



THE UNIVERSITY OF
NEW SOUTH WALES

School of Medical Sciences

PATH 3206

Cancer Pathology

Course convenor: Dr Christine van Vliet

SEMESTER I, YEAR III

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PATH3206 Cancer Pathology

2013

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PATH3206 Cancer Pathology

Integrated Timetable 2013

Week	Date	Time	Location	Lecturer	Title
2	11/03/2013	9	LG03	van Vliet	Lecture - Introduction & revision of neoplasia
		10	LG03	van Vliet	Lecture - Regulation of cell cycle
	11/03/2013	12-2	WW106/108	van Vliet	Practical - Neoplasia & regulation of cell cycle
3	18/03/2013	9	LG03	van Vliet	Lecture – Cancer pathology
		10	LG03	van Vliet	Lecture – Viral carcinogenesis
	18/03/2013	12-2	WW 106/108	van Vliet	Practical - Histopathology of neoplastic tissue
		12-2	WW 109/110	Kumar	Practical – Histopathology of neoplastic tissue
4	25/03/2013	9	See allocated	See allocated	Tutorial – Neoplasia
		10	LG03	Stewart	Lecture - Carcinogenesis
	25/03/2013	12-2	Museum	van Vliet	Practical - Poster Project
MID SESSION					BREAK
5	8/04/2013	9	See allocated	See allocated	Tutorial - Carcinogenesis
		10	LG03	Hawkins	Lecture - Colorectal Carcinogenesis I
	8/04/2013	12-2	WW 106/108	van Vliet	Practical - Colorectal carcinogenesis I
12-2		WW 109/110	Hawkins	Practical - Colorectal carcinogenesis I	
6	15/04/2013	9	see allocated	Dziegielewski	Lecture - Prostate carcinoma
		10	LG03	Hawkins	Lecture - Colorectal Carcinogenesis II
	15/04/2013	12-2	WW 106/108	van Vliet	Practical - Colorectal carcinogenesis II
		12-2	WW 109/110	Hawkins	Practical - Colorectal carcinogenesis II
7	22/04/2013	9	see allocated	See allocated	Tutorial - Colorectal carcinogenesis
		10	LG03	Velan	Lecture - Skin neoplasms
	22/04/2013	12-2	WW 106/108	van Vliet	Practical - Skin neoplasms
12-2		WW 109/110	Velan	Practical - Skin neoplasms	
8	29/04/2013	9	see allocated	See allocated	Tutorial - Skin neoplasm's
		10	LG03	van Vliet	Lecture – Cervical carcinogenesis
	29/04/2013	12-2	WW G2/G4	van Vliet	Practical – Cervical carcinogenesis
9	6/05/2013	9	LG03	Velan	Lecture - Intracranial neoplasms
		10	LG03	van Vliet	Lecture - Upper GI neoplasms
	6/05/2013	12-2	WW 106/108	van Vliet	Tutorial - Cervical carcinogenesis
		12-2	WW 106/108	van Vliet	MID SESSION EXAMINATION
10	13/05/2013	9	see allocated	See allocated	Tutorial - Upper GI neoplasms
		10	LG03	Kumar	Lecture – Pulmonary neoplasms
	13/05/2013	12-2	WW 106/108	Velan	Practical - Pulmonary neoplasms
12-2		WW 109/110	Kumar	Practical - Pulmonary neoplasms	
11	20/05/2013	9	see allocated	See allocated	Tutorial - Pulmonary neoplasms
		10	LG03	van Vliet	Lecture – Breast carcinoma
	20/05/2013	12-2	WW 106/108	van Vliet	Practical - Breast carcinoma
12-2		WW 109/110	Dziegielewski	Practical - Breast carcinoma	
12	27/05/2013	9	see allocated	See allocated	Tutorial - Breast carcinoma
		10	LG03	Lindeman	Lecture – Lymphoma & leukaemia
	27/05/2013	12-2	WW 106/108	See allocated	Poster presentations
12-2		WW 109/110	See allocated	Poster presentations	

13	3/06/2013	9	LG03	van Vliet	Lecture – Thyroid & pancreatic neoplasms
		10	LG03	van Vliet	Lecture – Feedback session
	3/06/2013	12-2	WW 106/108	van Vliet	PRACTICAL EXAMINATION
		12-2	WW 109/110	van Vliet	PRACTICAL EXAMINATION

Pathology Teaching Laboratory

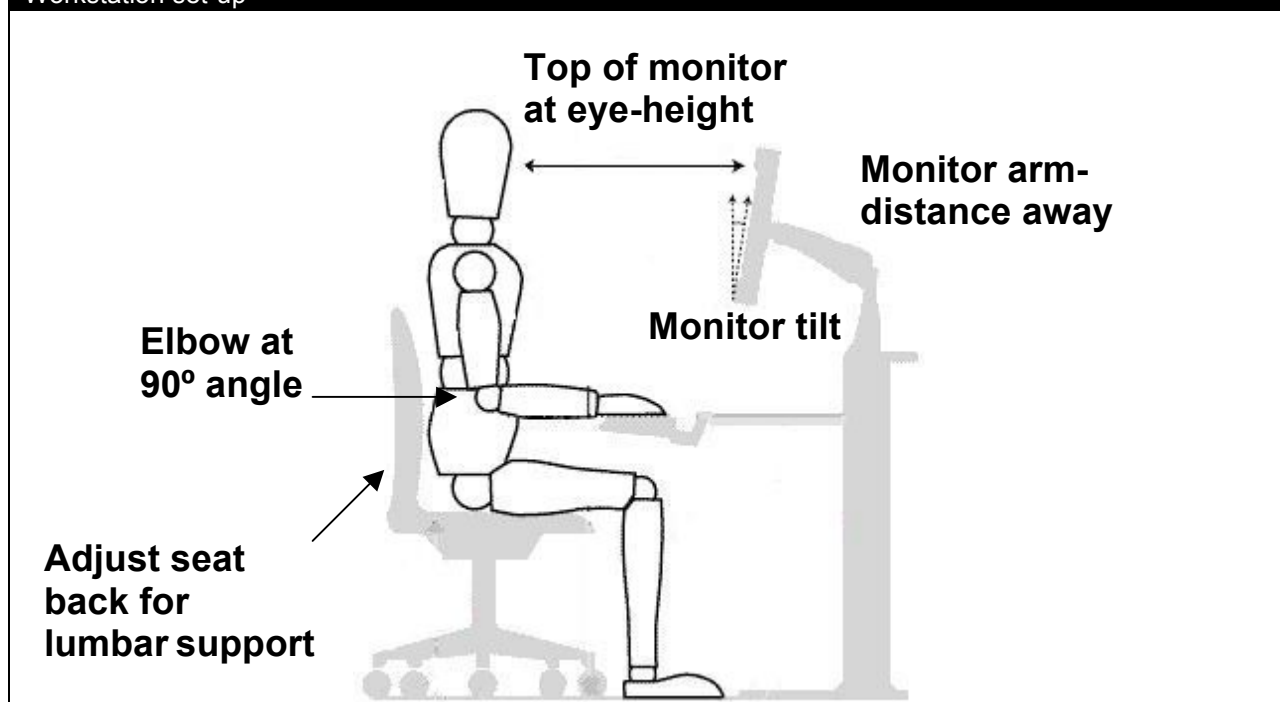
Student Risk Assessment


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 Pathology practicals in G2/G4
 & 106/108 & 109/110 in
 Wallace Wurth

Hazards	Risks	Controls
Ergonomics	Musculoskeletal pain.	Correct workstation set-up.
Electrical	Electrical shock/fire	Check electrical equipment in good condition before use. All portable electrical equipment tested and tagged.
Handling pots	Chemical spillage	Instructions on correct manual handling of pots

Workstation set-up



Manual handling of pots

- 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

1. It is recommended that all students wash their hands thoroughly as they leave practical class. Chemical residues may be present on pots.
2. **Carry one pot at a time.** Use two hands at ALL TIMES and support the base of pot.
3. **Avoid rough handling and/or tilting of pots.** This can cause leaking joints or tear tissue in specimen.
4. 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 a member of academic staff or the Museum Manager. A spill kit will then be used to absorb the fumes.

Personal Protective Equipment

Not necessary in these practicals.
Enclosed shoes must be worn to all Practicals.

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 and the Wallace Wurth security office.

Clean up and waste disposal

Spill kit

Declaration

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

Signature:.....Date:.....

Student Number:.....

Staff contacts in the Department of Pathology

Name	<i>Title</i>	<i>E-mail</i>
Dr Christine van Vliet	Lecturer and PATH3206 Convenor, Department of Pathology	C.vanVliet@unsw.edu.au Rm 1506 lvl 15 Mathews Bldg
A/Prof Gary Velan	Head Dept of Pathology	G.Velan@unsw.edu.au
Prof Nicholas Hawkins	Professor of Pathology and Head of School of Medical Sciences	N.Hawkins@unsw.edu.au
Prof Denis Wakefield	Professor, Department of Pathology	D.Wakefield@unsw.edu.au
Prof Rakesh Kumar	Professor, Department of Pathology	R.Kumar@unsw.edu.au
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Prof Carolyn Geczy	Professor, Department of Pathology	C.Geczy@unsw.edu.au
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Mr Thuan Thai	Lecturer, Department of Pathology	Thuan@unsw.edu.au
Dr Cristan Herbert	Lecturer, Department of Pathology	C.Herbert@unsw.edu.au

Technical and support staff

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: Room 355 3rd floor Wallace Wurth Building

Ms Chup 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 Carmen Robinson

Position: Student Advisor

Location: Room G27 Biosciences building

Ms Robinson is responsible for general administration and student support within the School of Medical Sciences.

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 if there are any broken or leaking specimens in the Museum.

Ms Bridget Murphy and Ms Ruth Miller

Position: Museum Education Officers

Location: Room G04 Ground Floor Samuels Building, Building F25

Ms Murphy and Ms Miller provides support for all undergraduate teaching programs, and assists in delivering an integrated learning program for senior high school students and community interest groups.

Mr Fergus Grieve

Position: SOMS Web, TELT and Information System Administrator

Location: Room 355 3rd floor Wallace Wurth Building

Mr Grieve maintains materials uploaded to *Blackboard*. Please contact Mr Grieve if you have any inquiries related to PATH3206 online resources, including lectures, assignments, timetables and communications.

PATH3206 Cancer Pathology

Introduction

Welcome to PATH3206 Cancer Pathology (previously Molecular Basis of Disease B).

PATH3206 aims to promote understanding of recent advances in the pathogenetic mechanisms underlying neoplasia. There is detailed discussion of molecular carcinogenesis, the metastatic process and techniques for diagnosis. Topics covered include neoplasia of the colon, breast, prostate, oesophagus, stomach, skin, lung cervix, thyroid and lymphoma and leukaemia.

To understand these processes, you will draw on your knowledge of normal anatomy, histology, biochemistry and physiology.

This course is offered during semester 1 and counts for six units of credit. PATH2201/2 (Processes in Disease) is a prerequisite for the course. This course complements PATH3208 Cancer sciences: Research Design, Measurement and Evaluation offered in semester 2.

The UNSW Handbook contains information for students wishing to undertake a major in Pathology.

For those wishing to pursue a career in research or hospital based laboratory work, 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 neoplasia.

The staff of the Department of Pathology join me in wishing you an interesting and enjoyable semester 1.
Dr Christine van Vliet (PATH3206 Convenor)

Course administration

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 Christine van Vliet (c.vanvliet@unsw.edu.au) by e-mail. Students wishing to see other members of staff should email and **make an appointment**. If students have difficulties of a personal nature, they should contact the School's Grievance Officer, Dr P. Pandey, or Prof Nick 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, 2013 will be held in the week commencing Monday 8th July, 2013. 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://medalsciences.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/>

Official communication by email

All students in course PATH3206 are advised that email is the official means 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). Students must use their official UNSW email address for all correspondence. 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.

Resources for students

Recommended text

You are expected to use the following text available online via the UNSW library Sirius website - <http://sirius.library.unsw.edu.au> (zID and zPass required). Search for the database MD Consult, then search for Robbins Basic Pathology.

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

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. V. Kumar, A.K. Abbas & N. Fausto (2010) Elsevier Saunders.

PATH 3206 Blackboard

Students enrolled in PATH3206 will be able to access the timetable, lecture notes and related information via [Blackboard](http://telt.unsw.edu.au): <http://telt.unsw.edu.au>

Images of disease (IOD) database

This database is a collection of images used for teaching within the Department. The latest version is available on online, optimized 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>. The following information might help you understand more about the 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

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 program that will run on every computer
Your computer must meet the minimal requirements or you will have trouble.
- 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.

Interactive images of disease

This is a collection of “hotspotted” images from the Department’s database on the Museum of Human Disease page. Images containing clickable “hotspots” allow identification of the normal features and pathological changes within each specimen. At present this is a limited selection, intended for the education of senior high school students and interested members of the public. Hence the accompanying clinical histories, descriptions and comments are written in plain English, with an emphasis on the prevention of these diseases.

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://web.med.unsw.edu.au/pathology/pathmus/>".

Additional learning resources

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:

Medline Plus ('health topics' index of disease with information)

<http://www.nlm.nih.gov/medlineplus/healthtopics.html>

University of Iowa (on-line histological slides on many of the topics covered)

http://www.path.uiowa.edu/virtualslidebox/nlm_histology/

http://www.path.uiowa.edu/virtualslidebox/iowa_histopathology/index.html

The Cancer Council New South Wales

<http://www.nswcc.org.au/>

The NSW Cancer Institute

<http://www.cancerinstitute.org.au/>

National Cancer Institute

<http://www.cancer.gov/>

Research opportunities

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

<http://medicallsciences.med.unsw.edu.au/somsweb.nsf/page/Research>

Student support services

Those students who have a disability that requires some adjustment in their teaching or learning environment are encouraged to discuss their study needs with the course convenor prior to, or at the commencement of, their course, or with the Equity Officer (Disability) in the Equity and Diversity Unit at <https://my.unsw.edu.au/student/atoz/Disability.html>. Issues to be discussed may include access to materials, note-takers, the provision of services and additional exam and assessment arrangements. Early notification is essential to enable any necessary adjustments to be made.

Course evaluation and development

Student evaluative feedback on the course is gathered each year using UNSW's Course and Teaching Evaluation and Improvement (CATEI) Process. Student feedback is taken seriously, and continual improvements are made to the course based in part on such feedback.

Student learning outcomes and graduate attributes

For the cancer topics covered:

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

1. Describe and explain the molecular and cellular pathogenetic mechanisms;
2. Describe the macroscopic and microscopic appearances;
3. Correlate the clinical features with the underlying pathogenetic mechanisms;
4. Discuss recent advances in knowledge pertaining to the molecular pathogenesis;
5. Develop written and oral skills in scientific communication.
6. Develop skills in collaborative teamwork

You are encouraged to develop the following Graduate Attributes by undertaking the learning activities in this course. These attributes will be assessed within the prescribed assessment tasks (see Assessment):

1. An in-depth engagement with the relevant disciplinary knowledge in its interdisciplinary context.
2. The capacity for analytical and critical thinking and for creative problem-solving.
3. The ability to engage in independent and reflective learning.
4. The skills required for collaborative and multidisciplinary work

Learning and Teaching approach.

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.
- 2) A series of **lectures** introduce you to pathological processes, as well as specific examples of those processes affecting organs and tissues;
- 3) **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 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) Learning is supported via **Blackboard**. Announcements, timetables, lecture slides and other resources will be made available during the course.

Prize

A prize will be awarded for Cancer Pathology:

1. Best team performance in tutorial quizzes (based on both team and individual scores)

Assessment

Students will undertake multiple forms of assessment during semester:

- Online progress assessment 5%
- Individual and team performance in tutorial quizzes 15%
- Mid-session examination (objective items + short answer) 10%
- Team project: poster and oral defence 20%
 - *Team member peer evaluation* 5%
 - *Academic staff evaluation* 15%
- Practical examination 10%
- Final examination (short answers) 40%

Team project: Poster and oral defence

Each team will be given a set of specimens, which illustrate pathological changes which may occur as a result of a neoplasm or set of predisposing factors.

The students are to **create a poster which:**

1. Briefly describes the macroscopic specimens
2. Describes how the specimens are linked e.g. all specimens may be related to the same cause or the specimens may be complications of a primary condition
3. Explains the underlying pathobiological mechanisms of the disease(s) present
4. Relates the pathobiological mechanisms to the clinical manifestations

Particular emphasis on explaining the pathobiological mechanisms should be made. Students should read their Robbins textbook and journal review articles.

Each group will have 10 minutes to present an **oral defence** of their poster. The spokesperson for the group (nominated by the students themselves) should deliver an overview of the poster in the first 2-3 minutes and in the remaining time all members of each group must 'defend' their poster to a Department of Pathology staff member.

The aim of the group project is to provide an in-depth understanding of the pathobiological mechanisms of individual neoplasms. The project will encourage students to think critically and engage in problem solving in order to determine the interrelatedness of pathological specimens. The presentation and oral defence will enhance students' skills in effective communication and teamwork.

SEMESTER I

- Week 4: Students allocated into groups of five. The specimens for the group project will be allocated to each group during the practical class.
- Week 11: **Group poster due electronically no later than 5pm Monday 20/5/2013.** Posters must be submitted electronically as a PowerPoint slide, using the poster submission icon on the PATH3206 Blackboard website. In addition the text of the posters must be submitted as a separate, fully referenced Word document, using the Turnitin icon on the PATH3206 Blackboard website, no later than 5pm Monday 20/5/2013, (see Submission of Team project).
- Week 12: Team poster presentation and oral defence semester.

Assessment criteria

Team member peer evaluation

Each student in the Team will complete an online evaluation form for each member of their Team. The student's peer evaluation will be marked out of 5 and will contribute 5% of the final course mark. The mark will be an average of all the Team members' assessments of the student. The Team member peer evaluation form is available for completion on the PATH3206 Blackboard website.

Team member peer evaluation form

Student name and student ID:

Team number:

Assessor's name and student ID:

Place a cross in the appropriate mark box for each of the five criteria listed. Total the score at the bottom of the table. Please justify your marks in the comments section.

	0	0.5	1.0
1. Participation in the planning of the presentation			
2. Execution of allocated tasks effectively and on time			
3. Attendance to meetings called on by Team members			
4. Contribution to Team discussion			
5. Scientific quality of contribution			

TOTAL: /5

Comments:

Team poster and oral defence evaluation

Teams will be marked on their presentations by a staff member from the Department of Pathology according to the following criteria:

- 1) The Team gives a macroscopic description of the specimens and demonstrates an understanding of the interrelatedness of the specimens.
- 2) The Team demonstrates an understanding of the underlying pathobiological mechanisms leading to the disease(s) present in the specimens and relates these to the clinical manifestations.
- 3) The Team demonstrates an ability to utilize the current medical literature to support their arguments.
- 4) The poster shows a high standard of design and effectively communicates key concepts to the audience.
- 5) Team members answer questions clearly and directly.

The presentation will be marked out of 15 and will contribute 15% of the final mark for the course. For **each** of the above objectives, marks will be distributed as follows:

- | | |
|--|---|
| • Did not address the objective | 0 |
| • Attempted to address the objective but did not achieve satisfactory standard | 1 |
| • Satisfactorily addressed the objective | 2 |
| • Addressed the objective well | 3 |

Submission of Team project

Posters must be submitted electronically as a PowerPoint slide, using the poster submission icon on the PATH3206 Blackboard website **no later than 5pm Monday 20/5/2013**.

In addition the text of the posters must be submitted as a separate fully referenced Word document, using the Turnitin icon on the PATH3206 Blackboard website **no later than 5pm Monday 20/5/2013**. Figures, diagrams and tables used in the poster must also be referenced in the Word document. All posters will be assessed for plagiarism by use of Turnitin software. Please use the American Psychological Association (APA) referencing style (see http://info.library.unsw.edu.au/biomed/skills/direct/Info_Skills_Docs/apa/apa1.htm).

IMPORTANT: The PowerPoint slide and word document must have PATH3206 and the Team number in the file name, e.g. PATH3206_Team1.ppt and PATH3206_Team1.doc

Late Team projects

Students will be penalised 5% of the mark for each day the poster is late. **Posters submitted later than 5pm Friday 24/5/2013 will receive a zero grade.**

Academic honesty and 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://my.unsw.edu.au/student/academiclife/assessment/AcademicMisconduct.html>

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.”

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.

Team and individual quizzes (TIQ)

There will be quizzes held in the tutorial sessions consisting of MCQs. Some tutorial quizzes will be undertaken by the individual student and then by the team, others just individually. Pre-reading for the quizzes is specified in the tutorial outlines of the manual. Students need to provide a reason to Dr van Vliet for a missed tutorial via email. If the reason is approved then the student will receive the average of their team's individual quiz mark and the team mark. If the reason is not approved the student will receive zero for both the individual and team quiz however the team will not be penalised.

Online progress assessment

An **online progress assessment** (5% of the final mark) consisting of MCQs will be provided. This assessment is to be completed during the 10 days in which it is available (**an announcement will be made on Blackboard**). This assessment encourages independent and reflective learning as the student may attempt the assessment as often as they wish, within the time allowed, until they receive a satisfactory score (>90%). Students will receive 5% of the final mark for satisfactory completion of the assessment.

Mid-session examination

A **mid-session exam in Week 9** (10% of the final mark) consisting of MCQs and short answer questions, will be conducted. The examination will include material covered in Weeks 1-8 of PATH3206. The skills achieved by mastering the tutorial quizzes will be assessed in this exam. The short answer questions are preparation for the end of course exam.

Practical examination

A **practical examination in Week 13** (10% of the final mark), will be conducted. This will consist of a series of stations each with questions based on material presented during the practical sessions and lectures.

Final written examination

A **2-hour end of course examination** (40% of the final mark) which will comprise **four** short-answer / essay style questions. The questions assess all the learning outcomes. This exam encourages an in-depth engagement with pathology within a clinical context. The questions vary in style; some questions may have two parts.

Missed exams

If in any circumstances you unavoidably miss an examination, you must inform the Registrar and also contact the relevant Course Office immediately. Normally, if you miss an exam (without medical reason) you will be given an absent fail. If you arrive late for an exam no time extension will be granted. It is your responsibility to check timetable and ensure that you arrive with sufficient time.

Supplementary examination

A supplementary examination may be awarded at the discretion of the Department of Pathology to students who have provided evidence for special consideration according to the UNSW guidelines. The deferred exam may include a significant oral element. Students who believe that they are eligible for further assessment must contact Dr van Vliet to seek further information. It is intended that supplementary exams for the School of Medical Sciences in Semester 1, 2013 will be held in the week commencing Monday 8th July, 2013.

Medical certificates

If you miss any examination for medical reasons you must lodge a medical certificate with New South Q within **3 DAYS** (refer to UNSW Student Gateway@ www.student.unsw.edu.au for further details). **Special considerations sought outside the 3 day time period WILL NOT be accepted except in TRULY exceptional circumstances.**

Attendance requirements

Attendance at tutorials and practical sessions is compulsory. An 80% attendance is required for you to be eligible to sit the final examination. Students need to provide a reason to Dr van Vliet for a missed tutorial via email. If the reason is approved then the student will receive the average of their team's individual quiz mark and the team mark. If the reason is not approved the student will receive zero for both the individual and team quiz however the team will not be penalised.

Sample examination paper

THE UNIVERSITY OF NEW SOUTH WALES
EXAMINATION

PATH 3206
CANCER PATHOLOGY

TIME ALLOWED – **2 HOURS**

TOTAL NUMBER OF QUESTIONS - **4**

ANSWER ALL QUESTIONS. ALL QUESTIONS ARE OF EQUAL VALUE

THIS PAPER MAY NOT BE RETAINED BY THE CANDIDATE.

NO HANDWRITTEN OR TYPED NOTES OR TEXTS MAY BE BROUGHT INTO THE EXAMINATION ROOM.

ANSWER EACH QUESTION IN A SEPARATE BOOK. ALL ANSWERS MUST BE WRITTEN IN INK. PENCILS MAY ONLY BE USED FOR DRAWING.

Question 1

- (a) Write notes on factors which can help determine the prognosis of a woman with carcinoma of the breast
- (b) Compare and contrast the predisposing factors, clinical features and biological behaviours of melanoma and basal cell carcinoma of the skin

Question 2

- (a) Discuss the clinical consequences of colorectal neoplasia, including the effects of benign colorectal neoplasms.
- (b) Discuss genetic changes that characterise development and progression of colorectal neoplasms. Highlight the ways in which understanding of hereditary bowel cancer syndromes has helped to explain the different genetic pathways involved in sporadic colorectal cancers.

Question 3

- (a) Write notes on **one** of the following:
 - (i) Role of oncogenes and apoptosis-related genes in the development of cancer
 - or*
 - (ii) Role of viruses in carcinogenesis
- (b) Describe the macroscopic features that may allow differentiation between benign and malignant neoplasms.

Question 4

A 38 year old woman presented to her local doctor with a 2 month history of bleeding after intercourse. More recently she had a spontaneous bloodstained discharge. After a series of investigations the woman underwent a hysterectomy.

- i) What is the likely diagnosis? How could this have been confirmed preoperatively?

- ii) Discuss the pathogenesis of the disease listed in part i. How might his disease have been prevented?

- iii) If this woman had not undergone treatment how might have her disease progressed?

The Museum of Human Disease

The Donald Wilhelm Museum of Human Disease is located on the ground floor of the Samuels Building (Building F25). Originally located on the 5th floor of the Wallace Wurth 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 until 2004), 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 pots!** 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 during weekdays from 8 a.m. to approximately 8 p.m. The Museum is security locked, and can only be entered by using your student card to enable the doors to be opened.

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.
- The specimens are preserved in Perspex and contain a range of preserving chemicals that may be harmful. Chemicals used may include formalin, pyridine and sodium dithionate. A full list of chemicals and associated information is available at the Health and Safety (H&S) station in the Museum 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.
- For more information on matters related to health and safety policies of UNSW visit this web site.
<http://www.ohs.unsw.edu.au/>

Lecture, practical and tutorial outlines

Neoplasia

Teaching provided

Lecture: Introduction and revision of neoplasia

Tutorial: Neoplasia

Practical: Neoplasia and regulation of the cell cycle

Practical: Histopathology of neoplastic tissue

Aim

These practicals and the associated lecture are designed to revise the topics as listed below:

- Epidemiology and classification of tumours
- Modes of tumour spread
- Clinical effects of tumours

Learning outcomes

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

1. Define the terms “metaplasia”, “neoplasia”, “anaplasia”, “dysplasia”, “carcinoma”, “sarcoma”, “benign”, “malignant”, “metastatic”.
2. Discuss the relative frequencies of common human tumours.
3. Explain the basis of the nomenclature used in the classification of neoplasms.
4. Recognise and describe the macroscopic features that may allow differentiation between benign and malignant neoplasms.
5. Recognise and describe common macroscopic growth patterns of neoplasms.
6. Discuss in general terms the methods available for the diagnosis of neoplasia.
7. Discuss the grading and staging of cancer

Pre-reading for tutorial quiz

1. Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 5 pp. 161-172, 207-213.

Practical: Neoplasia and regulation of the cell cycle

See handout.

Practical: Histopathology of neoplastic tissue

A. Case study (Virtual slides can be found at: <http://vslides.unsw.edu.au/>)

A 65 year old man presented to his local doctor complaining of worsening cough for the past 6 weeks. The previous day, he had coughed up a small amount of blood. On further questioning, he also gave a history of increasing shortness of breath on exertion for a few weeks. He had smoked a packet of cigarettes per day for over 40 years.

1. What diagnostic possibilities do you think would have been considered at the initial clinical presentation?

2. What investigations would have been useful in attempting to establish a diagnosis?

The patient subsequently underwent surgery. Virtual slide 69 was prepared from tissues removed at operation.
(Virtual slides can be found at: <http://vslides.unsw.edu.au/>)

3. What is your diagnosis? How do the microscopic findings help to explain the patient's clinical manifestations?

4. Why is the tumour of this particular histopathological type? Does the type of tumour matter?

5. Examine virtual slide 32. Could this specimen have been from this patient? Why or why not?

B. Additional slides

Your tutor will project and discuss virtual slides 56, 85 and other relevant examples as part of this class.

Carcinogenesis

Teaching provided

Lecture: Regulation of the cell cycle
 Practical: Neoplasia and regulation of the cell cycle
 Lecture: Cancer Pathology
 Lecture: Carcinogenesis
 Lecture: Viral carcinogenesis
 Tutorial: Carcinogenesis

Aim

The aim of these lectures and the associated practical and tutorial is to review current concepts of carcinogenesis, including the molecular basis of cell growth and neoplastic transformation.

Learning outcomes

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

1. Define the terms “carcinogen”, “proto-oncogene”, “oncogene”, “tumour suppressor gene”, “apoptosis-related genes”, “mismatch repair (MMR) genes”, “microsatellite”, “microsatellite instability (MSI)”.
2. Demonstrate a basic understanding of the molecular basis of cell growth and its regulation.
3. Demonstrate an understanding of oncogenes, tumour suppressor genes, apoptosis-related genes and mismatch repair genes and their roles in carcinogenesis. Give examples of each.
4. List important carcinogenic agents, including viruses, and discuss their proposed mechanisms of action.
5. Describe the multistep theory of carcinogenesis, utilising examples based on common human tumours.

TUTORIAL

Pre-reading for tutorial quiz

National Cancer Institute Understanding cancer series: Cancer Genomics
<http://www.cancer.gov/cancertopics/understandingcancer/cancergenomics>

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 5 pp. 177-188.

Recommended reading

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 5 pp. 173-207.

Past exam questions: Your Team should work together to develop answers to these questions

- Role of oncogenes and apoptosis-related genes in the development of cancer (2004)
- Role of viruses in carcinogenesis (2006)
- Discuss the role of tumour suppressor genes and apoptosis-related genes in the development of neoplasms.
- Discuss the role of oncogenes and tumour suppressor genes in the development of neoplasms. Illustrate your answer with examples from common human malignancies. (2007)

Practical: Poster project and teamwork

Students will be allocated to their team and given their specimens for their poster project.

Prostate carcinoma

Teaching provided

Lecture: Prostate carcinoma

Practical: Breast and prostate carcinoma

Aim

The aim of this lecture and associated practical and tutorial is to facilitate a basic understanding of prostate carcinoma

Learning outcomes

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

1. List the known risk factors for the development of prostate carcinoma.
2. Describe and contrast the clinical manifestations of prostate carcinoma and hyperplasia.
3. Outline procedures used commonly in the diagnosis of prostate cancer, and discuss the role of screening in the early diagnosis, in particular the use of prostate specific antigen (PSA) testing as a screening tool for prostate carcinoma.
4. Discuss factors that influence prognosis in prostate carcinoma, including responsiveness to hormonal manipulations.
5. Describe the modes of progression of prostate carcinoma, and recognise these appearances in macroscopic specimens of each disease.
6. Discuss the causes, consequences and investigation of skeletal metastases.

Recommended reading

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 17 pp. 663-668.

Additional resources

1. Smith DP, Supramaniam R, Marshall VR and Armstrong BK. Prostate cancer and prostate-specific antigen testing in NSW. *MJA* 2008; 189(6): 315-318.
(This is a useful article looking at the pros and cons of PSA as a screening tool.)
2. Lam Q.T. and Frydenberg M. *Strategies for Detecting Prostate Cancer*. March 2009 Common Sense Pathology cancer.
<http://www.rcpa.edu.au/static/File/Asset%20library/public%20documents/Publications/Common%20Sense%20Pathology/Prostate%20Cancer%20-%20March%202009%20edition.pdf>
Linked from <http://www.rcpa.edu.au/Publications/CommonSensePathology.htm>

Past exam questions: Your Team should work together to develop answers to these questions

- Factors which can help determine the prognosis of a man with carcinoma of the prostate (2004)

- A 72 year old man presented to the Emergency department with recent onset of a burning sensation while passing urine. This was associated with fever and loin pain. For the past 6 months he had suffered from poor urinary stream and in the past fortnight he had also developed increasingly severe lower back pain. Multiple radio-opaque lesions were present on x-ray of the lumbar spine.
 - i Which underlying disease is the most likely cause of the longer term urinary symptoms? What complications have ensued? What would be the probable finding on physical examination? What further investigations would be useful and what would be their likely results?
 - ii Discuss the pathogenesis of the underlying long-standing disease in part (i)?
 - iii Explain the pathophysiological mechanism underlying the clinical features and radiological finding in this case.

Colorectal carcinoma

Teaching provided

Lecture: Colorectal carcinogenesis I
Practical: Colorectal carcinogenesis I
Lecture: Colorectal carcinogenesis II
Practical: Colorectal carcinogenesis II
Tutorial: Colorectal carcinogenesis

Aim

The aim of these lectures and associated practicals and tutorial is to facilitate an understanding of this common malignancy.

Learning outcomes

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

1. List the factors and conditions known to predispose to the development of colorectal carcinoma.
2. Discuss the development of colorectal neoplasia as an example of the multistep theory of carcinogenesis.
3. Compare and contrast the “classical” (or chromosomal instability, CIN) pathway and microsatellite instability (MIN) pathway for colorectal carcinogenesis
4. Describe the common clinical manifestations of colorectal carcinoma and relate the manifestations to the macroscopic appearance of each disease and the site (i.e. left vs. right sided lesions).
5. List the investigations used commonly in the diagnosis of this malignancy.
6. Describe the common modes of spread of colorectal carcinoma, and relate the stage of each disease to its prognosis.

TUTORIAL

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 14 pp. 592-600.

Additional resources

1. Cancer Council Australia. *Guidelines for the prevention, early detection, and management of colorectal cancer: a guide for GPs*, 3rd edition (updated July 2008).
<http://www.cancer.org.au/Healthprofessionals/clinicalguidelines/colorectalcancer.htm>

Past exam questions: Your Team should work together to develop answers to these questions

- Discuss the clinical consequences of colorectal neoplasia, including the effects of benign colorectal neoplasms. (2001)
- Discuss the genetic changes that characterise development and progression of colorectal neoplasms. Highlight the ways in which the understanding of hereditary bowel cancer syndromes has helped to explain the different genetic pathways involved in sporadic colorectal cancer (2001)
- A 63 year old woman presented to her local doctor with a history of alternating constipation and diarrhoea for six months, assoc with a feeling of incomplete evacuation following defaecation and several episodes of bright blood coating her bowel motion. A diagnosis was made and a portion of her left colon and rectum was removed surgically. (2004)
 - Discuss the pathogenesis of the disease and what you consider the most likely cause of the presenting signs and symptoms (2004)
 - If the lesion had not been excised what complications might have ensued? (2004)
- Discuss the role of APC proteins and neoplastic transformation and progression (2005)
- Discuss factors which can determine the prognosis of a person with colorectal ca. (2006)
- Approximately 10-15% of colorectal carcinomas are associated with microsatellite instability. What features distinguish these cancers? How can the presence of microsatellite instability be diagnosed? (2007)

Practical: Colorectal carcinogenesis I

Diagnosis and treatment of colorectal cancer

Practical class aims

- To consider approaches to screening and diagnosis of colorectal carcinoma
- To revise the histological features of colorectal cancer
- To understand the role of pathology and imaging in the staging of colorectal cancer, and to recognise the relationship between stage and treatment.

Task 1: Teamwork

<p>Table 1 Screening</p>	<p>What is a Faecal Occult Blood test?</p> <p>How does the test work?</p> <p>Can it give a false positive result?</p> <p>Can it give a false negative result?</p>
<p>Table 2 Diagnosis</p>	<p>What is a colonoscopy, and how is it performed?</p> <p>Is a colonoscopy dangerous?</p> <p>What is a virtual colonoscopy?</p> <p>What is a capsule endoscopy, and can that be used to diagnose bowel cancer?</p> <p>How is a biopsy taken of a bowel tumour, and is it dangerous?</p> <p>What information does the pathologist provide to the surgeon regarding the tumour?</p>

Clinical History

A previously healthy 62 year old man presented following a single episode of bright red bleeding per rectum. Over the preceding month he had noted increasing constipation. He had not had any other problems with his health.

Task 2

1. Based on the history and clinical examination outlined above what are the possible causes of this problem (differential diagnosis), and what is the most likely diagnosis (provisional diagnosis)?

2. What investigations, if any, would you undertake to reach a definitive diagnosis?

Task 3

In due course, the man was referred to a colorectal surgeon who performed a sigmoid colectomy (removal of a portion of colon). A section of the tissue removed at surgery is shown on virtual slide 3. Virtual slides can be found at: <http://vslides.unsw.edu.au/>

Examine the virtual slide, and try to arrive at a diagnosis. To do this properly, you will have to answer each of the following questions

1. Is this a neoplasm? To answer this question you will have to decide

1A. Is there an abnormal mass of cells?

1A (i). Can you find a segment of normal bowel wall (mucosa, submucosa, muscularis propria, serosa) on the slide? If not, look up your histo texts! Can you see normal colonic epithelial cells in the mucosa – you will need these to answer question 1B?

1A (ii). Can you identify a mass of cells (bluish) that are abnormal here? Hope so. If you can, go to 1B. If not, ask your mother.

1B. Are the cells that make up this mass abnormal themselves? To answer this you need to compare the cells of the mass with normal cells. When you can compare with normal, decide;

1B (i). Is there increased variation (pleomorphism) in the size and shape of cells and their nuclei in the abnormal bits?

1B (ii). Is the nuclear to cytoplasmic ratio of these cells decreased?

1B (iii). Is the chromatin pattern different (more dark or coarse) within the nucleus? Don't know what chromatin is – look it up!

1B (iv). Do the cells have prominent nucleoli? Don't know what a nucleolus is – look it up!

1B (v). Do the cells have irregular nuclear membranes (with dents, folds, notches etc)?

2. What pattern of differentiation are the abnormal cells showing?

2A. Are they showing epithelial differentiation?

2B. If they are epithelial, is the pattern of differentiation squamous, neuroendocrine, secretory, transitional or what?

3. Is there evidence of spread of the abnormal cells beyond their normal anatomical location?

Based on your answers above, state your diagnosis

How would you stage this tumour, and what further information is required for accurate staging?

Task 4

The final task involves staging of bowel cancer, and the importance of this process not only to prognosis, but treatment as well. First, a few notes on how bowel cancer is currently treated in Australia.

Surgery: Bowel cancer is nearly always treated by surgical removal (*resection*) of the tumour. This is done usually a few weeks after a definitive diagnosis of cancer is made by biopsy of a suspicious lesion. To resect a carcinoma, the surgeon will usually remove a segment of bowel containing the tumour, as well as the mesentery containing the blood vessels, lymphatics and lymph nodes related to that part of the colon. As well as determining how far through the bowel wall a cancer has spread, the pathologist will also examine the lymph nodes to see if any are involved, and the edges where the surgeon has cut (resection margins) to determine if all the cancer has been removed. Generally in bowel cancer most cancer can be removed. The main exceptions are when the tumour is very low in the rectum and the surgeon tries to preserve the anal sphincter, or when tumour has spread through the bowel wall and is adherent to other adjacent structures (bladder, uterus, vagina, pelvic wall, small bowel etc). Fortunately these events are fairly uncommon.

Chemotherapy: Anticancer drugs have two roles in bowel cancer management. The first is where the tumour appears to have been removed by the surgeon, and no metastases are seen, but it has spread to the lymph nodes that have been removed at operation (Stage III). In this case, drugs are given to kill any tumour cells that may have spread further in the body. This is called *adjuvant chemotherapy*. For every 100 or so people with stage III bowel cancer, about 50 will die because their disease comes back (tumour recurrence). If all these people were given adjuvant chemotherapy, only about 35-40 would die. The second reason drugs are given is to relieve symptoms in people with metastatic disease. This is called *palliative chemotherapy*. It is not a cure, and so is only given if the person is unwell or symptomatic because of their disease. Palliative chemotherapy may prolong the life of someone with metastatic bowel cancer for several years.

Radiotherapy: Radiotherapy is used less commonly in bowel cancer. It is a focal form of treatment – you cannot irradiate the whole body or the person will die. Its main role is to control local disease, and in this regard it is often used with surgery for rectal cancers. It is also increasingly being used to shrink tumours prior to surgery (neoadjuvant radiotherapy) to make it easier for the surgeon to remove the tumour. Radiotherapy can also be used for isolated metastases, particularly in bone.

With that in mind, and having revised the TNM staging of bowel cancer, consider the following cases of colorectal neoplasia. For each case, indicate the stage of the cancer (using TNM terminology, and the nature of any further treatment that may be required (if any).

Case	Patient and site	Tumour details	Involved nodes	Distant mets	Indicate stage and further therapy.
1	77 female Caecum	35 x 35 x 20 mm Extends beyond outer aspect of muscularis propria but does not involve serosa	0 of 35	None seen	
2	68 male Sigmoid colon	22 x 19 x 12 mm Extends to involve serosal surface	4 of 19	None seen	
3	73 male Rectum	10 x 5 x 5 mm. Does not invade muscularis mucosae	0 of 15	None seen	
4	68 male Sigmoid colon	18 x 14 x 7 mm Involves submucosa but does not invade muscularis propria	2 of 7	None seen	

Case	Patient and site	Tumour details	Involved nodes	Distant mets	Indicate stage and further therapy.
5	43 female Transverse colon	30 x 20 x 20 mm Extends through muscularis propria and involves serosal surface.	8 of 10	Multiple hepatic mets	
6	82 male Ascending colon	18 x 15 x 5 mm Invades to within 2 mm of outer aspect of muscularis propria	0 of 14	None seen	
7	56 female Transverse colon	50 x 40 x 20 mm Extends through muscularis propria but does not involve serosa	4 of 17	Single hepatic met	

Practical: Colorectal carcinogenesis II

Pathways of colorectal carcinogenesis

Aim

To highlight the differences in the two common pathways of colorectal carcinogenesis, namely the “classical pathway” and the “microsatellite instability pathway”.

Resources

This powerpoint slide provides two clinical histories, as well as typical results of investigations relevant to the case, including histology, immunohistochemistry and microsatellite analysis of the tumours from both cases.

Learning outcomes

- State in simple terms the molecular basis of the detection of MSI
- State the basic principles of immunohistochemistry
- In your Teams, **match the various test results with the two histories (available on Blackboard)**. In doing the final task, you should be able to:
 - Identify the similarities and differences in the macroscopic appearance of colorectal carcinomas arising from the different pathways
 - Identify the similarities and differences in the macroscopic appearance of colorectal carcinomas arising from the different pathways
 - Discuss ways in which microsatellite instability may develop in colorectal cancer
 - Interpret staining for MLH1 and p53 in tissue sections

Case History A1

A 29 year old man presented with bleeding per rectum, that had been present for the past four weeks. He also complained of some constipation for the past 3 months, that had been intermixed with occasional episodes of diarrhea. He had not suffered any weight loss, and was otherwise in good health.

He had no other significant health problems. He stated that he had a history of bowel cancer in the family, with his father and his grandfather both dying from the disease at a relatively early age.

Case History A2

A 78 year old woman presented with a vague history of increasing tiredness. She stated that she had felt run down for the past three months, and lately had become increasingly short of breath when she went to the local shops – so much so that her daughter now had to do the shopping.

Because she looked pale, her doctor ordered a full blood count, which showed anaemia. Subsequently, she underwent a colonoscopy that showed a large polypoid carcinoma in the caecum. It was resected and she made an uneventful recovery.

Haematoxylin and Eosin stained tumour sections (available on <http://vslides.unsw.edu.au/>)

- #H1 (4 images from one slide)
- #H2 (4 images from one slide)

P53 immunohistochemistry of tumour (available on <http://vslides.unsw.edu.au/>)

- #P1 (4 images from one slide)
- #P2 (4 images from one slide)

MLH1 immunohistochemistry of tumour (available on <http://vslides.unsw.edu.au/>)

- #M1 (5 images from one slide)
- #M2 (5 images from one slide)

Results of microsatellite analysis (available on <http://vslides.unsw.edu.au/>)

(printout showing BAT25, BAT26 and BAT40 loci for tumour and normal tissue)

- #X1 (one page)
- #X2 (one page)

Skin neoplasms

Teaching provided

Lecture: Skin neoplasms

Aim

The aim of this lecture is to provide students with an overview of the epidemiology, prevention, pathogenesis, clinical features and biological behaviour of common skin neoplasms. .

Learning outcomes

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

1. Define the terms “UVA”, “UVB”, “hyperkeratosis”, "solar elastosis".
2. Describe the effects on skin of prolonged UV exposure.
3. Describe the epidemiology and risk factors for the development of common skin tumours, as well as preventative measures.
4. Recognise and describe common skin tumours, including melanoma, basal cell carcinoma and squamous cell carcinoma.
5. Describe the modes of progression of each of these tumours.
6. With respect to melanoma, describe factors that may be used to predict prognosis.

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 23 pp. 862-869.

Past exam questions: Your Team should work together to develop answers to these questions

- Compare and contrast the predisposing factors, clinical features and biological behaviours of melanoma and basal cell carcinoma of the skin (2005)
- Compare and contrast the predisposing factors, clinical features and biological behaviours of melanoma and squamous cell carcinoma of the skin. (2007)

Practical: Skin neoplasms

The images and virtual slides linked to this class are available at <http://vslides.unsw.edu.au/>

Task 1

The class will be divided into 4 groups. Each group will be assigned one of virtual slides 45, 38, 89 and 91. The members of the group should work together to prepare a presentation based on their slide. The presentation should include a histopathological description and microscopic diagnosis of the lesion, followed by a discussion of clinical features, risk factors and predisposing conditions, prognostic indicators and any other relevant information. Each group will have 15 minutes in the 2nd hour of the class to deliver their presentation; group members may elect their own spokesperson(s).

Virtual slide 45



Virtual slide 38



Virtual slide 91



Virtual slide 89



Cervical carcinoma

Teaching provided

Lecture: Cervical carcinoma

Practical: Cervical carcinoma

Tutorial: Cervical carcinoma

Aim

The aim of this lecture and associated practical and tutorial is to provide students with an appreciation of a common malignancy in women and the role of screening in preventing the disease.

Learning outcomes

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

1. Define the terms “cervical intraepithelial neoplasia (CIN), CIN I, CIN II CIN III”, “carcinoma in situ”, “koilocytosis”, “transformation zone”.
2. Contrast the epidemiology of carcinoma of the cervix in Australia with that seen in developing countries.
3. Discuss the pathogenesis of squamous cell carcinoma of the cervix, including the role of risk factors, with particular reference to human papilloma virus (HPV) infection.
4. Discuss the benefits and limitations of current methods used to detect precancerous lesions of the cervix.
5. Describe the clinical presentation and mode of spread of carcinoma of the cervix.
6. Discuss the role of vaccines in the prevention of carcinoma of the cervix in females and males.

TUTORIAL

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 18 pp. 685-688.

Additional resources

1. Farnsworth A. *Understanding Cervical Pathology*. Common Sense Pathology. September 2007.
<http://www.rcpa.edu.au/Publications/CommonSensePathology.htm>
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Past exam questions: Your Team should work together to develop answers to these questions

- Discuss the benefits and limitations of current methods used to detect precancerous lesions of the cervix (2004)
- A 38 year old woman presented to her local doctor with a 2 month history of bleeding after intercourse. More recently she had a spontaneous bloodstained discharge. After a series of investigations the woman underwent a hysterectomy.(2005)
 - i What is the likely diagnosis? How could this have been confirmed preoperatively?
 - ii Discuss the pathogenesis of the disease listed in part i. How might his disease have been prevented?
 - iii If this woman had not undergone treatment how might have her disease progressed?

Practical: Cervical carcinoma

See handout

Intracranial neoplasms

Teaching provided

Lecture: Intracranial neoplasms

Aim

The aim of this lecture is to facilitate an understanding of intracranial neoplasms.

Learning outcomes

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

1. Classify common primary intracranial neoplasms, and name the primary sites that most commonly give rise to intracranial metastases.
2. Describe the macroscopic and microscopic appearances of common intracranial neoplasms, and correlate these appearances with the clinical manifestations.
3. Explain the common clinical presentations of intracranial neoplasms.
4. Discuss the pathogenesis and pathophysiology of raised intracranial pressure.
5. Outline the investigations used in the diagnosis of intracranial neoplasms.

Recommended reading

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 22 pp. 842-847.

Upper Gastrointestinal Tract neoplasms

Teaching provided

Lecture: Upper GI neoplasms

Tutorial: Upper GI neoplasms

Aim

The aim of this lecture and associated tutorial is to gain an understanding of the pathogenesis of carcinoma of oesophagus and stomach.

Learning outcomes

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

1. Describe Barrett oesophagus and its role in the pathogenesis of oesophageal adenocarcinoma.
2. Compare and contrast the pathogenesis and clinical features of squamous cell carcinoma and adenocarcinoma of the oesophagus.
3. Recognise and describe the features of duodenal ulceration, gastric ulceration and gastric carcinoma.
4. Discuss factors that are implicated in the pathogenesis of gastric carcinoma including *Helicobacter pylori* infection
5. Explain the role of endoscopy and biopsy in the management of gastric disease.

TUTORIAL

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 14 pp. 558-572.

Past exam questions: Your Team should work together to develop answers to these questions

- Factors that are implicated in the pathogenesis of gastric carcinoma

Pulmonary neoplasms

Teaching provided

Lecture: Pulmonary neoplasms

Practical: Pulmonary neoplasms

Tutorial: Pulmonary neoplasms

Aim

The aim of this lecture and associated practical and tutorial is to facilitate an understanding of pulmonary neoplasms.

Learning outcomes

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

1. Discuss the pathogenesis of cough, haemoptysis and dyspnoea.
2. Describe the microscopic appearance of common neoplasms of the lung, and correlate this with the clinical manifestations.
3. Explain the basis of the extra-pulmonary clinical manifestations of lung neoplasms.
4. Describe the mode of spread of primary carcinomas of the lung.
5. Outline the investigations used in the diagnosis of lung neoplasms.

TUTORIAL

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 12 pp. 505-511.

Practical Pulmonary neoplasms

The images and virtual slides linked to this class are available at <http://vslides.unsw.edu.au/>

Case 1 - Clinical History

A 65 year old man presented to his local doctor complaining of worsening dyspnoea, cough and haemoptysis over the past 6 weeks. He had smoked a packet of cigarettes per day for over 40 years.

Task 1

Suggest possible causes for this man's symptoms. What might you expect to find on physical examination?

His GP organized a chest X-ray (see CXR linked from <http://vslides.unsw.edu.au/>).

Task 2

Describe the abnormalities seen on the chest X-ray. What is your differential diagnosis? What further investigations would you organize?



The following table shows the results of biochemical tests.

Clinical Chemistry			
Sodium (mmol/L)	141	135-145	
Potassium (mmol/)	3.7	3.4-4.5	
Chloride (mmol/L)	105	95-110	
Bicarbonate (mmol/L)	27	22-32	
Calcium (mmol/L)	3.2	2.10-2.55	*
Phosphate (mmol/L)	0.57	0.8-1.50	*
Urea (mmol/L)	3.8	3.0-8.0	
Creatinine (mmol/L)	0.09	0.05-0.12	
Bilirubin (mmol/L)	15	2-20	
Alkaline phosphatase (U/L)	85	38-126	
γ-glutamyltransferase (U/L)	27	<50	
AST (U/L)	31	<45	
ALT (U/L)	33	<45	
Tot Protein (g/L)	64	62-80	
Albumin (g/L)	43	33-48	

Task 3

What is the likely basis of the electrolyte abnormalities?

Your tutors will project the results of sputum cytology and an aspirate of pleural fluid. Bronchoscopy and biopsy of a lesion in the left main bronchus was also performed. On the basis of all the investigations undertaken it was decided the patient should have a pneumonectomy.

Virtual slide 1 was obtained from the tissues removed at operation.

Task 4

In the space below write a brief histopathological report on this slide and state your diagnosis.



Case 2 - Clinical History

A 59 year old woman presented with unproductive cough and chest pain. Bronchoscopy and biopsy revealed small cell carcinoma of the bronchus. It was decided that an operation was not appropriate and the woman was treated with chemotherapy. In spite of this treatment she gradually deteriorated over the following 3 months.

The following biochemical profile was obtained:

Clinical Chemistry			
Sodium (mmol/L)	144	135-145	
Potassium (mmol/)	2.9	3.4-4.5	*
Chloride (mmol/L)	108	95-110	
Bicarbonate (mmol/L)	37	22-32	*
Calcium (mmol/L)	2.4	2.10-2.55	
Phosphate (mmol/L)	1.2	0.8-1.50	
Urea (mmol/L)	4.6	3.0-8.0	
Creatinine (mmol/L)	0.10	0.05-0.12	
Bilirubin (mmol/L)	35	2-20	*
Alkaline phosphatase (U/L)	156	38-126	*
γ-glutamyltransferase (U/L)	78	<30	*
AST (U/L)	70	<45	*
ALT (U/L)	68	<45	*
Tot Protein (g/L)	65	62-80	
Albumin (g/L)	37	33-48	
Glucose (mmol/L)	10.3	3.0-6.0	*

The patient died 1 week after the above investigation.

Task 5

What abnormalities would you expect at autopsy? Examine the virtual slides showing metastatic small cell carcinoma in the lung and liver. Can you also suggest the basis of the biochemical abnormalities observed one week prior to her death?

**Task 6**

In the light of the histopathological findings, can you explain why she was not treated surgically?

Task 7

The lesion shown in virtual slide 2 was discovered on chest X-ray as a “coin” lesion in the peripheral lung. Summarise the histopathological features in the space below. What is your diagnosis?

Task 8

Examine virtual slide 3 (linked from <http://vslides.unsw.edu.au/>), which was prepared from tissue removed at autopsy from a 56 year old woman who died from disseminated malignancy, with the primary tumour unknown. Write down the major abnormalities in virtual slide 3. How might a pathologist determine the primary site of such a tumour?

Breast carcinoma

Teaching provided

Lecture: Breast carcinoma

Practical: Breast and prostate carcinoma

Tutorial: Breast and prostate carcinoma

Aim

The aim of this lecture and associated practical and tutorial is to facilitate a basic understanding of breast carcinoma.

Learning outcomes

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

1. List the known risk factors for the development of breast carcinoma and the molecular mechanisms for pathogenesis.
2. List and describe breast lesions which may produce a palpable mass, including carcinoma, fibroadenoma and fibrocystic change.
3. Outline procedures used commonly in the diagnosis of breast carcinoma including “triple assessment”, and discuss the role of screening in the early diagnosis of breast carcinoma.
4. Discuss factors that influence prognosis in breast carcinoma, including responsiveness to hormonal manipulations.
5. Describe the modes of progression of breast carcinoma

TUTORIAL

Pre-reading for tutorial quiz

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 18 pp. 704-714.

Additional resources

1. National Breast and Ovarian Cancer Centre. Clinical best practice. This site contains many useful articles on breast disease. <http://www.nbocc.org.au/bestpractice/>
2. Breast Screen NSW. www.bsns.org.au
(There are some interesting brochures on this website. One in particular that is informative and brief is “I’m under 40. Is it time for a screening mammogram?”)

Past exam questions: Your Team should work together to develop answers to these questions

- Factors which can help determine the prognosis of a woman with carcinoma of the breast (2001)

Practical: Breast and prostate carcinoma

Case 1 - Clinical History

A 45 year old woman presented to her local medical officer with a lump in the right breast. The lesion, which was in the lower outer quadrant of her breast, had been noticed to be enlarging over the last two weeks. There was no change in the size or nature of the lump in relation to her menstrual cycle. On examination there was a 3 cm stony hard lump, which was tethered to the overlying skin. There was dimpling of the overlying skin on contraction of the pectoralis muscle. There were firm, but mobile axillary lymph nodes on the right side.

Task 1

Each Team will be assigned one of Virtual Slides 29, 31, 36 and 32. The members of the Team should work together to prepare a presentation based on their slide. The presentation should include a histopathological description and microscopic diagnosis of the lesion, **followed by a discussion of whether the lesion is consistent with the clinical features in this case**. Risk factors and predisposing conditions, investigations, prognostic indicators and any other relevant information should be presented. Teams will be chosen to deliver a 10-minute presentation on their slide; other Teams will be asked to add and comment; Virtual Slides may be found at <http://vslides.unsw.edu.au/>

Virtual slide 29

Virtual slide 31

Virtual slide 36

Virtual slide 32

Case 2 - Clinical History

A 58 year old man presented with back pain. On further questioning he reported that he had had increasing problems with urination over the last 3 months. His stream had become poor and he had terminal dribbling.

Task 1

What further signs and symptoms would you seek from this patient?

Task 2

Outline the investigations that you would perform to confirm your provisional diagnosis.

Investigations	Expected Result

Task 3

Comment on the results of the biochemistry tests outlined in the table below.

Clinical Chemistry			
Sodium (mmol/L)	138	135-145	
Potassium (mmol/)	3.5	3.4-4.5	
Chloride (mmol/L)	103	95-110	
Bicarbonate (mmol/L)	26	22-32	
Calcium (mmol/L)	2.3	2.10-2.55	
Phosphate (mmol/L)	1.3	0.8-1.50	
Urea (mmol/L)	6.0	3.0-8.0	
Creatinine (mmol/L)	0.11	0.05-0.12	
Bilirubin (mmol/L)	12	2-20	
Alkaline phosphatase (U/L)	250	25-100	*
γ-glutamyltransferase (U/L)	35	<50	
AST (U/L)	25	<40	
ALT (U/L)	28	<35	
Tot Protein (g/L)	73	62-80	
Albumin (g/L)	45	35-47	
Prostatic-Specific Antigen (U/L)	51	0-0.4	*
Glucose (mmol/L)	4.7	3.5-6.0	

Task 4

Based on the investigations outlined above, what is the likely cause of this man's back pain? Your tutor will project an X-ray of this man's spinal disease. Is this consistent with your provisional diagnosis?

Task 5

A biopsy of the lesion was obtained. Examine Virtual slides 61c and 27. Which of these slides best explains all of the man's symptoms?

Lymphoma and leukaemia

Teaching provided

Lecture: Lymphoma and leukaemia

Aim

The aim of this lecture is to facilitate an understanding of lymphoma and leukaemia

Learning outcomes

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

1. Discuss the classification of lymphoma and leukaemia
2. Describe the macroscopic and microscopic appearances of lymphoma and leukaemia
3. Explain the common clinical presentations of lymphoma and leukaemia
4. Discuss the pathogenesis and pathophysiology of lymphoma and leukaemia
5. Outline the investigations used in the diagnosis of lymphoma and leukaemia

Recommended reading

Kumar V, Abbas AK, Aster JC (2012). *Robbins Basic Pathology* (9th Ed). Philadelphia, PA:Elsevier Saunders. Chapter 11 pp. 428-448.

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: lack of 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 – "*chemoattractant cytokines*"

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

Sepsis: 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