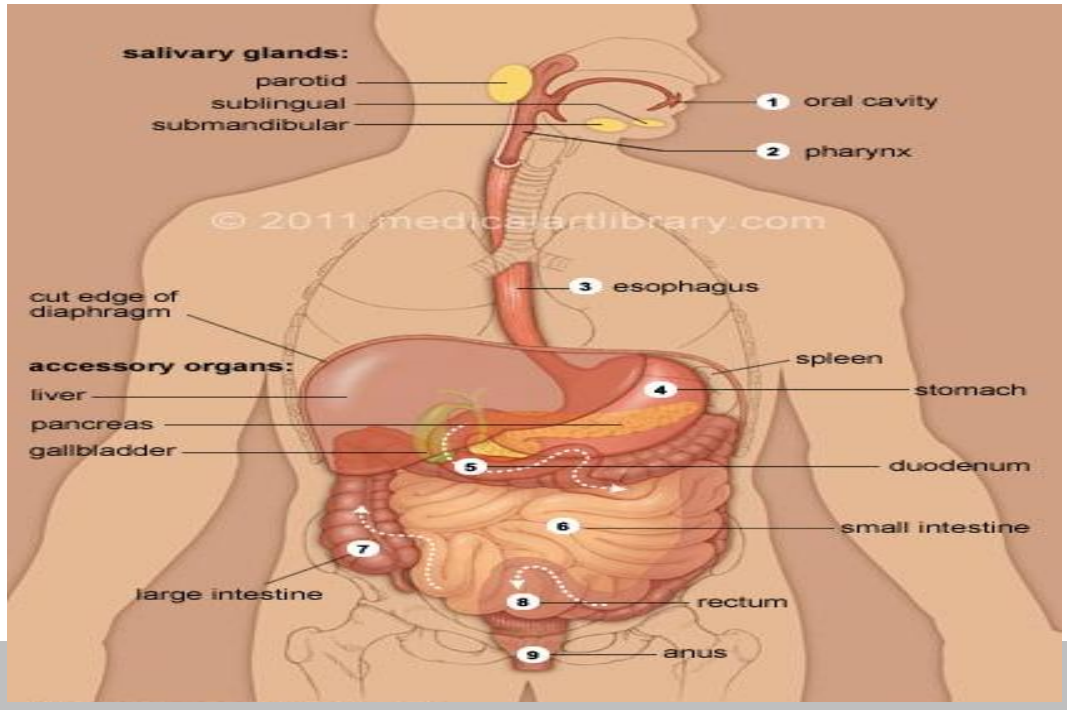
COLLEGE OF MEDICINE
Department of Medical Education
Curriculum Development & Research Unit



STUDENT'S BOOK

GASTROINTESTINAL & NUTRITION BLOCK (GNT 223)

YEAR 2 (Female)

2017-2018
(1438-1439)

COLLEGE OF MEDICINE
Department of Medical Education
Curriculum Development & Research Unit

THE GASTROINTESTINAL & NUTRITION BLOCK

Year Two

BLOCK BOOK AND STUDENT GUIDE

Female Group

(19 November 2017 to 11 January 2018)

(2017-2018) 1438-1439

WELCOME ADDRESS

Dear Students,

We are pleased to welcome you in the college of Medicine, Gastro Intestinal & Nutritional Block attachment. We hope you will find this block both useful and enjoyable.

A message from the Dean

We are pleased with your progress in the medical program and your achievements. Being a second year medical students is a great opportunity for you to consolidate what you have learnt in the preparatory year and prepares you for the clinical skills and competencies needed in the clinical years. The Department of Medical Education through its different units is working hard to create an integrated and innovative curriculum that builds on the changes introduced in the preclinical years and enforces best teaching/learning approaches in the design of the new medical curriculum. As you are aware, the College of Medicine at King Saud University is one of the best colleges not just in the Kingdom of Saudi Arabia but proved to be one of the best in the gulf region, and the Middle East. It also has its international influence among the best colleges of medicine worldwide. This makes us proud of our achievements and provides you with an insight about the quality of teaching and research that we have reached and our continuous work to maintain our standards.

Therefore, the medical curriculum aims at preparing you and equipping you with the best training and clinical skills to become a medical graduate that fulfills the highest international standards. Therefore, the focus of the curriculum is to enhance a number of skills such as case-based learning, critical thinking, self-directed learning, deep understanding of concepts, application of knowledge learnt, and how to make decisions on the basis of evidence. The curriculum also aims at enhancing your skills in areas such as professionalism, e-learning, task-based learning, and preparing you for life-long learning. The design of the curriculum encourages small group learning, use of cases for discussion, lectures, student-led seminars, bed-side teaching, task-based learning, use of multimedia and e–learning as modes for teaching and learning. The use of wide range of teaching and learning modes and small group discussion will help you to become active learners, and work with other students in your group as a team.

I wish you all the best during your academic year and would encourage all of you to get the best out of the teaching and learning opportunities provided to you during this year. Our teaching staff and clinicians would be very happy to help you on any issue that you need help with.

Professor Khalid A Fouda Neel

Dean, College of Medicine and the Supervisor of University Hospitals

A Message from the Vice Dean for Academic Affairs

It is my pleasure to welcome you all to the second year of Medicine. I would like to take this opportunity to congratulate you all on your success and achievements. There is no doubt that you have worked hard during the first year to adapt to the university system and our new integrated curriculum. In the meantime, we would like you to remember that success is not a destination, success is a journey and there will be many challenges during your journey of success. A successful person would turn these challenges into opportunities for success.

As you might be aware, our faculty under the leadership of our Dean is moving into an integrated curriculum that encourages small group learning and student-centered approaches for learning. To achieve these goals we have established the Department of Medical Education under the leadership of Dr Mona Soliman and his teams to develop the new integrated curriculum. The design of the new curriculum is focused on the students not the teachers. Our aim is to equip each of you with the current teaching and learning strategies that are used in the best universities worldwide and ensure that you will be an excellent medical doctor who will be committed to the profession and willing to serve patients in our country, our region, and wherever our government and our professional bodies would ask you for help.

On these bases, our aim is not just to graduate more doctors; our aim is to ensure that doctors graduating from our university are equipped with knowledge, skills, behavior, and competencies needed for best practice of medicine anywhere in the world. This goal makes a lot of responsibility from your end and we would like you to take this opportunity and work effectively to achieve your goals. Our academic and clinical staff are expert in their areas and very eager to help and support you to achieve your dreams. I would encourage you to ask for help when needed and our support team would work with you on any challenges you might face during the course. I wish you all the best.

Dr. Saleh Fahed Adhehri

Vice Dean for Academic Affairs

College of Medicine

A Message from the Gastro Intestinal & Nutrition Block Chair

Dear students,

It gives me a great pleasure to welcome you to GIT block which will consists of 6 weeks period integrating the basic science of gasterointestinal, hepatology and hematology systems. The objective of this block is to provide you a clear understanding of the physiology, anatomy and pathology of the GI tract and nutrition system. Moreover, it will enhance your skills of self- directed learning, critical thinking and the ability to analyse the acquired knowledge to build up your clinical orientation
I encourage you to maximize your learning experience from this block by proper utilization of the available resources provided to you during this rotation to achieve your goals and objectives and to strengthen the basic foundation of your knowledge for your future career.

Dr. Othman Al-Harbi

Gastrointestinal and Nutrition Block Chair

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General Information

Block Title	Gastrointestinal and Nutrition Block
Block Code & Number	GNT 223
Credit Hour	4
Block Duration	8 Weeks
Block Dates	19th November 2017 to 11th January 2018
Block Chairman	Dr. Othman Al Harbi
Block Co-Chair	Prof. Ali Somily
Members of the Committee	Prof. Samy Azer
	Dr. Hayam Gad
	Prof. Saeed Abuelmakarem
	Dr. Rana Hasanato
	Dr. Ahmed Al-Humaidi
	Prof. Hanan Hagar
	Dr. Fatma AlQahtani

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Year 2 - Female Group

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	Dr. Mona Al Safadi	0503117199	monasafadi@gmail.com; melsafadi@ksu.edu.sa
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	Dr. Alia Al Faedi	Ext. 72504	aliaalfaraedi@gmail.com
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	Prof. Aljohara Almenessier	0505274489	aalmeneessier@KSU.EDU.SA
	Prof. Hala Kfoury	0504127781	halakfoury@hotmail.com

List of the Problem-Based Learning Cases

The table below summarizes the PBL cases to be discussed in the Gastrointestinal & Nutrition Block.

Week	Case Number	Case Title
W1	**NO CASE**	
W2	**NO CASE**	
W3 (Monday & Thursday)	Case 1	"…Not Gaining Weight"
W4 (Monday & Thursday)	Case 2	"…Unexpected Outcomes"
W5 (Monday & Thursday)	Case 3	"…Vomited Dark Blood "
W6 (Monday & Thursday)	Case 4	"… Pale and feels tired"

Instructions:

The cases listed above will be discussed by students in their small groups. Each group is about 8 to 12 students. Each case will be discussed in two tutorials, on Sunday and Wednesday. Each tutorial is two hours long.

Attendance of Small Group Learning tutorials:

Students must attend all small group learning tutorials. If a student is not well, she needs to provide a medical certificate from their family doctor. If a student misses out to attend four tutorials without acceptable reason, he/she might not be allowed to attend the final examination.

Students Roles in Small Group Learning Tutorials:

The design of the curriculum encourages small group discussion and student-centered learning. To achieve these goals there is a need for establishing good group dynamics, interpersonal skills, and effective communication. These elements will ensure that learning is an enjoyable process and rewarding to each member in the group. Therefore, students play a vital role in making a difference in their groups. To achieve these changes and improve your learning outcomes, We recommend that you use the paper by Professor Samy Azer, titled "Becoming a Student in a PBL Tutorial", a copy is enclosed in the Appendix. Your continuous reflection on these tips and working on identifying your role in your group will help you in reaching these goals and building up your group.

General Objectives of the Gastrointestinal & Nutrition Block

By the end of the Gastrointestinal and Nutrition block, students should be able to:

- Correlate the relationship between the anatomical structures of the gastrointestinal and hepatobiliary system and their functions.
- Use knowledge learnt from basic sciences to interpret symptoms, signs and investigation results of patients with common gastrointestinal and hepatobiliary disorders.
- Understand the pathology, microbiology and pathogenesis of the common disorders affecting the gastrointestinal and hepatobiliary system.
- Discuss the pharmacological basis of drugs used in the management of patients with gastrointestinal and hepatobiliary diseases.
- Demonstrate knowledge of epidemiology and preventive approaches in common gastrointestinal and hepatobiliary disorders.
- Revisit epidemiological parameters such as body mass index and discuss the macro and micro nutritional requirements of a population.
- Discuss normal haemopoiesis and the functions of different haemopoietic cells.
- Discuss common disorders affecting the haemopoietic system, with particular emphasis on anaemia.
- Understand the role of haemoglobin, types of haemoglobin, and iron metabolism.
- Develop communication and professional skills at the level of a medical student.

1.0 Knowledge

1.1 Describe the anatomy of the gastrointestinal organs (Salivary glands, oropharynx, esophagus, stomach, pancreas, small intestine, large intestine, peritoneum) and hepatobiliary system (liver, gallbladder, biliary tract) and other intra-abdominal structures (aorta, inferior vena cava, lymphatics and lymph nodes, spleen, blood supply and venous drainage of abdominal organs) and correlate each structure to their functions.

1.2 Discuss the ultrastructure of each of the gastrointestinal organs and hepatobiliary system and discuss the significance of these structures at cellular and molecular levels in key biological processes such as digestion, absorption, metabolism of fats, carbohydrates and proteins, as well as drug metabolism and execration.

1.3 Correlate normal anatomical structures of the gastrointestinal and hepatobiliary systems to their radiological findings/features and discuss the differences between different radiological modalities and their uses in investigating common gastrointestinal and hepatobiliary disorders.

1.4 Discuss the pathology and the pathogenesis of common diseases affecting the gastrointestinal system including reflux oesophagitis, chronic gastritis, peptic ulcer, malabsorption, chronic pancreatitis, pancreatic cancer, irritable bowel syndrome, inflammatory bowel disease, diverticulitis, colon cancer, liver cirrhosis, cholestasis, portal hypertension, liver failure, cholecystitis, and biliary stones.
1.5 Discuss the normal flora of the intestine and discuss the microbiology of diseases affecting the gastrointestinal and hepatobiliary system including viral hepatitis, food poisoning, infectious diarrhea, and diverticulitis.
1.6 Discuss the normal physiology and biochemistry of the gastrointestinal system including gastrointestinal innervation, motility, secretion, gastrointestinal hormones, regulation, absorption, metabolism, and execration.
1.7 Discuss the pharmacology of drugs used in the management of peptic ulcer, gastritis, liver cirrhosis, viral hepatitis, inflammatory bowel disease, portal hypertension, irritable bowel syndrome, as well as antispasmodics, and chemotherapeutic agents.
1.8 Discuss the nutritional requirements of macro- and micronutrients (of children, adolescents, pregnant women, adults, elderly people and people with chronic diseases) as well as discuss common nutritional disorders.
1.9 Discuss the formation, secretion and metabolism and functions of bilirubin and bile salts and their enterohepatic circulation.
1.10 Discuss the liver functions and the liver function tests and the role of the cytochrome system in drug metabolism.
1.11 Discuss the haemopoietic system its components and functions, and discuss the physiology of blood, blood cells and common disorders affecting the haemopoietic system.
1.12 Discuss and apply the principles of self-directed learning.
1.13 Discuss the role of social, cultural, behavioural and genetic factors in the development of diseases affecting the gastrointestinal system.
1.14 Discuss the impact of chronic diseases affecting the gastrointestinal and haemopoietic systems on the patient and family.
1.15 Briefly discuss health promotion, health education, and prevention of diseases affecting the gastrointestinal and haemopoietic system.

20. Cognitive Skills

2.1 Identify problems, generate hypotheses, make an enquiry plan, weigh evidence for and against a hypothesis, and make a decision on the basis of available evidence.

2.2 Apply knowledge learnt from anatomy, physiology, biochemistry, pathology, microbiology, and pharmacology to problem-based learning cases and use knowledge learnt to justify their views and in making decisions.

2.3 Identify learning needs, search for new information and use new information to solve problems.

2.4 Work out how to handle uncertainty and decide on appropriate approaches to handle such situation.

2.5 Integrate knowledge learnt from different disciplines such as anatomy, physiology, biochemistry, pathology, microbiology, and pharmacology to discuss a problem, make priorities, and define their action plan, and learning needs.

1.0 Interpersonal Skills & Responsibility

3.1 Communicate effectively and demonstrate the ability to build rapport, work as a member of a small group and contribute to the learning of others.
3.2 Demonstrate the ability to monitor their progress, apply time management rules, and use feedback in improving their performance.
3.3 Demonstrate the ability to take medical history from patients and demonstrate the ability to present their findings, and communicate with patients using simple language without technical jargon.
3.4 Demonstrate accountability in their work with others in small groups (e.g., in problem-based learning).

2.0 Communication, Information Technology, Numerical

4.1 Use computer programs in searching for new information, sharing information and analyzing data.

5.0 Psychomotor Skills

5.1 Demonstrate the ability to take history from patients with common problems affecting the gastrointestinal system.

5.2 Demonstrate the ability to conduct clinical examination of the gastrointestinal system and demonstrate the ability to show correct techniques, correct sequence of examination.

Teaching and Learning Modes:

In an integrated curriculum like our curriculum, we use a wide range of teaching and learning strategies to ensure that learning meets the different needs of the students. These strategies include:

- Small group discussion
- Lectures
- Student-led seminars
- Practical classes
- Clinical skills
- Self-directed learning
- Writing an essay or mini thesis
- E-learning sessions

Objectives of the Lectures

Title of the lecture:	Anatomy of the oral cavity oseophagus and stomach
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	galmadani@ksu.edu.sa

Objectives:

- Describe the anatomy the oral cavity, (boundaries, parts, nerve supply).
- Describe the anatomy of the palate, (parts, muscles, nerve & blood supply).
- Describe the anatomy of the tongue, (structure, muscles, motor and sensory nerve supply, blood supply, and drainage).
- Describe the anatomy of the esophagus; extent, length, parts, strictures, relations, blood & nerve supply and lymphatic.
- Describe the anatomy of the stomach; location, shape, parts, relations, blood & nerve supply and lymphatic.
- Discuss the microscopic structure in correlation with the function of the following organs: Esophagus and Stomach.

Title of the lecture: Histology of the oesophagus and stomach	
Lecturer's Name:	Prof. Raeesa Abdultawab
Department	Anatomy
Block / Week	Gastrointestinal & Nutrition Block / 1
E-mail Address:	rmohammad@ksu.edu.sa

Objectives:

By the end of this lecture, the student should be able to discuss the microscopic structure, in correlation with the function, of the following organs:

- Esophagus.
- Stomach.

Title of the lecture: General principles of GIT physiology	
Lecturer's name	Dr. Hana Al Zamil
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	hanazamil@yahoo.comn

Objectives:

- Physiologic anatomy of gastrointestinal wall
- The general characteristics of smooth muscle
- The specific characteristics of smooth muscle
- Control of gastrointestinal function (ENS)
- Functional types of movements in the gastrointestinal tract
- Gastrointestinal blood flow (Splanchnic circulation)
- Effects of gut activity and metabolic factors on GI blood flow

Title of the lecture:	Anatomy and Histology of the salivary glands
Lecturer's name	Prof. Raeesa Abdultawab
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	rmohammad@ksu.edu.sa

Objectives:

- Describe the anatomy of the *parotid* gland: position, shape, structures within it, innervation and parotid duct.
- Describe the anatomy of the submandibular and sublingual salivary glands: location, shape, parts, ducts and innervation of the glands.
- Describe the microscopic structure of the major salivary glands in correlation with function.

Title of the lecture:	Role of salivary and stomach in digestion
Lecturer's name	Dr. Rana Hasanato
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	rhasanato@KSU.EDU.SA

Objectives:

- Understand the principle and importance of digestion of dietary foodstuffs
- Understand the role of salivary glands in digestion
- Understand the role of stomach in digestion

Title of the lecture:	Oesophageal motility and pathophysiology of reflux disease
Lecturer's name	Dr. Hana Al Zamil
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	hanazamil@yahoo.com

Objectives:

- Mastication & chewing
- Salivary glands
- Secretion of saliva
- Contents of saliva
- Functions of saliva
- Control of salivary secretion
- Swallowing
- Types of esophageal peristalsis
- Function of lower esophageal sphincter

Title of the lecture:	Physiology of the stomach and regulation of gastric secretions
Lecturer's name	Dr. Hana Al Zamil
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	hanazamil@yahoo.com

Objectives:

- Functions of stomach
- Gastric secretion
- Mechanism of HCl formation
- Gastric digestive enzymes
- Neural & hormonal control of gastric secretion
- Phases of gastric secretion
- Motor functions of the stomach
- Stomach Emptying

Title of the lecture: GERD Gastro oesophageal reflux disease	
Lecturer's Name:	Dr. Maha Arafa
Department	Pathology
Block / Week	Gastrointestinal & Nutrition Block / 1
E-mail Address:	marafa@hotmail.com

Objectives:

Upon completion of this lecture the students will:

- To describe the definition of reflux esophagitis.
- To know the pathogenesis of reflux esophagitis.
- To list the clinical features of reflux esophagitis.
- To describe the Pathology (gross and microscopic features) of reflux esophagitis.
- To list the complication of reflux esophagitis.
- To describe the definition of Barrett esophagus:
- To know the pathogenesis of Barrett esophagus:
- To list the clinical features of Barrett esophagus:
- To describe the Pathology (gross and microscopic features) of Barrett esophagus:
- To list the complication of Barrett esophagus including dysplasia and adenocarcinoma
- To know the cause and features of squamous cell carcinoma

Introduction:

The squamous lining of the oesophagus is easily damaged by regurgitated gastric contents and soon becomes chronically inflamed. A defective sphincter mechanism at the cardia predisposes to such gastro-oesophageal reflux, which is therefore an invariable accompaniment of hiatus hernia. It may also be a consequence of increased intra-abdominal pressure without herniation, or of gastric surgery. Other patients appear to have an underlying abnormality of upper gastro-intestinal motility which leads to gastro-oesophageal reflux and/or duodeno-gastric reflux. The characteristic symptom is an awareness of acid regurgitation with central chest pain or discomfort ('heartburn').

Key Outlines:

- Introduction on Physiology of esophageal motility
- Pathophysiology and causes of reflux esophagitis
- Theological features (gross and microscopic) of reflux esophagitis
- Other causes of esophagitis

- Clinical features and complications of reflux esophagitis

Take home message:

- Reflux esophagitis predisposes to Barrett's esophagus that leads to dysplasia and adenocarcinoma

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotran

Title of the lecture:	Pathology and pathophysiology of peptic ulcer disease
Lecturer's Name Department	Dr. Maha Arafa Pathology
Block / Week	Gastrointestinal & Nutrition Block/ 1
E-mail Address:	marafa@hotmail.com

Objectives:

Upon completion of this lecture the students will :

- Understand the Pathophysiology of acute and chronic peptic ulcer
- Know the possible causes of gastric and duodenal ulcers with emphasis on most common causes (H pylori and drugs)
- Recognize the gross and microscopic features of peptic ulcer
- Recognize the clinical features and consequences of acute and chronic peptic ulcer

Keywords:

- peptic, ulcer, gastric, duodenal, *Helicobacter* pylori, NSAIDs, Defensive factor, Aggressive factor.

Background:

- *Ulcers are defined as a breach in the mucosa of the alimentary tract that extends through the muscularis mucosae into the submucosa or deeper.* Under normal conditions, a physiologic balance exists between peptic acid secretion and gastroduodenal mucosal defense. Mucosal injury and, thus, peptic ulcer occur when the balance between the aggressive factors and the defensive mechanisms is disrupted.. Peptic ulcers are ulcers occurring in any part of the gastrointestinal tract exposed to the action of acidic gastric juice. They occur principally in the duodenum (duodenal ulcer) and stomach (gastric ulcer). Peptic ulcer disease is common all over the world. Many aetiological factors contribute to peptic ulcer but most commonly are *Helicobacter* pylori gastritis and NSAIDs.

Main concepts in the lecture:

- Chronic gastritis is defined as the presence of chronic inflammatory changes in the mucosa leading eventually to mucosal atrophy and epithelial metaplasia.
- By far the most important etiologic association is chronic infection by the bacillus *H. pylori*.
- *Ulcers* of the alimentary tract are defined histologically as a breach in the mucosa that extends through the muscularis mucosae into the submucosa or deeper. This is to be contrasted to *erosions*, in which there is a breach in the epithelium of the mucosa only.

- Peptic ulcers are chronic, most often solitary, lesions that occur in any portion of the gastrointestinal tract exposed to the aggressive action of acidic peptic juices.
- At least 98% of peptic ulcers are either in the first portion of the duodenum or in the stomach, in a ratio of about 4:1.
- *H. pylori* infection is the most important condition in the pathogenesis of peptic ulcer. *NSAIDs are the major cause of peptic ulcer disease in persons who do not have H. pylori infection.*
- *Stress ulcers (acute gastric ulcers):* associated with severe trauma, burns, CNS trauma or hemorrhage; usually small, multiple, hemorrhagic ulcers that are often shallow.

Conclusion:

- The major cause of *Chronic gastritis* is the infection by *Helicobacter pylori*, less commonly autoimmune in origin. *Peptic ulcer is a* breach in the epithelium caused most commonly by *H. pylori* infection and mucosal exposure to gastric acid and enzymes (pepsin), or less frequently by use of NSAIDs. peptic ulcers are created by an imbalance between the gastroduodenal mucosal defenses and the damaging forces that overcome such defenses.

Key Outlines:

- The Defensive and Aggressive Factors of the gastric mucosa.
- Pathophysiology and aetiology of acute and chronic peptic ulcer.
- Gross and microscopic features of gastric and duodenal ulcer
- Clinical features and complications of peptic ulcer disease.

Take home message:

- H pylori infection of the pyloric antrum is present in nearly all patients with chronic duodenal ulcer and approximately 75% of patients with chronic gastric ulcer.
- The main cause of peptic ulcer disease is *H. pylori* infection followed by NSAIDs

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotran

Title of the lecture: H2 blockers and proton pump inhibitors	
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	hananhhagar@yahoo.com

Objectives:

- Understand the key points of pathophysiology of the peptic ulcer disease
- Enumerate various classes of drugs used in peptic ulcer disease
- Know the characteristic pharmacokinetics, pharmacodynamics and side effects of drugs used in peptic ulcer disease.
- Know the cytoprotective drugs mainly misoprostol and its use in NSAIDs-induced peptic ulcer.

Title of the lecture:	Introduction to pleuripotent stem cell
Lecturer's name	Dr. Mona Al Safadi
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	melsafadi@ksu.edu.sa / monsafadi@gmail.com

Objectives:

- Stem Cell – Definition
- Stem Cell – main function within the body
- Where can we fined Stem Cells?
- Classifications of stem cells
- Embryonic Stem Cell
- Adult stem cells (Tissue Specific Stem Cell)
- Induced Pluripotent Stem Cell (iPS) cells
- Different approaches for isolation of pluripotent stem cells.
- The Promise of Stem Cell Technology.

Title of the lecture:	Anemia
Lecturer's name	Dr. Fatma Al Qatani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	fatqahtani@gmail.com

Objectives:

- To understand the normal control of red cell production
- To understand the mechanisms of which anaemia may arise
- To appreciate the signs and symptoms of anaemia
- To understand how anaemia can be classified by red cell size
- To be able to suggest causes of microcytic, normocytic and macrocytic anaemia
- To understand normal iron metabolism, how iron deficiency and anaemia of chronic disease may arise and how to investigate it.

Title of the lecture:	**Structure and function of haemoglobin**
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	sumbulfatma@gmail.com

Objectives:

- The structure and function of hemoglobin.
- The factors affecting oxygen binding to hemoglobin.
- Examples of normal and abnormal hemoglobin structures.

Title of the lecture:	**Transfusion and cross- matching**
Lecturer's name	Dr. Fatma Al Qahtani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	falqahtani@gmail.com

Objectives:

- To understand the inheritance and significance of the ABO system
- To understand the nature and significance of the Rh blood group system including RhD
- To know the principles involved in the selection of donor blood of suitable ABO and Rh groups for a recipient, and the principles of the cross-match, including the antiglobulin test
- To understand the hazards of blood transfusion.
- To know how to investigate a patient suspected of receiving an incompatible transfusion
- To know the basis of blood fractionation and the rationale for the use of specific blood products
- To know the pathogenesis, clinical features and the principles underlying the treatment and prevention of haemolytic disease of the newborn (HDN) due to anti-D.

Title of the lecture:	Heamoglobinopathy
Lecturer's name	Dr. Fatma Al Qatani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	falqahtani@gmail.com

Objectives:

- To understand the normal structure and function of haemoglobin
- To understand how the globin components of haemoglobin change during development, and postnatally
- To understand the mechanisms by which the thalassaemias arise
- To appreciate the clinical presentations and complications of thalassaemia
- To appreciate the contribution of haemolysis and ineffective erythropoiesis to the pathophysiology of thalassaemia
- To understand the pathophysiology of sickle cell anaemia
- To be able to describe the clinical presentation and complications of sickle cell anaemia
- To understand the role of haemoglobin electrophoresis and high performance liquid chromatography in the investigation of globin disorders
- To appreciate the many other haemoglobin variants associated with disease.

Title of the Lecture:	Role of H pylori in peptic ulcer and drugs used in treatment
Lecturer's name	Dr. Fawzia Al Otaibi
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 1
Email address	

Objectives of the lecture:

- To define peptic ulcer disease and assess its distribution among patients.
- To briefly indicate the signs and symptoms.
- To know impact of the discovery of *H.pylori* on the change of diagnosis and management of peptic ulcer.
- To understand the various gastric and duodenal diseases caused by *H.pylori*.
- To learn laboratory characteristics of *H. pylori* and its identification and diagnosis.
- To know the pathophysiology of *H.pylori* inside the stomach and duodenum.
- To learn the prevention methods used for *H.pylori* infection.
- To explore and learn the epidemiology and transmission ways of the disease.
- Finally, to know the management and treatment regiments used for eradication of *H.pylori*. .

Background:

After the Australian Easter holiday in 1982 an incubator was opened in Microbiology department of the Royal Perth Hospital, revealing the first ever culture of a spiral-shaped bacterium from gastric biopsies of patients with gastritis and peptic ulcers. The discovery of Helicobacter pylori, by Warren and Marshall has revolutionized gastro-enterology and has forced a reappraisal of many fundamental concepts not only of gastroduodenal disease but also of basic gastric pathophysiology. H pylori causes gastritis in more than half the world's population and is the etiologic agent of 95% of duodenal ulcers, 70-80% of gastric ulcers, and has a causal role in probably up to 60-70% of gastric cancer, a disease that remains one of the most common malignancies worl-wide.

Main concepts in the lecture:

- Peptic ulcer disease is an ulcer define as mucosal erosions associated with the over usage of NSAIDs.
- Peptic ulcer is created in an acidic area with more Peptic ulcers are arise in duodenum than stomach.
- 1982 in Perth (Australia),Warren and Marshal. Discovery revolutionized the treatment of duodenal and gastric ulcers.
- *H. pylori* are found in the human stomach. Molecular studies suggest transmission from an animal source.
- *Helicobacter pylori* is found closely associated with gastric mucosa and causes chronic active gastritis, gastric and duodenal ulcer (Peptic ulcer) and could develop adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphoma.

- *H.pylori* plays a role in gastric and duodenal ulceration and probably also gastric cancer. Over 80% of individuals infected with the bacterium are asymptomatic.
- More than 50% of the world's population harbour *H. pylori* in their upper gastrointestinal tract. Infection is more prevalent in developing countries.
- The route of transmission is unknown, although it is known individuals typically become infected in childhood.
- To colonize the stomach, *H pylori* must survive acidity. Using flagella, *H pylori* moves through stomach lumen and drill into the mucoid lining of stomach.
- Produces adhesions that binds to the epithelial cells. Produces large amounts of *urease* enzyme that break down urea into co2 +ammonia. This in-turn neutalizes gastric acid.
- Ammonia is toxic to epithelial cells along with *proteases*, *vacA* protein and *phospholipases* produced by *H pylori* and could damage epithelial cells.
- Colonization of stomach or duodenum results in chronic gastritis (inflammation of stomach lining). Inflammation stimulate more production of gastric acid. This leads to gastric and duodenal ulcers, atrophy and later cancer.
- CagA protein was found to contribute to peptic ulcer. Neutrophil-Activating Protein (NAP) recruits neutrophils to gastric mucosa causing inflammation. Free radical production in the gastric lining due to *H pylori,* increases host cell mutation.
- *H pylori* induces the production of TNF-α and Interleukin 8 that leads to host cells mutation.
- In vitro *H.pylori* is sensitive to amoxycillin, tetracycline, metronidazole, macrolides (clarithromycin).
- However, in vivo their efficacy is often poor due to the low pH of the stomach, their failure to penetrate the gastric mucus and the low concentration of antibiotic obtained in the mucosa of the stomach.
- Recently, Metronidazole in developing countries is becoming resistance (80-90%).

Conclusion:

There is now no doubt that H pylori is actively involved in the pathogenesis of peptic ulceration and not related to non steroidal anti-inflammatory agents. Infection is prerequisite for ulceration and elimination of infection allows healing of ulcers, in contrast to short term treatment with agents that suppress gastric acidity. Any recurrence of ulceration is almost always associated with recrudescence of infection. The pathogenic mechanism is not clear but several factors may contribute such as; the production of ammonia by urease which damage the mucus layer, the production of toxins or other substances (lipopolysaccharides) that activate inflammatory cells, the stimulation of auto-immune response by the production of antigens that cross-react with antral gastric antigens. Finally, the degradation of mucus by protease. Infected patients also have hyper-gastrinemia that contribute to the progression of ulcer. The eradication of H pylori requires the use of at least two antimicrobial agents in combination. The most used regimen is bismuth subcitrate (or subsalicylate), tetracycline (or amoxicillin) and metronidazole given for two weeks with an eradication rate of 90%.

Take home messages:

- learn about the description and characteristic of H pylori and how it is best diagnosed.
- Investigate the pathogenic mechanism and pathophysiology of H pylori in formulating ulcers.
- Explore the epidemiology of the disease and ways of transmissions of H pylori and ways of preventions.
- Read into the latest findings of immunization and vaccinations against H pylori.
- Know more about the treatment regimen used to eradicate h pylori and heal ulcers.

Further reading:

- Sherris Medical Microbiology , An Introduction to Infectious Diseases, Kenneth Ryan, George Ray, Latest edition; Chapter 22, page 380-385.

Title of the lecture:	Anatomy and histology of pancreas and biliary system
Lecturer's name	Prof. Raeesa Abdultawab / Dr. Jamilah El Medany
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	rmohammad@ksu.edu.sa / galmadi@ksu.edu.sa

Objectives:

- Location, surface anatomy, parts, relations & peritoneal reflection of the pancreas and gall bladder.
- Blood supply, nerve supply and lymphatic drainage of pancreas and gall bladder.
- Course of each of common hepatic, cystic and common bile duct and pancreatic ducts.
- Identify & describe the histological features of:
- Intrahepatic biliary passages.
- Extrahepatic bile ducts.
- Gall bladder.
- Exocrine pancreas.

Title of the lecture:	Embryology of the pancreas and small intestine
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	salsharawi@ksu.edu.sa

Objectives:

- Understand the process of digestion of dietary lipids including the organs involved, the enzymes and the end products.
- Discuss the hormonal control of lipid digestion by CCK and secretins.
- Comprehend the role of chylomicrons as the carriers of dietary lipids including their assembly and secretion by enterocytes.
- Understand the clinical manifestations of the diseases associated with defective lipid digestion and absorption.

Title of the lecture:	Physiology of the pancreas
Lecturer's name	Dr. Hana Al Zamil
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 1
Email address	hanazamil@yahoo.com

Objectives:

- Pancreatic acini
- Pancreatic secretion
- Pancreatic enzymes
- Control of pancreatic secretion, Neural, Hormonal (Secretin, Cholecystokinin)

Title of the lecture:	Biochemical aspects of digestion of proteins and carbohydrates
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	sumbulfatma@gmail.com

Objectives:

- Understand the overall process of dietary proteins' and carbohydrates' digestion, the organs involved, the enzymes required, and the end products.
- Implement the basic science knowledge of the process of proteins & carbohydrates digestion to understand the clinical manifestations of diseases that involve defective proteins' or carbohydrates' digestion &/or absorption.

Title of the lecture:	Biochemical aspects of digestion of lipids
Lecturer's name	Dr.Sumbul Fatma
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	sumbulfatma@gmail.com

Objectives:

- Understand the process of digestion of dietary lipids including the organs involved, the enzymes and the end products.
- Discuss the hormonal control of lipid digestion by CCK and secretins.
- Comprehend the role of chylomicrons as the carriers of dietary lipids including their assembly and secretion by enterocytes.
- Understand the clinical manifestations of the diseases associated with defective lipid digestion and absorption.

Title of the lecture:	Antiemetic drugs
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block / week	Gastrointestinal & Nutrition Block / 2
Email address	hananhhagar@yahoo.com

Objectives:

- Classify the main different classes of antiemetic drugs according to their mechanism of action.
- Know the characteristic pharmacokinetics & dynamics of different classes of antiemetic drugs.
- Identify the selective drugs that can be used according to the cause of vomiting.
- Learn the adjuvant antiemetics.
- Describe the major side effects for the different classes of antiemetics.

Title of the lecture:	Pathology and pathogenesis of acute and chronic pancreatitis
Lecturer's Name Department	Dr. Hala Kfoury Pathology
Block / Week	Gastrointestinal & Nutrition Block / 2
E-mail Address	hkfoury@ksu.edu.sa; halakfoury@hotmail.com

Objectives:

At the end of the two lectures the students will be able to:

- Recognize the predisposing factors of pancreatitis.
- Describe the different types of pancreatitis.
- Understand the pathogenesis of acute and chronic pancreatitis..

Introduction:

- Pancreatitis is well known in the medical practice. Systemic and/or local factors play a major role in the initiation, occurence and outcome of pancreatitis. The predisposing factors will be discussed. The pathogenetic mechanisms will also be addressed . A pathological description of the different types of pancreatitis will be considered.

Key Outlines:

- Pancreatitis : causes and clinical manifestations.
- Predisposing factors : stones, tumors etc...
- Pathology of Acute and Chronic pancreatitis including the complications of pancreatitis (i.e pseudocyst..).
- Pathogenetic mechanisms.

Summary:

- Pancreatitis may have an acute or a chronic form. The predisposing factors are widespread. An understanding of the causative mechanisms, clinical presentation, pathology and treatment will be covered.

Take home message:

The student will investigate into the causes of pancreatitis, the pathogenetic mechanisms and the histological findings related to acute and chronic pancreatitis

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotra

Further Reading:

- Harshmohan

Title of the lecture: GERD (Practical Sessions)	
Lecturer's Name:	Prof. Hala Kfoury
Department	Pathology
Block / Week	Gastrointestinal & Nutrition Block / 2
E-mail Address:	hkfoury@ksu.edu.sa; halakfoury@hotmail.com

Objectives:

At the end of these practical sessions, the students will be able to recognize, describe and understand the morphological appearance (both macroscopic and microscopic) of some of the common diseases of the GIT, hepatobiliary system and pancreas.

- Introductory presentation (using computer power point projector) of the gross pathology and histopathology slides used in teaching.
- The students will be asked to examine museum specimens , histopathology slides and pictures of both gross and microscopic sections relevant to the topics studied in GIT block lectures.
- All students will be given a CD containing pictures and photomicrograph of the gross pathology and histology to encourage self directed learning
- The students will be given the opportunity to visit the laboratories and familiarize themselves with the lab procedures.

Contents:

Gross pathology and histopathology section pictures of the following:

- **Salivary Glands:**
  - Pleomorphic adenoma of the salivary gland

- **II. Esophagus:**
  - Reflux/GERD.
  - Barrett's esophagus.
  - Squamous carcinoma of the esophagus.

- **Stomach:**
  - Chronic gastric ulcer.
  - Gastritis: Helicobacter-induced.
  - Adenocarcinoma of the stomach.

- **Small intestine :**
  - Chronic duodenal ulcer.
  - Celiac disease.
  - Carcinoid tumor

- **Large intestine:**
  - Crohn's disease.
  - Ulcerative colitis.
  - Adenomatous polyp of rectum/colon.
  - Familial polyposis.
  - Adenocarcinoma of the colon.

- **Hepatobiliary system:**
  - Chronic hepatitis.
  - Hepatic cirrhosis.
  - Hepato-cellular carcinoma.
  - Chronic cholecystitis with stones.

- **Pancreas:**
  - Chronic pancreatitis.
  - Pancreatic adenocarcinoma.

Title of the lecture: Nutritional requirements	
Lecturer's names	Sumbul Fatma
Department	Pathology (Biochemistry)
Block / Week	Gastrointestinal & Nutrition Block / 2
Email address	sumbulfatma@ksu.edu.sa

Objectives of the lecture:

Upon completion of this lecture, the students should be able to:

- Understand the basic terms of nutritional requirements that are important for establishing nutritional intake of a nutrient in a population.
- Interpret the food pyramid that recommends daily serving size from each food group for vegetarians and non-vegetarians.
- Identify dietary guidelines and goals that are necessary for good health
- Discuss energy requirement in humans including basic energy expenditure and the factors that affect it.
- Understand total parenteral nutrition (TPN) and its applications.

Background:

- Nutrients are the constituents of food necessary to sustain normal functions of the body.
- This lecture focuses on the kinds and amounts of macronutrients (fats, carbohydrates and proteins) that are needed to maintain optimal health and prevent chronic disease in adults.

Keywords:

- Nutritional requirements, EAR, RDA, AI, UL, malnutrition assessment, food pyramid, energy requirement, energy expenditure, dietary guidelines, dietary goals, total parenteral nutrition

Main concepts in the lecture:

- What is nutrition?
- Assessment of malnutrition.
- Dietary reference intakes (DRIs).
- Estimated Average Requirement (EAR).
- Recommended Dietary Allowance (RDA).
- Adequate Intake (AI).
- Acceptable Macronutrient Distribution Ranges (AMDR).
- Energy content of food, protein quality, negative and positive nitrogen balance.
- The Food Pyramid: dietary guidelines and goals.
- Energy requirement and expenditure in humans.

Conclusion:

- Basic standards of nutritional requirements are important for malnutrition assessment.
- Establishing these standards is essential for a population in order to avoid disease and maintain good health.
- Committees of American and Canadian experts organized by the Food and Nutrition Board of National Academy of Sciences have established Dietary Reference intakes (DRIs).
- The DRIs replace and expand on the recommended Dietary Allowances (RDA).

Take home messages:

- Understanding the basic terminology of human nutrition is essential for grasping the concept of assessing malnutrition and establishing nutritional requirement in a population.
- The primary purpose of setting these standards is to curb nutritional deficiencies in a population and hence the diseases associated with them.
- Establishing these requirements considers important factors including energy requirements and expenditure.
- There are four standards based on which these requirements are defined: Estimated Average Requirement (EAR), Recommended Daily Allowance (RDA), Adequate Intake (AI) and Tolerable Upper Intake Level (UL).
- Once these standards are set, recommendations are disseminated to public in the form of tools such as the food pyramid and dietary guidelines and goals, etc.
- The standards also cater to nutritional requirements of patients who are otherwise unable to feed normally.

Further reading:

- Lippincott's Biochemistry. 5th Edition, pp. 357-360. Lippincott Williams & Wilkins, New York, USA.

Title of the lecture: Macro and micro nutrients	
Lecturer's names	Dr. Sumbul Fatma
Department	Pathology (Biochemistry)
Block / Week	Gastrointestinal & Nutrition Block / 2
Email address	sumbulfatma@gmail.com

Objectives of the lecturer:

- Understand the nutritional importance of dietary macro and micronutrients.
- Identify major dietary sources and RDAs of macro and micronutrients.
- Evaluate the nutritional quality of proteins, the types of dietary carbohydrates, fibers and fats and their benefits.
- Discuss the role of macronutrients in causing diseases or conditions such as nitrogen imbalance, diabetes, obesity, atherosclerosis and heart disease.
- Understand the functions of micronutrients (vitamins, minerals and trace elements) and the diseases due to their deficiencies.

Background:

- Nutritional importance, RDAs and dietary sources of macro and micronutrients.
- Functions of macro and micronutrients.
- Diseases or conditions associated with malnutrition and excessive intake of these nutrients

Keywords:

- Dietary proteins, dietary carbohydrates, dietary lipids, dietary fibers, protein quality, nutrition, malnutrition, nitrogen balance, recommended daily allowance, essential amino acids, essential fatty acids, vitamins, minerals, trace elements

Main concepts in the lecture:

Macronutrients, as the name suggests, are nutrients that are required by the body in gram quantities in order to maintain body functions avoid disease. These include proteins, carbohydrates and fats. Macronutrients provide energy to the body; the major source being carbohydrates and fats. Dietary intake of macronutrients according to their RDAs is essential for maintaining good health. However, their excessive intake can lead to diseases or conditions such as diabetes, obesity, atherosclerosis and heart disease.

Micronutrients such as vitamins, minerals and trace elements are required in smaller amounts mainly in milligram or microgram quantities. They do not provide energy to the body rather they help in various body functions such as metabolism. Vitamins act as coenzymes for various biochemical reactions in the body. The RDA values for majority of micronutrients have been defined that ensures proper intake either from diet or supplements.

Conclusion:

- The macronutrients are the major source of energy for the body whereas micronutrients support the body functions.
- Malnutrition of macro and micronutrients is associated with a number of diseases and clinical conditions.

Take home messages:

- Macro and micronutrients are essential for energy and maintaining good health.
- Various diseases are associated either with malnutrition or excessive intake of these nutrients.

Further reading:

- Lippincott's Biochemistry, 6th Edition, pp 357-394, Lippincott Williams & Wilkins, New York, USA.
- Textbook of Biochemistry with Clinical Correlations 6th Edition, pp 1071-1096, Thomas M. Devlin, Wiley, USA.

Title of the Lecture: Plasma proteins	
Lecturer's name	Dr. Sumbul Fatma
Department	Pathology (Biochemistry)
Block/ Week	Gastrointestinal & Nutrition Block / 2
Email address	sumbulfatma@gmail.com

Objectives of the lecture:

- Identify types and various functions of plasma proteins.
- Discuss the role of plasma proteins in the diagnosis of diseases and conditions
- Interpret the normal and abnormal electrophoretic patterns of plasma proteins
- Identify the role positive and negative acute phase proteins in various diseases

Background:

- Plasma proteins perform many important functions in the body including maintaining oncotic pressure, transporting substances, defense, clotting and fibrinolysis
- The electrophoretic pattern of plasma proteins provides important information for the diagnosis of various diseases or conditions
- Positive and negative acute phase proteins are of diagnostic significance in differentiating various processes in the body such as infection and inflammation.

Keywords:

- Plasma proteins, electrophoresis, transport, oncotic pressure, albumin, globulins, thrombin, multiple myeloma, acute phase proteins

Main concepts in the lecture:

There are more than 300 different plasma proteins that play essential roles in various body functions including maintaining oncotic pressure, fighting infections, transporting hormones and other substances, clotting and fibrinolysis. Since many pathological conditions affect the levels of plasma proteins, their measurement provides important clues to diagnosis of those conditions. Each type of plasma protein exhibits a typical electrophoretic pattern that can be used to compare with corresponding abnormal pattern to diagnose a disease or condition. Several plasma proteins are used as markers for the diagnosis of multiple myeloma, infection, pulmonary emphysema, malignancies and inflammation. Some plasma proteins are found to be in high concentration because of infection, trauma, surgery and inflammation. These are called positive acute phase proteins. The levels of certain plasma proteins decrease in inflammation to compensate amino acids for positive acute phase proteins. These are called negative acute phase proteins.

Conclusion:

- Plasma proteins play important roles in various body functions.
- Many pathological conditions affect the levels of these proteins.

- The electrophoretic pattern of plasma proteins is a diagnostic tool for identifying various diseases.

Take home messages:

- Plasma proteins play essential roles in a number of cellular functions.
- They possess diagnostic significance in identifying various pathological conditions.

Further reading:

- Lecture Notes in Clinical Biochemistry, 9th Edition, AF Smith, pp 86-97, Blackwell Publishing, UK.
- Clinical Diagnosis and Management by Laboratory Methods, 19th Edition, John Bernard Henry, Saunders, USA.

Title of the lecture:	Anatomy and histology of the small intestine
Lecturer's name	Prof. Raeesa Abdultawab / Dr. Jamilah El Medany
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 3
Email address	rmohammad@ksu.edu.sa / galmadani@ksu.edu.sa

Objectives:

- List the different parts of small intestine.
- Describe the anatomy of duodenum, jejunum & ileum regarding: the shape, length, site of beginning & termination, peritoneal covering, arterial supply & lymphatic drainage.
- Differentiate between each part of duodenum regarding the length, level & relations.
- Differentiate between the jejunum & ileum regarding the characteristic anatomical features of each of them.
- Describe the microscopic structure of the three regions of the small intestine: Duodenum, Jejunum, Ileum.

Title of the lecture:	Physiology of the small intestine: motility and secretion
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 3
Email address	Hayam_gad@hotmail.com

Objectives:

- Motility in the small intestine.
- Control of intestinal motility.
- Secretions of the small intestine
- Digestion of carbohydrates, proteins and fats**.**
- Basic principles of gastrointestinal absorption, Absorption of carbohydrates, Absorption of proteins, Absorption of fats, Absorption of vitamins, Absorption and secretion of electrolytes and water.

Title of the Lecture:	Normal flora introduction to infectious diarrhea
Lecturer's name	Prof. Hanan Al Habib
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	hahabib@ksu.edu.sa

Objectives of the lecture:

- Define and recognize the various types of acute diarrheal illness
- Describe the epidemiology the host defenses in preventing the gastrointestinal infection
- Explain pathogenesis by which *Escherichia coli* ,*Campylobacter* and Y*ersinia* and their management
- Discuss the microbiological methods used for diagnosis of each of the bacterial agents including microscopy, selective media for maximal recovery
- Describe the pathogens, risk factors, clinical presentation and prevention of food poisoning travelers and antibiotic associated diarrhea.
- Name the etiological agents causing food poisoning and their clinical presentation

Background and Summary:

Acute diarrheal illness is one of the most common problems evaluated by clinicians. It is a major cause of morbidity and mortality world wide. Most of healthy people have mild illness but other might develop serious squeals so it is important to identify those individuals who require early treatment.

Main concepts in the lecture:

- Acute Diarrhea

  Diarrhea defined as loss motion more than three times per day. There various type of diarrheal illnesses presentations according to clinical presentations and etiological agents which include:

  - Infectious diarrhea: caused by :viral , Bacterial organisms (*Campylobcator, Shigella, Solmnella, Yersinea, Cholera & E. coli.*
  - Food poisoning : caused by :*Staphylococcus aureus, Clostridium perferinges & Bacillus.*
  - Traveler diarrhea: caused by : Enterotoxogenic *E-coli* .
  - Antibiotic associated diarrhea: caused by *Clostridium defficile.*

- Risk Factors

  Some patients will be at risk for diarrheal diseases more than the others eg. eating from restaurant, family member with Gastrointestinal symptoms, recent travel to developing countries, patient underlying illness and mediation and patients on antibiotics

- Clinical Presentation and Pathogenic Mechanism

  Gastroenteritis can be either due to enterotoxin production or invasion of microorganisms. Enterotoxin will lead mainly to vomiting and later diarrhea with no pus or mucus (no inflammation) and it will be rapid onset .*Vibreo cholerae, Staphylococcus aureus, Clostridium perfringens and Bacillus cereus* and other viral and some parasitic infections are the major cause of this disease . Invasive disease will lead to pus and blood in the stool and fever due to inflammation *caused by Shigella, Salmonella* spp., *Campylobacter*, some E-coli and *Entameba histolytica* which can lead to dysentery syndrome gross blood and mucous affect colonic mucosal surface of the bowel with extension to lymph nodes.

- Campylobacter

  The source of this organism can be dogs, cats, birds and poultry which can contaminate water milk and meat and person to person transmission is possible. Clinical the incubation period is 2-6 days and the patient present with abdominal cramp, bloody diarrhea, nausea and vomiting is rare. Usually it is self-limiting in 2-6 days and some time can lead to chronic carrier status or complication like *Guillian Barrie'* syndrome or reactive arthritis. Specific media needed to cultivate this organism that contain antibiotics and they grow in general in higher temperature than other enteric bacteria. Treatment needed usually they sensitive to ciprofloxacin, erythromycin or tetracycline

- E.coli

  Based on virulence factors, clinical manifestation, epidemiology and different O and H Serotype. There are five major categories of diarrheagenic *E.coli*:

  a. Enterotoxigenic *E.coli*
  It is a major cause of traveler's diarrhea affecting infant and adult in developing countries. It has heat-labile toxin (LT) and heat-stable toxin (ST) each has two fragments (A and B). Patient presents with watery diarrhea, abdominal cramps and some time and no routine diagnostic method.

  b. Enteroinvasive *E-coli*
  It produces dysentery similar to *Shigella* spp. Fever, severe abdominal cramps, malaise and watery or bloody diarrhea.

  c. Enteropathogenic *E-coli*
  Infantile diarrhea and causes outbreak in hospital nurseries and day- care centers.

  Patient will present with low grade fever, malaise, vomiting and diarrhea and the tool mucous but no blood.

  d. Entero hemorrhagic *E-coli*
  E.coli -0157H7 is the most common cause of hemorrhagic diarrhea, colitis and hemolytic uremic syndrome (HUS) which is low platelet count, hemolytic anemia and kidney failure. It is fetal disease in young and elderly persons in nursing homes.

Undercooked hamburgers, unpasteurized dairy products, apple cider, cookie dough. A subunit is active component of the toxin and B subunit is cytotoxic

Vertoxin I and vertoxin II are similar to Stx_1 (Shiga-toxin I & II). Other than 0157H7 can cause HUS.

e. Enteroadherant *E-coli*
Can cause pediatric diarrheal disease by adhering to the surface of the intestinal mucosa. Patient presents with watery diarrhea, vomiting, dehydration and abdominal pain that can last two or more weeks.

- Yersinia enterocolitica
  It can cause mesenteric lymphadenitis in children and septicemia in immunocompromized hosts. It is common in Europe, USA, and Canada. Source Cats, dogs, swine (chitterlings).It can survive cold temperatures and transfusion of packed red blood cells. Patient It presents with enteritis, arthritis and erythema nodosum and generalize infection in adult and children 1-5 years old. It is usually mild in old children and adult and mimic appendicitis. This organism growth at 25°c-30°c media Cefsulodin-Igrasan-Novabiacin

- C.difficile
  It can cause antibiotic associated diarrhea due to disruption of the endogenous bacterial flora of the colon. Transmit from person to person via Fecal-Oral route. Have been cultured from in animate hospital surfaces. *C.difficile* produces toxin A and B that can bind to surface epithelial cell receptors leading to inflammation mucosal injury and diarrhea. Pseudomembrane can result (neutrophils, fibrin, and cellular debris in the colonic mucosa) and toxic megacolon. Patient presents with fever, leukocytosis, abdominal pain and diarrhea. It can be diagnosed by toxin detection by EIA (enzyme immunoassay) and treated by Metronidazole± Vancomycin in addition to supportive care and surgery in severe cases

Take home messages:

- Gasteroenteritis is a common a serious medical condition which can lead to major complication
- Understanding the various clinical presentations can help in identifying the etiological agents.
- Diagnosis depends on the epidemiology, clinical presentation and confirmed by isolation of the organism from the stool.
- Hand washing and other preventive measures are needed to prevent and control the outbreak.
- Antibiotic treatment is not indicated in most of the cases but fluid replacement and other supportive care usually needed.

Further reading:

- *Sherries* Medical Microbiology, an Introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher: McGraw Hill.

Title of the lecture:	Pathology and the mechanisms of malabsorption
Lecturer's Name	Prof. Hala Kfoury
Department	Pathology
Block / Week	Gastrointestinal & Nutrition Block / 3
E-mail Address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture the students will :

- Understand that the malabsorption is caused by either abnormal digestion or small intestinal mucosa
- Know that malabsorption can affect many organ systems (alimentary tract, hematopoietic system, musculoskeletal system, endocrine system, epidermis, nervous system)
- Concentrate on celiac disease and lactose intolerance as two examples of malabsoption syndrome.

Introduction:

Malabsorption is a clinical term that encompasses defects occurring during the digestion or absorption of food nutrients. The digestion or absorption of a single nutrient component may be impaired, as in lactose intolerance due to lactase deficiency. When a diffuse disorder, such as celiac disease or Crohn's disease, affects the intestine, the absorption of almost all nutrients is impaired. The presenting symptom is usually Steatorrhea (passage of pale, bulky, and malodorous stools) as a result of fat malabsorption.

Key Outlines:

- Definition and pathophysiology of malabsorption
- Clinical features of malabsorption
- Pathology, pathogenesis, clinical features and complications of Celiac disease
- Pathophysiology and clinical features of lactose intolerance

Take home message:

- Malabsorption is due to a defect in
- small intestinal mucosa,
- pancreas or
- bile

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotran
- Harrison's Principles of Internal Medicine 16th Edition.

Title of the lecture:	Cholera
Lecturer's name	Dr. Fawzia Al Otaibi
Department	Pathology (Microbiology)
Block / week	Gastrointestinal & Nutrition Block / 3
Email address	Ofawzia04@ksu.edu.sa / ofawzia@ksu.edu.sa

Objectives:

- Know the epidemiology of cholera and history of cholera
- Recognize the microbiological characteristics of cholera
- Define the pathogeneses of cholera
- Describe the clinical features of cholera
- Outline the methods for laboratory diagnosis
- Discuss the management of cholera
- List major strategies for prevention and control of outbreak

Title of the lecture:	Anatomy of the omentum
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	salsharawi@ksu.edu.sa

Objectives:

- Brief knowledge about peritoneum as a thin serous membrane and its main parts; parietal and visceral.
- The peritoneal cavity and its parts the greater sac and the lesser sac (Omental bursa).
- The peritoneal folds: omenta, mesenteries, and ligaments.
- The omentum, as one of the peritoneal folds
- The greater omentum, its boundaries, and contents.
- The lesser omentum, its boundaries, and contents.
- The Omental bursa, its boundaries.
- The Epiploic foramen, its boundaries.
- Mesentery of the small intestine, and ligaments of the liver.
- Nerve supply of the peritoneum.
- Clinical points.

Title of the lecture:	Radiology of the abdomen
Lecturer's name	Dr. Faten Muhaideb
Department	Radiology
Block / week	Gastrointestinal & Nutrition Block / 3
Email address	fatenalmohideb@gmail.com

Objectives:

- To know various radiological investigations used for GIT.
- To understand step wise approach in requesting GIT radiology investigations.
- To be familiar with radiological appearance (anatomy) seen in various imaging modalities.
- To Interpret plan x-ray radiograph of abdomen with common pathologies.
- To know the common GIT pathologies presentation.
- To understand step wise approach in requesting GIT radiology investigations.
- To know common radiological pathologies in GIT

Title of the Lecture: Intestinal Helminthes	
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	monabadr@hotmail.com

Objectives of the lecture:

- Name the 3 main groups of parasitic Helminths and their characteristic morphological features.
- Know the 5 common examples of Nematodes with their scientific and common names.
- Describe the life cycle of these 5 examples of Nematodes with pathology, diagnosis and treatment.
- Describe the life cycle of Taenia saginata and T. solium and Hymenolepis nana
- Describe the life cycle Echinococcus granulosus and diagnosis
- Know treatment of Tapeworms.

Background:

- Foundation block will give students the required background

Title of the Lecture:	Viral Gastroenteritis
Lecturer's name	Dr. Malak El Hazmi
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	melhazmi@ksu.edu.sa

Objectives of the lecture:

- Identify and compare between the main viral etiology of gastroenteritis (rotavirus, adenovirus, calicivirus, astrovirus)
- Describe the essential characteristics of these viruses.
- Explain the epidemiology and the mode of transmission of these viruses.
- Explain the clinical manifestations of gastroenteritis.
- Describe and interpret the laboratory methods used to diagnose these viral infections.
- Describe the treatments and the prevention measures available for these viral infections

Keywords:

- (Viral gastroenteritis , rotavirus, enteric adenoviruses , calicivirus, astrovirus, norovirus)

Background:

- Viral gastroenteritis(GE) remains one of the major causes of death in young children. The main factors for high incidence and mortality are unsafe water or inadequate sanitation, A range of bacteria and parasites has been identified, but these account less than half of investigated cases in children . A number of different viruses such as rotavirus , enteric adenoviruses , astro and caliciviruses are the major cause of GE especially in young infants and children.

Main concepts in the lecture:

- Viral gastroenteritis

  Gastroenteritis means inflammation of the stomach and small and large intestines. Viral GE is an infection caused by a variety of viruses that results in vomiting or diarrhea.

- Viral etiology

  Many different viruses can cause gastroenteritis, including rotaviruses; noroviruses; adenoviruses, types 40 and 41; sapoviruses; and astroviruses.

- Epidemiology

  viral GE is contagious. They are transmitted by the fecal-oral route.

Viral GE affects people in all parts of the world. Each virus has its own seasonal activity Viral GE occurs in people of all ages and backgrounds. However, some viruses tend to cause diarrheal disease primarily among people in specific age groups. Rotavirus infections are the most common cause of diarrhea in infants and young children under 5 years old. Adenoviruses and astroviruses cause diarrhea mostly in young children, but older children and adults can also be affected. Noroviruses infect persons of all ages, including older children and adults.

- Clinical features

  The main symptoms of viral gastroenteritis are watery diarrhea and vomiting. The affected person may also have headache, fever, and abdominal cramps ("stomach ache").

  People who get viral gastroenteritis almost always recover completely without any long-term problems. Immune compromised persons are at risk for dehydration.

- Diagnosis

  None of theses viruses grows readily in routine cell cultures but all have morphological characteristic recognizable by electron microscopy . Currently common viruses causing gastroenteritis can be rapidly detected in a stool by immunoassay .

- Treatment

Most cases of viral gastroenteritis resolve over time without specific treatment. The primary goal of treatment is to reduce the symptoms, and prompt treatment may be needed to prevent dehydration. Severe dehydration may require intravenous fluids and hospitalization. Untreated severe dehydration can be life threatening.

- Prevention
  - Viral gasteroentritis

    can be prevented by improving hygiene meaures and sanitation. Rotavirus gastroenteritis can also be prevented by vaccines. Currently there are two licensed rotavirus vaccines available .These vaccines are given to children in their first year of life with other childhood vaccines.

Conclusion:

Rotavirus , enteric adenoviruses , astroviruses and caliciviruses are the major cause of GE especially in young infants and children. They can be rapidly detected in a stool by immunoassay No specific treatment or prevention are available except rotvirus vaccines to prevent rotavirus GE.

Take home messages:

- Viral gastroenteritis is a highly contagious infection of the intestines caused by one of several viruses.
- Gastroenteritis affects all age group, but it is mainly disease of infants and children.
- The main symptoms are watery diarrhea, abdominal cramping and vomiting.
- Lab diagnosis depends on detection of these viruses in stool.
- Vaccine is available for rotaviruses.

Further reading:

- Notes on Medical Microbiology. By; Morag C. Timbury, A. Christine McCartney, Bishan Thakker and Katherine N. Ward. 2002. Pages; 338 - 344.
- Medical Microbiology. By: David Greenwood, Richard C.B. Slack, John F Peutherer and Mike Barer, 17th Edition, 2007. Pages;545-551,565-571

Title of the lecture: Pathophysiology and mechanisms of diarrhea	
Lecturer's Name:	Prof. Hala Kfoury
Department	Pathology
Block / Week	Gastrointestinal & Nutrition Block / 3
E-mail Address:	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture the students should:

- Understand the physiology of fluid in small intestine
- Describe the pathophysiology and causes of various types of diarrhea (Secretory, osmotic, Exudative, Motility-related)
- Define acute diarrhea and enumerate its common causes
- Define chronic diarrhea and enumerate its common causes

Introduction

Diarrhea is the passage of fluid feces and occurs when the rate of movement of intestinal contents is increased so that complete digestion and absorption of fluid in the intestine fails to occur. The volume of feces is usually greatly increased in diarrhea, leading to increased frequency of evacuation and increased loss of water and electrolytes. Diarrhea may result from (1) increased fluid secretion into the intestine (secretory diarrhea); (2) the presence of increased amounts of osmotically active substances in the intestinal lumen; (3) inflammation of the intestine—mainly the small intestine; (4) increased peristalsis, eg, resulting from stimulation of smooth muscle by serotonin in carcinoid syndrome and in irritable bowel syndrome; (5) failure of colonic water absorption, eg, after surgical removal of the colon;

Key Outlines:

- Introduction to physiology fluid in small intestine
- Mechanisms of various types of diarrhea (osmotic, secretory, inflammatory, and motility related)
- Acute diarrhea, definition, causes, and approach
- Chronic diarrhea, definition, causes, and approach

Take home message:

More than 90% of cases of acute diarrhea are caused by infectious agents; these cases are often accompanied by vomiting, fever, and abdominal pain. Diarrhea lasting >4 weeks warrants evaluation to exclude serious underlying pathology. In contrast to acute diarrhea, most of the many causes of chronic diarrhea are noninfectious. The classification of chronic diarrhea by pathophysiologic mechanism facilitates a rational approach to management

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotran
- Harrison's Principles of Internal Medicine 16th Edition

Title of the Lecture:	Shigella & salmonella
Lecturer's name	Prof. Hanan Al Habib
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	hahabib@ksu.edu.sa

Objectives of the lecture:

- Develop an algorithm using biochemical to identify and classify *Salmonella* and *Shigella.*
- Describe the antigenic structures and virulence factors of *Salmonella* and *Shigella*.
- Compare the pathogenesis of various species of *Salmonella* and *Shigell*a.
- Describe the clinical features and risk factor for the infection with two organism
- Describe the general concepts for management of Gastroenteritis caused by these two organisms.

Background:

- *Shigella* and *Salmonella* are major etiological agents of infectious diarrhea worldwide.
- *Salmonella* inhabit the GI tracts of animals and human acquired the infection by ingesting the organisms in contaminated animal food products or poultry, milk and eggs. Some *Salmonella* transmitted by human carries.
- *Shigella* spp. are associated with human carriers due to improper sanitary condition and poor personal hygiene

Main concepts in the lecture:

- Salmonella are motile gram negative rods.
  - There are over 2500 serovars based on O and H antigen.
  - Non-typoidal Salmonella spp. are found in animals and is acquired from food or pets
  - While Salmonella typhi is strictly human pathogens and is acquired from food or water contaminated with organisms from another case or a carrier.
  - *Salmonella typhimurium* and *Salmonella enteritidis* are the two most frequent cases of non -typhoidal *Salmonella* spp. gastroenteritis
  - *Salmonella typhi* penetrates the gastrointestinal epithelial and is disseminated by the blood stream.
  - Non- typhoidal *Salmonella* spp. generally remain with the gastrointestinal tract, although some serovars may disseminate in the blood.
  - *Salmonella typhi* stimulate a monocytic response with gastrointestinal symptoms.
  - Non -typhoidal *Salmonella s*pp. induce a granulocytes response with pronounced gastrointestinal symptoms.
  - *Salmonella typhi* localized to the cells and distributed to all body organs by the blood, giving raise the organ specific disease.
  - Enteric fever might present as a pyrexia of unknown origin (PUO) and abdominal symptoms with incubation period of 5-21 days.

- Rash (rose spots), relatively bradycardia, leucopenia and hepatosplenomegaly may be present.
- Complication of enteric fever include cholecystis, intestinal perforation, endocarditis and oesteomylitis.
- Non -typhoidal gastroenteritis is self -limiting its incubation period 12-48 hours and it presents with fever, abdominal pain and diarrhea.
- Enteric fever is diagnosed by isolation and identification of the organisms and highest yield of organism from the bone marrow.
- Identification of *Salmonella* spp. depends on biochemical reaction and serological reaction using the Kaufmann-white scheme.
- Today many of *Salmonella* are resistant to first line treatment which Chloramphenicol, Cotrimoxazol, Ampicillin and Ciprofloxacin.
- Treatment with Azithromycin or ceftriaxon in patients from India and SE Asia and treatment with ciprofloxacin in patients from other area.
- Protection from enteric fever is provided by vaccination.
- Antibiotic are not usually required in case of gastroenteritis caused by non-typhoidal *Salmonella.*
- Prevention of gastroenteritis is by adequate hygiene standards at all points in the food chain.

- Shigella is non motile gram negative bacilli does not ferment lactose.

  - *Shigella c*an cause bacillary dysentery due to inflammation and invasion of intestinal colonic mucosa leading to cell death.
  - The term normal flora is used to describe microorganisms that are frequently founds in body sites in normal healthy individuals.
  - It can escapes to the cytoplasm and spread from one cell to another accelerates cell death and forming of necrotic cells.
  - *Shigella* causes marked inflammation and invasion but rarely invade the blood stream.
  - *Shigella* transmitted from person to person via Fecal- oral route (the infective dose is as few as 10-100 bacilli); spread via poor hygiene and contaminated water /foods (salads, sandwiches, fruits)
  - Incubation period is 2-7 days and symptoms begins with abdominal pain, watery diarrhea and after 1-3 days, small volume of blood- stained and mucous diarrhea develops with tenesmus. Patient may become toxic, with fever ,headache, malaise hemodynamic instability.
  - Diagnosis through isolation of the organism from the stool specimen and definite identification depends on biochemical reaction and serological testing.
  - *Shigella* infection is self- limited in some cases and is treated with oral rehydration and electrolytes replacements.
  - Antibiotics can be used to reduce the duration of illness and carrier state
  - IV ceftriaxone and ampicillin, oral TMP-SMX or ciprofloxacin or Doxycycline
  - Most infections resolve with 1-2 weeks but complication like hemolytic uremic syndrome, Reiter's syndrome and ankylosing spondylitis can occur.

Conclusion:

- Shigella and Salmonella are major etiological agents of infectious diarrhea worldwide.
- Salmonella spp. inhabit the GIT of animals and human acquired the infection by ingesting the organisms in contaminated animal food products or poultry, milk and eggs. Some salmonella transmitted by human carries.
- Shigella spp. are associated with human carriers due to improper sanitary condition and poor personal hygiene

Take home messages:

- The source of non- typhoidal *salmonella* is animals while human is the reservoir for *salmonella typhi*.
- Non- typhoidal *Slmonella* can cause gastroenteritis. Which self- limiting illness and does not require any antibiotic treatment in most of the cases.
- *Salmonella typhi* leads mostly to systemic infection with dissemination through blood to RES and any organ can be affection antibiotic treatment is a must.
- Stool sample is the best specimen for the diagnosis of enteric pathogens and blood and bone marrow might be need to diagnose enteric fever.
- *Shigella* source is human similar to *Salmonella typhi* but they rarely disseminate to blood instead they invade the colonic mucosa leading to severe necrosis of the colonic epithelial cell and treatment indicated in some cases.

Further reading:

- Sherries Medical Microbiology, an introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher: Mc Graw Hill.

Title of the lecture:	Nutrition Education
Lecturer's name	Dr. Rufaida AdDabagh
Department	Family and Community Medicine
Block / week	Gastrointestinal & Nutrition Block / 3
Email address	rdabbagh@ksu.edu.sa

Objectives:

At the end of the lecture you should gain the ability to:

- Define nutrition education.
- Recognize the importance of nutrition education.
- Understand methods used in nutrition education.

Title of the lecture:	Anatomy and histology of the large intestine
Lecturer's name	Prof. Raeesa Abdultawab / Dr. Jamilah El Medany
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	rmohammad@ksu.edu.sa / galmadani@ksu.edu.sa

Objectives:

- Describe the anatomy of the *parotid* gland: position, shape, structures within it, innervation and parotid duct.
- Describe the anatomy of the submandibular and sublingual salivary glands: location, shape, parts, ducts and innervation of the glands.
- Describe the microscopic structure of the major salivary glands in correlation with function.

Title of the Lecture:	Intestinal protozoa
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	monabadr@hotmail.com

Objectives of the lecture:

- Know morphology of cysts and trophozoits of Giadia lamblia parasites
- Describe life cycle of Giardia parasites
- Describe Giardia trophozoits in tissue sections
- Discuss the clinical picture of Giardia parasites (Typical and Atypical).
- How to diagnose Giadia in the labs
- Know the chemotherapy against Giardia parasites.
- Summarize general features of Intestinal Entamoebae.
- Know the six types of Entamoebae.
- Compare between E. histolytica and E. dispar.
- Describe Life cycle of E. histolytica
- Discuss Pathology of E. histolytica (intestinal and extra-intestinal).
- Diagnosis and treatment of Amoebae
- Life cycle of Cryptosporidium and diagnosis

Keywords:

- (Trophozoites, Cyst., metronidazole., E. histolytica., E. dispar., Amoeboma-Amoebic dysentery, Cryptosporidium, lesion.)

Background:

- Foundation Block is enough to give prior knowledge for this block

Title of the lecture:	Physiology of the colon: motility
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	Hayam_gad@hotmail.com

Objectives:

- Parts of the Colon
- Functions of the Colon
- The physiology of Different Colon Regions
- Secretion in the Colon
- Nutrient Digestion in the Colon
- Absorption in the Colon
- Bacterial Action in the Colon
- Motility in the Colon
- Defecation Reflex

Title of the lecture:	Schistosomiasis
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	monabadr@hotmail.com

Objectives:

- know the global distribution of schistosomiasis
- describe the life cycle of schistosomiasis
- compare relation between chronic schistosomiasis and portal hypertension
- know pathology, diagnosis and treatment of schistosomiasis
- know life cycle of Fasciola hepatica
- know pathology , diagnosis and treatment of Fasciola hepatica
- compare between true infection and sheep liver infected with Fasciola hepatica which lead to false infection

Title of the lecture: Colonic tumours and polyps	
Lecturer's name	Dr. Maha Arafa
Department	Pathology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	marafa@hotmail.com

Objectives:

- Upon completion of this lecture the students will :
- Differentiate between the neoplastic and non-neoplastic polyps and to know common types of intestinal polyps
- Know the clinical presentation of left and right sided colon cancer, and the environmental factors that increase its risk
- Understand the Pathology and pathogenesis of colon cancer

Introduction:

Many different pathologic processes involving the mucosa result in polyps that project into the lumen of the intestine. Intestinal polyps may be foci of epithelial hyperplasia, epithelial neoplasms, hamartomas, or retention polyps. Not all polyps are associated with epithelial proliferation. Inflammation (inflammatory polyps), lymphoid hyperplasia (lymphoid polyps), and mesenchymal neoplasms (lipoma, leiomyoma) may also result in polyps. Malignant epithelial neoplasms (ie, carcinomas) account for 95% of intestinal malignancies. Most of these occur in the colon and rectum. They represent one of the prime challenges to the medical profession, because they almost always arise in adenomatous polyps that are generally curable by resection. With an estimated 134,000 new cases per year and about 55,000 deaths, this disease accounts for nearly 15% of all cancer-related deaths in the United States.

Key Outlines:

- Neoplastic and Non-neoplastic intestinal polyps
- Relationship of Neoplastic Polyps to Carcinoma
- Familial Polyposis Syndrome
- Risk factors and Carcinogenesis of colon cancer
- Clinical presentation of colon cancer

Take home message

- Intestinal Adenoma (adenomatous polyp) is a preneoplastic lesion that predispose to colorectal carcinoma.

Further reading:

- Pathologic Basis of Disease, Robbins and Cotran.

Title of the lecture: Treatment of dysentery and amoebiasis	
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block / Week	Gastrointestinal & Nutrition Block / 4
E-mail Address	hananhhagar@yahoo.com

Objectives:

- To understand different causes of dysentery.
- To describe different classes of drugs used in treatment of both bacillary dysentery and amebic dysentery.
- To be able to describe actions, side effects of drugs for treating bacillary dysentery.
- To understand the pharmacokinetics, actions, clinical applications and side effects of antiamebic drugs.
- To be able to differentiate between types of antiamebic drugs; luminal amebicides, and tissue amebicide.

Keywords:

- Amebic, dysentery, bacillary dysentery, oral rehydration solution .anti-amebic and antibiotics in dysentery.

Background:

- Pathogenesis of amebic dysentery
- Pathogenesis of Bacillary dysentery
- Clinical presentation of different types of dysentery
- Management of amebic dysentery

Main concepts in the lecture:

- The lecture will focus at the brief over view of the pathogenesis and clinical presentation of the amebic and bacillary dysentery.
- Management of dysentery using appropriate pharmacologic therapy.
- Mechanism of action and adverse effects of drugs used in the management of dysentery
- Discuss the selection of drugs according to patients age and disease conditions.

Conclusion:

- Pharmacologic management of amebic and bacillary dysentery
- Mechanism and toxicity profile of drugs
- Rationale of selection of drugs in the management of dysentery

Take home messages:

- Amebic and bacillary dysentery are treatable conditions.
- Judicial use of pharmacologic therapy could be safe and effective
- Oral rehydration is the first step to manage dysentery

Further reading:

- Bertram G. Katzung, Anthony J. Trevor (2014).13th Edition. Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

Title of the lecture:	Inflammatory bowel Disease in Ulcerative colitis
Lecturer's name	Dr. Maha Arafah
Department	Pathology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	marafa@hotmail.com

Objectives:

- Know the two forms of idiopathic inflammatory bowel disease (IBD).
- Describe Ulcerative Colitis with respect to: clinical features and extra-intestinal manifestations, pathogenesis, pathology (gross and microscopic features), complications (especially adenocarcinoma preceded by dysplasia)

Title of the lecture:	Irritable bowel syndrome
Lecturer's name	Dr. Othman Al Harbi
Department	Medicine
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	alharbiothman@hotmail.com

Objectives:

- Understand the hypothesis explain the pathphysiology of IBS.
- Common sign and symptoms
- Rome III criteria of diagnosis
- Introduction to management of IBS

Title of the lecture:	Drugs used in treating constipation and IBS
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block/ Week	Gastrointestinal and Nutrition Block / 4
E-mail Address	hananhhagar@yahoo.com; hhagar@ksu.edu.sa

Objectives:

- Define constipation
- Know the different symptoms of constipation
- Know the different lines of treatment of constipation
- Identify the different types of laxatives
- Discuss the pharmacokinetics, dynamics, side effects and uses of laxatives
- Discuss the difference between different treatment including bulk forming laxatives, osmotic laxatives, stimulant laxatives
  And stool softeners (lubricants).

- Define irritable bowel syndrome (IBS).
- Identify the pharmacokinetics, dynamics, side effects and uses of drugs used for IBS.

Keywords:

- Constipation, laxatives, bulk forming laxatives, osmotic laxatives, stimulant laxatives, lubricants, IBS, Alosteron, Tegaserod).

Background:

- Physiology of gastrointestinal motility including small intestine and colon.
- Causes and pathogenesis of constipation
- Causes and pathogenesis of IBS

Main concepts in the lecture:

- The lecture will focus at the brief overview of the causes and clinical presentation of the constipation and IBS
- Management of constipation and IBS using appropriate pharmacologic therapy.
- Mechanism of action and adverse effects of drugs used in the management of constipation and IBS
- Discuss the selection of drugs according to disease conditions.

Conclusion:

- Pharmacologic management of constipation and IBS
- Mechanism and toxicity profile of drugs
- Rationale of selection of drugs in the management of constipation and IBS

Take home messages:

Constipation is a treatable condition but can be avoided using special precautions.

- Judicial use of pharmacologic therapy could be safe and effective.

Further reading:

- Bertram G. Katzung, Anthony J. Trevor (2014).13th Edition. Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

Title of the lecture:	Introduction of inflammatory disease-Crohn's disease
Lecturer's name	Dr.Maha Arafah
Department	Pathology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	

Objectives:

- Know the two forms of idiopathic inflammatory bowel disease (IBD).
- Compare and contrast Crohn disease with respect to: clinical features and extra-intestinal manifestations, pathogenesis, pathology (gross and microscopic features), complications (especially adenocarcinoma preceded by dysplasia)

Title of the lecture:	Drugs used in IBD and biological and immune therapy of IBD
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	hananhhagar@yahoo.com

Objectives:

- Define inflammatory bowel disease.
- Differentiate between ulcerative colitis and Crohn' disease.
- Define the stepwise treatment of IBD.
- Discuss the pharmacokinetics, pharmacodynamics, uses and adverse effects of 5-amino salicylic acid compounds (5-ASA), glucocorticoids, immunomodulators and biological therapy (TNF-α inhibitors).
- Compare between drugs used for induction of remission and those used for maintenance of remission.

Title of the lecture: Anatomy, histology& radiology of the small & large intestine -Practical	
Lecturer's name	Dr.Sanaa Al Sharawani / Dr. Faten Al Mohaideb
Department	Anatomy / Radiology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	salsharawi@ksu.edu.sa/ fatenalmohideb@gmail.com

Objectives:

- List the different parts of small and large intestine.
- Describe the anatomy of duodenum, jejunum & ileum regarding: the shape, length, site of beginning & termination, peritoneal covering, arterial supply & lymphatic drainage.
- Describe the anatomy of different parts of large intestine regarding: *the* surface anatomy, peritoneal covering, relations, arterial & nerve supply
- Differentiate between each part of duodenum regarding the length, level & relations.
- Differentiate between the jejunum & ileum regarding the characteristic anatomical features of each of them.
- Describe the microscopic structure of the three regions of the small intestine: Duodenum, Jejunum, Ileum.
- List the characteristic features of colon.
- Identify the histological structure of the 4 layers of colon.
- Identify the histological structure of the 4 layers of appendix.

Title of the lecture:	Biliburin metabolism
Lecturer's name	Dr.Hayam Gad
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	Hayam_gad@hotmail.com

Objectives:

- Definition of bilirubin
- Bilirubin metabolism
- Bilirubin formation
- Transport of bilirubin in plasma
- Hepatic bilirubin transport
- Excretion through intestine
- Other substances conjugated by glucuronyl transferase.
- Differentiation between conjugated & unconjugated bilirubin
- Other substances excreted in the bile

Title of the lecture:	Liver function tests (Lfts)
Lecturer's name	Dr. Sumbul Fatma
Department	Pathology (Biochemistry)
Block / Week	Gastrointestinal & Nutrition Block / 5
Email address	sumbulfatma@gmail.com

Objectives:

Upon completion of this lecture, the students should be able to:

- Understand the major metabolic functions of the liver and causes of liver dysfunction.
- Discuss markers of liver function tests such as liver enzymes, bilirubin, albumin and prothrombin time that can diagnose hepatic injury and assess hepatic function.

Key words:

- Liver function tests, hepatocellular injury, bilirubin, albumin, jaundice, alanine aminotransferase, aspartate aminotransferase

Background:

Liver function tests (LFTs) are an important array of tests that help detect and assess liver injury and function. These are broadly classified into two categories: (i) tests to detect hepatic injury and (ii) tests to assess hepatic function. The former include measurement of liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) for detecting hepatocellular injury, and alkaline phosphatase (ALP) and □-glutamyltransferase (GGT) enzymes to detect cholestasis. The latter include markers of assessing hepatic function such as serum bilirubin (total and conjugated), bile salts and urobilinogen, total proteins, serum albumin and albumin/globulin ratio, and prothrombin time.

Abnormal levels of conjugated and unconjugated serum bilirubin are important in diagnosing pre-hepatic, hepatic and post-hepatic jaundice. Markers such as serum albumin and globulin are used for assessing the synthetic function of the liver. Despite their specificity, LFTs do have some limitations. Normal LFT values do not always indicate absence of liver diseases since the liver has very large reserve capacity. On the contrary, asymptomatic people may have abnormal LFT results. Therefore diagnosis should also include clinical examination.

Key principles to be discussed:

- LFTs are important tests that include enzymatic and non-enzymatic markers to detect and assess liver injury and function.
- Although very useful in diagnosis, LFTs have some limitations that make it necessary to include clinical examination.

Take home messages:

- LFTs help detect liver injury and function.
- LFTs do have some limitations

Further reading (Prescribed book):

- Lippincott's Biochemistry. Lippincott Williams & Wilkins, New York, 2011. pp 282-283.
- Lecture notes: Clinical Biochemistry, 8th edition, Geoffrey Beckett, Simon Walker, Peter Rae, Peter Ashby. January 2010, © 2010, Wiley-Blackwell.

Title of the lecture:	Reticuloendothelial system and function of the spleen
Lecturer's name	Dr. Nervana Bayoumi
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	drnerv@hotmail.com

Objectives:

- Describe Monocyte macrophage system (RES)
- Functions of monocytes/macrophages in different tissues
- Mechanism of chemotaxis, phagocytosis and microbial killing
- Explain functions of spleen
- Understand the basic concept of the indications and risks of spleenectomy.

Title of the lecture:	Coagulation mechanism
Lecturer's name	Dr. Nervana Bayoumi
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 4
Email address	drnev@hotmail.com

Objectives:

- Describe different stages of haemostasis
- Recognize different clotting factors & cascade of clotting.
- Explain the intrinsic, extrinsic and common pathway.
- Recognize the role of thrombin in coagulation
- Explain process of fibrinolysis and function of plasmin
- Enumerate clotting disorders

Title of the lecture:	Anatomy of the liver and spleen
Lecturer's name	Dr. Sanaa Alsharawani
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	salsharawi@ksu.edu.sa

Objectives:

- Location, subdivisions and relations and peritoneal reflection of liver.
- Blood supply, nerve supply and lymphatic drainage of liver
- Location, subdivisions and relations and peritoneal reflection of spleen.
- Blood supply, nerve supply and lymphatic drainage of spleen.
- Describe: The histological structure of liver with special emphasis on:
- Classical hepatic (liver) lobule.
- Hepatocytes.
- Portal tract (portal area).
- Hepatic (liver) blood sinusoids.
- Space of Disse (perisinusoidal space of Disse)
- Bile canalculi.
- 2. The histological structure of spleen with special emphasis on:
- The white pulp and the red Pulp.

Title of the lecture:	Platelets structure and functions
Lecturer's name	Dr. Abeer Alghomals
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	abeerkg@yahoo.com

Objectives:

- Describe formation and development of platelets
- Explain platelet normal ultrastructure
- Understand the functions of different platelets organelles and surface receptors
- Explain the role of platelets in haemostasis.
- Describe thrombocytopenia

Title of the Lecture:	Trypanosomiasis and filariasis
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 5
Email address	monabadr@hotmail.com

Objectives of the lecture:

- Know stages of Haemoflagellates
- Know geographical distribution of African sleeping sickness
- Describe life cycle of African trypanosomiasis
- Discuss pathology and diagnosis of African sleeping sickness
- Describe life cycle of American trypanosomiasis.
- Know signs and symptoms and how to diagnose American trypanosomiasis
- discuss the treatment of trypanosomiasis.
- Summarize major filarial infections of Humans.
- Describe life cycle of Onchocerca volvulus.
- Know pathology, diagnosis and treatment of onchocerciasis.
- Discuss pathology caused by lymphatic filariasis
- Describe life cycle of Wuchereria bancrofti.
- Know about diagnosis and treatment of lymphatic filariasis.
- Describe life cycle of Loa loa and how it diagnosed and treated

Keywords:

- (African sleeping sickness, Tsetse fly., Chancre., CNS., CSF., Chagas disease- Reduviid bug, Chagoma, Romana' sign, C- shape, xenodiagnoses, river blindness, loiasis.

Background:

- Foundation Block is enough to give brior knowledge for this block

Title of the lecture:	Approach to bleeding disorders
Lecturer's name	Dr. Fatma Al Qatani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	falqahtani@gmail.com

Objectives:

- To know the function of platelets and the relationship between the platelet count in peripheral blood and the extent of abnormal bleeding.
- To know about the diseases associated with
- A failure of platelet production
- A shortened platelet lifespan, especially immune thrombocytopenic purpura(ITP).
- To know the principles of investigation of patient suspected of having a haemostatic defect.
- To understand the role of platelets, blood vessel wall and coagulation factors in normal haemostasis.
- To know the classification of haemostatic defects.
- To know the platelet morphology and life span.
- To know the platelet function and diseases due to platelet function disorders.
- To know the causes of thrombocytopenic purpura and non-thrombocytopenic purpura.

Title of the lecture:	Biochemical aspects of bile acids and salts
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	sumbulfatma@gmail.com

Objectives:

- To know the structure of primary bile acids and salts
- To know the structure of secondary bile acids and salts
- To know the functions of bile salts
- Enterohepatic circulation
- Malabsorption syndrome
- Cholelithiasis

Title of the lecture:	Urea cycle
Lecturer's name	Dr. Rana Hasanato
Department	Biochemistry
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	rhanasanato@ksu.edu.sa

Objectives:

- Understand the reactions for removal of α-amino group of amino acids and formation of ammonia.
- Identify the importance of blood transport of ammonia to the liver in the form of glutamine/alanine.
- Understand the importance of conversion of ammonia into urea by the liver through urea cycle
- Identify urea as the major form for the disposal of amino groups derived from amino acids
- Identify the causes (hereditary & acquired), clinical manifestations and management of hyperammonemia

Title of the lecture:	Pathology and pathogenesis of liver Cirrhosis
Lecturer's name	Dr. Maha Arafah
Department	Pathology
Block / week	Gastrointestinal & Nutrition Block / 5
Email address	marafa@hotmail.com

Objectives:

- Define Cirrhosis.
- Recognize the types of cirrhosis.
- Recognize the causes and the pathogenetic mechanisms leading to cirrhosis.
- Describe the pathological findings in cirrhotic livers.

Title of the Lecture:	Viral hepatitis II (enteric hepatitis, EBV, CMV & YFV)
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 6
Email address	monabadr@hotmail.com

Objectives of the lecture:

- Distinguish the etiology of enteric viral hepatitis (HAV, HEV) from other viruses causing hepatitis such as EBV, CMV, Yellow fever virus.
- Describe the main characteristics of HAV ,HEV,EBV, CMV, and Yellow fever virus
- Describe the epidemiology and the mode of transmission of these viruses.
- Describe the clinical manifestations of enteric viral hepatitis.
- Describe the laboratory methods used to diagnose enteric hepatitis.
- Describe the treatments and the prevention measures available for these viral infection

Keywords:

- (Hepatitis, HAV, HEV, Herpes viruses, EBV, CMV, arboviruses, Yellow fever virus)

Background:

Viral hepatitis is a common infectious disease, caused by many viruses .The clinical manifestations of hepatitis are virtually the same regardless of which hepatitis virus is the cause. It presents as a febrile systemic upset with gastrointestinal symptoms followed in many cases by jaundice. Thus, a specific diagnosis can only be made in the laboratory.

Main concepts in the lecture:

- Many viruses cause hepatitis , the medically important viruses are commonly described as viral hepatitis, because their main site of infection is the liver. They are divided in to two groups based on their major mode of transmission:
    - Parenterally transmitted hepatitis (blood -borne) (hepatitis B,C,D and G viruses)
    - Enterically transmitted hepatitis (faecal- borne) (hepatitis A and E viruses)
    - Enterically transmitted hepatitis are caused by HAV & HEV. They are transmitted mainly by the fecal-oral route but they can be transmitted by other routes . Most infections are asymptomatic or cause mild illness especially in children but the severity of the disease increases with age.
    - Enterically transmitted hepatitis is an acute, self limited disease, resolve spontaneously within weeks , however HEV has a higher mortality rate especially in pregnant women ,and is more likely to cause fulminant disease than HAV. There is no carrier state and no tendency to chronically or malignancy (hepatocellular carcinoma)
    - The diagnosis is made on the detection of Virus- specific IgM in the patients blood.
    - No antiviral therapy is available. Active immunization with vaccine and passive immunization with human immunoglobulin are available to prevent hepatitis A but

not E. Personal hygiene such and public health measures such sewage disposal are of prime importance .
    - Hepatitis may occur in the course of glandular fever, cytomegalovirus infection and very rarely in a wide variety of other virus diseases e.g. herpes simplex, entero and adeno virus infections..
- Epstein- bar virus (EBV) is the etiologic agent of glandular fever or infectious mononucleosis IM . It is an acute, self-limited infectious disease of children and young adults. It is identified by its characteristic clinical, hematologic and serologic manifestations.
- Cytomegalovirus (CMV) causes a febrile illness with splenomegaly impaired liver function (sometimes with jaundice) and the appearance of abnormal lymphocytes in the blood. In contrast to IM caused by EBV, heterophil antibodies are absent ,in addition to pharyngitis and cervical adenopathy are uncommon.
- Several arboviruses can cause hepatitis as a part of general infection. Yellow fever occurs primarily in the tropical areas of Africa and South America. Two distinct epidemiological patterns are recognized with different reservoirs.
- No antiviral therapy is available, prevention of yellow fever involves mosquito control and immunization with vaccine containing live, attended yellow fever virus. It is recommended mainly to traveler to endemic area.

Conclusion:

Hepatitis results from infection with many viruses. In contrast to blood-borne hepatitis, enterically transmitted hepatitis, they are caused by non-enveloped RNA viruses. They are acute self-limited diseases. There is no carrier state and no tendency to chronically or hepatocellular carcinoma.

No specific treatment or prevention are available except HAV can be prevented by passive and active immunization .In addition, Yellow fever virus can be prevented by vaccine.

Take home messages:

- The clinical manifestations of hepatitis are more or less the same regardless the cause.
- Diagnosis is made by the detection of specific IgM antibody in serum against specific virus.
- Enterically transmitted hepatitis are mainly but not only transmitted by fecal-oral route.
- CMV and EBV causing hepatitis as well as other diseases.
- The differences between infectious mononucleosis and infectious mononucleosis–like syndrome.
- Yellow fever virus is transmitted by mosquito, is restricted to endemic area and.

Further reading:

- Medical Microbiology. David Greenwood ,Richard Slack, John Peutherer and Mike Barer. 17th Edition, 2007. Pages; 428-435, 484-485, 507-523, 533-534.

- *Review of Medical Microbiology and Immunology*. By: Warren Levinson. 10th Edition, 2008. Pages; 257-259, 292-294, 301, 305-306.

Title of the Lecture:	Leishmaniasis
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / Week	Gastrointestinal & Nutrition Block / 5
Email address	monabadr@hotmail.com

Objectives of the lecture:

- Know different stages of Leishmania parasites
- Describe life cycle of Leishmania parasites
- Discuss what diseases caused by Leishmania parasites and what is endemic in Saudi Arabia
- Geographical distribution of Leishmania in the world either cutaneous or visceral Leishmaniasis
- Know what are the vectors of Leishmania
- Discuss clinical types of Leishmaniasis
- Know uncommon types of the diseases
- How Leishmaniasis is diagnosed in the labs
- What is the best treatment for Leishmaniasis

Keywords:

- (Promastigotes, Amastigotes., macrophages., cutaneous., Visceral., Kala-azar NNN medium, oriental sore., sand fly, lesion., Pentostam., PKDL)

Background:

- Foundation Block is enough to give brior knowledge for this block

Title of the Lecture: Biochemistry of vitamin K	
Lecturer's name	Dr. Sumbul Fatma
Department	Pathology (Biochemistry)
Block / Week	Gastrointestinal & Nutrition Block / 3
Email address	sumbulfatma@gmail.com

Objectives of the lecture:

- Identify the types and sources of vitamin K.
- Understand the role of vitamin K in blood coagulation.
- Recognize the importance of γ-carboxylation of glutamic acid in coagulation proteins.
- Understand the role of anticoagulant drugs in affecting vitamin K function.
- Discuss the causes and disorders of vitamin K deficiency.

Background:

- Vitamin K is essential for blood coagulation process.
- It is a coenzyme for γ-carboxylation of glutamic acid (Glu) residues of prothrombin and coagulation factors II, VII, IX and X.
- γ-carboxyglutamate (Gla) is highly important for the prothrombin-platelet interaction.
- Anticoagulant drugs such as warfarin and dicoumarol are structural analogs of vitamin K that inhibit the activation of vitamin K to its hydroquinone form.
- A number of bleeding disorders are associated with vitamin K deficiency.

Keywords:

- Vitamin K, phylloquinone, menaquinone, menadione, coenzyme, coagulation factors, prothrombin, platelets, calcium, γ-carboxylation, γ-caroxyglutmate, deficiency, deficiency, ecchymotic patches, bleeding disorders, coumarin, warfarin, dicoumraol,

Main concepts in the lecture:

Vitamin K is a fat-soluble vitamin essential for normal blood coagulation function. There are several forms of vitamin K from different sources. Phylloquinone or vitamin K_1 is abundant in green leafy vegetables. The intestinal flora of humans synthesizes Menaquinone or vitamin K_2. Synthetic form includes menadione, also known as vitamin K_3.

Vitamin K is essential for the synthesis of prothrombin and blood clotting factors II, VII, IX and X. Their synthesis requires conversion of their glutamic acid (Glu) residues to γ-carboxyglutamate (Gla). This carboxylation reaction is catalyzed by a carboxylase enzyme that requires vitamin K as a coenzyme that enables prothrombin to bind to platelets to initiate the coagulation process. Recent research has shown that vitamin K is also essential for γ-carboxyglutamate (Gla) synthesis in proteins not related to coagulation such as osteocalcin, which appears to play a role in bone formation and mineralization.

Anticoagulant drugs such as warfarin and dicoumarol affect the functions of vitamin K by inhibiting the synthesis of its active hydroquinone form of thus increasing blood-clotting time. The major disorder due to vitamin K deficiency is prolonged blood coagulation time. Causes of vitamin K deficiency include lipid malabsorption, prolonged antibiotic therapy and gastrointestinal infections with diarrhea. Although deficiencies are rare in adults, these are more common in newborn infants.

Conclusion:

- Vitamin K is a coenzyme for γ-carboxylation of glutamic acid residues in prothrombin and clotting factors.
- γ-carboxyglutamate is required for blood coagulation process.
- Vitamin K deficiency causes bleeding disorders.

Take home messages:

- Vitamin K is essential for blood coagulation process.
- It mediates the process by γ-carboxylation of glutamic acid residues of prothrombin and coagulation factors.

Further reading:

- Lippincott's Biochemistry. 6th Edition, pp 389-391, Lippincott Williams & Wilkins, New York, USA.
- Textbook of Biochemistry with Clinical Correlations. Thomas M. Devlin 6th Edition, pp. 1099-1101, John Wiley & Sons, Inc. USA.

Title of the lecturer: Hepatotoxic drugs	
Lecturer's name	Prof. Yieldez Bassiouni
Department	Pharmacology
Block/ Week	Gastrointestinal & Nutrition Block / 5
E-mail Address	Yieldez@yahoo.com

Objectives:

- Define the role of liver in drug detoxification
- Discuss the types (patterns) of hepatotoxicity
- Classify hepatotoxins
- Explain how a drug can inflict hepatotoxicity
- State the pathological consequences of hepatic injury
- Contrast the varied clinical presentation of hepatotoxicity
- Enlist the possible treatment

Keywords:

- hepatoxicity, hepatotoxin, cytochrome p450, inflammatory cholestasis. Idiosyncratic, isoniazid erythromycin ALT, ALP

Background:

- Liver is the main organ for drug metabolism
- In the result of drug metabolism active and toxic metabolite of drug formed
- Toxic metabolite may leads to liver toxicity

Main concepts in the lecture:

- The main concept is in the lecture is to introduce the consumer about the detoxic effect of liver.
- Discuss the drugs which may cause various types of hepatoxicity.
- Clinical presentation and lab findings ad management of hepatotoxicity.

Conclusion:

- what is drug induce hepatotoxicity..
- The knowledge of drugs which cause liver toxicity and their type.
- Identify the clinical manifestation and laboratory results variation in hepatoxicity

Take home messages:

- Role of liver in drug metabolism.
- Drugs produce liver toxicity
- Types of hepatotoxicity and its clinical and laboratory manifestation.

Further reading:

- Bertram G. Katzung, Anthony J. Trevor (2014).13th Edition. Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

Title of the lecture: Complication of the liver cirrhosis	
Lecturer's Name	Dr. Maha Arafa
Department	Pathology
Block / Week	Gastrointestinal & Nutrition Block / 5
Email address	marafa@hotmail.com

Objectives:

Upon completion of this lecture the students will be able to:

- Recognize the major complications of cirrhosis.
- Understand the pathogenetic mechanisms underlying the occurrence of the complications.
- Recognize the clinical features inherent to the above mentioned complications.
- Describe the pathological findings of the different complications.

Introduction:

- Cirrhosis is a major cause of morbidity and mortality all over the world. The lecture is meant to stress upon the different types of complications which may occur in the clinical setting of cirrhosis. The complications include mainly ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, portal hypertension, variceal bleeding, and hepatorenal syndrome.as well as hepatocellular carcinoma.

Key Outlines:

- Ascites,
- Spontaneous bacterial peritonitis,
- Hepatic encephalopathy,
- Portal hypertension,
- Variceal bleeding
- Hepatorenal syndrome.
- Hepatocellular carcinoma.
- Pathophysiology.
- Clinical manifestations with diagnostic approach.
- Pathological evaluation
- Conclusion

Summary:

Cirrhosis is the end stage of different patterns of injury to the liver leading to a common pathway characterized by the death of hepatocytes, extracellular matrix deposition and vascular reorganization. The complications of cirrhosis are varied and the majority are of a

dismal prognosis, therefore, the early recognition for a prompt treatment is of utmost importance; therefore, the student should be aware of this critical issue.

Take home message:

The student will investigate into the clinical presentation, the pathogenetic mechanisms and the pathology of the possible complications secondary to cirrhosis.

Prescribed reading:

- Pathologic Basis of Disease, Robbins and Cotran

Further Reading:

- Textbook of pathology
  Harshmohan

Title of the lecture:	Megaloblastic anemia
Lecturer's name	Dr. Alia Al Faraedi
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- To understand the mechanisms by which macrocytic anaemia may arise
- To appreciate the signs and symptoms of macrocytic anaemia
- To understand how macrocytic anaemia can be classified
- To be able to know the causes of macrocytic anaemia
- To understand the normal metabolism of vitamin B12 and folic acid, and to appreciate how megaloblastic anaemia may arise
- To suggest some normoblastic causes of macrocytosis.

Title of the lecture:	Histology of the liver and spleen
Lecturer's name	Prof. Raeesa Abdultawab
Department	Anatomy
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	rmohammad@ksu.edu.sa

Objectives:

- Location, subdivisions and relations and peritoneal reflection of liver.
- Blood supply, nerve supply and lymphatic drainage of liver
- Location, subdivisions and relations and peritoneal reflection of spleen.
- Blood supply, nerve supply and lymphatic drainage of spleen.
- Describe: The histological structure of liver with special emphasis on:
- Classical hepatic (liver) lobule.
- Hepatocytes.
- Portal tract (portal area).
- Hepatic (liver) blood sinusoids.
- Space of Disse (perisinusoidal space of Disse)
- Bile canalculi.
- 2. The histological structure of spleen with special emphasis on:
- The white pulp and the red Pulp.

Title of the lecture:	Acute leukemia I
Lecturer's name	Dr. Fatma Al Qahtani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- To understand the definition of acute leukemia and recognize the general features of leukemia
- To understand the general concepts of leukemia pathogenesis
- To understand the clinical presentation and recognize the importance of early diagnosis of acute leukemia
- To understand the general themes of classification and the basic tool of diagnosis
- To recognize the most common presenting features of acute myeloid leukemia and their significance in therapeutic approaches
- To know the most important indicators implicated in prognosis of acute myeloid leukemia.

Title of the lecture:	Acute leukemia II
Lecturer's name	Dr. Fatma Al Qathani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- To emphasize on the general aspects of leukemia including definition, common feature and general classification and the basic diagnostic tool for acute leukemia
- To understand the clinical features of acute lymphoblastic leukemia
- To understand the difference between T-ALL and B-ALL in term of clinical and pathological features
- To recognize the most important prognostic factor for ALL.

Title of the lecture:	**Chronic Leukemia**
Lecturer's name	Dr. Fatma Al Qathani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- To understand the general features of Myeloproliferative neoplasms
- To understand the clinicopathological differences between acute myeloid leukemia and chronic myeloid leukemia (CML)
- To understand the diagnostic approach for chronic leukemia and the major differential diagnosis of CML
- To recognize the importance of genetic study in diagnosis and treatment of CML.
- To understand the general aspect of myelodysplastic syndrome (MDS) including definition, pathogenesis, clinical features and prognosis
- To understand the general aspect of chronic myelomonocytic leukemia CMML including definition, pathogenesis, clinical features and prognosis.

Title of the lecture:	**Polycytemia**
Lecturer's name	Dr. Alia Faraedi
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- To understand the physiological mechanisms that regulate erythropoiesis
- To recognize the secondary and primary causes of polycythemia
- To understand the clinicopathological features of polycythemia vera
- To recognize the importance of genetic studies in diagnosis and management of polycythemia vera
- To understand the general aspects of essential thrombocythemia and primary myelofibrosis

Title of the lecture:	Malaria
Lecturer's name	Dr. Mona Badr
Department	Pathology (Microbiology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	monabadr@hotmail.com

Objectives:

- Know the 5 species of malaria that infect humans
- Describe the life cycle of malaria, morphology and clinical picture
- Compare pathogenesis of different malaria species
- Know endemic countries of malaria species
- Know malaria paroxysm
- Know complications of malaria
- Describe methods for laboratory diagnosis of malaria
- Know action of anti malarial drugs in different life stages of malaria parasite

Title of the lecture:	**Approach to haemolysis**
Lecturer's name	Dr. Fatma Al Qahtani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	falqahtani@gmail.com

Objectives:

- To be able to define haemolysis and haemolytic anaemia
- To be able to classify haemolytic anaemias into congenital and acquired types, and to know the aetiological factors in each division
- To understand the difference between intravascular and extra-vascular haemolysis, and to recognise the laboratory features of each
- To appreciate that disorders of globin function such as sickle cell disease are subtypes of haemolytic anaemia
- To understand the role of autoantibodies in the production of haemolytic anaemias and to know the types of disease with which they are associated
- To understand some causes of non-immune acquired haemolytic anaemias.

Title of the lecture:	Lymphoploriferative disorder
Lecturer's name	Dr. Fatma Al Qahtani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	falqahtani@gmail.com

Objectives:

- To understand the general features of lymphoproliferative disorders (LPD)
- To understand some benign causes of LPD such as infectious mononucleosis
- To understand the general classification of malignant LPD
- To understand the clinicopathological features of chronic lymphoid leukemia
- To understand the general features of the most common (LPD) (Burkitt lymphoma, Follicular lymphoma, multiple myeloma and Hodgkin lymphoma).

Title of the lecture: G6PD	
Lecturer's name	Dr. Rana Hasanato
Department	Pathology (Biochemistry)
Block / Week	Gastrointestinal & Nutrition Block / 6
Email address	rhasanato@ksu.edu.sa

Objectives:

By the end of this lecture, the students should be able to:

- Comprehend the biochemical basis of G6PD deficiency anemia
- Enumerate the precipitating factors for G6PD deficiency anemia
- Differentiate between the classes of G6PD deficiency anemia (variant enzymes)
- Recognize the diagnostic methods for G6PD deficiency anemia

Key words:

- Hexose monophosphate pathway (HMP). G6PD. Oxidative stress.

Background:

Hexose monophosphate pathway (HMP) or Pentose Phosphate Pathway (PPP) is characterized by:

- It is an alternative oxidative pathway for glucose
- No ATP production
- It is a major pathway for NADPH production
- It produces ribose-5-phosphate for nucleotide synthesis

Main concept in the lecture:

- Overview of the Hexose monophosphate pathway (HMP) or Pentose Phosphate Pathway (PPP)
- Uses of NADPH in general
- Uses of NADPH as an antioxidant
- Other antioxidant systems
- Oxidative stress: definition, consequences, associated diseases.
- G6PD overview
- Biochemical basis of G6PD deficiency hemolytic anemia
- Precipitating factors for G6PD deficiency hemolytic anemia
- Different classes of G6PD deficiency hemolytic anemia
- Variant enzymes of G6PD deficiency hemolytic anemia
- Diagnosis of G6PD deficiency hemolytic anemia

Take home messages:

- Students are prepared to recognize the biochemical basis and the different forms of G6PD together with the precipitating factors and the diagnostic methods of G6PD deficiency hemolytic anemia.

Further reading:

- Lippincott's Illustrated Reviews; Biochemistry, 4th edition, Editors: Pamela Champe, Richard A. Harvey, 2008, chapter 9, pp 109-116.

Title of the lecture:	**Anti- Platelet Drugs**
Lecturer's name	Prof. Yieldez Bassiouni
Department	Pharmacology
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	Yieldez@yahoo.com

Objectives:

- To describe different classes of anti-platelet drugs and their mechanism of action
- To explain the pharmacological effects, pharmacokinetics, clinical uses and adverse effects of anti-platelet drugs

Title of the lecture:	**Anti- malarial Drugs**
Lecturer's name	Dr. Alia Alshanawani
Department	Pharmacology
Block / week	Gastrointestinal & Nutrition Block / 6
Email address	

Objectives:

- Classify the main antimalarial drugs depending on their goal of therapy
- Detail the pharmacokinetics & dynamics of main drugs used to treat attack or prevent relapses
- State the WHO therapeutic strategy for treatment
- Hint on the CDC recommendations for prophylaxis in travelers to endemic areas

Title of the lecture:	Ultrasound of the liver and gallstones
Lecturer's name	Dr.Faten Al Muhaideb
Department	Radiology
Block / week	Gastrointestinal & Nutrition Block / 7
Email address	fatenalmohideb@gmail.com

Objectives:

- Identify normal appearance of hepatobiliary system on different radiology modalities.
- Know advantages and disadvantages of each radiology modality in general as well as in regard to imaging hepatobiliary system.
- Select the appropriate imaging modality that can help in answering the clinical concern.

Title of the lecture:	Bleeding disorders
Lecturer's name	Dr. Fatma Al Qahtani
Department	Pathology (Haematology)
Block / week	Gastrointestinal & Nutrition Block / 7
Email address	falqahtani@gmail.com

Objectives:

- To know the main sequence of events in the coagulation pathways
- To know the principles underlying the prothrombin time (PT), activated partial thromboplastin time (APTT) and thrombin time (TT)
- To know the principles of investigation of a patient suspected of having a haemostatic defect
- To know the mode of inheritance, clinical presentation, method of diagnosis and principles of treatment of haemophilia A (factor FVIII deficiency), haemophilia B (factor IX deficiency), haemophilia C (factor XI deficiency) and von Willebrand disease (VWD)
- To know the alterations in the haemostatic and fibrinolytic mechanisms associated with disseminated intravascular coagulation (DIC) and the causes of DIC
- To understand normal fibrinolysis and the principles of fibrinolytic therapy
- To understand the principles of anticoagulant therapy with unfractionated heparin, low molecular weight heparin and warfarin and to know about the laboratory control of such therapy
- To be aware of the natural anticoagulant mechanisms in blood and some of the prothrombotic states (thrombophilia)
- To know the effects of vitamin K deficiency and liver disease on the clotting mechanisms.

Title of the lecture:	Physiology of bile salts and enterohepatic circulation
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block / week	Gastrointestinal & Nutrition Block / 7
Email address	Hayam_gad@hotmail.com

Objectives:

- Functions of the bile and stages of bile secretion.
- Characteristics of bile.
- The main constituents of bile.
- Functions of gall bladder.
- Differences between hepatic bile and gall bladder bile.
- Control of biliary system.
- Primary and secondary bile acids.
- Enterohepatic circulation of bile salts.
- Absorption and uptake of bile acids.
- Functions of bile acids.

Title of the lecture:	Pathology and pathogenesis of gallstones and cholecystitis
Lecturer's name	Dr. Maha Arafa
Department	Pathology
Block / week	Gastrointestinal & Nutrition Block / 7
Email address	marafa@hotmail.com

Objectives:

- Recognize the predisposing factors of cholecystitis.
- Describe the different types of cholecystitis.
- Understand the pathogenesis of acute and chronic cholecystitis

Title of the lecture: Anatomy, histology& radiology of liver, spleen, pancreas &biliary system -Practical	
Lecturer's name	Dr. Jamilah El Medany/ Dr. Faten Al Mohaideb
Department	Anatomy / Radiology
Block / week	Gastrointestinal & Nutrition Block / 7
Email address	galmadani@ksu.edu.sa / fatenalmohideb@gmail.com

Objectives:

- Location, subdivisions and relations and peritoneal reflection of liver.
- Blood supply, nerve supply and lymphatic drainage of liver
- Location, subdivisions and relations and peritoneal reflection of spleen.
- Blood supply, nerve supply and lymphatic drainage of spleen.
- Describe: The histological structure of liver with special emphasis on:
- Classical hepatic (liver) lobule.
- Hepatocytes.
- Portal tract (portal area).
- Hepatic (liver) blood sinusoids.
- Space of Disse (perisinusoidal space of Disse)
- Bile canalculi.
- The histological structure of spleen with special emphasis on:
- The white pulp and the red Pulp.

Schedule – Female Group

WEEK 1 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (1) Starting: 19/11/2017 to 23/11/2017

OESOPHAGUS AND STOMACH

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 19 November 2017	Monday 20 November 2017	Tuesday 21 November 2017	Wednesday 22 November 2017	Thursday 23 November 2017
8:00 - 9:00 am Histology of the esophagus and stomach **(Anatomy)** **Prof. Raeesa Abdultawab**	**8:00 -9:00am** Role of salivary gland and stomach in digestion **(Biochemistry)** **Dr. Rana Hasanato**	**8:00 - 9:00 am** **Self-Directed Learning**	**8:00 - 10:00 am** **Practical** Anatomy, histology and radiology of the eosophagus & stomach **F2** **(Anatomy, Histology & Radiology)** **Dr. Jamilah El-Medany** **Dr. Faten Al Mohideb** **All Staff**	**8:00 - 9:00 am** Structure and function of haemoglobin **(Biochemistry)** **Dr. Sumbul Fatma**
9:00 – 10:00 am Anatomy of the oral cavity oseophagus and stomach **(Anatomy)** **Dr. Jamilah El-Medany**	**9:00-10:00am** Oesophageal motility and pathophysiology of reflux disease **(Physiology)** **Dr. Hana Alzamel**	**9:00-10:00am** Pathology and pathophysiology of peptic ulcer **(Pathology)** **Dr. Maha Arafah**		**9:00 - 10:00 am** **Self-Directed Learning**
10:00 - 11:00 am General principles of GIT physiology **(Physiology)** **Dr. Hana Alzamel**	**10:00 -11:00am** Physiology of the stomach and regulation of gastric secretions **(Physiology)** **Dr. Hana Alzamel**	**10:00-12:00nn** **Practical** Anatomy, histology and radiology of the eosophagus & stomach **F1** **(Anatomy, Histology & Radiology)** **Dr. Jamilah El-Medany** **Dr. Faten AL Mohideb** **All Staff**	**10:00 - 11:00am** **Self-Directed Learning**	**10:00 - 11:00am** **Self-Directed Learning**
11:00 – 12:00 nn Anatomy and histology of the salivary glands **(Anatomy)** **Dr. Jamilah El-Medany** **Prof. Raeesa Abdultawab**	**11:00 – 12:00 nn** Gastro Esophageal Reflux Disease (GERD) **(Pathology)** **Dr. Maha Arafah**		**11:00 - 12:00 nn** Anemia **(Haematology)** **Dr. Fatma Al Qahtani**	**11:00 - 12:00 nn** *H pylori* and drugs used in treatment **(Microbiology)** **Dr. Fawzia Al Otaibi**
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 - 3:00 pm **Inter-professional education and collaboration (1)** **(Professionalism)** **Dr. Hana Al Zamil**	**1:00 – 2:00pm** **Self-Directed Learning**	**1:00 – 2:00pm** H2 blockers and proton pump inhibitors **(Pharmacology)** **Prof. Hanan Hagar**	**1:00 - 2:00 pm** **Self-Directed Learning**	**1:00 - 3:00 pm** **Salam**
	2:00-3:00 pm **Self-Directed Learning**	**2:00 – 3:00pm** **Self-Directed Learning**	**2:00 -3:00 pm** **Self-Directed Learning**	

WEEK 2 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (2) Starting: 26/11/2017 to 30/11/2017

PANCREAS

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 26 November 2017	Monday 27 November 2017	Tuesday 28 November 2017	Wednesday 29 November 2017	Thursday 30 November 2017
8:00 -9:00 am Anatomy and histology of pancreas and biliary system **(Anatomy)** **Prof. Raeesa Abdultawab** **Dr. Jamilah El-Medany**	**8:00-9:00 am** **Self-Directed Learning**	**8:00-9:00 am** **Self-Directed Learning**	**8:00-9:00 am** **Self-Directed Learning**	**8:00 – 9:00 am** **Self-Directed Learning**
9:00 - 10:00 am Embryology of the pancreas and small intestine **(Anatomy)** **Dr. Sanaa Al-Shaarawi**	**9:00-10:00 am** Introduction to the block **Prof. Ali Somily**	**9:00 - 10:00 am** Pathology and pathogenesis of acute and chronic pancreatitis **(Pathology)** **Prof. Hala Kfoury**	**9:00 - 10:00 am** Introduction to pleuripotent stem cell **(Anatomy)** **Dr. Mona Al Safadi**	**9:00-10:00 am** **Self-Directed Learning**
10:00 - 11:00am Physiology of the pancreas **(Physiology)** **Dr. Hana Alzamel**	**10:00 - 11:00am** Antiemetic drugs **(Pharmacology)** **Prof. Hanan Hagar**	**10:00 - 12:00 nn** Pathology Practical GERD & Peptic Ulcer **(Pathology)** **Prof. Hala Kfoury**	**10:00 - 11:00 am** Macro and micro nutrients **(Biochemistry)** **Dr. Sumbul Fatma**	**10:00 - 11:00 am** Plasma protein **(Biochemistry)** **Dr. Sumbul Fatma**
11:00 – 12:00 nn Biochemical aspects of digestion of lipids **(Biochemistry)** **Dr. Sumbul Fatma**	**11:00 – 12:00 nn** Biochemical aspects of digestion of proteins and carbohydrates **(Biochemistry)** **Dr. Sumbul Fatma**		**11:00 – 12:00 nn** Transfusion & cross-matching heamoglobinopathy **(Haematology)** **Dr. Fatma Al Qahtani**	**11:00 – 12:00 nn** Haemoglobinopathy **(Haematology)** **Dr. Fatma Al Qahtani**
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 - 3:00 pm Leadership and management skills **(Professionalism)** **Prof. Lulu Al Nuaim**	**1:00 - 3:00 pm** Stress management **(Professionalism)** **Dr. Johara Al Me neser**	**1:00 - 2:00pm** Nutritional Requirements **(Biochemistry)** **Dr. Sumbul Fatma** **2:00-3:00 pm** **Self-Directed Learning**	**1:00 - 2:00pm** **Self-Directed Learning** **2:00-3:00 pm** **Self-Directed Learning**	**1:00 - 3:00pm** **Salam**

WEEK 3 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (3) Starting : 03/12/2017 to 07/12/2017

SMALL INTESTINE

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 03 December 2017	Monday 04 December 2017	Tuesday 05 December 2017	Wednesday 06 December 2017	Thursday 07 December 2017
8:00 -9:00 am Anatomy and histology of the small intestine **(Anatomy)** **Dr. Jamilah El-Medany/ Prof. Raeesa Abdultawab**	**8:00-10:00am** Problem-Based Learning (PBL) **Case 1 Part 1**	**8:00 -9:00 am** **Self-directed Learning**	**8:00-9:00am** Taking history from a patient with acute diarrhea **F1** **(Clinical Skills)**	**8:00 -10:00 am** Problem-Based Learning (PBL) **Case 1 Part 2**
9:00 - 10:00 am Physiology of the small intestine: motility and secretion **(Physiology)** **Dr. Hayam Gad**		**9:00 - 10:00 am** Radiology of the abdomen **(Radiology)** **Dr. Faten Al Mohideb**	**9:00 - 10:00 am** Taking history from a patient with acute diarrhea **F2** **(Clinical Skills)**	
10:00 - 11:00 am Normal flora and introduction to infectious diarrhea **(Microbiology)** **Prof. Hanan Habib**	**10:00 - 11:00 am** Cholera **(Microbiology)** **Dr. Fawzia Al Otaibi**	**10:00 - 11:00 am** Intestinal helminthes **(Microbiology)** **Dr. Mona Badr**	**10:00 - 11:00 am** Pathophysiology and mechanisms of diarrhea **(Pathology)** **Prof. Hala Kfoury**	**10:00 - 12:00 nn** **Practical** Clinical chemistry and pathology practical about malabsorption, acute and chronic pancreatitis **(Biochemistry)** **Prof. Hala Kfoury** **All Staff**
11:00 – 12:00 nn Shigelle and salmonella **(Microbiology)** **Prof. Hanan Habib**	**11:00 – 12:00 nn** Viral gastroenteritis **(Microbiology)** **Dr. Malak El Hazmi**	**11:00 – 12:00 nn** Intestinal protozoa **(Microbiology)** **Dr. Mona Badr**	**11:00 – 12:00 nn** Pathology and the mechanisms of malabsorption **(Pathology)** **Prof. Hala Kfoury**	
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 - 3:00 pm **Inter-professional education and collaboration** **(2)** **(Professionalism)** **Dr. Hana Al Zamil**	**1:00 - 2:00 pm** Nutrition Education **(Family and Community Medicine)** **Dr. Rufaida AdDbagh**	**1:00 - 2:00 pm** Biochemistry of vitamin K **(Biochemistry)** **Dr. Sumbul Fatma**	**1:00 - 3:00 pm** Professionalism in different cultural context (sensitivity to other belief and world views **(Professionalism)** **Prof. Hala Kfoury**	**1:00 - 3:00pm** **Salam**

WEEK 4 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (4) Starting: 10/12/2017 to 14/12/2017

COLON

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 10 December 2017	Monday 11 December 2017	Tuesday 12 December 2017	Wednesday 13 December 2017	Thursday 14 December 2017
8:00 - 9:00 am Anatomy and histology of the large intestine **(Anatomy)** **Dr. Jamilah El-Medany** **Prof. Raeesa Abdultawab**	**8:00 - 10:00 am** Problem-Based Learning (PBL) **Case 2 Part 1**	**8:00-10:00 am** **Practical** Anatomy, histology and radiology of the small and large intestine **F2** **(Anatomy & Histology** **Dr. Sanaa Al-Shaarawi** **Dr. Faten Al Mohided**	**8:00 -9:00 am** **Per rectal (PR)** **F1** **(Clinical Skills)**	**8:00 - 10:00 am** Problem-Based Learning (PBL) **Case 2 Part 2**
9:00 - 10:00 am Anatomy of the omentum **(Anatomy)** **Dr. Sanaa Al-Shaarawi**			**9:00 - 10:00 am** **Per rectal (PR)** **F2** **(Clinical Skills)**	
10:00 - 11:00am Physiology of the colon: motility **(Physiology)** **Dr. Hayam Gad**	**10:00 - 11:00am** Colonic tumours and polyps-1 **(Pathology)** **Dr. Maha Arafah**	**10:00 - 11:00am** Treatment of dysentery and amoebiasis **(Pharmacology)** **Prof. Hanan Haggar**	**10:00 - 11:00am** Irritable bowel syndrome **(Medicine)** **Dr. Othman Al Harbi**	**10:00 - 11:00am** Introduction to inflammatory bowel disease- Crohn's disease **(Pathology)** **Dr. Maha Arafah**
11:00- 12:00nn Schistosomiasis **(Microbiology) Parasitology** **Dr. Mona Badr**	**11:00- 12:00nn** Colonic tumors and polyps-2 **(Pathology)** **Dr. Maha Arafah**	**11:00- 12:00nn** Inflammatory bowel disease and ulcerative colitis **(Pathology)** **Dr. Maha Arafah**	**11:00- 12:00nn** Drugs used in treating constipation and IBS **(Pharmacology)** **Prof. Hanan Hagar**	**11:00- 12:00nn** Drugs used in IBD and biological and immune therapy of IBD **(Pharmacology)** **Prof. Hanan Hagar**
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 - 2:00 pm **Self -directed learning**	**1:00 - 3:00 pm** **Practical** Anatomy, histology and radiology of the small and large intestine **F1** **(Anatomy & Histology** **Dr Sanaa Al-Shaarawi** **Dr. Faten Al Mohideb**	**1:00 - 2:00 pm** Reticuloendothelial system and function of the spleen **(Physiology)** **Dr. Nervana Bayoumi**	**1:00 - 3:00 pm** Community Service and volunteer work (1) **(Professionalism)** **Dr. Hana Al Zamil**	**1:00 -3:00 pm** **Salam**
2:00-3:00 pm **Self –directed learning**		**2:00-3:00 pm** Coagulation mechanisms **(Physiology)** **Dr. Nervana Bayoumi**		

WEEK 5 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (5) Starting: 17/12/2017 to 21/12/2017

LIVER & HEMATOPOIETIC SYSTEM

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 17 December 2017	Monday 18 December 2017	Tuesday 19 December 2017	Wednesday 20 December 2017	Thursday 21 December 2017
8:00 – 10 :00am **MIDBLOCK** **EXAMINATION**	**8:00 - 10:00am** Problem-Based Learning (PBL) **Case 3 Part 1**	**8:00 - 9:00am** Biochemical aspects of bile acids and salts **(Biochemistry)** **Dr. Sumbul Fatma** **9:00 – 10:00am** Urea cycle **(Biochemistry)** **Dr. Rana Hasanato**	**8:00- 9:00 am** **Abdominal examination** **F1** **(Clinical Skills)** **9:00 – 10:00am** **Abdominal examination** **F2** **(Clinical Skills)**	**8:00 - 10:00am** Problem-Based Learning (PBL) **Case 3 Part 2**
10:00 - 11:00am Biliburin metabolism **(Physiology)** **Dr. Hayam Gad**	**10:00 - 11:00am** Anatomy of the liver and spleen **(Anatomy)** **Dr. Sanaa Alshaarawi**	**10:00 -11:00am** Pathology and pathogenesis of liver Cirrhosis **(Pathology)** **Dr. Maha Arafah**	**10:00 - 11:00 am** Cytochrome system and drug metabolism **(Pharmacology)** **Dr. Alia Alshanawani**	**10:00 -11:00am** Complication of liver cirrhosis **(Pathology)** **Dr. Maha Arafah**
11:00- 12:00nn Liver function tests **(Biochemistry)** **Dr. Sumbul Fatma**	**11:00- 12:00nn** Platelets structure and functions **(Physiology)** **Dr. Abeer Alghomals**	**11:00- 12:00nn** Viral hepatitis B, C, D and G **(Microbiology)** **Parasitology** **Dr. Mona Badr**	**11:00- 12:00nn** Leishmaniasis **(Microbiology)** **Parasitology** **Dr. Mona Badr**	**11:00- 12:00nn** Cancer of the liver and pancreas **(Pathology)** **Dr. Maha Arafah**
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 - 3:00 pm Community Service and volunteer work (2) **(Professionalism)** **Dr. Hana Al Zamil**	**1:00 - 2:00 pm** Trypanosomiasis **(Microbiology)** **Parasitology** **Dr. Mona Badr**	**1:00 - 3:00 pm** **Practical** Liver function test **(Integrated Biochemistry & Pathology)** **Dr. Rana** **Dr. Sumbul** **Dr. Maha** **All Staff**	**1:00 – 2:00 pm** **Self -directed learning**	**1:00 – 3:00pm** **Salam**

WEEK 6 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (6) Starting: 24/12/2017 to 28/12/2017

SPLEEN & HEMATOPOIETIC SYSTEM

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 24 December 2017	Monday 25 December 2017	Tuesday 26 December 2017	Wednesday 27 December 2017	Thursday 28 December 2017
8:00 - 9:00am Megaloblastic anaemia **(Haematology)** **Dr. Alia Al-Faraedi**	**8:00 - 10:00am** Problem-Based Learning (PBL) **Case 4 Part 1**	**8:00 - 9:00am** Malaria **(Microbiology)** **Parasitology** **Dr. Mona Badr**	**8:00 - 9:00am** Taking history from a patient with anaemia **F1** **(Clinical Skills)**	**8:00 - 10:00 am** Problem-Based Learning (PBL) **Case 4 Part 2**
9:00 – 10:00am Histology of the liver and spleen **(Anatomy)** **Prof. Raeesa Abdultawab**		**9:00 – 10:00am** Chronic Leukemia **(Haematology)** **Dr. Fatma Al Qahtani**	**9:00 – 10:00am** Taking history from a patient with anaemia **F2** **(Clinical Skills)**	
10:00 - 11:00am Acute leukemia I **(Haematology)** **Dr. Fatma Al Qahtani**	**10:00 - 11:00am** Acute leukemia II **(Haematology)** **Dr. Fatma Al Qahtani**	**10:00 -11:00am** Anti-coagulant drugs **(Pharmacology)** **Prof. Yieldez Bassiouni**	**10:00 - 11:00 am** Lymphoploriferative disorder **(Haematology)** **Dr. Fatma Al Qahtani**	**10:00 -11:00am** Anti-Platelet Drugs **(Pharmacology)** **Prof. Yieldez Bassiouni**
11:00- 12:00nn Viral hepatitis A and E **(Microbiology)** **Dr. Malak El Hazmi**	**11:00- 12:00nn** Polycythemia **(Haematology)** **Dr. Alia Al-Faraedi**	**11:00- 12:00nn** Approach to Haemolysis **(Haematology)** **Dr. Fatma Al Qahtani**	**11:00- 12:00nn** G6PD **(Biochemistry)** **Dr. Rana Hasanato**	**11:00- 12:00nn** Anti-Malarial Drugs **(Pharmacology)** **Dr. Alia Alshanawani**
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**	**Lunch** **12:00-1:00pm**
1:00 -3:00pm Continuous professional development, lifelong learning and professionalism through mentoring **(Professionalism)** **Dr. Johara Al Meneser**	**1:00 – 3:00pm** **Practical** Liver, biliary system and pancreas **(Pathology)** **Dr. Maha Arafah**	**1:00 – 3:00pm** **Practical** Haemoglobinopathis **(Haematology)** **Dr. Fatma Al Qahtani**	**1:00 – 3:00pm** **Practical** Blood Parasites **(Microbiology)** **Parasitology** **Dr. Mona Badr**	**1:00 - 3:00 pm** **Salam**

WEEK 7 – GASTROINTESTINAL & NUTRITION BLOCK (GNT 223) (Female)

Week (7) Starting: 31/12/2017 to 04/01/2018

GALLBLADDER & BILIARY SYSTEM

CHAIR PERSON : Dr. Othman Al Harbi

CO-CHAIR: Prof. Ali Somily

Sunday 31 December 2017	Monday 01 January 2018	Tuesday 02 January 2018	Wednesday 03 January 2018	Thursday 04 January 2018
8:00 - 9:00 am Pathophysiology of ascitis **(Medicine)** **Dr. Waleed Al Hamoudi**	**8:00 -9:00 am** **Self -directed learning**	Consolidation	Consolidation	Consolidation
9:00 – 10:00am Physiology of bile salts and enterohepatic Circulation **(Physiology)** **Dr. Hayam Gad**	**9:00 – 11:00am** **Practical** Anatomy, histology and radiology of liver, spleen, pancreas and biliary system **F2** **(Anatomy, Histology & Radiology)** **All Staff**			
10:00- 11:00 am Ultrasound of the liver and gallstones **(Radiology)** **Dr. Faten Al Mohideb**				
11:00- 12:00nn Bleeding disorders **(Haematology)** **Dr. Fatma Al Qahtani**	**11:00- 12:00nn** Pathology and pathogenesis of gallstones and cholecystitis **(Pathology)** **Dr. Maha Arafah**			
Lunch **12:00-1:00pm**	**Lunch** **12:00-1:00pm**			
1:00 – 3:00pm **Practical** Anatomy, histology and radiology of liver, spleen, pancreas and biliary system **F1** **(Anatomy, Histology & Radiology) All Staff**	**1:00 –3:00 pm** **Practical** Hepatitis **(Microbiology)** **Dr. Malak El Hazmi**			

Week 7: Consolidation from 02 January 2018 to 04 January 2018
Week 8: Examination week from 07 January 2018 to 11 January 2018

Plagiarism

Plagiarism is a voluntary act to copy sentences and give a misleading impression that the text is created by the person whose name appears on the work. For example an assignment submitted as part of the requirements of assessment of a subject.

Plagiarism may include plagiarism of ideas and or plagiarism of text (sentences or paragraphs). It also may include the use of diagrams, tables, images, cartoons etc without acknowledging the original creator of the work.

The act of copy-and-paste writings even if the aim is to produce a good assignment with well-structured English statements is unethical and when discovered could cause serious consequences including disciplinary action. Students need to construct statements in their own words and refer to the correct references related to what they have written and included in their assignment/work. Giving credit and acknowledgement to the original authors/creators are valued by the academic community as it reflects an ethical and professional attitude.

Why is plagiarismwrong?

Universities, higher education institutes and scientific communities consider plagiarism as a major problem for a number of reasons:

- It is an act of stealing ideas and the work of original authors/creators.
- It does not represent acceptable professional, ethical or scientific behaviour.
- It raises doubts about the credibility of the person/group of people who committed such act.

How can teachers/college discover an act of plagiarism?

There are a number of software programs such as iThentcate and many others available to detect the act of plagiarism. Some of these programs are available free online.

These tools can locate the places and sentences where students have copied and the original resource (articles, manuscripts, papers, books, websites) for such statements/paragraphs or images.

What are the consequences of plagiarism?

Students who commit plagiarism will be exposed to disciplinary actionincluding the failure of the subject concerned provided that such act has been confirmed with evidence.

Assessment of Students in the Block (year 2)

Dear Student, in order to pass the block, you must obtain a minimum final block grade of D (the grading guide attached as appendix[1]), this grade is a composition from several block requirements, which can be subdivided as:

1- Attendance
2- Tutor assessment
3- Written Exams (MCQs, SAQs)
4- OSPE (Objective Structured Practical Examination)

Marks distribution for the CNS, GIT, Endocrine & Reproduction Blocks

The final grade is a composition of the grades obtained for the specified block requirements, calculated as follows:

Continuous Assessment (Tutor Assessment in PBL sessions) : 5%

- Written Examinations:
  - SAQs : 20%
  - MCQ : 50%
    - Mid-Block Exam (20%)
    - Final Block Exam (30%)
- OSPE : 25 %

TOTAL : 100 %

1. <u>Attendance :</u>

Students are required to attend not less than 75% of all educational activities during the block. These include small group teaching, lectures, practical sessions, skills training sessions and integrated clinical sessions.

Your attendance will be recorded during all sessions. Failure to meet this requirement without a valid explanation will result in exclusion from the final examination. On the other hand, your presence will be rewarded by assigned marks.

2. <u>Tutor Assessment in Large and Small groups (Continuous Assessment):</u>

During each session, your individual efforts will be evaluated by your tutor. The tutors are instructed to evaluate two aspects:

a. The extent to which you demonstrate that you study and prepare yourself thoroughly between the two sessions (i.e., preparation).

b. The extent to which you actively contribute during group discussion (i.e., participation). Your grade for each session depends upon both your preparation and your participation. The grade will be on the scale from "5", "4", "3", "2", or "1". Which have the following general descriptors:

5 = Outstanding (Excellent)
4 = Very good
3 = Good

2 = Average
1 = Poor

The block contains two sessions each week, so the maximum amount of 'participation points' you are able to obtain will be from two sessions multiplied by the number of weeks. The total participation points will be recalculated according to the weight for each participation in the total assessment.

Your tutor can give you more information about the evaluation of your participation. The details of these evaluation also given in "Tutor Assessment of Student" form.

3. Written Examination:

   a. Mid-block exam for Year 2 comprises 20% marks: In the form of MCQs, these are prepared mainly from sessions presented to the students in large group. This exam will consist of 50 - 60 MCQs that will assess factual knowledge.

   b. Final written exam comprises 30% marks for the Year 2 blocks: at the end of the block in form of MCQs, that are prepared mainly from sessions and presented to the students. This exam will consist of 80 -100 MCQs that will assess factual knowledge too.

4. Objective Structured Practical Examination (OSPE):

This comprises 25% of the marks. It is a practical examination at the end of the block. The OSPE examination will consist of 15-20 OSPE stations. Each station will take about 5 minutes, which contains a mix of slide show and some practical sessions. The purpose of the OSPE stations is to test your deeper understanding of the basic sciences. The OSPE will take place at the end of each block.

5. Short Answer Questions (SAQs)

This comprises 20% of the marks. It contains 3 to 5 cases which include 12-20 integrated SAQs. The time allocated for each question is 3 minutes. The purpose of SAQs is to test the knowledge and its application in integrated manner and to increase the validity and reliability of the written exam. The SAQ will take place at the end of each block.

Block Evaluation

The block evaluation uses the following three data sources:

1. Student Feedback
2. Tutor Feedback
3. Student Results

Methods of student's formative assessment:

- Self evaluation
- Peer evaluation
- Tutor evaluation (both summative & formative)
- Assignments

LEARNING RESOURCES

Medical Dictionary

Prescribed :

Martin EA (2016). Oxford Concise Medical Dictionary.9th Ed. Oxford: Oxford University Press.

Recommended textbooks:

Dorland (2012). Dorland's Pocket Medical Dictionary with CD-ROM, 29th Edition, Elsevier, UK.

Dorland (2011). Dorland's Illustrated Medical Dictionary with CD-ROM, 32nd Edition, Elsevier, UK.

Anatomy & Embryology

Prescribed textbook:

Drake RL, Vogl W and Mitchell AWM (2014). Gray's Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.

Snell RS (2005). Clinical Anatomy for Medical Students. 7th ed. Philadelphia: Lippincott Williams & Wilkins.

Schoenwolf GC, Breyl SB, Baurer PR, Fancis-West PH. (2014). Human Embryology. New York: Churchill Livingstone.

Recommended textbooks:

McMinn RH (2004). McMinn's Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.

Moore KL and Dalley AF (2005). Clinically Oriented Anatomy. Philadelphia: Lippincott Williams & Wilkins.

Netter FH (2006). Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.

Agur AMR and Dalley AF (2005). Grant's Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

More KL (2002). The Developing Human. Philadelphia: Saunders WB.

Sadler TW. (2005) Langman's Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

Sadler TW. (2006) Langman's Medical Embryology. 10th ed. Philadelphia: Lippincott Williams & Wilkins.

Histology

Prescribed textbook:

Gartner LP (2016). Color Textbook of Histology. 4th ed. Philadelphia: Saunders WB.

Recommended textbooks:

Young B, O' Dowd G, Woodford P (2013). Wheater's Functional Histology. 6th ed. London: Churchill Livingstone.

Physiology

Prescribed textbook:

Hall JE. Guyton and Hall Textbook of Medical Physiology (2015). 13th Edition. Churchill Livingstone, UK.

Recommended textbooks:

Koeppen BM and Stanton BA. (2010) Berne & Levy Physiology, updated Edition. 5th ed. London: Mosby

Sherwood L. (2006). Human Physiology: From Cells to Systems. 4th ed.Brooks/Cole Pub.Co: Sydney.

Fox SI. (2015). Fundamentals of Human Physiology. 14th ed. McGraw-Hill: Boston.

Saladin KS (2011). Anatomy and Physiology The Unity of FORM and FUNCTION. McGraw Hill Lange, USA

Barrett KE, Barman SM, Boitano S, Brooks HL (2015). Ganong's Review of Medical Physiology. 25th Edition. McGraw-Hill Publisher, UK.

Carroll RG (2007). Elsevier's Integrated Physiology. Mosby, Elsevier, UK.

Pharmacology

Prescribed textbook:

Rang HP, Ritter JM, Flowei RJ, Henderson G. (2016). Range & Dale's Pharmacology. 8th Edition. Churchill Livingstone, Elsevier, UK.

Recommended textbooks:

Bertram G. Katzung, Anthony J. Trevor (2014).13th Edition. Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

Medical Biochemistry

Prescribed textbook:

Gaw A, Murphy MJ, Cowan RA, O'Reilly DJ, Stewart MJ, Sheperd J, (2009). Clinical Biochemistry: An Illustrated Colour Text. 4th ed. Churchill Livingstone, Elsevier.

Ferrier D, (2014). Lippincott's Illustrated Review Biochemistry. 6th ed. Lippincott Williams & Wilkins.

Recommended textbooks:

Murray RK, Roolwell VW, Bender D, Botham KM, Weill A, Kennelly PJ (2009). Harper's Illustrated Biochemistry. 28th Editions. McGraw Hill, Lange, New York.

Baynes J and Dominiczak M (2014). Medical Biochemistry. Elsevier.

Lieberman M, (2013).4th Edition. Mark's Basic Medical Biochemistry: A Clinical Approach. Lippincott Williams & Wilkins, New York.

Champe PC, Harvey RA, Ferrier DR (2008). Lippincott's Illustrated Reviews Biochemistry. 3th ed. Philadelphia: Lippincott Williams & Wilkins.

Microbiology & Parasitology

Prescribed textbook:

Murray P, Rosenthal K, Pfaller M, (2013). Medical Microbiology: Study smart with Student Consult. 7th ed. Elsevier.

Recommended textbooks:

Goering R, DoCkrell H, Zuckerman M, Wakelin D, Riott I, Mims C (2012). Mims' Medical Microbiology. 5th Edition. Mosby, UK.

John DT, Petri Jr (2006). Markell and Voge's Medical Parasitology. Ninth Edition. Elsevier, UK.

Greenwood D, Slack RC, Peutherer JF, Barer MR (2007). Medical Microbiology. Seventh Edition. Churchill Livingstone, UK.

Strohol WA. Lippincotts Illustrated Review Microbiology (2006). Second Edition. Lippincott Williams & Wilkins, New York.

Brooks GF, Butel JS, and Morse SA. (2004). Jawetz, Melnick, and Adelberg's Medical Microbiology. 23rd ed. New York: McGraw-Hill Co and Lange Appleton.

Engleberg NC (2013). Schaechter's Mechanisms of Microbial Disease. 5th ed. Philadelphia: Lippincott Williams & Wilkins.

Neva FA, Brown HW. (1994). Basic Clinical Parasitology. 6th ed. Connecticut: Prentice-Hall International Inc.

Chamberlain NR (2008). Medical microbiology & immunology. McGraw Hill Lange Publisher, UK.

Levinson WE (2010). Review of Medical Microbiology and Immunology. Eleventh-Edition, McGraw-Hill Publisher, UK

Pathology & Genetics

Prescribed textbook:

Kumar V, Abbas A, Aster L, (2013). Robbins Basic Pathology. 9th ed. Saunders. Philadelphia Elsevier

Hoffbrand V, Moss PAH, (2016). Hoffrand's Essential Hematology. 7th ed. Wiley Blackwell.

Nusbaum RL, McInnes RR, Willar HF, (2015). Thompson & Thompson Genetics in Medicine. 8th ed. Elsevier.

Recommended textbooks:

Kumar V, Abbas AK, and Fausto N (2004). Robbins and Cotran Pathologic Basis of Disease. 7th ed. Philadelphia: Saunders WB.

Young B, Stewart W. (2009). 5th Edition. Wheaters Basic Histopathology. A Colour Atlas and Text. Churchill Livingstone, Elsevier, UK.

Immunology

Prescribed textbook:

Owen J, Punt J, Stranford S, (2013) Kuby Immunology: Kindt, kuby Immunology. 7th ed. W.H. Freeman.

Recommended textbooks:

Delves PJ, Martin SJ, Burton DR, Riott IM (2012). Riott's Essential Immunology. 8th Edition. Elsevier.

Male D, Brostoff J, Roth DB, and Roitt I. (2006). Immunology. 7th ed. Edinburgh: Mosby.

PBL and Learning Skills

Prescribed textbook:

Azer SA (2006). Core Clinical Cases in Basic Biomedical Sciences. Hodder-Arnold, UK.

Azer SA (2008). Navigating Problem-Based Learning. Elsevier Australia, Australia.

Recommended textbook:

Kushner TK and Thomasma DC (2001). Dilemmas for Medical Students and Doctors in Training. Cambridge: University Press.

Communication Skills & Introduction to Clinical Medicine

Prescribed textbook:

Lloyd M, Bor R (2009). Communication Skills for Medicine. Elsevier.

Munro JF, Campbell IW (2006). Macleod's Clinical Examination. Tenth Edition. Churchill Livingstone, UK.

Talley NJ and O'Connor S. (2006). Pocket Clinical Examination. Melbourne: Blackwell Science.

Medicine

Kumar P and Clark M (2012). Clinical Medicine. 7th ed. Edinburgh: Elsevier Saunders.

Walker B.R, Colledge Nicki.R, Ralston Stuart.H, Penman I. (2014). Davidson's Principles and Practice of Medicine. 22nd ed. Edinburgh: Churchill Livingstone.

(In the preclinical years these two textbooks may help you in the preparation of your learning issues, you will also need them in the clinical years).

Professionalism

Prescribed textbook:

Feldman MD, Christensen JF (2014). Behavioural Medicine. A Guide for Clinical Practice. McGraw-Hill Lange, UK.

Stern DT (2006). Measuring Medical Professionalism. Oxford University Press, UK.

Spandorfer J, Pohl CA, Rattner SL, Nasca TJ (2010). Professionalism in Medicine. A case-based Guide for Medical Students. Cambridge University Press, UK.

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KING SAUD UNIVERSITY
College of Medicine
Department of Medical Education
Feedback to Students on PBL Performance
Year 2 (Academic Year 2016-2017)

Student's name:……Group number…….
Tutor's name…….Block: GASTROINTESTINAL & NUTRITION BLOCK

The feedback items are grouped under two main headings.
1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement, 4= developed; 5=well developed (marks are allocated as follows: 1 mark for rank 1, 2 marks for rank 2, 3 marks for rank 3, 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

1. Learning and cognitive skills:

Ability to:	1	2	3	4	5
Identify problems in the case					
Generate hypotheses					
Build mechanisms					
Collect new information					
Interpret findings					
Identify learning issues					
Apply knowledge learnt					

Mark= /5

2. Interaction and participation to group function:

Ability to:	1	2	3	4	5
Work collaboratively with other members					
Take active roles such as scribing					
Communicate effectively					
Arrive to tutorials on time					
Demonstrate good manners					
Keep the group focused					
Share resources with others					

Mark = /5

Comments
……
……
……
……

Tutor's Name: Signature: Total Mark= /10

KING SAUD UNIVERSITY
College of Medicine
Department of Medical Education
Assessment of Student in PBL
Year 2 (Academic Year 2016-2017)

Student's name: …….Group number:……..
Tutor's name: …….Block: GASTROINTESTINAL & NUTRITION BLOCK

The assessment items are grouped under two main headings.
1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement, 4= developed; 5=well developed (marks are allocated as follows: 1 mark for rank 1, 2 marks for rank 2, 3 marks for rank 3, 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

1. Learning and cognitive skills:

Ability to:	1	2	3	4	5
Identify problems in the case					
Generate hypotheses					
Build mechanisms					
Collect new information					
Interpret findings					
Identify learning issues					
Apply knowledge learnt					

Mark= /5

2. Interaction and participation to group function:

Ability to:	1	2	3	4	5
Work collaboratively with other members					
Take active roles such as scribing					
Communicate effectively					
Arrive to tutorials on time					
Demonstrate good manners					
Keep the group focused					
Share resources with others					

Mark = /5

Comments
……
……
……
……

Tutor's Name: Signature: Total Mark= /10

KING SAUD UNIVERSITY
COLLEGE OF MEDICINE
MEDICAL EDUCATION DEPARTMENT

STUDENTS' EVALUATION OF THEIR PBL TUTOR

Date:_____
Tutor's Name:_____ **Group No.:**_____
Student: ☐ **Peer:** ☐ **Other:** ☐ **Name (Optional):**_____

How well did the tutor facilitate group process in the following regards? Please put a check (✓) in the box.

1.	Appropriately facilitated the brainstorming sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
2.	Appropriately facilitated the hypothesis reorganization sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
3.	Appropriately facilitated the reporting sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
4.	Appropriately manage the time flow.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
5.	Help to keep the group focused on its task	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
6.	Provided a well balanced intervention within the group process, but avoided dominating.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
7.	Intervened when chairman or reporter needed.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
8.	Provided constructive positive and constructive feedback to the group as needed.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
9.	Encouraged positive and constructive feedback within the group about its performance	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
10.	Showed enthusiasm.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
11.	Helped to create a supportive group climate.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
12.	Encouraged logical and critical thinking.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
13.	Overall rating of the tutor.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐

Number Code Values:

5- EXCELLENT **4- VERY GOOD** **3-GOOD** **2- FAIR** **1- POOR**

KING SAUD UNIVERSITY
COLLEGE OF MEDICINE
MEDICAL EDUCATION DEPARTMENT

STUDENT RATING OF LECTURE

Date:_____**Subject:**_____**Instructor:**_____

Purpose:
This form is designed as an observation tool to rate the performance of each instructor in the different sessions. It is intended to provide a tool for lecturer improvement.

Directions:
Using the anchors below, check (✓) your rating for each item below. Check (✓) N/A for items that do not apply.

No.	Standard Procedure	5	4	3	2	1	N/A
1	Started and ended class on time.						
2	Presented overview of content and objectives.						
3	Presented information according to objectives.						
4	Used relevant examples and illustrations (graphs, etc.) to explain major ideas						
5	Used alternative explanations when necessary.						
6	Made efficient use of questions with students.						
7	Covered all contents/objectives.						
8	Exhibited enthusiasm.						
9	Encouraged students to express themselves.						
10	Asked questions prior to closure						
11	Summarized major points/related contents to objectives.						
12	Amount you learned in the class was:						

Mention 3 strong points in this lecture:

1. _____
2. _____
3. _____

Mention 3 points for Improvement:

1. _____
2. _____
3. _____

Your name: (optional)_____