable, lecture notes and related information online via Moodle (login with zPass):

http://moodle.telt.unsw.edu.au/

This page provides links relating to the course such as:

- Timetables.
- Copies of lecture notes or slides (where available).
- Student self assessment questions.

It is expected that students will access this module regularly. Feedback on what additional material students would find useful on this site is encouraged.

Online progress assessments

Two progress assessments in PATH3205 will be conducted online. Further details of these assessments are provided in the Assessment section of this manual.

Links to these assessments will be provided from the Molecular Basis of Disease Moodle module at the appropriate times.

The Museum of Human Disease page contains links to some excellent undergraduate and postgraduate educational resources, of which we would encourage you to make full use.

The address is: **http://medicalsciences.med.unsw.edu.au/community/museum-human-disease/home**

Reference section

Glossary of terms used in Pathology

Abscess: a localised collection of pus in an organ or tissue

Acquired: a lesion occurring due to an event after birth (cf. congenital)

Acquired immunodeficiency syndrome (AIDS) :a disease caused by the human immunodeficiency virus (HIV), resulting in progressive depletion of T-cells necessary for cell-mediated immunity, leading to susceptibility to opportunistic infections and tumours

Adenoma: a benign neoplasm derived from glandular (secretory) epithelial cells

Acute respiratory distress syndrome (ARDS): respiratory failure caused by diffuse damage to type 1 pneumocytes and alveolar capillaries, often secondary to shock (hence the term "shock lung")

Aetiology: cause of a disease

Agenesis: congenital absence of an organ or structure

Allele: one of two alternative genes at a locus that controls a particular characteristic

Allergen: antigen which gives rise to allergic reactions, usually mediated by IgE antibody

Allograft: a tissue graft between two individuals of the same species (synonymous with homograft)

Amyloidosis: extracellular deposition of an insoluble protein complex, usually derived from serum proteins, with a fibrillar structure and a characteristic conformation (twisted beta-pleated sheet); deposits stain homogeneously pink with H & E, and brick-red with a Congo red stain

Anaemia: a significant reduction in the level of circulating haemoglobin below the normal range

Anaphylaxis: an acute hypersensitivity reaction, characterised by bronchospasm, peripheral vasodilatation, hypotension (shock) and oedema (especially laryngeal oedema)

Anaplasia: less than normal differentiation of cells; an important feature of malignant neoplasms

Anergy: the inability to react to a number of common skin test antigens; usually denotes depressed cell-mediated immunity (CMI)

Aneurysm: a localised abnormal dilatation of a vessel due to weakness of its wall

Anorexia: loss of appetite

Antibody: immunoglobulin specifically reactive with a particular antigen

Antigen: a substance which can induce a detectable immune response

Aplasia: congenital disturbance leading to failure of development of a part (synonymous with agenesis)

Apoptosis: a form of individual cell death, particularly observed in physiological turnover, in which the morphological changes consist of nuclear condensation and fragmentation (cf. necrosis)

Arteriosclerosis: refers to a group of processes in which there is thickening and loss of elasticity ("hardening") of arterial walls; it includes atherosclerosis, Monckeberg medial calcific sclerosis, and arteriolosclerosis

Arthralgia: pain (of any cause) in a joint or joints

Arthritis: inflammation of a joint or joints; usually signified by pain (arthralgia), erythema and swelling

Ascites: abnormal accumulation of fluid in the peritoneal cavity

Atelectasis: failure of normal degree of expansion of lung or segments of lung tissue

Atheroma: deposition of lipid in the intimal lining of systemic arteries accompanied by reactive changes in the vessel wall

Atherosclerosis: the commonest disease of arteries, characterised by focal or eccentric thickening of the intima by inflammatory and fibrotic lesions associated with the deposition of lipids; a circumscribed elevated lesion is referred to as an atheromatous plaque

Atrophy: diminution in size of an organ or tissue which had previously reached mature size, due to a decrease in size and/or number of its constituent specialised cells (cf. agenesis, aplasia and hypoplasia)

Autoimmunity: a disease caused by failure of normal immunological tolerance, such that the immune system identifies "self" antigens as foreign

Autolysis: post-mortem digestion of tissue by its own intracellular enzymes

Bacteraemia: the presence of bacteria in the blood (cf. pyaemia and septicaemia)

Benign: in reference to neoplasms, the term indicates strict localisation, growth by expansion, and frequent encapsulation (synonymous with innocent)

Biopsy: sampling of tissue for diagnosis, includes excisional, incisional and needle procedures, and also subsumes many cytological procedures

Boil: a small abscess of the skin, usually originating in a hair follicle or sweat gland (synonymous with furuncle)

Bronchiectasis: abnormal permanent dilatation of the bronchi, which may be localised or diffuse, congenital or acquired; associated with a chronic productive cough and recurrent pulmonary infections

Bulla: a large abnormal thin-walled cavity filled with liquid or gas

Cachexia: extreme wasting of the body, accompanied by weakness, anorexia and anaemia; most commonly seen in the terminal phase of malignancy

Calculus: a stone formed in a hollow tube or viscus, e.g. gallbladder, renal pelvis

Cancer: often used synonymously with carcinoma (see below); also a general term for all malignant neoplasms

Carbuncle: a multilocular abscess resulting from extension of a boil into the subcutaneous tissues

Carcinogen: an agent which can cause a cell to undergo neoplastic transformation, or which may initiate such a process by permanently altering cellular DNA

Carcinoma: a malignant neoplasm derived from epithelium

Carcinoma in situ: a malignant epithelial neoplasm which has not yet invaded through the basement membrane

Catarrh: inflammation of a mucosal surface associated with a mucoid exudate, e.g. nasal catarrh

Cell-mediated immunity (CMI): immune response in which T-cells and macrophages predominate

Cellular differentiation: process of development of phenotypic characteristics of a mature tissue by selective gene expression

Cellular swelling: a mild degenerative change of cells in which the affected tissues appear somewhat pale and swollen, resulting from failure of the 'sodium pump', permitting the entry of sodium and water into the cell

Cellulitis: a diffuse inflammation of subcutaneous tissue extending along connective tissue planes

Chemokines: peptide molecules that induce chemically-directed migration of inflammatory cells – "*chemo*attractant cyto*kines*"

Chemotaxis: chemically-directed cellular migration

Chronic inflammation: an inflammatory response evoked by a persistent stimulus and characterised by aggregation of inflammatory cells and tissue proliferation rather than exudation

Circumscribed: well defined or demarcated, e.g. circumscribed lesion

Complement: a series of plasma proteins involved in many aspects of the inflammatory response, including opsonisation, chemotaxis and cytotoxicity

Congenital: literally, "born with" a disease; a condition attributable to events prior to birth

Cirrhosis: a chronic diffuse condition of the liver in which necrosis of hepatocytes is accompanied by fibrosis and regeneration, resulting in destruction of liver architecture and ultimate conversion of the parenchyma into numerous nodules separated by fibrous septa

Clone: a group of cells, all of which are the progeny of a single cell

Clot :a semi-solid mass formed from constituents of the blood after death (post-mortem clot), following haemorrhage, or in vitro (cf. thrombus)

Congestion: an excess of blood in the vessels, resulting from too much blood being delivered by the arteries (active congestion; synonymous with hyperaemia), or too little being drained by the veins (passive congestion, as in congestive cardiac failure)

Consolidation: becoming firm or solid: usually applied to the lung in which the alveolar spaces are filled to varying degrees with inflammatory exudate, retained secretions, neoplastic tissue or scar tissue

Cyst: a sac with a distinct wall lined by flattened cells enclosing fluid or other material

Cytokines: protein or peptide molecules mediating pathologically significant cellular reactions

Degeneration: a change in structure and function caused by injury to cells; the change is often reversible

Delayed hypersensitivity (DTH): cell-mediated immune response elicited by the subcutaneous injection of an antigen, with subsequent oedema and inflammation which are maximal between 24 and 48 hours (cf. immediate hypersensitivity)

Desmoplasia: induction of connective tissue growth, usually refers to the stroma of tumours (synonymous with fibroplasia)

Disseminated intravascular coagulation (DIC): widespread thrombosis in the microvasculature arising secondary to another illness, resulting in consumption of platelets and clotting factors (often leading to severe haemorrhage), traumatic damage to red cells and ischaemia to vital organs; common causes include septicaemia, obstetric emergencies and malignancy

Diverticulum: a pouch or sac arising from a hollow organ or structure

Dysentery: an inflammation of the colon characterised by pain, rectal tenesmus, profuse diarrhoea, with mucus and blood in the faeces (stool)

Dysplasia: atypical cellular differentiation; may be observed histopathologically within neoplasms or pre-neoplastic lesions

Dystrophic calcification: the localised deposition of calcium salts in dead or degenerate tissue (in the presence of normal plasma levels of calcium and phosphorus)

Dysuria: pain or difficulty with urination

Ecchymosis: a large area of discolouration of skin caused by extravasation of blood into subcutaneous tissues (synonymous with bruise)

Effusion: abnormal collection of fluid in a body cavity

Embolism: the transportation by the blood of abnormal material and its impaction in a vessel at a point remote from its entry into the circulation

Empyema: the presence of pus in a cavity or hollow organ, e.g., empyema of gall bladder

Epidemiology: the study of the incidence, distribution, and determinants of disease in a population, and its application to the control of health problems

Epistaxis: bleeding from the nose

Erythema: redness of the skin resulting from vasodilatation

Exudate: proteinaceous fluid resulting from the selective extravasation of intravascular plasma in response to an inflammatory stimulus; exudate usually has a specific gravity exceeding 1.020 due to its relatively high content of protein and cellular debris (cf. transudate)

Fatty change: the abnormal accumulation of lipid within parenchymal cells

Fibrinoid: a descriptive term for a variety of microscopic changes that occur in various tissues under dissimilar circumstances, in which the affected tissues stain brightly with eosin

Fibrinous: the adjectival form of fibrin - the protein formed by interaction of thrombin and fibrinogen

Fibrous: literally, containing fibres; but often used in Pathology to refer to collagenous connective tissue

Fine needle aspiration (FNA): a form of biopsy in which a fine needle (usually 25 gauge) is inserted into an area of tissue and a number of cells are collected, then expelled onto a slide and stained for cytological examination

Fistula: an abnormal communication between two body surfaces (cf. sinus)

Fracture: a break in the continuity of bone

Free radicals: highly reactive molecular forms capable of causing injury

Gangrene: necrosis with putrefaction of macroscopic portions of tissue

Goitre: an enlarged thyroid gland

Grade: degree of malignancy of a neoplasm, judged from histological features

Graft versus host disease: the rejection of host tissues that are recognised as foreign by transplanted immunocompetent cells which are capable of replication - usually a complication of bone marrow transplantation; typical manifestations include skin rash, jaundice, vomiting and diarrhoea

Granulation tissue: consists of newly formed blood vessels, fibroblasts and their products, and inflammatory cells: the tissue of repair

Granulomatous inflammation: a form of chronic inflammation; characterised by focal aggregations of chronic inflammatory cells, principally macrophages and their derivatives, e.g., epithelioid cells; these focal lesions are known as granulomas, and may exhibit central necrosis

Hamartoma: a developmental malformation consisting of an overgrowth of tissue(s) proper to the part, sometimes resembling a neoplasm (cf. haematoma)

Haemangioma: a developmental malformation of blood vessels (i.e. an example of a hamartoma)

Haematemesis: vomiting of blood

Haematoma: localised collection of blood or clot in solid tissues

Haematuria: blood in the urine

Haemoptysis: coughing up of blood-stained sputum or gross blood

Healing: the process by which the body replaces damaged tissue with living tissue; healing includes both regeneration and repair

Hernia: the abnormal protrusion of the whole or part of a viscus or other internal structure through an opening

HLA (Human Leucocyte Antigen): the major histocompatibility (MHC) genetic region in man; important in control of immune responses and graft rejection

Humoral immunity: immune response in which the predominant effector mechanism involves antibodies

Hyaline: a descriptive term for homogeneous, somewhat glassy or refractile microscopic appearance exhibited by various extracellular tissue elements or by the cytoplasm of cells

Hydronephrosis: abnormal dilatation of the renal pelvis and calyces, often associated with renal cortical atrophy

Hyperaemia: an increased volume of blood within actively dilated vessels in an organ or part of the body (cf. congestion)

Hyperplasia: an increase in size of an organ or tissue due predominantly to an increase in the number of its constituent specialised cells

Hypertrophy: an increase in size of an organ or tissue due predominantly to increase in size of its constituent specialised cells

Hypoplasia: the failure of development of an organ to a full, mature size (cf. aplasia)

Iatrogenic: implies 'caused by doctors', incorrectly derived from Greek root

Immediate hypersensitivity: immune response elicited within a few minutes after exposure to an antigen (allergen) due to the presence of preformed IgE antibodies; demonstrable after intradermal injection as a wheal with surrounding vasodilatation

Immunity: a state of reactivity following exposure to an antigen

Infarct: circumscribed ischaemic necrosis of tissue resulting from interference to blood flow, usually arterial

Infection: the invasion of the body by pathogenic micro-organisms

Inflammation: the process by means of which exudate and cells accumulate in irritated tissues and usually tend to protect them from further injury; may be acute or chronic –when unqualified, the term "inflammation" usually refers to acute inflammation

Inspissated: thickened, e.g. inspissated mucus obstructing an airway

Interleukins: a subset of cytokines originally construed to mediate leucocyte interactions

Ischaemia: a state of inadequate blood supply to a tissue or organ - potentially reversible

Karyolysis: loss of basophilic staining of the nucleus due to the action of DNase, often seen in necrotic cells (cf. pyknosis, karyorrhexis)

Karyorrhexis: fragmentation of the nucleus of a necrotic cell (cf. pyknosis, karyolysis)

Keloid: hypertrophic cutaneous scar, in excess of that necessary to heal the original defect

Lesion: an alteration of structure or of functional capacity due to injury or disease

Leucocytosis: an elevated number of circulating white blood cells

Leucopenia: a decreased number of circulating white blood cells

Leucoplakia: a lesion characterised by whitish thickening of mucosal epithelium

Lithiasis: formation of stones (calculi), e.g., nephrolithiasis, cholelithiasis

Lymphokines: soluble products of lymphocytes (especially T-cells) involved in cell-mediated immune responses (cf. cytokines)

Malignant: literally means virulent or life-threatening; in reference to neoplasms, the term indicates rapid growth, invasion of neighbouring tissues, potential for spread by metastasis, and frequently a fatal outcome; the single most important histopathological criterion of malignancy is tissue invasion

Melaena: tarry black coloured faeces due to altered blood from haemorrhage into the bowel, usually from the stomach or duodenum

Metaplasia: an adaptive substitution of one type of differentiated cell(s) by another type of differentiated cells

Metastasis: in reference to malignant neoplasms, the term refers to the development of secondary growths which arise from, but are discontinuous with, the primary lesion; such is termed a metastasis or metastatic lesion (synonymous with secondary)

Metastatic calcification: precipitation of calcium salts in apparently normal tissue as a result of disturbed calcium-phosphorus metabolism (e.g., hypercalcaemia) (cf. dystrophic calcification)

Monoclonal: attributable to a single clone of cells, and so more characteristic of a neoplastic than a reactive process (polyclonal)

Morphology: the structure of tissues and organs

Mutagen: an agent capable of damaging the DNA structure of cells; initiators of neoplastic transformation are mutagenic

Necrosis: death of cells in a restricted portion of tissue, recognisable by the autolytic changes undergone after the cells have died

Neoplasm: an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues, and persists in the same excessive manner after cessation of the stimuli which evoked the change

Occult: hidden, concealed, not evident; as 'occult blood in faeces' requiring special techniques for detection

Oedema: excessive accumulation of fluid causing swelling of tissues

Oliguria: abnormally low urine output (< 400 mL/day)

Organisation: a part of the healing process, occurring after an injury that has destroyed tissue which is unable to regenerate; involves the ingrowth of granulation tissue

Paraneoplastic: effects of a neoplasm not related to either the primary tumour mass or metastatic tumour deposits, e.g. abnormal hormone production, cachexia, etc.

Paraprotein: an abnormal band on serum protein electrophoresis, due to a monoclonal immunoglobulin and often associated with B cell neoplasia

Pathognomonic: characteristic/diagnostic of a particular disease

Pathogenesis: mechanism(s) by which the cause (aetiology) of a disease produces the clinical manifestations

Pathology: the scientific study of diseases

Peptic ulcer: an ulcer occurring in a portion of the alimentary tract exposed to the effect of gastric acid and pepsin

Petechiae: minute rounded spots of haemorrhage on skin, mucous membrane or cut surface of an organ; singular = petechia

Phagocytosis: ingestion of foreign or particulate matter by cells

Phlebothrombosis: formation of a thrombus in a vein

Polymerase chain reaction (PCR): a molecular diagnostic technique based on amplification by DNA polymerase of a known sequence of genomic DNA isolated from cells, or of DNA reverse-transcribed from mRNA or viral RNA; permits rapid, sensitive and specific detection of e.g. genetic mutations

Polyp: a projecting mass of tissue arising from an epithelial surface; may be composed of neoplastic, inflammatory or other tissues, found especially on mucous membranes

Prognosis: forecast of the outcome of an illness, based on the natural history of the disease and the likely response to treatment

Promoter: an agent, not acting as a mutagen, which causes an initiated cell or cell population to complete the process of neoplastic transformation

Proto-oncogene: a gene present in the normal cell (e.g. *RAS*, *MYC*); when one allele is inappropriately activated may cause or accompany the onset of cellular neoplastic transformation

Purpura: bleeding into the skin and/or mucous membranes, e.g. petechiae (pinpoint),ecchymoses (bruises)

Pus: typically a semi-fluid of creamy colour, pus is composed of necrotic and living neutrophils, together with necrotic tissue cells and exudate

Putrefaction: decomposition of organic matter by micro-organisms, accompanied by the development of disagreeable odour

Pyaemia: the presence of pus-inducing micro-organisms in the circulation with resultant formation of abscesses at sites of their lodgment (cf. bacteraemia and septicaemia)

Pyknosis: shrinkage and increased basophilic staining of the nucleus in a necrotic or apoptotic cell, caused by reduced pH (cf. karyolysis, karyorrhexis)

Regeneration: replacement of parenchymal cells by multiplication of similar surviving cells

Repair: replacement of lost tissue by connective tissue elements and parenchymal cells in varying proportions; when replaced completely by granulation tissue, which later matures to fibrous tissue, the result is referred to as a scar

Resolution: the return of a diseased tissue or organ to normal without residual scarring

Sarcoma: a malignant neoplasm arising from mesenchymal tissue

Sclerosis: hardening of tissue, especially from overgrowth of fibrous tissue

Septicaemia: severe infection with marked systemic clinical features; septicaemia is usually the expression of rapid and continuous invasion of the blood stream by microorganisms from the tissues, or multiplication in blood stream (cf. bacteraemia and pyaemia)

Shock: a clinical state in which there is widespread inadequate perfusion of tissues

Sign: a clinical feature identified by observation or examination of the patient (cf. symptom)

Sinus: in Pathology, this relates to an abnormal communication between a lesion (e.g., an abscess) in an organ, and an overlying surface (e.g., skin) (cf. fistula)

Staging: assessment of the size and extent of spread of a malignant neoplasm, important in determining the treatment and prognosis

Stem cell: a primitive cell from which differentiated cells arise during development, renewal and maintenance

Suppuration: the formation or discharge of pus

Symptom: a manifestation of disease which the patient may be aware of, or describe

Syndrome: a group of symptoms and signs which, when considered together, characterise a disease or lesion

Telangiectasis: a cluster of dilated malformed blood vessels (usually capillaries) producing a small red focal lesion, most common in skin or mucous membranes

Teratogen

an environmental agent which acts in utero to cause abnormal development, resulting in malformation of the fetus; teratogens include infective agents, radiation, drugs and chemicals

Teratoma: a true neoplasm arising from totipotential cells and therefore composed of numerous tissues which may not be indigenous to the part in which it occurs

Thrombophlebitis: inflammation of a vein (phlebitis) with associated thrombosis

Thrombus: a solid or semi-solid mass formed from the constituents of blood within the intact vascular system during life (cf. clot)

Tolerance: a state of non-responsiveness of cells of the immune system to a particular antigen

Toxaemia: the presence in the blood of toxic products produced by bacteria or formed in body cells

Transudate: fluid accumulated in tissue planes or spaces which is low in protein and which has leaked into the tissues from the micro-circulation; it occurs in non-inflammatory disorders such as congestive cardiac failure and venous obstruction (cf. exudate)

Tumour: a lump or swelling; however, the term is frequently used as a synonym for neoplasm

Tumour suppressor gene: a gene present in normal cells, which acts to suppress cellular proliferation (e.g. *TP53, RB*); when both alleles are inactivated, may cause or accompany the onset of neoplastic transformation

Ulcer: a lesion resulting from a circumscribed loss of surface epithelium of variable depth, often accompanied by inflammation of the adjacent tissue

Vesicle: a small blister

Western blotting (immunoblotting): a molecular diagnostic technique involving separation of proteins by gel electrophoresis, transferring them to a solid membrane via a blotting procedure, incubating with specific antibodies and applying a sensitive technique for detection of bound antibody; often used to detect specific proteins (e.g. viral) present in the serum

Zoonosis: a disease "accidentally" transmitted to humans from an animal host

PATH3205 Introduction

Course details

This course is offered during Semester I and counts for six units of credit (6OC). PATH2201/PATH2202 (Processes in Disease/Processes in Disease for Health and Exercise Science) are prerequisites for the course. It is also advantageous for students to have undertaken previous or concurrent study in ANAT3231 Cell Biology and BABS3041 Immunology I.

Course aims

The course **PATH3205 Molecular Basis of Inflammation and Infection** aims to:

1. Promote understanding of the molecular basis of inflammation, responses to infection, allergy, autoimmunity, and diseases of the cardiovascular and respiratory systems. These concepts are introduced in the context of common human diseases or disease processes.
2. Relate the above concepts of processes in human disease to biomedical research via the '*Research Lecture and Lab Series*' which provides introductory lectures on start-of-the-art areas of medical research, as well as associated laboratory-based workshops.
3. Develop oral and written communication skills which underpin dissemination of discoveries in human disease via medical research.

These aims will be achieved by specialist teaching of core concepts and research techniques by academic pathologists who are clinically and/or scientifically trained.

The course aims integrate molecular aspects of human disease into the context of histopathology and macroscopic specimens for each above mentioned disease topics. Furthermore, course aims mesh well with other disciplines including Anatomy, Biochemistry, Immunology, Microbiology, Pharmacology and Physiology.

Student learning outcomes

At the completion of this course, you should be able to:

1. Describe the causes, pathogenetic mechanisms, macroscopic and microscopic appearances and clinical consequences of inflammation, responses to infection, allergy, autoimmunity, and diseases of the cardiovascular and respiratory systems.
2. Work in collaborative teams to communicate concepts of disease in an oral presentation to non-specialist audiences.
3. Work independently to communicate, report and evaluate '*Research Lecture and Laboratory Topics*' in the written form by using specialist scientific journal articles and information from the *Research Lecture and Laboratory Series* and recording findings and interpretations in ePortfolios and as Laboratory Reports.
4. Understand the relevance of laboratory techniques in the diagnosis of human disease.

Learning and teaching rationale

The intended learning outcomes are achieved through study of the common patterns of response to injury, which are often referred to as pathological processes. To understand these processes, you will draw on your knowledge of normal anatomy, histology, biochemistry and physiology. PATH2201 Processes in Disease has introduced the fundamental concepts for the specific diseases to be addressed in PATH3205. This will involve more detailed discussion of recent advances in knowledge pertaining to the molecular basis of inflammation and infection, autoimmunity and both research and diagnostic techniques.

Future directions

The course complements PATH3206 *Cancer Pathology*, ANAT3212, BABS3041, PATH3207 *Musculoskeletal Diseases* and PATH3208 *Cancer Sciences: Research Design, Measurement and Evaluation* which also run in the same academic year. For those wishing to pursue a career in research or hospital based laboratory work, PATH3205 will not only develop your basic knowledge of molecular processes, but also provide a framework for understanding how these processes link to the modern practice of medicine and medical research. Similarly, for those who may wish to pursue a career in the health sciences, the course will provide an understanding of the cellular and molecular processes underlying the clinical manifestations of disease. Furthermore, development of ePortfolios will assist students in understanding career pathways in medical research.

Teaching strategies

The course comprises of lectures, tutorials, practical classes, '*research lectures and laboratories*' and assignments, which cover the general and specialist aspects of the molecular basis of disease.

The course employs a variety of teaching modes in order to facilitate your learning:

1) A **collaborative, team-based approach** to learning. It is anticipated that students will have an enhanced learning experience through the use of team quizzes, peer teaching and team projects. You are also encouraged to utilise your allocated teams as study groups and build your e-Portfolios.
2) A series of **lectures** introduce you to pathological processes, as well as specific examples of those processes affecting organs and tissues. The core lecture series focuses on specific diseases such as meningitis, HIV and diabetes. The tutorials are designed to be complementary to lectures and place these topics in the larger context of human disease. A list of aims and objectives is included for each lecture and tutorial, along with points for discussion and a list of suggested additional resources available on the web.
3) Small group **tutorials** are intended to extend and amplify your understanding of material presented in lectures in an interactive format, where you are encouraged to clarify any difficulties regarding the concepts discussed. Students will be allocated into teams and will complete individual and team quizzes and work collaboratively on interpretation of clinical problems and/or investigation results. Pre-reading will be assigned for each tutorial;
4) **Practical classes** employ computer-based virtual microscopy, in order to permit correlation between disease processes, changes in cells and tissues at the microscopic level and the manifestations of disease. Practical classes and tutorials in Molecular Basis of Inflammation and Infection are aimed at amplifying and extending your understanding of the topics gleaned from attendance at lectures and reading of the recommended text, as well as correcting any misconceptions. Practical and tutorial classes will reinforce the clinico-pathological correlations associated with each topic. They are intended to help you to acquire the ability to recognize the macroscopic and microscopic features of pathology specimens and to relate the pathology to clinical application. Macroscopic "pots" will be generally used in conjunction with projected microscopic slides, x-rays and other materials;
5) The course also includes several **'*Research Lecture and Laboratory Series*'** topics that, as the name would suggest, focus on the most recent research-led advances in molecular medicine. This section of the course is an innovation for the Department of Pathology and introduces the 'world of medical research' by way of specialist lectures that directly relate to research workshop laboratories; demonstrating 'state-of-the-art' molecular techniques that are key in diagnosis of disease. We hope it will provide you with an exciting and challenging glimpse of current approaches in medical research. Learning is supported via Moodle and Mahara (e-Portfolios). Announcements, timetables, lecture slides and other resources will be made available during the course.

Prizes

Two prizes will be awarded for Molecular Basis of Inflammation and Infection:

1. Best team performance in tutorial quizzes (based on both team and individual scores);
2. Best performance by team members in a combination of mid-session and end of course exam.

Assessment

The breakdown of assessmentsin the course is as follows:

Group presentation	15%
ePortfolios: LabBooks	10%
Individual and team performance in tutorial quizzes	15% (4 x 3.75%)
On-line progress assessments (x2)	5%
Practical Examination	10%
Final examination (2 hours)	45%

Group project *(15%)*

Students will work in groups to prepare a 15 minute PowerPoint presentation on a topic to be allocated in week 2, S1. Several one-hour sessions will be set aside for students to present their work to the rest of the group. One student from each group will designated to deliver the presentation by random draw (so all students must come prepared), and the remaining students in the group will be responsible for answering questions relating to the presentation.

Prior to the formal student presentations, The Learning Centre will run two presentation skills sessions and a follow-up session. This is an important part of developing skills for the group project.

The group project will be assessed by peers and academics. The peer assessment mark will weigh 25% and the academic assessment mark will weigh 75% of the total mark for this assessment. Sample assessment forms are included below.

Students will be asked to reflect on their group project using their ePortfolios.

ePortfolios: Lab Books *(10%)*

Students will be required to complete questions based on research labwork within their ePortfolios. Completed laboratory reports must be returned to the BSB office (room G27, ground floor, Biological Sciences) in **Week 12** by 2pm on Friday 30th May. Topics will be allocated by a random draw. Late Lab Reports will attract a penalty of 10% of the report mark per week or part thereof.

Marking criteria are shown below:

SECTION	COMPONENT PARTS	
Expected Writing Conventions		
Presentation	Neat, margins, 1.5 spacing, 12 point font. Simple staple. Page numbering	
Word Limit	1000 words	
Total Marks to be awarded		**/30**
Introduction		
The introduction gives an overview of the whole report and a brief literature review or orientation to the topic	Demonstrates an understanding of the topic and how it fits into the broader research area	/4
Hypothesis	Provides a statement of the expected rationale, purpose and outcome of the research area	/2
Aim	Provides a statement on how to address the proposed hypothesis **Word count 350**	/2
Materials and Methods		
	Demonstrates knowledge of research methodology and correct application to the research question **Word count 100**	/2
Results		
	Clearly states experimental outcomes (3) Demonstrates evidence of experimental outcomes (3) **Word count 300**	/6
Discussion		
	Critical evaluation of the chosen texts and methodology Demonstrates an ability to access the current medical research literature on the topic to gain information and use this in support of an argument (3) Directly addresses the question in the topic (2) **Conclusion statement: re-state key findings and state position about the identified issue (1)** **Word count 250**	/6
Reference List	Correctly uses references in the report ie. in text citations and provides and appropriate reference list	/2
ePortfolios	Demonstrates engagement and reflective practice : eg. building an awareness of skills portfolio, covering subject related skills, transferable skills, personal values, work experience, strengths and weaknesses, self reflection eg. on learning	/6

PATH3205 Group Presentations - Peer Assessment Form

Group No. AA

Topic Autoimmune haemolyticanaemia

Group members

XXX
XXX
XXX

Student Assessors (Group Y)

Name…… **Sign**……

Name…… **Sign**……

Name…… **Sign**……

	0	1	2	3	4
Clear explanation of disease process					
Structure of content – introduction, logical flow, conclusions					
Effective use of PowerPoint to deliver presentation					
Ability to answer questions					
Overall impression					
Subtotal					
TOTAL					

Comments:

PATH3205 Group Presentations – Academic Assessment Form

Group No. AA

Topic Autoimmune haemolytic anaemia

Group members

	0	1	2	3	4
Clear explanation of disease process					
Structure of content – introduction, logical flow, conclusions					
Effective use of PowerPoint to deliver presentation					
Ability to answer questions					
Overall impression					
Subtotal					
TOTAL					

Comments	
Strengths	
Improvement	
Points for clarification (if necessary)	

Assessor: …… (sign). **Date:** ……

On-line progress assessments *(5%)*

Students will be offered two online assessments during the course. These are to be completed **during the 10 days in which each is available (students will be notified in lectures when this will be)**. The assessments will include objective items in the same style as the final examination. Students may attempt the assessments as often as they wish within the time allowed until they receive a satisfactory score (>90%). The aim of these assessments is to provide students with feedback on their progress rather than to rank students. Students will receive 2.5% of the total mark for satisfactory completion of **each** of the assessments.

Individual and team performance in tutorial quizzes *15% (4 x 3.75%)*

Small group **tutorials** are intended to extend and amplify your understanding of material presented in lectures in an interactive format, where you are encouraged to clarify any difficulties regarding the concepts discussed. Students will be allocated into teams and will complete individual and team quizzes and work collaboratively on interpretation of clinical problems and/or investigation results. Pre-reading will be assigned for each tutorial.

Practical examination *(10%)*

Students will complete a practical examination during the **final week** of term (scheduled in normal teaching time). This will consist of a series of 6 stations each with questions based on material presented during term. Students will rotate around the stations, spending 5 minutes per station.

Final examination *(45%)*

Students will complete a two-hour written exam at the **end of session** that will contribute 45% of your overall mark. This will include objective items and five short answer questions. Some of the short answer questions may be directly from the Trial Examination Questions in the manual, the learning objectives or the on-line self-assessment. Marks will be weighted as follows:

Short answer	75%	(5 x 15 mins each)
Objective items	25%	

The short answer questions vary in style, but are intended to provide you with the opportunity to demonstrate your understanding of the topic and your ability to integrate ideas rather than simple "regurgitation of facts".

Supplementary examination

If required, it is intended that supplementary exams for the School of Medical Sciences in Semester 1, 2014 will be held 15th, 16th and 17th July, 2014. Special considerations sought outside the 3 day time period WILL NOT be accepted except in TRULY exceptional circumstances.

Students who believe that they are eligible for further assessment must contact Dr Polly to seek further information.

Sample examination paper

SAMPLE EXAMINATION FORMAT FOR 2012

(1) TIME ALLOWED : 2 HOURS.

(2) ANSWER ALL QUESTIONS.

(3) ANSWER PART A QUESTIONS ON SEPARATE PAGES. WRITE CLEARLY IN INK.

(5) ANSWER PART B USING THE GENERALISED ANSWER SHEET PROVIDED.

(6) THIS PAPER MAY NOT BE RETAINED BY THE CANDIDATE.

PART A (75 marks)

1. Discuss the pathways by which bacterial toxins produce fever.

(15 marks)

2. Why do patients develop such a rapid response to bacteria such as *Staphylococcus aureus* while the response to *Mycobacterium tuberculosis* is delayed?

(15 marks)

3. How do antigen-antibody complexes cause inflammation in systemic lupus erythematosus?

(15 marks)

4. Discuss the following statement:

   If everyone else in Australia vaccinates their children, it is best not to vaccinate yours (as long as you are the only person who thinks this way).

(15 marks)

5. Discuss the following statement:

   Heart disease is primarily a disease of middle aged and older people. Therefore, people should not be concerned about exercise, blood pressure or diet until they reach their forties.

(15 marks)

PART B (25 marks)

This part of the examination consists of 12 questions, each containing 5 statements. For each question, select the **BEST or MOST APPROPRIATE** answer from among the alternatives. On the supplied generalised answer sheet, **FILL IN** the corresponding circle. **USE PENCIL**. You will score 2 for each correct answer and 0 for each incorrect or omitted answer. An example is shown:

1. **The most important effects of meningococcal septicaemia result from:**

   (A) Formation of antigen-antibody complexes and initiation of the complement cascade.

   (B) The ability of the bacteria to exit the blood stream and colonise the meninges.

   (C) The effects of LPS on macrophages promoting the release of TNF.

   (D) A direct toxic effect of LPS on the myocardium.

   (E) Formation of septic infarcts in multiple organs including the kidney.

Answer: C

PATH3205 Course Timetable

NOTE: Changes in the timetable will be announced onMoodle.

Week	Date	Time	Location	Lecturer	Title
1	Tue 4/3	9	BioMed E	Polly	Introduction to Molecular Basis of Inflammation and Infection ** *presentation topics announced***
Module: Infective Inflammation					
		10	BioMed E	Lloyd	Viruses, hosts, and infectious diseases
	Fri 7/3	9 10-11 11-12	BioMed F BioMed F Teaching Labs: G6/G7	Luciani Bull Bull / Luciani	Hepatitis C – understanding the virus Hepatitis C and host immunity Hepatitis C - virology/immunology lab
2	Tue 11/3	9	BioMed E	Velan	Meningitis
		10	BioMed E	McNeil	Researching Rheumatoid Arthritis
	Fri 14/3	9 10-11 11-12	BioMed F Teaching Labs: G6/G7 Tutorial rooms: G6/G7 G16/G17	Jones/Polly Dziegielewski *see allocation*	Science communication I: Presentation and collaborative learning skills Museum Study Session 1 – Acute and Chronic inflammation Tutorial 1 – Acute and Chronic inflammation
Module: Immune Responses in Inflammation, Asthma and IBD					
3	Tue 18/3	9	BioMed E	Sewell	Molecular basis of allergy
		10	BioMed E	Kumar	Molecular basis of asthma
	Fri 21/3	9 10-12	BioMed F Teaching Labs: G6/G7	Kumar Kumar/Herbert	Smoking and the lung *Asthma Research Lecture; Asthma Lab*
4	Tues 25/3	9	BioMed E	Grimm	Immune-mediated Bowel Diseases – Ulcerative Colitis and Coeliac Disease
		10	BioMed E	Grimm	*IBD Research Lecture*
	Fri 28/3	9-11 11-12	Teaching Labs: G6/G7 Tutorial rooms: G6/G7 G16/G17	Grimm/Lee/Luo *see allocation*	*IBD Lab* Tutorial 2 – Immune Responses in Inflammation eg. Allergy, Asthma and IBD
Module: Disturbances of Immunity and Inflammation, HIV and Autoimmunity					
5	Tues1/4	9	BioMed E	Post	HIV, the virus and its effects - I
		10	BioMed E	Post	HIV, the virus and its effects - II
	Fri 4/4	9 10-12	BioMed F Teaching Labs: G6/G7	Jones/Polly McNeil/Bryant	Science communication II: Presentation and collaborative learning skills *Rheumatoid Arthritis Research Lecture ; Rheumatoid Lab*
6	Tues 8/4	9	BioMed E	Wakefield	Autoimmune disease I
		10	BioMed E	Wakefield	Autoimmune disease II ***On-line assessment I***
	Fri 11/4	9 10-12	BioMed F Teaching Labs: G6/G7	DiGirolamo DiGirolamo	Stem Cells, Inflammation and Immunology Research Lecture Stem Cells Research Lab
Mid Semester Break 18-27 April					
8	Tues 29/4	9	BioMed E	Tedla	Mechanisms that terminate immune responses
		10	BioMed E	Dziegielewski	Renal Disease - Principles/Examples
	Fri 2/5	9-11 11-12	Teaching Labs: G6/G7 Tutorial rooms: G6/G7 G16/G17	Wakefield/Tedla *see allocation*	*Practical class 1 - Autoimmunity* Tutorial 3 – HIV and Autoimmune disease
Module: Systemic Inflammation: Effects *Research Team Presentations*					
9	Tues 6/5	9	BioMed E	Polly	Student presentations
		10	BioMed E	Polly	Student presentations
	Fri 9/5	9	BioMed F	Polly	Inflammation and Musculoskeletal Effects- Cachexia

		10-12	Teaching Labs: G6/G7	Polly	Cachexia Research Lab
10	Tues 13/5	9	BioMed E	Polly	Student presentations
		10	BioMed E	Polly	Student presentations
	Fri 16/5	9	BioMed F	Phillips	Inflammation Effects-Pancreatitis and Pancreatic Cancer
		10-12	Teaching Labs: G6/G7	Phillips	Pancreatic Disease Research Lab
11	Tues 20/5	9	BioMed E	Polly	Student presentations
		10	BioMed E	Polly	Student presentations ***On-line assessment II***
	Fri 23/5	9 10-12	BioMed F Teaching Labs: G6/G7	Thomas Thomas/Thai	Inflammation and Cardiovascular Effects Inflammation and Cardiovascular Research Lab:
12	Tues 27/5	9	BioMed E	Polly	Student presentations
		10	BioMed E	Polly	Student presentations *****Lab reports due 2pm 30/05/2014-G27 Biological Sciences*****
	Fri 30/5	9 10-11	BioMed F Teaching Labs: G6/G7	Kumar Dziegiewlewski	Cardiovascular Disease Examples and Complications Museum Study Session 2 –Cardio-respiratory
		11-12	Tutorial rooms: G6/G7 G16/G17	*see allocation*	Tutorial 4 – Cardiovascular disease
13	Tues 3/6	8	BioMed E	Polly/Jones	'Feedback'
		9	BioMed E	Revision	Kumar
	Fri 6/6	9-12	G6/G7 G16/G17	Polly	** Practical examination **

KEY:

Di Girolamo	A/Prof Dr Nick Di Girolamo	Inflammation and Infection Research Centre (IIRC;Pathology)
Dziegielewski	Dr Mark Dziegielewski	Department of Pathology, UNSW
Grimm	Prof Michael Grimm	Inflammation and Infection Research Centre (IIRC;Pathology);St George Hospital
Jones	Ms Gwyn Jones	The Learning Centre, UNSW
Kumar	Prof Rakesh Kumar	Department of Pathology, UNSW
Lloyd	Prof Andrew Lloyd	Inflammation and Infection Research Centre (IIRC;Pathology);
Polly	Dr Patsie Polly	Inflammation and Infection Research Centre (IIRC); Department of Pathology
Post	Dr Jeffrey Post	Prince of Wales Clinical School, UNSW and Prince of Wales Hospital
Sewell	A/Prof Bill Sewell	St Vincent's Hospital
Tedla	A/Prof Nicodemus Tedla	Inflammation and Infection Research Centre (IIRC;Pathology);
Thomas	Dr Shane Thomas	Department of Pathology, UNSW (CVR, Lowy; Pathology)
Velan	A/Prof Gary Velan	Department of Pathology, UNSW
Wakefield	Prof Denis Wakefield	Department of Pathology, Associate Dean-Faculty of Medicine, UNSW

PATH3205 Lecture program
Additional learning resources:

The suggested textbook for the course is Kumar, Cotran and Robbins, Basic Pathology (8th Edition), Saunders Elsevier.

In addition, there are many resources available on the web, which vary from simple patient information brochures to on-line pathology courses to information on the latest research. Some general sites you may find useful are:

Centre for Disease Control (see especially 'health topics A-Z')
http://www.cdc.gov/

University of Utah (tutorials and images on many of the topics covered)
http://library.med.utah.edu/WebPath/webpath.html#MENU

Medline Plus ('health topics' index of diseases with information)
http://www.nlm.nih.gov/medlineplus/healthtopics.html

Other resources are indicated for some lectures.

Introduction to Molecular Basis of Inflammation and Infection

Aim

This lecture is designed to provide an overview of the course and revise your understanding of inflammation.

Learning objectives

At the completion of this lecture you should be able to:

1. Classify the host reaction to different classes of infectious agents into acute and chronic inflammatory responses.
2. Distinguish between the clinical manifestations of acute and chronic inflammation and provide examples of diseases resulting from each.
3. Outline the role of "immunopathology" in infectious disease, auto-immunity and transplantation.
4. Acquired and innate immunity

Points for discussion

Discuss the following statement:

Not all host responses to infection are beneficial. Sometimes too strong a response is dangerous.

Viruses, hosts and infectious diseases

Aim

This lecture provides an overview of the way in which different viruses interact with host immune responses to produce a spectrum of clinical infectious diseases. The lecture will review the characteristics of different viruses, and the key elements of host antiviral immune responses. The factors which influence the pattern of the host immune response to a given virus, including host genetics and viral subversion strategies will be discussed. Achievements, and remaining challenges, in vaccine design against viral infections will also be included.

Learning objectives

At the completion of this lecture you should be able to:

1. Understand the principles of how different viruses and host immune responses interact to influence infection outcomes.
2. Understand the role of host genetic factors and viral evasion factors modify antiviral immunity.
3. Understand the principles of antiviral vaccine design and development.

Points for discussion

Why were so many people affected by the recent swine flu pandemic, and why didn't many die?

Additional Resources

Roitt's essential immunology. Peter J Delves; Ivan M Roitt (Ivan Maurice) 12th ed. Chichester, West Sussex ; Hoboken, NJ: Wiley-Blackwell 2011.

Medical Immunology. Gabriel Virella. New York, Informa Healthcare 2007.
Available online at UNSW Library

Hepatitis C

Aim

This series of two lectures provides an overview of the virology and immunology of hepatitis C virus (HCV) infection, focusing on factors that contribute to clearance or persistence, and factors driving liver injury. The first lecture is an overview of the current understanding of the pathophysiology of HCV infection and the clinical outcomes, and how the virus replicates and finally how it interacts with the human immune response. The second part will be cover the immunology of hepatitis C infection. Both lectures will provide insights into research activities at the University of New South Wales in this topic, including studies of viral evolution and cellular immune responses against the virus.

Learning objectives

At the completion of this lecture you should:

1. Understand mechanisms driving HCV infection outcomes, and the role of viral evolution and host immune responses in these outcomes.
2. Understand clinical outcomes from HCV infection
3. Understand the assay systems used in the laboratory to diagnose HCV infection, and to investigate viral characteristics and host immune responses.
4. Be aware of current treatments and new treatment strategies
5. Understand the challenges in HCV vaccine development

Points for discussion

What are the major components that determine outcomes of HCV infection?

What are the future options for prevention of HCV infection?

Additional Resources

Immunological determinants of the outcomes from primary hepatitis C infection. Post J, Ratnarajah S, Lloyd AR. Cell Mol Life Sci. 2009 Mar;66(5):733-56.

Host and viral factors in the immunopathogenesis of primary hepatitis C virus infection.Lloyd AR, Jagger E, Post JJ, Crooks LA, Rawlinson WD, Hahn YS, Ffrench RA.Immunol Cell Biol. 2007 Jan;85(1):24-32. Epub 2006 Nov 28. Review.

Molecular basis of meningitis

Aim

This lecture is designed to familiarise you with the effects of bacterial products in triggering acute inflammation, as well as the complications of the acute inflammatory response in meningitis.

Learning objectives

At the completion of this lecture you should be able to:

1. Understand the importance of meningitis within our community and summarise the major causes.
2. Describe the roles of bacterial products in causing acute inflammation.
3. Outline the chemical mediators released by the host that take part in acute inflammation.
4. Discuss the roles of different cell types in an acute inflammatory response.
5. Summarise the time course of an acute inflammatory response, with reference to the appearance of different cells and inflammatory mediators.
6. Describe the mechanisms by which bacterial cells and products are destroyed and removed.
7. Outline the complications of meningitis and relate these to the process of acute inflammation together with healing and repair.
8. Discuss the pathogenesis and complications of meningococcal septicaemia.

Points for discussion

Discuss the following statement:
Acute inflammation usually involves "pattern recognition" of bacterial toxins and products. By contrast, responses to intracellular bacteria and viruses require specific immune recognition.

Discuss the pathways by which bacterial products may produce fever.

Discuss the pathways by which bacterial products may induce coagulation and the consequences of this.

Additional Resources:

Web site U. Utah (images) http://library.med.utah.edu/WebPath/INFLHTML/INFLIDX.html

Researching Rheumatoid Arthritis

Aim

This lecture provides an overview of approaches to undertake research into rheumatoid arthritis with a particular focus on the role of mast cells.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline broad concepts about the pathological processes underlying the development of rheumatoid arthritis (RA).
2. Understand different experimental approaches to research a disease such as RA – e.g., *in vivo*, *in vitro*, animal models.
3. Recognise basic scientific methods including the need for positive and negative controls.
4. Discuss the potential role of mast cells in the pathogenesis of RA.

Points for discussion

How could these approaches be used to study other human diseases?

Additional Resources

Web site Arthritis Australia www.arthritisaustralia.com.au

Molecular basis of allergy

Aim

This lecture is designed to introduce the concept of immunological hypersensitivity and its relationship to common allergic phenomena.

Learning objectives

By the completion of this lecture you should be able to:

1. Discuss the roles of allergens and antibodies in acute hypersensitivity reactions.
2. Define the following terms: “allergy”; “anaphylaxis”; “eczema”; and “atopy”.
3. Discuss the role of mast cells and their chemical mediators in type I hypersensitivity.
4. Understand the cellular interactions required for an IgE antibody response.
5. Outline common examples of type I hypersensitivity reactions and their clinical manifestations.
6. Describe the rationale for “desensitisation” therapy for common allergies.

Points for discussion

Many people lack the enzyme lactase and ingestion of milk results in stomach cramps and diarrhoea. Is this the same as milk allergy?

Discuss the following statement:

People don’t have allergic responses to their first bee sting.

Additional resources

Web sites	NIAID	http://www.niaid.nih.gov/topics/allergicdiseases/Pages/default.aspx
	ASCIA	http://www.allergy.org.au/health-professionals/hp-information/asthma-and-allergy

Molecular basis of asthma

Aim

This lecture provides an overview of the current understanding of the pathogenesis of asthma.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline the pathological processes underlying the development of asthma.
2. List common triggers for allergic airway disease and exacerbations of asthma.
3. Discuss the role of cytokines and other inflammatory mediators in asthma.
4. Briefly summarise the pathophysiological basis for the current approach to assessment and treatment of asthma.
5. Discuss the potential role of immunotherapy for asthma.

Points for discussion

Are all deaths from asthma avoidable with adequate therapy?

Additional Resources

Web site Asthma Australia http://www.asthmaaustralia.org.au/

Smoking and the lung

Aim

The aim of this lecture is to familiarise you with the common effects of smoking on the lung.

Learning objectives

By the completion of this lecture you should be able to:

1. Explain the terms “emphysema”, “chronic bronchitis” and “muco-ciliary escalator”.
2. Outline how smoking may predispose to lung infections.
3. Describe the pathogenesis of smoking-related chronic obstructive lung disease.
4. Discuss the effects of alpha-1 proteinase inhibitor deficiency, and its interaction with smoking.
5. Comment briefly on the relationship between smoking and lung cancer.

Points for discussion

Why don’t all heavy smokers develop emphysema?

Additional Resources:

Web sites: QUIT Australia http://www.quitnow.info.au/

Immune-mediated Bowel Diseases – Inflammatory bowel disease and Coeliac Disease

Aim

This lecture is designed to introduce you to the complexities of the gut mucosal immune system, using the inflammatory bowel diseases and coeliac disease as examples.

Learning objectives

By the completion of this lecture you should be able to:

1. Describe the importance of the mucosal immune system in maintaining tolerance to food and bacterial antigens and responding to pathogens.
2. Compare the role of commensals and pathogens within the gut microflora.
3. Explain what is meant by "oral tolerance".
4. Describe the pathogenesis of inflammatory bowel diseases and coeliac disease.
5. Discuss the clinical course and treatment of inflammatory bowel diseases and coeliac disease.

Points for discussion

Many brands of yoghurt promote the idea of providing live bacteria. Recent case reports describe the use of faecal transplants in recurrent colon infections. Discuss whether these may be useful strategies to prevent intestinal infection or treat intestinal inflammation.

Additional Resources:

Web sites	NIDDK	http://digestive.niddk.nih.gov/ddiseases/pubs/celiac/
		http://digestive.niddk.nih.gov/ddiseases/topics/IBD.aspx
Reviews	*Nature* 2011; 474(7351):307-17 (an excellent overview of IBD pathogenesis)	
	International Reviews of Immunology 2011. 30(4):219-31 (a simple review of pathogenesis, clinical presentation and treatment of coeliac disease)	

HIV: The virus and its effects I

Aim

This lecture aims to provide an overview of the epidemiology and clinical course of HIV.

Learning objectives

By the completion of this lecture you should be able to:

1. Define the terms “HIV”, “AIDS”, “CD4 count”, “viral load”, “quasi-species” and “AIDS-defining illness”.
2. Discuss the modes of transmission of HIV.
3. Compare and contrast the modes of transmission and course of the HIV epidemic in Africa with HIV infection in the developed world.
4. Outline the clinical course of HIV infection, including common causes of mortality.
5. Outline the changes in the immune response to HIV, as well as changes in general immune function over the course of infection with HIV.

Points for discussion

Why are AIDS patients at particular risk of tuberculosis but not usually at significantly increased risk of infection with extracellular bacteria?

Discuss the modes of transmission of HIV and how these may differ between Africa and the USA.

Transmission of HIV by blood transfusion is an emotive issue. Explain why it may be hard to completely eliminate the possibility of transmission by this route.

Additional Resources

Web-sites	UCSF information site	http://hivinsite.ucsf.edu/
	CDC	http://www.cdc.gov/hiv/resources/factsheets/index.htm
	U.Utah (HIV tutorial)	http://library.med.utah.edu/WebPath/TUTORIAL/AIDS/HIV.html

HIV: The virus and its effects II

Aim

This lecture aims to review the host response to HIV infection and the mechanisms of viral pathogenesis.

Learning objectives

By the completion of this lecture you should be able to:

1. Explain the role of antibody and cell-mediated immunity to HIV.
2. Discuss the role of cytotoxic T-lymphocytes (CTL) in reducing viral load.
3. Compare and contrast the possible roles of viral cytopathic effect versus CTL killing of infected cells in reducing CD4 T-cell numbers.
4. Discuss the importance of cellular receptors in permitting HIV entry into the cell.
5. Summarise the mechanisms of viral mutation, and relate this to viral evasion of the immune response and viral drug resistance.
6. Briefly summarise the different vaccine strategies and antiviral drugs now being used/studied for use against HIV.

Points for discussion

Mycobacterium tuberculosis and the Human Immunodeficiency Virus (HIV) may both infect macrophages. Discuss why this may be a useful strategy for pathogens.

Discuss the mechanisms of host damage during viral infection.

Suggest reasons why development of a vaccine against HIV has proved so difficult.

Additional Resources

Web-sites	UCSF information site	http://hivinsite.ucsf.edu/
	CDC	http://www.cdc.gov/hiv/resources/factsheets/index.htm
	U.Utah (HIV tutorial)	http://library.med.utah.edu/WebPath/TUTORIAL/AIDS/HIV.html

Autoimmune Disease I

Aim

This lecture provides an introduction to the mechanisms of autoimmunity, using autoimmune thyroid disease as an example.

Learning objectives

By the completion of this lecture you should be able to:

1. Outline the concepts of "self-tolerance" and autoimmunity.
2. Describe how self-tolerance is maintained in B cells and T cells.
3. Suggest possible mechanisms for "breaking" self-tolerance.
4. List the different types of hypersensitivity reactions and briefly discuss the molecular basis of these.
5. Discuss the pathogenesis of autoimmune thyroid disease and relate this to: (i) loss of self tolerance; and (ii) the different types of hypersensitivity reactions.

Points for discussion

Discuss the following statement:

Auto-immunity is a diagnosis of exclusion. When we find a pathogen we describe immunopathology as infectious, otherwise we call it autoimmune.

Additional Resources

Web sites	NIAID	http://www.niaid.nih.gov/publications/autoimmune.htm
	U.Utah (images)	http://library.med.utah.edu/WebPath/IMMHTML/IMMIDX.html

Autoimmune Disease II

Aim

This lecture provides an outline of some of the systemic manifestations of autoimmune disease, using systemic lupus erythematosus (SLE) as an example.

Learning objectives

By the completion of this lecture you should be able to:

1. Discuss the common clinical manifestations of systemic lupus erythematosus (SLE).
2. Outline the proposed aetiology and pathogenesis of SLE.
3. Explain the molecular basis of the abnormalities found in the kidney*, skin, heart and CNS in SLE.
4. Briefly summarise the treatment and complications of SLE.
5. Discuss different types of investigations used to diagnose autoimmune disease.

Points for discussion

How do antigen-antibody complexes cause inflammation?

Additional resources

Web sites	NIAID	http://www.niaid.nih.gov/publications/autoimmune.htm
	U.Utah (images)	http://library.med.utah.edu/WebPath/IMMHTML/IMMIDX.html

Stem Cells and Stem Cell Deficiency - Relation to Ocular Inflammation

Aim

This lecture provides an overview of the current understanding of human stem cells. It will focus mainly on how stem cells maintain the eye (cornea) in a healthy and transparent state, and will describe the pathological processes that develop in stem cell deficient disorders of the eye.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline the importance (function) of the stem cell niche
2. List common characteristics of stem cells
3. Discuss the pathological processes that characterise stem cell deficiency in the cornea.
4. Summarise the treatment options for patients with severe corneal blindness
5. Understand the basis of immunohistochemistry as a technique that is widely used to identify stem cells in tissue

Points for discussion

List the types of stem cells that have been identified. Does the use of all types of stem cells raise moral/ethical/religious, other issues?

Additional Resources

Web site Australian Stem Cell Centre www.**stemcellcentre**.edu.au/

Renal Disease

Aim

This lecture elucidates the pathogenesis of the nephritic and nephrotic syndromes and reviews the causes and clinical consequences of acute kidney injury and chronic kidney disease.

Learning objectives

By the completion of this lecture you should be able to:

1. Summarise the common causes of acute kidney injury (AKI).
2. Explain the pathophysiological basis of the clinical features of the nephritic syndrome and the nephrotic syndrome.
3. Discuss the indications for renal biopsy and the way that the pathologist assesses the biopsy.
4. Summarise the common causes of chronic kidney disease (CKD) in Australia.
5. Discuss the aetiology and pathogenesis of important causes of chronic kidney disease, including diabetic nephropathy and reflux nephropathy.
6. Outline the common systemic complications that occur in chronic kidney disease.

Points for Discussion

Why are indigenous Australians at particular risk of developing chronic kidney disease?

Additional Resources

Australian Institute of Health and Welfare.*Chronic Kidney Disease in Australia in 2005.*
http://www.aihw.gov.au/publication-detail/?id=6442467782

Inflammation and Musculoskeletal Effects - Cachexia

Aim

This lecture provides an overview of the current understanding of how muscle wasting occurs during cancer progression. This lecture will address transcriptional events that underlie how skeletal and cardiac muscle 'breaks down' due to inflammation in the setting of cancer.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline the importance of cytokines in cancer related cachexia.
2. Understand what is meant by the term 'cachexia' and understand which tissues are affected.
3. Discuss the types of cancers that appear to have the strongest cachectic effect.
4. Understand the basis of qRT-PCR and Western blotting as routine molecular techniques to measure gene and protein expression.

Points for discussion

Is nutritional supplementation beneficial for 're-building' muscle?

Additional Resources

Cancer cachexia mechanisms and clinical implications.Gastroenterol Res Pract. 2011;2011:601434.

Cancer Cachexia: a systematic literature review of items and domains for involuntary weight loss in cancer. Crit Rev OncolHematol. 2011; 80(1):114-44.

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Inflammation and Pancreatic Cancer

Aim

This lecture will provide a current overview of pancreatic cancer and how the associated stromal reaction (composed of dense extracellular matrix, blood vessels and stromal cells including fibroblasts, inflammatory cells and endothelial cells) contributes to pancreatic cancer progression.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline the importance of the stromal reaction in pancreatic cancer progression and chemoresistance.
2. Summarise the role of a key stromal cell (Pancreatic stellate cell) in creating a hypoxic microenvironment which results in poor drug delivery into pancreatic tumours.
3. Discuss future treatment options for pancreatic cancer which include targeting both the cancer cells and stromal cells.
4. Understand the importance of *in vivo* animal models which accurately mimic human pancreatic cancer.

Inflammation and Cardiovascular Dysfunction

Aim

This lecture provides an overview of the current understanding of how arteries become dysfunctional during cardiovascular disease. It will primarily focus on the importance of endothelial cells in maintaining a healthy blood vessel, and will outline the pathological processes that cause these cells to become dysfunctional resulting in the promotion of cardiovascular disease.

Learning objectives

At the completion of this lecture you should be able to:

1. Outline the importance of the vascular endothelium for the maintenance of cardiovascular homeostasis.
2. Summarise how nitric oxide is produced by endothelial cells and list its beneficial activities.
3. Understand what is meant by the term 'endothelial dysfunction' and the methods available to measure the degree of such dysfunction in cardiovascular disease patients.
4. Define what the term 'oxidative stress' is and how this process can promote endothelial cell dysfunction during cardiovascular disease.
5. Recall potential treatment options available to treat endothelial dysfunction during cardiovascular disease.
6. Understand the basis of Western blotting as a routine biochemical technique to measure protein phosphorylation and cell signalling.

Points for discussion

Are antioxidant supplements as beneficial as they are made out to be in the media?

Additional Resources

Redox Control of Endothelial Function and Dysfunction: Molecular Mechanisms and Therapeutic Opportunities. *Antiox.Redox. Signal*. 2008, 10: 1713-1766

Cardiovascular Disease

Aim

This lecture provides an overview of the basis of the clinical features of cardiovascular disease, focusing on ischaemic heart disease and cerebral infarction.

Learning objectives

By the completion of this lecture you should be able to:

1. Describe the natural history of ischaemic heart disease
2. Summarise the major manifestations and complications of MI.
3. Explain the basis of tests used to diagnose MI.
4. Describe the pathogenesis and features of cerebral infarction.
5. Discuss the basis of peripheral vascular disease.

Points for discussion

Discuss the following statement:

Both recent and old myocardial infarction may be associated with an increased risk of stroke. Explain the reasons for each.

Additional Resources:

Web sites: Australian Heart Foundation http://www.heartfoundation.org.au/

PATH3205 Tutorial program

Guide to the tutorials

Tutorials in Molecular Basis of Disease are NOT mini-lectures or didactic sermons with you as the passive observers. The tutorials ARE aimed at amplifying and extending your understanding of the topic gleaned from attendance at lectures and reading of the recommended text, as well as correcting any misconceptions. Hence, adequate preparation and active participation are essential.

Remember, it is much better to make a mistake in the relative safety of a tutorial, than to make a critical error in an essay or exam because of a misconception of basic pathological principles. Any student experiencing difficulty with the course should discuss this either with their tutor, the Convenor of PATH3205 (Dr Polly) or the Grievance Officer (Dr Pandey).

The tutorials are also intended to help you to acquire the ability to recognise the macroscopic features of pathology specimens, which exemplify the topic of each tutorial. By participating, you will be given adequate opportunity to practice and improve your communication skills, as well as receiving constructive feedback from your tutor.

Material discussed in tutorials is examinable, and should be considered as an adjunct to lectures, practical classes and museum study sessions. Each of these forms of teaching is intended to complement and amplify the others. For this reason, the lectures, tutorials and practical classes on each topic are coordinated as closely as possible.

Tutorial 1 – Acute and Chronic Inflammation

Aims:

This tutorial aims to:

Revise and integrate your knowledge of the molecular basis of acute and chronic inflammation, with reference to acute bacterial meningitis and chronic viral hepatitis as examples.

Learning objectives:

At the completion of this tutorial you should be able to:

1. Outline how the acute inflammatory response helps to eliminate bacterial infection.
2. Contrast the host response to intracellular bacteria and viruses with the response to pyogenic bacteria.
3. Describe the pathogenesis of acute bacterial meningitis.
4. Describe the microscopic and macroscopic appearances of acute bacterial meningitis.
5. Explain the common local and systemic complications associated with acute bacterial meningitis.
6. Describe the features of chronic inflammation that distinguish it from acute inflammation.
7. Outline the pathogenesis of acute versus chronic hepatitis.

Points for discussion

Staphylococcus aureus tends to cause abscesses in the skin, lung and brain. Discuss the reasons for this.

Describe the molecular pathogenesis of septic shock in acute bacterial meningitis.

What features of chronic viral hepatitis indicate that it is an example of a chronic inflammatory response?

Additional Resources:

Web site	U. Utah	http://library.med.utah.edu/WebPath/INFLHTML/INFLIDX.html

Tutorial 2 – Immune Responses in Inflammation:Allergy, Asthma and IBD

Aims:

This tutorial is designed to reinforce your knowledge of asthma and common allergic responses.

Learning objectives:

At the completion of this tutorial you should be able to:

1. List common triggers for asthma and describe how these lead to the clinical manifestations.
2. Describe the abnormalities in the airways of an asthmatic.
3. Outline the molecular targets and aims of asthma therapy.
4. Discuss common allergic responses and their triggers.
5. Summarise the common treatments for allergies and the ways in which they alter the underlying pathological processes.
6. Compare the role of commensals and pathogens within the gut microflora.
7. Describe the pathogenesis of inflammatory bowel diseases and coeliac disease. Discuss differences and similarities.

Points for discussion

Discuss the following statement:

Asthma is the price we pay for economic development, fewer childhood infections and modern housing.

Additional Resources:

Web site	Asthma Australia	http://www.asthmaaustralia.org.au/
	NIAID	http://www.niaid.nih.gov/publications/allergies.htm

Tutorial 3 – HIV and Autoimmunity

Aims:

This tutorial aims to:

1. Broaden your understanding of the molecular basis for the pathology seen in HIV infection and link this knowledge to the current strategies for HIV prevention and treatment.
2. Outline the pathological basis of common autoimmune conditions.

Learning objectives:

At the completion of this tutorial you should be able to:

1 Outline the pathways of cell entry, mechanisms of viral replication, and host response to HIV.

2 Describe at which points current therapies affect the above pathways.

3 Summarise the current role of drug therapy in HIV treatment and prevention.

4 Discuss the role of other sexually transmitted diseases in the spread of HIV.

5 Discuss the potential use of vaccines in HIV, outlining some of the problems that could be anticipated.

6 List common auto-immune diseases and outline the pathological basis for these conditions.

7 Compare the roles of T cells and antibodies in auto-immunity.

8 Discuss the pathogenesis of autoimmune thyroid disease in terms of the different types of "hypersensitivity reactions".

Points for discussion

Discuss the following statement:

HIV is mutating too fast for drugs to catch up with it.

Discuss the following statement:

All the clinical effects of autoimmune diseases are examples of type II or type III hypersensitivity.

Additional Resources:

Web-sites	UCSF information site	http://hivinsite.ucsf.edu/
	Centre for Disease Control	http://www.cdc.gov/hiv/resources/factsheets/index.htm
	U.Utah (tutorial)	http://library.med.utah.edu/WebPath/TUTORIAL/AIDS/HIV.html

Tutorial 4 – Cardiovascular disease

Aims:

This tutorial aims to review your understanding of the causes of heart disease.

Learning objectives:

At the completion of this tutorial you should be able to:

1 List the health consequences of smoking and discuss the molecular basis for these.

2 Discuss how lifestyle may affect the risk of heart disease.

3 Discuss the controversy surrounding the potential role of infectious agents in causing heart disease.

4 Compare the roles of prevention and treatment in ischaemic heart disease.

Points for discussion

Discuss the following statement:

Industrialised societies may increase the risk of heart disease through lifestyle factors, but decrease it through hygiene measures.

Additional Resources:

Web sites: Australian Heart Foundation http://www.heartfoundation.org.au/

PATH3205 Guide to the Research Lectures and Laboratories

The course includes several **'*Research Lecture and Laboratory Series*'** topics that, as the name would suggest, focuses on the most recent research advances in molecular medicine. This section of the course is an innovation for the Department of Pathology and introduces the 'world of medical research' by way of specialist lectures that directly relate to research workshop laboratories; demonstrating 'state-of-the-art' molecular techniques that are key in disease diagnosis. Attendance is compulsory and will be assessed in the form of written LabReports and ePortfolios. Learning is supported via Blackboard, Moodle and Mahara (e-Portfolios).

The format of the Research Laboratories may involve the following activities:

- An short introduction/orientation to the laboratory
- Work stations
- Demonstration laboratory stations
- Interactive vLabs
- State-of-the-art techniques

Suggested Research Laboratory formats include:

- Introductory Mini Lecture on Research Topic + Orientation to the Lab activities and their relevance to the research (20-30 mins)
- 2-3 work stations with demonstration labs/teamwork/vLabs showcasing one or two key techniques and associated images that are open for interpretation by lab 'Teams' (60-90 mins)

Research Laboratory Reports (1000 words; suggested breakdown below):

- Introduction + Review + Relevant Article (250 words)
- Hypothesis (25-50 words)
- Aim (25-50 words)
- Materials and Methods (100 words)
- Results (300 words)
- Discussion (250 words)

PATH3205 Guide to the Practical Classes

The practical class component of the course is included to provide a link between your knowledge of the theory of the molecular basis of human disease and the actual laboratory techniques with which this these mechanisms are discovered and measured. The practical classes are not geared towards a hands-on "wet bench" approach. This has the disadvantage that you do not gain the opportunity to manipulate the apparatus itself in most cases. On the other hand it allows the course to focus on the key issues of experimental design and interpretation – important factors throughout science.

Despite the modern trend towards molecular diagnostics, the traditional approach of microscopic examination of diseased tissues (histopathology) remains a key diagnostic tool for many diseases. The final practical class is devoted to histopathology and will use this to illustrate the correlation between molecular changes and structural lesions in tissue specimens. Whilst one practical class is not sufficient to make you into diagnostic pathologists, it is hoped that this class will introduce you to the aesthetics and challenges of histopathology.

The content of the practical classes will be examined in the practical exam at the end of semester. In addition, some questions in the written exam may be based upon components of the practical classes.

Science Teaching Laboratory Student Risk Assessment	UNSW THE UNIVERSITY OF NEW SOUTH WALES	Pathology practicals in G6/G7 in Wallace Wurth for PATH3205 MBII, 2014 Date for review: 13/2/2015

Hazards	Risks	Controls
Physical Sharp plastic (pipette tips)	'Stabbing' wound of hand	• Wear personal protective equipment Dispose of in sharps bin provided • Do not eat, drink, apply cosmetics or smoke in the teaching laboratory • Wear personal protective equipment • Low concentrations of chemicals used • Wear personal protective equipment • Safety data sheets for the chemicals available in the laboratory
Biological Antibody	Inoculation/Irritant	
Chemical Acrylamide	Corrosive/Flammable	
Azide	Irritant/neurotoxic	
PBS	Irritant	

Pipetting ergonomics

Pipetting can cause aches and pains. Here are some handy hints:

- Adjust your chair or stool so that your elbow is at a 90° angle while pipetting.
- Adjust the height and position of sample holders, solution container, and waste receptacle so that they are all approximately the same.
- Try to work with your hands below shoulder height.
- Let go of the pipette from time to time and give the fingers/hand a break
- Do not twist or rotate your wrist while pipetting
- Use minimal pressure while pipetting
- Try to switch periodically between different types of work.

Personal Protective Equipment required

Emergency Procedures

In the event of an alarm sounding, stop the practical class The initial sound is advising you to prepare for evacuation and during this time start preparing to leave, remove your PPE, wash your hands and pack up your bags. The second sound gives instruction to leave. The Wallace Wurth assembly point is in the lawn in front of the Chancellery. In the event of an injury inform the demonstrator. First aiders and contact details are on display by the lifts. There is a first aid kit in the laboratory and the Wallace Wurth security office. Remove your personal protective equipment,

Clean up and waste disposal

- Remove your gloves and dispose in the biowaste bins provided.
- Dispose of all pipette tips in the bin provided.

Ethics Approval

This type of practical does not require ethics approval.

Declaration

Practical class 1 – Autoimmunity

Aim

This practical class aims to provide insight into the common laboratory tests used in the diagnosis of autoimmunity.

Learning objectives

At the completion of this practical class you should be able to:

1 Outline the use of anti-nuclear antibody staining in the diagnosis of autoimmune disease.

2 Discuss rheumatoid factor and immune complexes and relate these to the pathogenesis of autoimmune disease.

3 Discuss the pathogenesis of autoimmune disease caused by anti-receptor antibodies.

4 Describe the principles of indirect immunofluorescence and how these tests are used in the diagnosis of autoimmune disease.

Practical class 2 – Cardiovascular and Respiratory disease

Aim

This practical class aims to demonstrate structure-function correlation in ischaemic heart disease and asthma and to provide an opportunity to discuss diagnostic methods and aspects of their interpretation.

Learning objectives

At the completion of this practical class you should be able to:

1 Discuss the effects of atherosclerosis of the aorta and lower limbs.

2 Explain the relationship between coronary artery atherosclerosis and myocardial infarction.

3 Discuss the basis of laboratory tests for myocardial infarction.

4 Understand how the concepts of sensitivity and specificity apply to such tests.

5 Relate the clinical features of asthma to the morphological changes in the airway wall.

6 Explain how a volume-time curve provides information about airflow obstruction.

PATH3205 Museum Study Sessions

Museum Study Session 1 – Acute and chronic inflammation

Aim:

The aim of this session is to familiarise you with the macroscopic and microscopic appearances of acute and chronic inflammation.

Objectives:

By the completion of this study session you should be able to:

1. Differentiate the microscopic appearances of acute and chronic inflammatory cells.
2. Recognise the microscopic appearance of necrosis, pus, and granulation tissue and relate this to the underlying pathological processes.
3. Differentiate the macroscopic and microscopic appearances of meningitis and cerebral abscess and discuss the underlying pathology of both.
4. Compare and contrast the pathology of lobar pneumonia, bronchopneumonia and lung abscess.
5. Recognise the macroscopic and microscopic appearance of chronic suppurative inflammation.

Additional resources:

Museum specimens (and descriptions).

Images of disease CD-ROM (both macro and micro specimens)

Station 1: (Slide 28)

Virtual Slide 28 is an example of acute suppurative meningitis.

Q1: What is the predominant cell type in the subarachnoid space?

Q2: Suggest some other substances would you expect to find here and relate their presence to the process of acute inflammation.

Q3: What abnormalities would you expect to find if you did a lumbar puncture and removed some cerebrospinal fluid?

Review the macroscopic appearance of meningitis in specimen 2446.29. Correlate the macroscopic and microscopic appearances.

Station 2: (Slide 48)

Virtual Slide 48 is an example of a cerebral abscess.

Q1: What features of the organism may have lead to this lesion as opposed to the lesion in slide 28?

Q2: What has happened to the tissue in the centre of the lesion?

Review the macroscopic appearance of cerebral abscess in specimen 1262.29. Correlate the macroscopic and microscopic appearances.

Q3: Comparing slide 28 with slide 48, suggest what processes might have caused the patient's death in each case.

Station 3: (Slide 99)

Virtual Slide 99 is a section of lung. Identify the pleura. This is an example of organisation and granulation tissue.

Q1: What are the large red spaces in the granulation tissue? Why are these required?

Q2: What cell types would you expect to see in the granulation tissue?

Q3: Discuss the role of different cell types in the formation of granulation tissue.

Station 4:

Q1: Compare specimen 1048.10 (lobar pneumonia), specimen 604.10 (bronchopneumonia) and specimen 1242.10 (lung abscess). Try to suggest where the pathogens involved would fall along the following spectra:

Most tissue destructive — Least tissue destructive

Most localised — Leastlocalised

Station 5: (Slide 16)

Specimen 305.5 and Virtual Slide 16 demonstrate the macroscopic and microscopic appearances of skin with a pilonidal sinus. This is an example of inflammation together with healing and repair.

Q1: What sort of cells are within the centre of the lesion?

Q2: What sort of tissue is the wall of the lesion composed of?

Q3: Can you see any foreign material within the cavity? Explain how this may have led to the features referred to in Q1 and Q2 above.

Museum study session 2 – Cardio-respiratory disease

Aim:

The aim of this session is to familiarise you with the macroscopic appearances of cardiovascular disease.

Objectives:

By the completion of this study session you should be able to:

1. Recognise the macroscopic appearance of atherosclerosis and discuss the underlying pathology and complications.
2. Describe the macroscopic appearance of myocardial infarction, its complications and the process of repair in the heart.
3. Recognise the appearance of a cerebral infarct.
4. Discuss the consequences of chronic rheumatic heart disease on cardiac valves.
5. Recognise and discuss the pathological changes in the lung of a smoker.

Additional resources:

Museum specimens (and descriptions).
Images of disease CD-ROM

Station 1: Specimen 2718.12

This specimen contains the aorta which exhibits a common pathological condition.

Q1: What is the widespread change affecting the lining of the aorta?

Q2: What other process is beginning to occur in the small pocket?

Q3: Describe how (1) leads to (2).

Q4: What other complications of (1) may occur?

Station 2: Specimen 1587.11

This specimen contains a specimen of heart with thrombosis in a coronary artery.

Q1: How might this lesion have killed the patient?

Station 3: Specimen 734A.11

This contains a heart with a 6 week old myocardial infarct.

Q1: Identify the abnormal tissue and describe the abnormalities of the endocardium and pericardium in the specimen.

Q2: What processes have led to the changes in (1)?

Q3: What changes has the infarcted region undergone in the last 6weeks?

Station 4: Specimen 453.29

This specimen contains a brain with a region of cerebral infarction.

Q1: Explain why a recent myocardial infarct may have predisposed the patient to this lesion.

Q2: Some dark areas within the lesion may represent haemorrhage. Suggest why this may be important in deciding how to treat the patient.

Station 5: Specimen 1230.11

This specimen contains a heart in which the orifice of the mitral valve is almost completely closed.

Q1: Identify the abnormal valve and suggest what type of tissue is causing this abnormality.

Q2: Describe the process that led to formation of this tissue.

Q3: Suggest one haemodynamic and one infective complication of rheumatic heart disease involving the mitral valve.

Station 6: Specimen 1221.9

This specimen contains a specimen of lung showing changes due to smoking.

Q1: Recall the definition of emphysema. Can you see any such changes in the lung?

Q2: How might this interfere with lung function?

Q3: What are the black spots in the centre of the cut lung?

Catalogue of Museum Specimens

305.5 Pilonidal Sinus

Clinical History :

A 26 year old man developed a tender "lump" on his sacral region which bled occasionally. For over a year he suffered periodic episodes of swelling, terminating in the discharge of pus. A pilonidal sinus was excised.

Macroscopic Pathology :

The specimen is a large ellipse of skin which has been cut open down to the natal cleft. A collection of hairs can be seen in a cavity deep in the subcutaneous tissue. From this a sinus tract runs to a raised nodule on the surface.

Microscopic Pathology :

Chronic purulent inflammation in the subcutaneous tissue associated with pilonidal sinus.

453.29 Cerebral Infarction

Clinical History :

During her evening meal, a 78 year old woman suddenly developed dysarthria and hemiparesis. On admission she was conscious but confused. Examination revealed a left-sided hemiparesis and a left upper motor neurone facial palsy. Her eyes were deviated to the right, and she had dysarthria and dysphasia. The left Babinski response was positive. BP 180/100. ECG showed atrial fibrillation. She received physiotherapy and general supportive treatment, and showed signs of improvement, but then collapsed and died of pulmonary embolus.

Macroscopic Pathology :

The specimen consists of one half of a hemisected brain whose right cerebral hemisphere exhibits an extensive area of cerebral softening and scattered foci of haemorrhage.

604.10 Bronchopneumonia

Clinical History :

Between 3.5.65 and 17.2.68, this man was admitted to hospital on 11 occasions for ischaemic heart disease, cardiac failure and pulmonary oedema. On 13.3.68, he was admitted with pyrexia. Chest X-ray showed large areas of consolidation in the left mid-zone. He had a haemoptysis on the day before he died on 20.3.68.

Macroscopic Pathology :

This section of lung shows a number of areas of confluent bronchopneumonia and early abscess formation. Some thrombus is present in small vessels.

Microscopic Pathology :

Some areas show early bronchopneumonia with exudate and neutrophils in the alveolar spaces, while others exhibit early organisation and carnification, suggesting that the condition has been present for some time.

734A.11 Myocardial Infarct

Clinical History :

A 74 year old woman was well until 4 weeks before her death when she became progressively tired and breathless. Blood count showed mild anaemia, which was treated with vitamin B12 and iron. Five days before admission, she was commenced on digoxin. On the day of admission, she became acutely breathless, and despite vigorous treatment, she failed to respond, and died 2 days later.

Macroscopic Pathology :

The horizontal section of the heart shows dilatation of the left and right ventricles. There is infarction of the anterior part of the interventricular septum and a considerable portion of the left ventricle (transmural). There has been considerable resorption of necrotic muscle, with replacement by scar tissue and consequent thinning of the ventricular wall. This infarct could be aged approximately 6 weeks, macroscopically, and this is in keeping with the clinical history. Overlying the infarcted area, there is organising fibrinous pericarditis, and small thrombi are seen on the endocardial surface.

Microscopic Pathology :

Sections show myocardial infarction with replacement granulation and scar tissue.

1048.10 Lung : Lobar Pneumonia

Clinical History :

A woman aged 67 years who had suffered Parkinson's disease for 15 years, developed cough with yellow sputum for 3 days, and breathlessness for 2 days. She was admitted in a stuporose condition with rales and rhonchi present in all areas of her chest, and died the same day.

Macroscopic Pathology :

The specimen is a slice of the left lung. In the upper lobe there is an apical tuberculous scar, with smaller satellite tubercles in the adjacent tissue, indicating active disease. The lower lobe is uniformly consolidated (grey hepatization) by lobar pneumonia.

Microscopic Pathology :

The lower lobe exhibits lobar pneumonia (grey hepatization), the upper lobe, interstitial fibrosis and active pulmonary tuberculosis.

1221.9 Pulmonary Emphysema

Clinical History :

A man aged 70 years had smoked 40 cigarettes a day for many years. He presented a year before his death with a 2 month history of persistent cough with profuse purulent sputum. Bronchoscopy was highly suggestive of carcinoma of the upper lobe bronchus, but biopsy was inconclusive. He had a hilar mass which was considered inoperable, and a course of radiotherapy gave only temporary relief of his symptoms, which recurred a few weeks before his death, following massive haemoptysis. Autopsy showed carcinoma of the left lung, with multiple metastases.

Macroscopic Pathology :

No tumour is apparent in this specimen of lung, but there is generalized centrilobular emphysema involving particularly the upper lobe. Some inhaled blood clot is present in the bronchial tree.

Microscopic Pathology :

Sections of the upper lobe show bullous and centrilobular emphysema, with the bronchioles and alveoli often filled with blood.

1230.11 Heart : Mitral Stenosis

Clinical History :

Four years before death, a 59 year old woman first noticed palpitations, dyspnoea on exertion, engorgement of the neck veins and cyanosis of the lips and fingers, together with pain in the right hypochondrium, abdominal swelling and oedema of the ankles. She was treated for congestive cardiac failure, and was thought to have mitral stenosis and regurgitation, with auricular fibrillation and tricuspid incompetence. There was no past history of rheumatic fever. She was admitted to hospital unconscious, with intense cyanosis and ankle oedema, and died 2 days later.

Macroscopic Pathology :

The specimen is part of the heart. The right ventricle is dilated and hypertrophied, the left is small. The lumen of the mitral valve has been reduced to a slit about 4 mm long, at the tip of a funnel-like deformity.

1242.10 Lung : Abscesses

Clinical History :

A 70 year old woman presented with a history of dyspnoea on effort, chest pain and swelling of her ankles for many years. These symptoms had become worse, and she was now dyspnoeic at rest, had developed ulcers on her feet, and was almost completely incapacitated. On admission, she was very pale and breathless, with signs of congestive cardiac failure and auricular fibrillation. Iron deficiency anaemia was diagnosed and treatment was commenced, but she died suddenly about 4 weeks later. Autopsy revealed bronchopneumonia with multiple lung abscesses.

Macroscopic Pathology :

The specimen is part of the upper lobe of the right lung, in the centre of which is a collection of abscesses, the largest being 2.5 cm in diameter. Surrounding this area is a zone of bronchopneumonia.

Microscopic Pathology :

Sections of the lungs show abscesses of various ages, the oldest being about 2 -3 weeks. The pus contains clumps of Gram-negative organisms. The surrounding parenchyma shows features of bronchopneumonia.

1262.29 Cerebral Abscess

Clinical History :

This specimen was obtained at autopsy on a patient who had suffered from chronic otitis media and died of bacterial meningitis.

Macroscopic Pathology :

There is a large abscess in the left temporal lobe, the inferior surface of which has been cut away to expose the abscess cavity. The blood vessels of the pia arachnoid are engorged, and the meninges at the base of the brain are ragged and opaque, due to meningitis.

1587.11 Coronary Artery Thrombosis

Clinical History :

A 45 year old woman suffered polyuria and polydypsia, and had lost 12 kg weight in 2 years, in spite of a good appetite. During the 6 days prior to admission, she was faint, weak and vomiting. On examination, glycosuria was noted. Fasting blood sugar was 20 mmol/l; BP 150/100. She died suddenly 2 days later.

Macroscopic Pathology :

The heart shows fatty change of the myocardium. Two centimetres from its origin, the right coronary artery was thrombosed for a distance of 5 cm.

2446.29 Brain : Pneumococcal Meningitis

Clinical History :

A 34 year old female complained of aching pains, severe frontal headache and cough with a moderate amount of sputum for two weeks before the terminal illness. She recovered sufficiently to return to work, but on 2.9.38 she complained of a heavy feeling in her legs, and headache, which was becoming worse. On 5.9.38 she was drowsy, lapsed into unconsciousness, and was admitted to hospital. On examination, she was febrile, hypertonic and her eyes were deviated upward and to the left. She had neck and spine stiffness. CSF contained 225 polymorphs per cm. Gram positive cocci were seen in a direct smear, and the culture yielded Strep. pneumoniae. She died on 7.9.38.

Macroscopic Pathology :

The brain shows meningitis, the superior surface being bathed in pus. There was very little pus at the base of the brain. The blood vessels show intense congestion, and a few small haemorrhages are present. Pneumococcus was recovered from culture of the pus. The lungs and pleural cavities showed no signs of congestion or inflammation.

2718.12 Early Aortic Aneurysm

This specimen of distal aorta has been opened to show moderately severe atherosclerosis, and a small saccular aneurysm, approximately 30 mm in diameter, below the origin of the renal arteries.