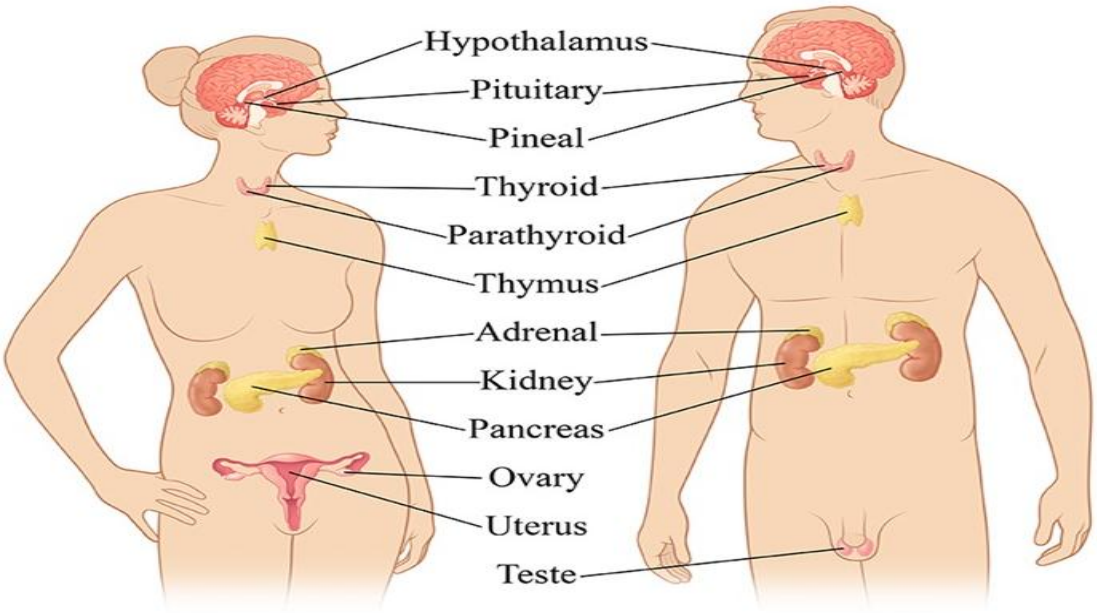
College of Medicine
Department of Medical Education
Curriculum Development & Research Unit



STUDENT'S BOOK

ENDOCRINE BLOCK (ENDO 255)

YEAR 2 (Male Group A)

2017-2018
(1438-1439)

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

THE ENDOCRINE BLOCK

Year Two

BLOCK BOOK AND STUDENT GUIDE

Male Group A

(21 January 2018 to 08 March 2018)

(2017-2018) 1438-1439

WELCOME ADDRESS

Dear Students,

We are pleased to welcome you in the college of Medicine, Endocrine 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 fulfils 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 mean time, 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 Adhehri

Vice Dean for Academic Affairs

College of Medicine

A Message from the Endocrine System Block Chair

Dear Students,

It gives me a great pleasure to welcome you to the Endocrine block which we hope you will enjoy. This block is designed to provide you with the necessary background in the areas of anatomy, physiology, pathology and pharmacology of the endocrine system. In addition to lectures, suitable practical sessions will be provided as well as interesting case studies to prepare you for the clinical aspects of this system. We are interested to get full interaction from you and continuous participation in all of the activities of the course.

Our best wishes to you for continuous success.

Prof. Riad Al Sulimani

Endocrine Block Chair

TABLE OF CONTENTS

General Information

Block Title	Endocrine System
Block Code & Number	ENDO 255
Credit Hour	6
Block Duration	7 Weeks
Block Dates	21st January 2018 to 08th March 2018
Block Chairman	Prof. Riad Al- Sulimani
Block Co-Chair	Dr. Usman Ghani
Members of the Committee	Prof. Samy Azer
	Dr. Essam Aldin Salama
	Dr. Khalid Al Regaiey
	Prof. Ammar Rikabi
	Dr. Abdulkarim F. Al Hetheel
	Prof. Abdulrahman Almotrefie

List of the teaching staff
Year 2 – Male Group A

Department	Name	Mobile	E-Mail
Anatomy	Prof. Ahmed Fathalla	0501562983	ahmedfathalla@gmail.com
	Prof. Saeed Abuelmakarem	0556439341	saeedmakarem@hotmail.com
	Dr. Essam Aldin Salama	0592871734	Essamco58@gmail.com
	Dr. Mohammed Vohra	0508845648	vohra@ksu.edu.sa
	Dr. Aly Mohamed	0556751503	alymahmed53@hotmail.com
	Dr. Mohammed Atteya	0560773248	atteya.m@gmail.com
Histology	Dr. Aly Mohammed	0556751503	alymahmed53@hotmail.com
Physiology	Prof. Abdulmajeed Al Drees	0506270531	adrees@ksu.edu.sa
	Dr. Mouaadh Abdelkarim	0596600802	Mouaadh.abdelkarim@gmail.com
	Dr. Ahmad Alsabeeh	-	-
	Dr. Khalid Al Regaiey	0505535005	kalregai@gmail.com
Pharmacology	Prof. Abdulrahman Al Motrefi	0545507838	motrefi@ksu.edu.sa
	Dr. Saeed Ahmed Shiekh	Ext.75351	saeedahmedshk@yahoo.com
	Prof. Mohammad Al Humayyd	0505475978	humayyd@yahoo.com
	Dr. Ishfaq Bukhari	0545507838	iabukhari@ksu.edu.sa
Pathology	Dr. Mohammed Swayyed	Ext. 71892	alswayyed@hotmail.com
	Dr. Abdullah Basabien		abasabein.c@ksu.edu.sa
Biochemistry	Dr. Usman Ghani	0551596921	ugresearch@hotmail.com ughani@ksu.edu.sa
	Dr. Ahmed Mujamammi	Ext.79512	mujamammi@gmail.com/aamujamammi@ksu.edu.sa
	Dr. Zeyad Kurdee	-	-
Immunology	Prof. Adel Almogren	Ext. 71843	almogren@ksu.edu.sa
Microbiology	Dr. Ahmed Al Barrag	92677	Aalbarrag2@hotmail.com Aalbarrag2@ksu.edu.sa
	Prof. Ali Somily	0532703322	Ali.somily@gmail.com/somily@ksu.edu.sa
Medicine	Dr. Mohammed Mujamammi	Ext.79512	mujamammi@gmail.com/aamujamammi@ksu.edu.sa
	Prof. Riad AlSulimani	0505415166	sulimanirs@yahoo.com/ rsulimani@ksu.edu.sa
Family & Community Medicine	Dr. Ebraheem Qusaidi	Ext. 92769	gossady@hotmail.com
Psychiatry	Dr. Mohammed Al Jaffer	89351	maljaffer@ksu.edu.sa
Patient Safety	Prof. Hamza Abdulghani	0505442859	hamzaabg@gmail.com
	Dr. Ahmed Aboshaiqah	-	-
	Dr. Hussein Saad	-	-

List of the Problem-Based Learning Cases

The table below summarizes the PBL cases to be discussed in the Endocrine System Block.

Week	Case Number	Case Title
W1	**NO CASE**	
W2 (Monday & Thursday)	1	"…Cannot tolerate hot weather"
W3	**NO CASE**	
W4 (Monday & Thursday)	2	"…Looking for hope"
W5 (Monday & Thursday)	3	"…Trying to loose body weight"
W6	**NO CASE**	

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, he/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-centred 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 Learning Objectives

By the end of this block the students will be able to:

- Correlate the histological structures of the endocrine glands (pituitary, thyroid, parathyroid, adrenal and the pancreas) and their functions.
- Consolidating the role of hypothalamic-pituitary axis in the regulation of thyroid, adrenal and gonads functions.
- Understand the biochemical structure, synthesis, metabolism and mechanism of action of hormones produced by pituitary, thyroid, parathyroid, adrenal and the pancreas.
- Understand the pathology and pathogenesis of pituitary, thyroid and adrenal gland disorders.
- Use knowledge learned from basic sciences to interpret the symptoms, signs and the investigation results of patients with common endocrine diseases.
- Understand the pathology and pathogenesis of type 1 diabetes mellitus, and understand the role of obesity and metabolic syndrome in the pathogenesis of type 2 diabetes mellitus.
- Emphasize the role of the parathyroid hormone and vitamin D in calcium metabolism.
- Understand the pharmacological basis of drugs commonly used in the management of patients with endocrine disorders.
- Understand the role of life style and patient education in the prevention of chronic diseases such as obesity and diabetes.

Learning Objectives in detail

1.0 Knowledge

1.1 Discuss the anatomy and ultrastructure of the endocrine glands (pituitary, thyroid, parathyroid, adrenal and endocrine pancreas) and correlate structure to their functions.

1.2 Discuss the biochemistry and physiology of the endocrine hormones (chemical structure, synthesis and secretion, target cell, receptor, function, metabolism, mechanism of action and regulation) including hormones of the anterior and posterior pituitary, hormones of the thyroid and parathyroid glands, adrenal hormones, and hormones secreted by the endocrine pancreas.

1.3 Discuss the role of the hypothalamic-pituitary axis and regulation mechanisms such as negative feedback mechanisms in hormonal regulation.

1.4 Discuss the embryology of the endocrine glands.

1.5 Correlate the anatomy of endocrine glands (e.g., pituitary gland and adrenal gland) to their radiological findings/features.

1.6 Discuss the pathology, pathogenesis and key clinical features of diseases affecting the endocrine glands including hypo and hyperpituitarism, hypo and hyperthyroidism,

Hashimoto's thyroiditis, thyroid nodules and thyroid cancer, hypo and hyperparathyroidism, Cushing disease/syndrome, Addison disease, adrenal adenoma, adrenal cancer, pheochromocytoma, type 1 and type 2 diabetes mellitus, obesity, and metabolic syndrome.

1.7 Discuss the pharmacology of drugs used in the management of common diseases affecting the endocrine system including drugs used in hypothyroidism, hyperthyroidism, parathyroid disorders, pituitary disorders, calcium and vitamin D disorders, and adrenal disorders. Also the pharmacology of insulin, oral hypoglycaemic agents, and corticosteroids.

1.8 Discuss the epidemiology, health promotion, and prevention of obesity, and diabetes mellitus in the community.

1.9 Discuss calcium and phosphate homeostasis and the role of vitamin D and parathyroid hormones and other factors in their regulation.

1.10 Discuss the pathology and pathogenesis, and key clinical features of type 1 and type 2 diabetes mellitus and identify key differences between these two diseases and common complications and infections in patients with diabetes.

1.11 Discuss impact of chronic diseases on patients and family members (e.g., type 1 diabetes in an adolescent, hypothyroidism, acromegaly).

1.12 Discuss the principles of self-directed learning and their applications.

1.13 Discuss the role of social, environmental, behavioural and genetic factors in the development of diseases affecting the endocrine system.

1.14 Briefly discuss health promotion, health education, and prevention of diseases affecting the endocrine system.

2.0 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 Use available information to differentiate between normal and abnormal changes (e.g., obesity versus Cushing disease).

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

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

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

3.0 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).

4.0 Communication, Information Technology, Numerical

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

5.0 Interpersonal Skills & Responsibility

5.1 Demonstrate the ability to take history from patient with an endocrine problem and present their findings.

5.2 Demonstrate the ability to conduct clinical examination of the endocrine 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 session.
- Lectures.
- Seminars.
- Laboratory based practical Class.
- Clinical skills Center.
- Independent learning.
- E-learning & Multimedia

Objectives of the Lectures

Title of the lecture:	Anatomy of the Pituitary glands- Practical
Lecturer's name	Dr. Aly Mohamed
Department	Anatomy
Block / week	Endocrine Block / week 1
Email address	Alymahmed53@hotmail.com

Objectives:

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

- Describe the position of the pituitary gland.
- List the structures related to the pituitary gland.
- Differentiate between the lobes of the gland.
- Describe the blood supply of pituitary gland & the hypophyseal portal system.

Title of the lecture: Histology of Pituitary Glands	
Lecturer's name	Dr Aly Mohammad
Department	Anatomy
Block / week	Endocrine Block / week 1
Email address	Alymahmed53@hotmail.com

Objectives:

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

1. The microscopic structure of pars distalis of the pituitary gland in correlation with its functions.
2. The microscopic structure of pars nervosa of the pituitary gland in correlation with its functions.
3. The hypophyseal portal circulation; components and significance.

Background:

- Stroma of the pituitary gland.
- Parenchyma of the pituitary gland.
- Blood supply of the pituitary gland.

Main concepts in the lecture:

Pars distalis contains:

- Acidophils: Somatotrophs and mammotrophs.
- Basophils: Thyrotrophs, gonadotrophs and corticotrophs.
- Chromophobes.
- Blood Capillaries.

Pars nervosa contains:

- Axons of hypothalamic-hypophyseal tract.
- Herring bodies.
- Pituicytes.
- Blood capillaries.

Conclusions:

- The Pituitary gland is important for the activity and viability of other endocrine glands.

Take-home messages:

- The microscopic structure of pars distalis of the pituitary gland in correlation with its functions.
- The microscopic structure of pars nervosa of the pituitary gland in correlation with its functions.
- The hypophyseal portal circulation; components and significance.

Further readings:

- Recommended Textbooks- Color Textbook of Histology (Gartner and Hiatt)- Latest Edition.
-

Title of the lecture:	Physiology of Posterior Pituitary Gland
Lecturer's name	Dr. Khalid Al Regaiey
Department	Physiology
Block / week	Endocrine Block / week 1
Email address	kalregai@gmail.com

Objectives:

- Hypothalamic control

- Posterior pituitary hormones
- ADH
  - Physiological functions
  - Control of secretion
    - ❖ Osmotic stimuli
    - ❖ Non-osmotic stimuli

- Oxytocin
  - Physiological functions
  - Control of secretion

Title of the lecture:	Physiology of Hypothalamo-Pituitary axis and regulatory mechanism
Lecturer's name	Prof. Abdulmajeed Aldress
Department	Physiology
Block / week	Endocrine Block / week 1
Email address	adrees@ksu.edu.sa

Objectives:

- **Structure of pituitary gland**
- Anterior pituitary cell types and hormones
- Posterior pituitary cell types and hormones

- **Hypothalamic control of pituitary gland**
- Hypothalamo-hypophysial portal system
- Hypothalamo-hypophysial tract

- **Feedback mechanisms**
- Positive feedback
- Negative feedback

Title of the lecture:	**Anterior pituitary disorders**
Lecturer's name	Dr. Mohammed Mujammami
Department	Medicine
Block / week	Endocrine Block / week 1
Email address	mujamammi@gmail.com

Objectives:

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

- a brief review of the embryological development of anterior and posterior lobes of the pituitary glands
- The function of the pituitary glands in relation to hypothalamus
- The hormones that are released from the anterior pituitary glands and their function
- The most common pathological disorders of anterior pituitary glands
- Pituitary adenoma and its related clinical manifestations:
    - non-functional pituitary adenoma
    - Functional pituitary adenoma: Acromegaly, cushing's disease, hyperprolactinemia, gonadotroph secreting adenoma, TSH-secreting adenoma
    - hypopituitarism: adrenal insufficiency, hypothyroidism, GH deficiency
- Diagnosis and management of the above conditions

Title of the lecture:	General mechanisms of action hormone
Lecturer's name	Dr. Zeyad Kurdee
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 1
Email address	

Objectives:

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

- Acquire the knowledge for general consequence of hormone-receptor interaction
- Understand different mechanisms of action of hormones
- Recognize the biomedical importance due to disturbance in the normal mechanisms of hormonal action

Keywords:

Intercellular communication, hormones, target, stimulus, receptors, plasma half-life, cAMP, cGMP, phosphatidyl inositol, tyrosine kinase, insulin

Background:

Multicellular organisms depend in their survival on their adaptation to a constantly changing environment. Intercellular communication is necessary for this adaptation to take place. Human body synthesizes many hormones that can act specifically on different cells of the body. More than one hormone can affect a given cell type. Hormones can exert many different effects in one cell or in different cells. A target is any cell in which the hormone (ligand) binds to its receptor.

Main concepts in the lecture:

- Background
- Factors determining the response of a target cell to a hormone
- General Features of Hormone Classes
- Hormone-receptor interaction
- General features of hormone classes
- Classification of hormones by mechanism of action
- Class I- Mechanism of Action of Steroid-Thyroid Hormones.

- Class II- Hormones that bind to cell surface receptors A. The second messenger is cAMP, cGMP, Ca/PI, tyrosine kinase.
- Mechanism of action of insulin and its effects.
- Abortion of Hormonal Stimulus.
- Biomedical importance

Take-home messages:

- Hormones are involved in responses to a stimulus, using a variety of signaling mechanisms to facilitate cellular adaptive responses.
- Group I hormones are lipophilic, while group II are hydrophilic. Other differences exist between both groups.
- Hormones can be classified according to their mechanism of action (specific examples of each category were discussed)
- Biomedically, studying hormones' actions in details helps to:
    - understand consequences of abnormal hormone releaserelated diseases (excessive, deficient or inappropriate)
    - design therapeutic approach for such diseases.

Further readings:

Lippincott's IllustratedReviewsBiochemistry: 6th edition,Chapters 8, 17 and 23. Buxton, Iain LO, and Dayue Duan. "Cyclic GMP/Protein Kinase G Phosphorylation of Smad3 Blocks Transforming Growth Factor-β–Induced Nuclear Smad Translocation."(2008): 151-153.

Title of the lecture:	**Anatomy and embryology of the thyroid and parathyroid glands.**
Lecturer's name	Prof. Saeed Abuemakarem
Department	Anatomy
Block / week	Endocrine Block / week 2
Email address	saeedmakarem@hotmail.com

Objectives:

- Describe the shape, position, relationships and the structure of the thyroid gland.
- List the blood supply & lymphatic drainage of the thyroid gland.
- List the nerves that may be injured during thyroidectomy operation.
- Describe the shape, position, blood supply & lymphatic drainage of the parathyroid glands.
- Describe the development of the thyroid & parathyroid glands.
- Describe common congenital anomalies of the thyroid gland.

Background:

- Importance of the anatomy of the thyroid and parathyroid glands for any clinical problems.
- Developmental anatomy of the thyroid and parathyroid glands and most common congenital anomalies.
- Most important nerves in relation with thyroidectomy.

Main concepts in the lecture:

The structure and location of the thyroid gland ; it consists of 2 lobes connected together by isthmus which is lying on the 2^{nd},3^{rd} and 4^{th} tracheal rings. Each lobe has apex extends at the thyroid cartilage and base at 4^{th} or 5^{th} tracheal ring.
The thyroid gland surrounded by 2 capsules : an inner fibrous connective tissue capsule and outer cervical fascial capsule.
The relation of thyroid gland : anterolateral; posterior and medial relations.
The posterior border of thyroid gland related to parathyroid glands and anastomosis between superior and inferior thyroid arteries.
The thyroid gland developed from the thyroid primordium of the endoderm of the primitive pharynx, passing along the the thyroglossal duct.
The superior parathyroid gland developed from the doral part of the 3^{rd} pharyngeal pouch.
The inferior parathyroid gland developed from the dorsal part of the 4^{th} pharyngeal pouch.

The most common congenital anomalies :
1-Thyroglossal duct cyst which lying inferior to the hyoid bone.
2- Ectopic thyroid gland.
The nerves endanger during thyroidectomy are :
External laryngeal nerve and
Recurrent laryngeal nerve.

Take-home messages:

- Anatomy of the thyroid and parathyroid glands.
- Development of thyroid gland.
- Common congenital anomalies of thyroid.
- Development of parathyroid gland.
- Clinical anatomy related to thyroidectomy operation.

Further readings:

Recommended textbooks:

- Clinical Anatomy for Medical Students by Richard S. Snell- Latest Edition.
- The Developing human by Moor and Persaud – Latest Edition.

Title of the lecture:	Histology of Thyroid glands.
Lecturer's name	Dr Aly Mohammad
Department	Anatomy
Block / week	Endocrine Block / week 2
Email address	Alymahmed53@hotmail.com

Objectives:

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

1. Describe the histological structure of the thyroid gland.

2. Identify and correlate the different endocrine cells in the thyroid gland with their functions.

Background:

- Stroma of the thyroid gland.
- Parenchyma of the thyroid gland.
- Thyroid follicles: components and structure..

Main concepts in the lecture:

Thyroid follicles are formed of:

- Follicular cells.
- Parafollicular cells.
- Colloid.

Interfollicular spaces contain:

- Connective tissue.
- Interfollicular cells.
- Blood capillaries.

Conclusions:

- Normal functions of the thyroid gland is important for the activity and viability of many other body organs .

Take-home messages:

- Describe the histological structure of thyroid gland.
- Identify and correlate between the different endocrine cells in thyroid gland and their functions.

Further readings:

- Recommended textbooks: Color Textbook of Histology (Gartner and Hiatt)- Latest Edition.

Title of the lecture: Hyper and Hypo Thyroidism	
Lecturer's name	Prof. Abdulmajeed Aldress
Department	Physiology
Block / week	Endocrine Block / week 2
Email address	adrees@ksu.edu.sa

Objectives:

At the end of this lecture the student should be able to:

- Identify the causes and consequences of hypocalcaemia.
- Recognize the common causes of hypoparathyroidism and describe the clinical tests used to demonstrate latent or overt tetany.
- Identify the Causes and consequences of hypercalcaemia.
- Identify consequences of vitamin D deficiency and excess.
- Classify the following disorders as causes of either hypercalcemia or hypocalcemia:
  a. Primary hyperparathyroidism e. Vitamin D deficiency
  b. PTH-secreting tumors f. Bony metastases
  c. Hypoparathyroidism g. Renal failure
  d. Vitamin D intoxication.
- Identify the pathophysiology of hyperparathyroidism, hypoparathyroidism, pseudohypoparathyroidism, rickets osteoporosis and osteomalacia.

Keywords:

(Hypocalcemia, Hypercalcemia, Hypoparathyroidism, Hyperparathyroidism, pseudohypoparathyroidism, rickets, osteoporosis, and osteomalacia.

Background:

The students are expected to have some background on the:

• Anatomy and histology of the parathyroid gland.

• Dietary sources and body requirements of calcium.

• Role of calcium in muscle contraction and in excitable tissues.

• Role of calcium as intracellular second messenger.

Main concepts in the lecture:

- In hypocalcemia, increased secretion of PTH increases the formation of 1,25 dihydroxycholicalciferol, and these two hormones cooperate to restore blood calcium by increasing mobilization from bone, decreasing loss by the kidney, and increasing absorbtion of dietary calcium .

- In response to hypercalcemia, shutdown of PTH secretion and 1,25 dihydroxycholicalciferol, synthesis allow calcium levels to decline slowly, while increased secretion of calcitonin promptly inhibits bone-resorbing activity of osteoclasts.
- Deficiency of estrogen or excessive thyroid hormone or glucocorticoids decreases skeletal mass.
- A typical attack of tetany due to hypocalcemia involves muscular spasms in the face and characteristic contractions of the arms and hands. Laryngeal spasm and contraction of respiratory muscles may compromise breathing. Pronounced hypocalcemia (low blood calcium) may produce more generalized muscular contractions and convulsions.
- Increased concentration of calcium in blood (hypercalcemia) may cause calcium salts to precipitate out of solution because of their low solubility at physiologic PH. "Stones" form, especially in the kidney, where they may produce severe painful damage (renal colic), which may lead to renal failure and hypertension.

Conclusion:

Students should be able to:

- The major consequences of altered concentrations of calcium phosphates in the body fluids.
- The consequences of vitamin D deficiency and excess.
- The causes and consequences of hypo- and hyper- parathyroidism.
- The pathophysiology of rickets, osteoporosis and osteomalacia.

Take-home messages:

- The causes and the clinical effects of hypocalcemia.
- The causes and the clinical effects of hypercalcemia.
- The pathophysiology of rickets, osteoporosis and osteomalacia
- The sources and the consequences of vitamin D deficiency and excess.
- The sources and effects of Parathyroid hormone related peptide (PTHrP).

Further readings:

- Review of Medical Physiology by: William F. Ganong, Chapter 21, Pages 369 – 382.
- Essential Medical Physiology by : Leonard R. Johnson, Chapter 43, Pages 597 – 615

Title of the lecture:	Biochemistry of Thyroid Hormones and Thermogenesis
Lecturer's name	Dr. Usman Ghani
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 2
Email address	Ughani@ksu.edu.sa

Objectives:

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

- Describe the types and biosynthesis of thyroid hormones.
- Discuss the thyroid hormone actions.
- Understand the regulation of thyroid hormones.
- List and interpret the thyroid function tests.
- Define goiter and differentiate between hypo and hyperthyroidism.
- Discuss the role of thyroid hormone in thermogenesis.

Keywords:

Thyroid hormone, T_3, T_4, synthesis, regulation deiodinase, metabolism, thyroid function tests, hypothyroidism, hyperthyroidism, Hashimoto's thyroiditis, Graves' disease, autoantibodies, thermogenesis, uncoupling proteins

Background:

- Thyroid gland synthesizes the thyroid hormones in two forms (T_3 and T_4). Their secretion is regulated by hypothalamic-pituitary-thyroid axis.
- Thyroid hormones are essential for metabolism, thermogenesis and development
- Thyroid function tests (TSH, T_3, T_4, autoantibodies) are important for the diagnosis of thyroid disorders.
- Goitre is the enlargement of the thyroid gland associated with hypo, hyper or normal function of the gland.
- Thyroid hormones regulate thermogenesis in the body by influencing metabolism and ATP synthesis.

Main concepts in the lecture:

Thyroid hormones are synthesized by the thyroid gland in two forms: T_3 (tri-iodothyronine) and T_4 (thyroxine). Their biosynthesis involves iodination, coupling of two tyrosine molecules and finally binding to thyroglobulin protein for transport. T_3 is more biologically active than T_4, which is synthesized from T_4 in the peripheral tissue by the deiodinase enzyme. Thyroid hormones are essential for body functions including metabolism, thermogenesis and development. The hypothalamic-pituitary-thyroid axis is responsible for its regulation. The hypothalamus secrets thyroid-releasing hormone (TRH), which stimulates pituitary to produce thyroid-stimulating hormone (TSH) that binds to the thyroid gland to produce thyroid hormone. T_3 and T_4 also exert feedback inhibition on the axis. Thyroid function tests are used to diagnose thyroid disorders such as hypo and hyperthyroidism. The tests include measurement of TSH, total or free T_4, total or free T_3 and autoantibodies. Goitre is the enlargement of thyroid gland associated with its hypo, hyper or normal function. Primary hypothyroidism is due to failure of the thyroid gland whereas secondary is because of failure of hypothalamic-pituitary-thyroid axis. The etiology may include Hashimoto's thyroiditis, congenital defects or severe iodine deficiency. Hyperthyroidism is due to excessive production and exposure of thyroid hormone to tissues (thyrotoxicosis). Some of the etiological factors include Graves' disease, thyroid adenoma and excessive intake of T_3 and T_4. Graves' disease is an autoimmune disorder in which antibodies against TSH receptors on thyroid cells mimic the action of pituitary hormone, therefore, releasing excessive thyroid hormone.

One of the most important actions of thyroid hormones is thermogenesis. They play an essential role in the obligatory and facultative thermogenesis by regulating metabolism in the brown and white adipose tissues, muscle, liver, pancreas and controlling hypothalamic-pituitary axis. One of the mechanisms of thermogenesis by thyroid hormones involves release of heat without ATP production mediated by UCPs (uncoupling proteins).

Conclusion:

- Thyroid hormones (T_3 and T_4) are synthesized by the thyroid glands and regulated by the hypothalamic-pituitary-thyroid axis.
- They play essential roles in a number of body functions including metabolism, thermogenesis and regulation.
- Thyroid function tests are important for the diagnosis of thyroid disorders such as goitre, hypo and hyperthyroidism.

Take-home messages:

- Thyroid hormones are synthesized in the thyroid gland by iodination, coupling and binding to thyroglobulin protein.
- Thyroid hormones regulate metabolism and thermogenesis in the body.
- It is regulated by hypothalamic-pituitary-thyroid axis.
- Thyroid function tests such as TSH, total and free T_4 and T_3, and antibodies help diagnose and follow up thyroid disorders.
- Goiter, hypo- and hyperthyroidism are due to abnormalities in thyroid functions.

Further readings:

1. Clinical Biochemistry: An Illustrated Colour Text, 5th Edition, Allan Gaw, pp. 88-93, Churchill Livingstone, UK.
2. Nedergaard, J and Cannon, B. Thyroid hormones: igniting brown fat via the brain. *Nature Medicine*, Volume 16, Number 9, pp. 965-967, 2010.

Title of the lecture:	Pathology of the Thyroid Glands
Lecturer's names	Dr. Mohammed Swayyed
Department	Pathology
Block / week	Endocrine Block / week 2
Email address	alswayyed@hotmail.com

Objectives of the lecturer:

- The ways in which thyroid disorders present.
- The major causes of both hypo and hyperthyroidism.
- The causes of the thyroid endemic goiter and its pathology.
- The causes and pathology of a solitary nodule in the thyroid gland.
- Understand the classification, histopathologic features and behavior of thyroid carcinoma.

Background:

- Anatomy, Histology and Physiology of the Thyroid Gland

Keywords:

- Hypothyroidism, pathological features, Hashimoto's thyroiditis, causes of simple multinodular goiter, solitary thyroid nodule, thyroid neoplasms.

Main concepts in the lecture:

The content of the two lectures is as follows:

- Etiology and pathologic features of hyperthyroidism (Grave's disease).
- Brief account on causes and features of hypothyroidism.
- Pathology and clinical features of Hashimoto's thyroiditis.
- Definition and causes of simple and multinodular goiter.
- Causes and pathology of solitary thyroid masses.
- Pathology of thyroid neoplasms including: follicular adenomas, papillary carcinoma, follicular, anaplastic and medullary carcinomas.

Conclusion:

(Please refer to learning objectives and contents).

Take home messages:

- Structure and function of thyroid gland.
- Classification and pathological features of the thyroid gland and disorders.

Further reading:

- Robbin's and Cotran, Pathologic Basis of Disease, 9th Edition

Title of the lecture:	Pharmacology of drugs used in hyperthyroidism
Lecturer's name	Prof. .Abdulrahman Al Motrefi
Department	Pharmacology
Block / week	Endocrine Block / week 2
Email address	motrefi@ksu.edu.sa

Objectives:

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

- To describe different classes of drugs used in treatment of hyperthyroidism
- To understand the mechanism of action, pharmacological effects, clinical uses and adverse effects of each drug.
- To recognize treatment of special cases such as hyperthyroidism during pregnancy, Graves' disease and thyroid storm

Keywords:

- (Thyroid gland, Thyroid dysfunction, Manifestation of hyperthyroidism, Drug treatment of hyperthyroidism Thioamides, Iodides, Radioactive iodine, Beta blockers)

Background:

- Prior knowledge about anatomy, physiology and function of thyroid gland.
- Prior knowledge about thyroid regulation by hypothalamus and anterior pituitary.
- Prior knowledge about iodine metabolism, synthesis and secretion of thyroid hormones.
- Prior knowledge about causes and clinical presentation of thyrotoxicosis.

Main concepts in the lecture:

- First brief introduction about the synthesis and secretion of thyroid hormones, causes, signs and symptoms of thyrotoxicosis. This is followed by classification of drugs used in treatment of hyperthyroidism. Detailed information about selected drugs; mechanism of action, pharmacokinetics, clinical uses, advantages and disadvantages of thioamides, iodides, radioactive iodine, beta blockers in treatment of hyperthyroidism.
- The student must compare between the Pharmacokinetic and adverse of action of antithyroid drugs.
- How to treat special cases of hyperthyroidism such as hyperthyroidism during pregnancy, Graves' disease and thyroid storm.

Conclusions:

- Antithyroid drugs (Thioamides), Iodides, Radioactive iodine, Beta blockers are used for treatment of hyperthyroidism; they have different mechanisms of action, pharmacokinetic profile, and adverse effects

Take home messages:

- Graves' Disease is an autoimmune disease and one of the commonest cause of hypethyroidism.
- Thioamides, iodides, radioactive iodine, beta blockers are used for treatment of hyperthyroidism.
- Propylthiouracil (PTU), methimazole, carbimazole are antithyroid drugs with different pharmacokinetic and pharmcodynamic profiles and adverse effects.
- Lugol's solution, potassium iodide are used to prepare patients with thyrotoxicosis for surgery.
- ^{131} I isotope is used for diagnosis and treatment of thyrotoxicosis.
- Propranolol, is used to reduce tremors, palpitation, heat intolerance and nervousness which accompany hyperthyroidism.
- Propylthiouracil is the drug of choice for treatment of hyperthyroidism during pregnancy.

Further reading:

- Basic and Clinical Pharmacology, Bertram Katzung ,Anthony Trevor
- Lippincott Illustrated Reviews: Pharmacology

Title of the lecture:	Pharmacology of drugs used in hypothyroidism
Lecturer's name	Prof.Abdulrahman Al Motrefi
Department	Pharmacology
Block / week	Endocrine Block / week 2
Email address	motrefi@ksu.edu.sa

Objectives:

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

- To describe different classes of drugs used in hypothyroidism and their mechanism of action
- To understand their pharmacological effects, clinical uses and adverse effects.
- To recognize treatment of special cases of hypothyroidism such as myxedema coma

Keywords:

Thyroid gland, thyroid dysfunction, manifestation of hypothyroidism, Thyroid hormone preparations, Levothyroxine, Liothyronine, Liotrix

Background:

- Prior knowledge about anatomy, physiology of thyroid gland
- Prior knowledge about synthesis and secretion of thyroid hormones
- Prior knowledge about causes and clinical presentation of hypofunction of thyroid gland

Main concepts in the lecture:

First brief introduction about the synthesis and secretion of thyroid hormones. This is followed by classification of drugs used in hypothyroidism. Detailed information about selected drugs; mechanism of action, pharmacokinetics, clinical uses, advantages and disadvantages of Levothyroxine, liothyronine, liotrix
How to treat special cases of hypothyroidism such as myxedema coma

Conclusions:

Thyroid hormones are used for treatment of hypothyroidism; they have the same mechanism of action but with different pharmacokinetic profile.

Take home messages:

- Hypothyroidism may be congenital, primary or secondary.
- Treatment of hypothyroidism is by hormone replacement therapy; Levothyroxine, Liothyronine, Liotrix

- Liothyronine is more potent and rapid onset of action but of shorter duration than levothyroxine, should be avoided in cardiac patients
- Myxedema coma is a life –threatening condition can be treated by I.V. liothyronine and I.V. hydrocortisone

Further reading:

- Basic and Clinical Pharmacology, Bertram Katzung ,Anthony Trevor
- Lippincott Illustrated Reviews: Pharmacology

Title of the lecture:	The Immune System and Endocrine disorders
Lecturer's names	Prof. Adel Almogren
Department	Immunology
Block / week	Endocrine Block / week 2
Email address	almogren@ksu.edu.sa

Objectives of the lecturer:

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

- To recognize that many endocrine disorders are organ-specific autoimmune diseases.
- To understand the mechanisms of damage which take place at endocrine glands and their consequences.
- To know the important examples of autoimmunity which affect different endocrine glands and the pathogenesis of these disorders.

Title of the lecture:	Histology of parathyroid glands
Lecturer's name	Dr Aly Mohammad
Department	Anatomy
Block / week	Endocrine Block / week 3
Email address	Alymahmed53@hotmail.com

Objectives:

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

- The microscopic structure of the parathyroid gland.
- The functional structure of the parathyroid cells.

Background:

- Stroma of the thyroid gland.
- Parenchyma of the thyroid gland.
- Functional significance of cells of parathyroid gland.

Main concepts in the lecture:

Microscopic components of parathyroid gland:

- Chief cells.
- Oxyphil cells.
- Fat cells.
- Blood Capillaries.

Conclusions:

The normal function of parathyroid gland is essential for the human life.

Take-home messages:

- The microscopic structure of the parathyroid gland.
- .The functional structure of the parathyroid cells.

Further readings:

- Recommended Textbooks-Color Textbook of Histology (Gartner and Hiatt)- Latest Edition.

Title of the lecture: Calcium Homeostasis	
Lecturer's name	Khalid Al Regaiey
Department	Physiology
Block / week	Endocrine Block / week 3
Email address	kalregai@gmail.com

Objectives:

- Identify and describe the primary sources and mechanism of actions in target tissues of the following calcitropic hormones where they exert their major effects:
  a. Parathyroid Hormone (PTH),
  b. Calcitonin,
  c. Vitamin D,
  d. Parathyroid hormone related peptide (PTHrP).
- Identify the actions of calcitropic hormones at bone (osteoblasts and osteoclasts), kidney and intestine.
- Identify the effect of the following factors on the measurement of free (ionized) calcium and total serum calcium:
  a. pH changes
  b. Protein abnormalities
- Identify and describe calcium homeostasis in pregnancy, lactation and in postmenopausal women (lack of estrogen).

Keywords:

- (Calcium, Phosphate, Parathyroid hormone, Calcitonin, Vitamin D,
- PTHrP, Osteoblasts and Osteoclasts , Kidney, Intestine, Estrogen)

Background:

The students are expected to have some background on the:

- Anatomy and histology of the parathyroid gland.
- Dietary sources and body requirements of calcium.
- Role of calcium in muscle contraction and in excitable tissues.
- Role of calcium as intracellular second messenger.

Main concepts in the lecture:

- Maintenance of the concentration of calcium in the extracellular fluid depends on the rate of calcium absorption from the intestine, excretion in the urine, and exchange with bone.

- Parathyroid hormone (PTH) increases blood calcium by stimulating calcium mobilization from bone and calcium reabsorption from the glomerular filtrate. It also indirectly simulates calcium absorption from the gut by increasing the synthesis of the active form of vitamin D.
- PTH lowers blood phosphate by decreasing the reabsorption of phosphate in the proximal tubules of the kidney.
- Secretory cells of the parathyroid glands directly monitor blood calcium concentrations and increase their rates of PTH secretion when calcium level decline. Conversely, high concentrations of blood calcium inhibit PTH secretion.
- Calcitonin is secreted by the C cells of the thyroid gland in response to increasing concentrations of blood calcium. Its principal physiologic effect is to inhibit the activity of osteoclasts in bone.
- Ultraviolet light catalyzes the conversion of 7-dehydrocholestrol to vitamin D3 in the skin. Successive hydroxylations in the liver at carbon 25 and in the kidney at carbon 1 results in the active form, 1,25dihydroxycholicalcifrol.
- 1,25dihydroxycholicalcifrol increases calcium absorption in the intestine and the kidney and promotes calcium mobilization from bone

Conclusion:

Students should be able to:

- Recall Calcium Metabolism, Sources, Distributions & Functions
- Understand Organ & Endocrinal Homeostasis of Calcium with recognition of the roles of:

  - Vitamin D

  - Parathyroid Hormone (PTH)

  - Calcitonin Hormone

  - Parathyroid hormone related peptide (PTHrP).

Take-home messages:

- The concentration of ionized calcium in blood is regulated within narrow limits.
- The parathyroid hormone and its effect on bone, kidneys, and the intestinal epithelium.
- The function of Calcitonin.
- Vitamin D compounds, the sequences of event leading to the formation of an active form of vitamin D3 and its regulatory role in calcium reabsorption.
- The sources and effects of Parathyroid hormone related peptide (PTHrP).

Further readings:

1. Review of Medical Physiology by: William F. Ganong, Chapter 21, Pages 369 – 382.

2. Essential Medical Physiology by : Leonard R. Johnson, Chapter 43, Pages 597 – 615.

Title of the lecture:	Vitamin D, and Rickets
Lecturer's name	Dr. Zeyad Kurdee
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	

Objectives:

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

- Understand the functions, metabolism and regulation of vitamin D
- Discuss the role of vitamin D in calcium homeostasis
- Identify the types and causes of rickets
- Correlate vitamin D and calcium deficiency in osteoporosis
- Identify biomarkers used for the diagnosis and follow up of osteoporosis

Keywords:

Vitamin D, 1,25-dihydroxycholecalciferol, cholecalciferol, □-hydoxylase, metabolism, calcium homeostasis, rickets, osteoporosis, biomarkers

Background:

- Vitamin D is a steroid hormone that performs essential functions in the body including calcium homeostasis, bone mineralization and calcium absorption.
- Skin, liver and kidneys play a major role in its synthesis whereas it is tightly regulated in the kidneys.
- Vitamin D deficiency is generally prevalent in the general population especially in Saudi Arabia.
- Rickets and osteoporosis are one of the diseases of vitamin D deficiency.

Main concepts in the lecture:

Vitamin D is considered a steroid hormone. Its mechanism of action is like steroid hormones. The steps of its synthesis involve skin, liver and kidneys where it is finally formed as active 1,25-dihydroxycholecalciferol. Vitamin D plays an important role in calcium homeostasis in the body by influencing calcitonin and parathyroid hormones. It regulates plasma calcium and phosphate levels and increases intestinal calcium absorption. Deficiency of vitamin D is common in the general population

especially in Saudi Arabia where deficiency prevails in the majority of the population. Vitamin D deficiency in children can lead to nutritional rickets, a disease mainly because of nutritional deficiency of vitamin D. Other type of rickets is due to genetic defects in vitamin D synthesis or its receptor. Rickets is characterized by bone demineralization that causes the bones to become soft leading to skeletal deformities. Osteoporosis is another disease in which deficiency of vitamin D partly contributes to the disease. Postmenopausal women are at high risk of developing osteoporosis It is characterized by reduction in bone mass per unit volume leading to bone fragility and fractures. Biomarkers for the diagnosis of osteoporosis include bone formation and resorption markers such as osteocalcin, bone-specific alkaline phosphatase, P1NP and CTX-1.

Conclusion:

- Vitamin D is an essential micronutrient that regulates calcium homeostasis and bone function.
- Rickets and osteoporosis are due to vitamin D deficiency.
- A number of biomarkers help diagnose osteoporosis.

Take-home messages:

- Overview of vitamin D metabolism and regulation.
- Importance of vitamin D functions.
- Vitamin D deficiency is common in populations.
- Rickets and osteomalacia are due to vitamin D deficiency.
- Various biochemical markers clinically important for assessment of osteoporosis.

Further readings:

- Lippincott's Biochemistry 6th Edition, pp. 386-389.
- Clinical Biochemistry: An illustrated colour text 5th Edition by Allan Gaw (Churchill Livingstone)
- Wheater, G. et al. The clinical utility of bone marker measurements in osteoporosis. *J. Trans. Med*. 2013, 11: 201-214.

Title of the lecture:	Pathology of thyroid and parathyroid gland- Practicals
Lecturer's name	**Dr. Abdullah Basabien /Dr. Mohammed Alswayyed**
Department	Pathology
Block / week	Endocrine Block / week 3
Email address	abasabein.c@ksu.edu.sa/alswayyed@hotmail.com

Objectives:

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

- Identify the gross macroscopic picture of thyroid and suprarenal nodules.
- Describe the microscopic picture of thyroid adenomas and carcinomas.
- Recognize the histopathological changes found in thyroid and suprarenal tumors.

Contents:

Gross pathology and histopathology section pictures of:

- Multinodular goiter.
- Hashimoto's thyroiditis.
- Papillary thyroid carcinoma.
- Addison's disease.
- Cushing syndrome.
- Follicular adenoma.
- Pheochromocytoma.

Title of the lecture: Pharmacology Vitamin D and Calcium	
Lecturer's name	Dr. Ishfaq Bukhari
Department	Pharmacology
Block / week	Endocrine Block / week 3
Email address	iabukhari@ksu.edu.sa

Objectives:

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

- By the end of lecture, the students will be able to :
- Recognize the common drugs used in calcium & vitamin D disorders
- Classify them according to sources & Pharmacological effects
- Detail the pharmacology of each drug , regarding , Mechanism, clinical utility in affecting calcium & vitamin D

Title of the lecture:	Embryology and Anatomy adrenal glands
Lecturer's names	Dr. Essam Salama / Dr. Dr. Jamila El Medany
Department	Anatomy
Block / week	Endocrine Block / week 4
Email address	Essamco58@gmail.com / galmadani@ksu.edu.sa

Objectives of the lecturer:

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

- Describe the position of the pituitary gland.
- List the structures related to the pituitary gland.
- Differentiate between the lobes of the gland regarding the structure and function.
- Describe the blood supply of pituitary gland & the hypophyseal portal system.

Title of the lecture:	Histology of adrenal gland
Lecturer's name	Dr Aly Mohammad
Department	Anatomy
Block / week	Endocrine Block / week 4
Email address	Alymahmed53@hotmail.com

Objectives:

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

1. Differentiate between adrenal cortex and adrenal medulla.
2. Identify the histological features of each cortical zone and its cells.
3. Identify the histological features of the medullary cells.

Background:

- Stroma of the adrenal gland.
- Parenchyma of the adrenal gland.
- Blood supply of the adrenal gland.

Main concepts in the lecture:

Adrenal cortex contains:

- Zona glomerulosa.
- Zona fasciculata.
- Zona reticularis.
- Blood Capillaries.

Adrenal medulla contains:

- Chromaffin cells (Pheochromocytes).
- Sympathetic ganglion cells.
- Blood capillaries.

Conclusions:

- The adrenal cortex contains steroid hormone secreting cells.
- The adrenal medulla contains catecholamine secreting cells

Take-home messages:

- Differentiate between adrenal cortex and medulla.
- Identify the histological features of each cortical zone and its cells.
- Identify the histological features of the medullary cells.

Further readings:

- Recommended textbooks: Color Textbook of Histology (Gartner and Hiatt)- Latest Edition.

Title of the lecture: Adrenal Gland hormones (Mineralocorticoids)	
Lecturer's name	Dr. Khalid AlRegaiey
Department	Physiology
Block / week	Endocrine Block / week 4
Email address	kalregai@gmail.com

Objectives:

- Describe the cellular arrangements and functional components of the adrenal gland.
- List the hormones secreted by the cortex of the adrenal gland.
- Summarize regulation of secretion of adrenocortical steroids.
- Discuss regulation of aldosterone secretion.
- List the major stimuli for aldosterone secretion.
- Explain how negative feedback regulates aldosterone secretion.

Title of the lecture: Adrenal Gland hormones- Glucocorticoids Part 1 and androgens Part 2	
Lecturer's name	Dr. Khalid AlRegaiey
Department	Physiology
Block / week	Endocrine Block / week 4
Email address	kalregai@gmail.com

Objectives:

- Explain regulation of glucocorticoid and adrenal androgen secretion.
- List the trigger(s) for cortisol secretion.
- Describe the physiological actions of aldosterone.
- Outline the actions of glucocorticoids.
- Summarize the actions of adrenal androgens.
- Describe the causes and major manifestations of hyperadrenocorticism and Hypoadrenocorticism

Title of the lecture:	Physiology of Adrenal Medulla and pheochromocytoma
Lecturer's name	Dr. Khalid AlRegaiey
Department	Physiology
Block / week	Endocrine Block / week 4
Email address	kalregai@gmail.com

Objectives:

- List the hormones secreted by the medulla of the adrenal gland.
- Describe circumstances in which catecholamines are released from the adrenal gland.
- List the major actions of catecholamines.
- Define and describe the major manifestation of pheochromocytoma

Title of the lecture: Addison Disease	
Lecturer's name	Dr. Ahmed Mujamammi
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 4
Email address	amujamammi@ksu.edu.sa

Objectives:

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

- To identify different causes of Addison's disease
- To identify secondary causes of adrenocortical hypofunction
- To understand the diagnostic algorithm for adrenocortical hypofunction
- To understand the interpretation of laboratory tests of Addison's disease

Background:

The adrenal gland consists of 2 distinct tissues of different embryological origin, the outer cortex and the inner medulla. Adrenal cortex secretes glucocorticoids, mineralocorticoids and sex hormones. Disorders of adrenal cortex are uncommon, but they can be readily treated. Diagnosis of disorders of adrenal cortex can be achieved by performing sensitive screening tests and followed by specific tests for confirmation.

Main concepts in the lecture:

- Regulation of hormonal secretion by adrenal cortex through hypothalamic/anterior pituitary/adrenal axis.
- Causes of Addison's disease
- Adrenocortical hypofunction secondary to pituitary disease
- Provocative (stimulation) tests for Addison's disease
- Diagnostic algorithm (flowchart) for a suspected case of adrenocortical hypofunction

Take-home messages:

- Addison's disease is due to destruction of adrenals by autoimmune, infection, or infiltrative lesions

- Adrenocortical hypofunction may occur secondary to pituitary disease, e.g., tumors, infection, trauma, or iatrogenic (surgery or radiation)
- Initial screening for Addison's disease by serum cortisol and ACTH. Other tests to support the diagnosis include serum urea, electrolytes and glucose
- Confirmatory tests for Addison's disease by short Synacthen test
- Diagnosis of secondary adrenocortical hypofunction by depot (long) Synacthen test

Further readings:

- Lecture notes, Clinical Biochemistry, Wiley BlackWell, 9th edition, 2013, chapter 9, page 116-133.
- Clinical Chemistry, Principles, Procedures, Correlations, Lippincott Williams & Wilkins, 7th edition, 2013, chapter 21, page 453-471.
- Lippincott's Illustrated Reviews: Biochemistry 6th edition, Unit III, Chapter 18, Pages 219-244.

Title of the lecture:	**Biochemistry of Obesity: Role of Hormones**
Lecturer's name	Dr. Usman Ghani
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 4
Email address	Ughani@ksu.edu.sa

Objectives:

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

- Define and characterize obesity in terms of BMI and risk factors.
- Compare the anatomic and biochemical differences in fat deposition.
- Understand the role of adipocytes in fat storage and release of hormones.
- Discuss the hormonal control of obesity by leptin, adiponectin and other hormones
- Discuss the management and treatment options for obesity.

Keywords:

Obesity, BMI, fat depots, subcutaneous, visceral fat, ectopic fat, leptin, adiponectin, ghrelin, CCK, insulin, orlistat, lorcaserin, bariatric surgery

Background:

- Obesity is a disorder of the body weight regulatory systems characterized by an accumulation of excess body fat.
- Sedentary lifestyle and abundance and wide variety of palatable, inexpensive foods has contributed to an obesity epidemic.
- Obesity is strongly associated with a risk for arthritis, diabetes, hypertension, cardiovascular disease and cancer.

Main concepts in the lecture:

- BMI, mortality risk associated with high BMI.
- Factors contributing to obesity such as environmental and genetic.
- Anatomic differences in fat deposition.

- Biochemical differences in different fat depots.
- Adipocytes as an endocrine organ.
- Hormonal control of obesity mainly by leptin and adiponectin.
- Role of other hormones such as ghrelin, CCK and insulin.
- Metabolic changes in obesity and fat loss.
- Treatment options include drugs and surgery.

Conclusion:

- Obesity is due to accumulation of body fat that results from excessive energy intake than expenditure.
- BMI can be calculated from height and weight that correlates well to body fat.
- The anatomic distribution of fat has a major influence on associated health risks.
- The afferent signals, circulating hormones, and metabolites influence appetite. Long-term signals are generated by leptin an adiponectin whereas short-term signals are generated by ghrelin and CCK to control hunger and satiety.
- Weight reduction is achieved best with a reduction in caloric intake, modest reduction with pharmacologic treatment. Surgical procedures are designed to limit food intake for the severely obese patients who do not respond to other treatments.

Take-home messages:

- Obesity is correlated to an increased risk for a number of chronic conditions and mortality.

Further readings:

- Lippincott's Biochemistry. 5th Edition, pp 349-356. Lippincott Williams & Wilkins, New York, USA.

Title of the lecture: Cushing Syndrome	
Lecturer's name	Dr. Ahmed Mujamammi
Department	Pathology
Block / week	Endocrine Block / week 4
Email address	amujamammi@ksu.edu.sa

Objectives:

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

- To identify different causes of Cushing's syndrome.
- To understand the diagnostic algorithm for Cushing's syndrome
- To understand the interpretation of laboratory tests of Cushing's syndrome
- To identify the importance of radiological investigations for diagnosis of Cushing's syndrome

Background:

The adrenal gland consists of 2 distinct tissues of different embryological origin, the outer cortex and the inner medulla. Adrenal cortex secretes glucocorticoids, mineralocorticoids and sex hormones. Disorders of adrenal cortex are uncommon, but they can be readily treated. Diagnosis of disorders of adrenal cortex can be achieved by performing sensitive screening tests and followed by specific tests for confirmation.

Main concepts in the lecture:

- Regulation of hormonal secretion by adrenal cortex through hypothalamic/anterior pituitary/adrenal axis.
- Causes of Cushing's syndrome
- Suppression and provocative (stimulation) tests for Cushing's syndrome
- Diagnostic algorithm (flowchart) for a suspected case of Cushing's syndrome

Take-home messages:

- ACTH-dependent Cushing: due to pituitary causes (Cushing's disease) and due to ectopic production of ACTH.

- ACTH-independent Cushing: due to adrenal adenoma or carcinoma and due to steroid therapy (iatrogenic).
- Initial screening for Cushing by 24 h urine free cortisol or low-dose dexamethasone suppression test
- Confirmatory tests for Cushing by diurnal rhythm of plasma cortisol and insulin-induced hypoglycemia
- Tests to determine the cause of Cushing: Plasma ACTH, high-dose dexamethasone suppression test, CRH stimulation test and radiological investigations

Further readings:

- Lecture notes, Clinical Biochemistry, Wiley BlackWell, 9th edition, 2013, chapter 9, page 116-133.
- Clinical Chemistry, Principles, Procedures, Correlations, Lippincott Williams & Wilkins, 7th edition, 2013, chapter 21, page 453-471.
- Lippincott's Illustrated Reviews: Biochemistry 6th edition, Unit III, Chapter 18, Pages 219-244.

Title of the lecture:	Pathology of the Adrenal Glands
Lecturer's names	Dr. Mohammed AlSwayyed
Department	Pathology
Block / week	Endocrine Block / week 4
Email address	alswayyed@hotmail.com

Objectives of the lecturer:

- Understand the structure and function of adrenal glands.
- Know the common disorders that can affect the adrenal medulla.
- Know the disorders that can cause hypo or hyper function of the adrenal cortex.

Background:

- Pathology, Anatomy, Histology and Physiology of Adrenal Glands

Keywords:

- Pathology of phaechromocytoma, adrenocortical neoplasms, Cushing's syndrome, Conn's syndrome and Addison disease.

Main concepts in the lecture:

The content of the two lectures is as follows:

- Pathology of phaechromocytoma.
- Pathology of adrenocortical neoplasms, Cushing's syndromes, Conn's syndrome and Addison disease.

Conclusion:

(Please refer to learning objectives and contents).

Take home messages:

- Structure and function of adrenal glands
- Classification and pathological changes of the major disorders seen in the adrenal glands.

Further reading:

- Robbin's and Cotran, Pathologic Basis of Disease, 9th Edition

Title of the lecture: Pharmacology of Corticosteroids	
Lecturer's name	Dr.Saeed Ahmed Shiekh
Department	Pharmacology
Block / week	Endocrine Block / week 4
Email address	sheikhsa63@gmail.com

Objectives:

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

- Define and classify corticosteroids
- To Compare Various Corticosteroids
- Concept of mechanism of action and pharmacological effects of corticosteroids
- Explain the clinical uses of corticosteroids
- To discuss the adverse effect profile of corticosteroids

Title of the lecture:	Epidemiology of Obesity
Lecturer's name	Dr. Ebraheem Qusadi
Department	Family and Community Medicine
Block / week	Endocrine Block / week 4
Email address	gossady@hotmail.com

Objectives:

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

- Describe the magnitude of the problem of obesity.
- Recognize the consequences of obesity.
- List the factors leading to obesity.
- Discuss the prevention of obesity

Title of the lecture:	Histology of pancreas
Lecturer's name	Dr Aly Mohammad
Department	Anatomy
Block / week	Endocrine Block / week 5
Email address	Alymahmed53@hotmail.com

Objectives:

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

4. The microscopic structure of pancreatic islets of Langerhans.
5. The microscopic structure of the cells of pancreatic islets of Langerhans in correlation with their functions.

Background:

- Stroma of the pancreatic islets of Langerhans.
- Parenchyma of the pancreatic islets of Langerhans.
- Blood supply of the pituitary gland.

Main concepts in the lecture:

Pancreatic islets of Langerhans contain:

1- Alpha cells.

2- Beta cells.

3- D cells.

4- PP cells

5- Blood Capillaries.

Conclusions:

- Pancreatic islets of Langerhans secrete glucagon, insulin, somatostatin and pancreatic polypeptide

Take-home messages:

- The microscopic structure of pancreatic islets of Langerhans.
- The microscopic structure of the cells of pancreatic islets of Langerhans in correlation with their functions

Further readings:

- Recommended textbooks: Color Textbook of Histology (Gartner and Hiatt)- Latest Edition.

Title of the lecture:	Anatomy of the Pancreas
Lecturer's names	Dr. Mohammed Vohra
Department	Anatomy
Block / week	Endocrine Block / week 5
Email address	vohra@ksu.edu.sa

Objectives of the lecturer:

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

- Describe the anatomical view of the pancreas regarding; location, parts relations, ducts
- Know the arterial supply & venous drainage

Title of the lecture: Physiology of Pancreas	
Lecturer's name	Dr. Ahmad Alsabeeh
Department	Physiology
Block / week	Endocrine Block / week 5
Email address	

Objectives:

- Describe the cellular arrangements and functional components of the pancreas
- List the hormones secreted by the pancreas.
- Outline the regulation of insulin secretion.
- Describe the mechanism of action of insulin.
- Describe actions of pancreatic Somatostatin.
- Outline the physiological and biochemical actions of insulin.
- Describe the consequences of insulin deficiency.
- Describe mechanism of action of glucagon.
- Outline regulation of glucagon secretion.
- Outline the physiological and biochemical actions of glucagon.
- Outline the effects of other hyperglycemic hormones.

Title of the lecture:	Biochemistry of Metabolic Syndrome
Lecturer's name	Dr. Usman Ghani
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	Ughani@ksu.edu.sa

Objectives:

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

- Define metabolic syndrome, insulin resistance and dyslipidemia.
- Discuss the risk factors for metabolic syndrome and other medical conditions associated with it.
- Define the diagnostic criteria for Metabolic syndrome.
- Discuss the management of metabolic syndrome and current treatment options.

Keywords:

- metabolic syndrome, obesity, insulin resistance, dyslipidemia, metformins, fibrates, TZDs

Background:

- Metabolic syndrome is a group of risk factors that raises the risk for heart disease and other health problems such as diabetes and stroke.
- The risk for heart disease, diabetes, and stroke increases with the number of metabolic risk factors.
- Metabolic syndrome is becoming more common because of increasing prevalence of diabetes and obesity.

Main concepts in the lecture:

- Definition and markers of metabolic syndrome.
- Metabolic changes observed in obesity, effects of insulin resistance and relation of Dyslipidemia with metabolic syndrome.
- Risk factors for metabolic syndrome and medical conditions associated with metabolic syndrome.
- Criteria for the diagnosis of metabolic syndrome.
- Management of metabolic syndrome- primary and secondary intervention.

- Current treatment options with metformins, fibrates and thiazolidinediones.

Conclusion:

- Metabolic syndrome is a cluster of conditions that includes hypertension, insulin resistance, hyperglycemia, obesity and dyslipidemia.
- These conditions increase the risk for heart disease, stroke and diabetes.

Take-home messages:

- Metabolic syndrome is a combination of metabolic abnormalities that increase the risk of heart disease, diabetes and other diseases.
- The features of metabolic syndrome include obesity, high serum triglycerides (TGs), low HDL cholesterol, hypertension, hyperglycemia and insulin resistance
- Obesity, alcoholism, sedentary lifestyle and smoking are some of the risk factors for metabolic syndrome.
- Management of the syndrome includes lifestyle modifications to reduce weight and medications.

Further readings:

- Textbook of Biochemistry with Clinical Correlations by Thomas M. Devlin, 6th Edition, pp 862-863.
- Lippincott's Biochemistry. 5th Edition, pp 353-355, Lippincott Williams & Wilkins, New York, USA.

Title of the lecture:	Metabolic changes in Diabetic Mellitus
Lecturer's name	Dr. Zeyad Kurdee
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	

Objectives:

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

- To understand the differences between type 1 and 2 DM
- To understand the natural course of type 1 and 2 DM
- To understand the diagnostic criteria for DM
- To identify the metabolic changes in DM including: increase of hepatic glucose output; decrease of glucose uptake; and inter-organ relationship in T1DM and T2DM
- To identify the mechanisms of diabetic complications

Background:

Diabetes mellitus (DM) is a heterogeneous group of multifactorial, polygenic syndromes characterized by an elevated fasting blood glucose levels caused by an absolute or relative insulin deficiency. DM is the leading cause of adult blindness and amputation and a major cause of renal failure, nerve damage, heart attacks, and strokes. Most cases of DM can be separated into two groups, type 1 DM (insulin-dependent DM) and type 2 DM (noninsulin-dependent DM). The incidence and prevalence of type 2 DM is increasing because of the aging of the population and the increasing prevalence of obesity and lifestyles. The increase in children with type 2 DM is particularly disturbing.

Main concepts in the lecture:

- Differences between type 1 and 2 DM.
- Natural course of type 1 and 2 DM.
- Diagnostic criteria for DM.
- Metabolic changes in DM and mechanisms of diabetic complications**.**

Take-home messages:

- DM is a group of disorders characterized by high blood glucose levels, as a result of faults in the production of insulin, action of the produced insulin, or both.

- Type 1 DM usually attacks children and young adults, but the onset of the disease can strike at any age.
- In type 1 DM, the islets of Langerhans become infiltrated with activated T lymphocytes, leading to insulitis. Over a period of years, this autoimmune attack on the β cells leads to gradual depletion of the β-cell population.
- Type 2 DM is the most common form of the disease and is associated with ethnicity, family history, impaired metabolism of glucose, older age, obesity and physical inactivity.
- The effects of DM involve long–term damage, dysfunction and failure of various organs.
- DM may present with thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a hyperosmolar hyperglycemic state (HHS) may develop.
- The long–term effects of DM include progressive development of the specific complications of nephropathy, neuropathy and/or retinopathy.

Further readings:

- Lippincott's Illustrated Reviews: Biochemistry 6th edition, Unit V, Chapter 25, Pages 337-348.
- Clinical Chemistry, Principles, Procedures, Correlations, Lippincott Williams & Wilkins, 7th edition, 2013, chapter 14, page 298-310.

Title of the lecture:	Biochemistry Diabetic Ketoacidosis (DKA)
Lecturer's name	Dr. Zeyad Kurdee
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	

Objectives:

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

- To understand diabetic emergencies including diabetic ketoacidosis (DKA), hyperosmolar hyperglycemic state (HHS) and hypoglycemia.
- To have a knowledge about DKA: definition; causes; mechanisms; manifestations and precipitating factors.
- To understand the terms of ketogenesis and ketolysis.
- To have a knowledge about HHS: definition; causes; mechanisms and manifestations.
- To know about hypoglycemia, its clinical presentation, hormonal mechanisms to prevent hypoglycemia and glycemic thresholds for various responses to hypoglycemia.

Background:

Diabetes mellitus (DM) is a heterogeneous group of multifactorial, polygenic syndromes characterized by an elevated fasting blood glucose levels caused by an absolute or relative insulin deficiency. DM is the leading cause of adult blindness and amputation and a major cause of renal failure, nerve damage, heart attacks, and strokes. Most cases of DM can be separated into two groups, type 1 DM (insulin-dependent DM) and type 2 DM (noninsulin-dependent DM). The incidence and prevalence of type 2 DM is increasing because of the aging of the population and the increasing prevalence of obesity and lifestyles. The increase in children with type 2 DM is particularly disturbing.

The diabetic emergencies include Diabetic ketoacidosis (DKA), Hyperosmolar Hyperglycemic State (HHS) and Hypoglycemia. DKA is a possible life-threatening complication of DM. It frequently occurs in patients with type 1 DM, but under certain circumstances, it can occur in patients with other types of diabetes. DKA consequences of an insulin deficiency and is characteristically diagnosed when testing finds show elevated blood glucose, low blood pH, and presence of ketone bodies in either the blood or urine. HHS is a complication of DM, chiefly type 2, in which high blood sugars cause severe dehydration, leading to an increment in osmolarity. HHS is related to DKA, but they can be distinguished by measuring ketone bodies, organic molecules that are typically undetectable in HHS. Hypoglycemia is characterized by central nervous system symptoms, including aberrant behavior, coma or confusion; the level of blood

glucose equals to or less than 40 mg/dl; and following the administration of glucose, the symptoms are resolved within minutes.

Main concepts in the lecture:

- Diabetic emergencies.
- Diabetic keto acidosis (DKA).
- Hyperosmolar Hyperglycemia State (HHS).
- Hypoglycemia.
- Ketogenesis and ketolysis.

Take-home messages:

- Acute complications of DM include: DKA, HHS, and hypoglycemia
- DKA is a triad of hyperglycemia, ketonemia and high anion gap
- metabolic acidosis, and can be precipitated by several stressful factors.
- Ketone bodies (KB) are synthesized in the liver (HMG CoA synthase is the rate limiting enzyme) and utilized by peripheral organs and not the liver (liver lacks thiophorase enzyme)
- KB can serve as energy source (this is important for the brain in case
- of hypoglycemia)
- In DKA there is excessive ketogenesis (more than ketolysis) *(details of the mechanisms and consequences are required)*
- HHS is a serious condition, usually occurs in elderly with T2DM, and has
- high mortality rate.
- Hypoglycemia is a medical emergency that might be caused by DM treatment (intensive) and impaired protective mechanisms against hypoglycemia. Its clinical manifestations are due to sympathetic overactivity and neuroglycopenia.
- Case presentation, examination of DKA can provide provisional diagnosis, and should be confirmed by comprehensive blood and urine lab investigation including measuring blood glucose, KB, pH, pCO2, electrolytes, osmolality, protein, and kidney function test; anion gap calculation; hematocrit; and urine glucose and KB.

Further readings:

- Lippincott's Illustrated Reviews: Biochemistry 6th edition, Unit V, Chapters 16, 23 and 25, Pages 195-197, 315-318 and 337-348.

Title of the lecture:	Glucose Homeostasis
Lecturer's name	Dr. Usman Ghani
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	ughani@ksu.edul.sa

Objectives:

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

- Define glucose homeostasis and the metabolic processes involved.
- Differentiate between different phases of glucose homeostasis.
- Discuss the primary sources of energy and major organs utilizing glucose during the five phases of homeostasis.
- Understand the role of hormones in maintaining glucose homeostasis.

Keywords

- glucose, homeostasis, glycogenolysis, gluconeogenesis, ketone bodies, phases, insulin, glucagon, fatty acid oxidation

Background:

Glucose is the main source of fuel for the cells in our bodies, but it's too big to simply diffuse into the cells by itself. Instead, it needs to be transported into the cells. The low blood concentrations of glucose can causes seizures, loss of consciousness, and death. On the other hand, long lasting elevation of blood glucose concentrations, can result in blindness, renal failure, vascular disease, and neuropathy. Therefore, blood glucose concentrations need to be maintained within narrow limits. The process of maintaining blood glucose at a steady-state level is called glucose homeostasis.

Main concepts in the lecture:

- What is glucose homeostasis?
- Different sources of glucose.
- The five phases of glucose homeostasis including the origin of glucose and the major fuel of brain during those stages.
- Hormonal involvement of glucose homeostasis including the role of insulin, Glucagon and others that antagonize the action of insulin like cortisol and growth hormone.

Conclusion:

- Glucose Homeostasis is the balance of insulin and glucagon to maintain blood glucose.
- Insulin is secreted by the pancreas in response to elevated blood glucose following a meal and lowers blood glucose by increasing glucose uptake in muscle and adipose tissue and by promoting glycolysis and glycogenesis in liver and muscle.
- A fall in blood glucose increases the release of glucagon from the pancreas to promote glucose production.

Take-home messages:

- Glucose homeostasis is a process that controls glucose metabolism and maintains blood glucose level in the body
- There are five phases of glucose homeostasis- Phase I (well-fed state), Phase II (glycogenolysis), Phase III (gluconeogenesis), Phase IV (glucose, ketone bodies (KB) oxidation), Phase V (fatty acid (FA), KB oxidation)
- Hormones that regulate glucose metabolism include insulin (lowers glucose level) and glucagon (increases glucose level.
- Other hormone such as cortisol, growth hormone and adrenaline are known to antagonize the actions of insulin thus increases the blood glucose level.

Further readings:

- Textbook of Biochemistry with Clinical Correlations by Thomas M. Devlin, 6th Edition, pp 862-863.
- http://www.vivo.colostate.edu/hbooks/pathphys/endocrine/pancreas/insulin_phys.html

Title of the lecture:	Measurement of glucose in blood and urine - Practical
Lecturer's name	Dr. Usman Ghani / Dr. Ahmed Mujammami
Department	Pathology (Biochemistry)
Block / week	Endocrine Block / week 5
Email address	Ughani@ksu.edu.sa /amujamammi@ksu.edu.sa

Objectives:

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

- Perform the measurement of glucose in blood and urine using glucometer and dipstick methods respectively.
- Understand the principle of the glucose tests.
- Record and calculate the results obtained from the experiments.
- Interpret the results.
- Diagnose conditions or diseases such as hyperglycemia, diabetes mellitus and associated complications using ADA guidelines.

References:

- Presentation slides
- American Diabetes Association (ADA) website

Title of the lecture:	Pathology of Diabetes Mellitus Type 1 and Type 2
Lecturer's names	Dr. Mohammed Alswayyed
Department	Pathology
Block / week	Endocrine Block / week 5
Email address	alswayyed@hotmail.com

Objectives of the lecturer:

- Understand the structure of the pancreas and have a basic understanding of its function.
- Have an understanding of the classification, pathogenesis, clinical features and complications of diabetes mellitus.

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:

- Diabetes mellitus, pancreas, classification, pathogenesis, clinical features and complications.

Main concepts in the lecture:

The content of the two lectures is as follows:

- Classification, pathogenesis and major clinical and histopathological changes seen in diabetes mellitus type 1 and 2.
- Complications of diabetes mellitus.

Conclusion:

(Please refer to learning objectives and contents).

Take home messages:

- Structure and function of pancreas.
- Classification and pathological changes in the pancreas in cases of diabetes mellitus.

Further reading:

- Robbin's and Cotran, Pathologic Basis of Disease, 9th Edition

Title of the lecture:	Common infections in Diabetes Mellitus
Lecturer's names	Prof. Ali Somily
Department	Microbiology
Block / week	Endocrine Block / week 5
Email address	Ali.somily@gmail.com/somily@ksu.edu.sa

Objectives of the lecturer:

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

- Define the term Diabetes Mellitus *(Diabetic foot).*
- Know the common infections in Diabetes Mellitus *(Diabetic foot).*
- Know the pathogenesis in the common infections in Diabetes Mellitus *(Diabetic foot).*
- Know the organisms that cause the common infections in Diabetes Mellitus *(Diabetic foot).*
- Know the clinical features of infections in Diabetes Mellitus *(Diabetic foot).*
- State the laboratory diagnostic tests and the radiological test of the infections in Diabetes Mellitus *(Diabetic foot).*
- State the complications of Diabetes Mellitus *(Diabetic foot),* mainly those common infections in Diabetes Mellitus *(Diabetic foot).*
- Know the management of infections in Diabetes Mellitus *(Diabetic foot)* including nursing management and anti-microbial management.

Title of the lecture:	Candidiasis
Lecturer's name	Dr. Ahmed Albarrag
Department	Pathology (Microbiology)
Block / week	Endocrine Block / week 5
Email address	aalbarrag@ksu.edu.sa

Objectives:

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

- Acquire the basic knowledge about *Candida* as a pathogen
- know the main infections caused by *Candida* species
- Identify the clinical settings of such infections
- Know the laboratory diagnosis, and treatment of these infections.

Keywords:

Candidiasis , *Candida* , Candidemia, Pneumonia, Infection

Background:

Candidiasis refer to infection caused by any species of the of the genus *Candida* . Candidiasis is the most common fungal infection. There are many species of *Candida*, the most common is *Candida albicans*. *Candida* is normal flora of skin, oral cavity, gut, vagina, and urethra. However, under certain conditions, it can cause infections, ranging from superficial and cutaneous disease to more life-threatening infections particularly in immunocompromised individuals.

In people with reduced immunity such as AIDS, Malignancy, Diabetes are frequently affected. This yeast can reach the bloodstream and the infection may spread to other organs. The invasive candidiasis is associated with high mortality if not treated early.

Main concepts in the lecture:

- *Candida* is a unicellular yeast fungus. reproducing by budding
  - Microscopy:Budding yeast cells, and Pseudohyphae.
  - Culture: Creamy colony, fast growing on Sabouraud Dextrose agar (SDA), Blood agar (48 hr)
- *Candida* is Human commensal (Oral cavity, Skin, Gastrointestinal tract, Genitourinary tracts)
- Alteration in immunity, normal flora, or damage in the barriers could lead to a disease.

Infection caused by any species of the genus *Candida* is referred to as candidiasis. Candidiasis is the most common fungal infection. There are many species of *Candida*, the most common

is *Candida albicans*. This yeast can reach the bloodstream and the infection may spread to other organs. The invasive candidiasis is associated with high mortality in case of delay in initiation of appropriate therapy.

Candidiais include:

- Mucous membrane infections include Thrush (oropharyngeal), Esophagitis, Vaginitis
- Cutaneous infections (Paronychia, Onychomycosis , Diaper rash, Chronic mucotaneous candidiasis)
- Urinary tract infection
- Candidemia
- Disseminated (systemic, invasive) infections

Candidiasis : Laboratory diagnosis

Specimen depend on site of infection.

1. Direct microscopy : Gram stain, Giemsa, GMS, stained smears.

Positive: Budding yeast cells and pseudohyphae will be seen in stained smear or KOH.

2. Culture: Culture on SDA and blood agar

Candida will grow on SDA within 24-48

Because *C. albicans* is the most common species to cause infection

The following tests are used to identify *C. albicans:*

1. Germ tube test : Formation of germ tube when cultured in serum at 37°C
2. Chlamydospore production in corn meal Agar
3. Resistance to 500 μg/ml Cycloheximide

- If these 3 are positive this yeast is *C.albicans,*
- If negative, then it could be any other yeast:
  - Use Carbohydrate assimilations. (Commercial kits available for this like: API 20C, API 32C
  - Culture on Chromogenic Media (CHROMagar™ Candida)

Treatment of Candidiasis

Treatment depends on the type and site of the infection

Oropharyngeal: Topical Nystatin suspension, Clotrimazole troches ,Miconazole, Fluconazole suspension.

Vaginitis: Miconazole, Clotrimazole, Fluconazole

Systemic treatment of Candidiasis (Selection is based on *Candida* species and the site)

Fluconazole

Voriconazole
Caspofungin
Amphotericin

In candidemia :Treat for 14 days after last positive culture and resolution of signs and symptoms

Remove catheters, if possible

Antifungal susceptibility testing in the following cases:

- For *Candida* isolated from sterile samples
- If the patient is not responding to treatment
- In case of recurrent infections

Points to consider:

- *C. glabrata* can be less susceptible or resistant to fluconazole
- *C. krusei* is intrinsically resistant to fluconazole

Conclusion:

- Candidiasis is the most common fungal infection.
- There are many species of *Candida*, the most common is *Candida albicans*.
- *Candida* is normal flora of skin, oral cavity, gut, vagina, and urethra. However, alteration in immunity, normal flora, or damage in the barriers could lead to a disease.
- It can cause infections, ranging from superficial and cutaneous disease to more life-threatening infections particularly in immunocompromised individuals.
- The invasive candidiasis is associated with high mortality if not treated early.
- Treatment can be topical or systemic.

Take-home messages:

- *Candida* is the most common fungal pathogen
- Infections range from superficial to more life-threatening invasive infections.
- Alteration in immunity, normal flora, or damage of the barriers could lead to the disease
- Diagnosis is based on direct microscopy and culture.
- Treatment selection is based on Candida species and type and site of the infection.

Further readings:

- Alhedaithy, S.S., Medical Mycology Lecture slides. 2009 (2nd Edition).
- •Sherries Medical Microbiology, an introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher: Mc Graw Hill.

Title of the lecture: Oral Hypoglycaemic drugs part 1 and part 2	
Lecturer's name	Prof. Al Humayyd
Department	Pharmacology
Block / week	Endocrine Block / week 5
Email address	

Objectives:

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

- Classify the different categories of oral hypoglycemic drugs
- Explain the mechanism of action, pharmacokinetics and pharmacodynamics of each class of oral hypoglycemic
- Describe the clinical uses of hypoglycemics
- Know the side effects, contradiction of each class of oral hypoglycemic.

Title of the lecture: Management of ketoacidosis and hypoglycemia	
Lecturer's name	Prof. Al Humayyd
Department	Pharmacology
Block / week	Endocrine Block / week 5
Email address	

Objectives:

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

- Identify the different characters of diabetic ketoacidosis.
- Know the different lines of treatment for hyperglycemia, dehydration, electrolyte deficits and ketoacidosis.
- Recognize the characters of hypoglycemia and how it can be prevented.
- Describe the different treatment of hypoglycemia
- Be able to differentiate between hypoglycemia and hyperglycemia coma.

Title of the lecture:	Epidemiology of Diabetes Mellitus
Lecturer's name	Dr. Ebraheem Qusadi
Department	Family and Community Medicine
Block / week	Endocrine Block / week 5
Email address	gossady@hotmail.com

Objectives:

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

- To list the types of Diabetes Mellitus
- To describe the prevalence of Diabetes Mellitus
- To recognize the importance of diagnostic criteria for estimating the prevalence of diabetes mellitus
- To discuss the risk factors and complications of type II diabetes mellitus

The College of Medicine and the Department of Medical Education are working on ensuring that our students receive optimal support to their learning. The list of academics shown below represents the departments involved in the teaching and learning of this block. If a student needs help in their teaching and learning they might consult one academic from the list. He/she might email them and arrange a time to see them if needed, otherwise email might be of help

ACADEMIC SUPPORT TEAM			
Names	**Department**	**Contact numbers**	**Email Addresses**
Prof. Riad Sulimani	Medicine	0505415166	rsulimani@ksu.edu.sa sulimanirs@gmail.com
Dr. Usman Ghani	Pathology	0551596921	ugresearch@hotmail.com
Prof. Samy Azer	Medical Education	0542307075	sazer@ksu.edu.sa
Dr. Essam Salama	Anatomy	0565252913	essamco58@gmail.com
Dr. Khalid Al Regaiey	Physiology	0505535005	kalregai@gmail.com
Prof. Ammar Rikabi	Pathology	0541842840	Ammar_rikabi12@yahoo.com rikabi@ksu.edu.sa
Dr. Abdulkarim F. Al Hetheel	Microbiology	0560793999	abdulkarimfahad@hotmail.com aalhetheel@ksu.edu.sa
Prof. Abdulrahman Al Motrefie	Pharmacology	0534761466	motrefi@ksu.edu.sa

Block Schedule– Male A Group

WEEK 1 –BLOCK ENDOCRINE (ENDO 225) (Male Group-A)				
Week (1) Starting: 21/01/2018 to 25/01/2018 **PITUITARY GLAND**				
CHAIR PERSON : Dr. Riad Al-Sulimani				
CO-CHAIR: Dr. Usman Ghani				
Sunday 21 January 2018	**Monday 22 January 2018**	**Tuesday 23 January 2018**	**Wednesday 24 January 2018**	**Thursday 25 January 2018**
8:00 - 9:00 am **Self- Directed Learning**	**8:00-9:00am** Introduction to the endocrine physiology **(Physiology)** **Prof. Abdulmajeed Aldress**	**8:00-9:00am** **Self- Directed Learning**	**8:00 - 9:00am** Physiology of the anterior pituitary gland (part 2**)** **(Physiology)** **Prof. Abdulmajeed Aldress**	**8:00 -9:00 am** Anterior pituitary disorders **(Medicine)** **Dr. Mohammed Mujammami**
9:00 - 11:00 am **PROFESSIONALISM FINAL EXAM**	**9:00– 10:00am** **Introduction to the Endocrine Block** **Prof. Riad Sulimani**	**9:00– 10:00am** **Self- Directed Learning**	**9:00 - 11:00 am** **(Practical)** **Anatomy and radiology of the pituitary gland** **(Anatomy, Dr Essam Salama and Radiology)** **All S taff**	**9:00 - 10:00 am** General mechanisms of actions of hormones **(Biochemistry)** **Dr. Zeyad Kurdee**
	10:00 - 11:00am **Anatomy & histology of the pituitary gland** **(Anatomy)** **Prof. Ahmed Fathalla** **Prof. Aly Mohammed**	**10:00 - 11:00am** **Self- Directed Learning**		**10:00 - 11:00am** **Self- Directed Learning**
11:00- 12:00 pm **Self- Directed Learning**	**11:00 -12:00pm** Physiology of hypothalamo-pituitary axis and regulatory mechanisms **(Physiology)** **Prof. Abdulmajeed Aldress**	**11:00- 12:00 pm** Physiology of the anterior pituitary gland (part 1) **(Physiology)** **Prof. Abdulmajeed Aldress**	**11:00- 12:00 pm** Physiology of the posterior pituitary **(Physiology)** **Dr. Khalid Al Regaiey**	**11:00- 12:00 pm** **Self- Directed Learning**
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 – 2:00pm **Self- Directed Learning**	**1:00 – 2:00pm** **Self- Directed Learning**	**1:00 - 3:00 pm** **Salam**	**1:00 – 2:00pm** **Self- Directed Learning**	**1:00 – 3:00pm** What is Patient Safety? **(Patient Safety)** **Prof. Hamza Abdulgahni**
2:00 – 3:00pm **Self- Directed Learning**	**2:00-3:00 pm** **Self- Directed Learning**		**2:00 – 3:00pm** **Self- Directed Learning**	

WEEK 2 – BLOCK ENDOCRINE (ENDO 225) (Male Group-A)

Week (2) Starting: 28/01/2018 to 01/02/2018

THYROID GLAND

CHAIR PERSON : Dr.Riad Al-Sulimani

CO-CHAIR: Dr.Usman Ghani

Sunday 28 January 2018	Monday 29 January 2018	Tuesday 30 January 2018	Wednesday 31 January 2018	Thursday 01 February 2018
8:00 - 9:00 am Histology of the parathyroid glands **(Anatomy)** **Prof. Aly Mohamed**	**8:00 - 10:00 am** **Problem-Based Learning(PBL)** **Case 1 Part 1**	**8:00 - 9:00am** **To take a history related to thyroid signs and symptoms** **A1** **(Clinical Skills)**	**8:00 - 10:00am** **(Practical)** **Anatomy and histology of thyroid & parathyroid Glands** **(Anatomy)** **Dr. Essam Salama** **All Staff**	**8:00 - 10:00 am** **Problem-Based Learning(PBL)** **Case 1 Part 2**
9:00 - 10:00 am Physiology of the thyroid gland **(Physiology)** **Prof. Abdulmajeed Aldrees**		**9:00 – 10:00am** **To take a history related to thyroid signs and symptoms** **A2** **(Clinical Skills)**		
10:00 - 11:00am Histology of the thyroid gland **(Histology)** **Prof. Aly Mohamed**	**10:00 - 11:00am** Biochemistry of thyroid hormones & thermogenesis **(Biochemistry)** **Dr. Usman Ghani**	**10:00 - 11:00am** Hypo and hyperthyroidism and hashimotos thyroiditis **(Pathology)** **Dr. Mohammed Alswayyed**	**10:00 - 11:00pm** Pharmacology of drugs used in hyperthyroidism **(Pharmacology)** **Prof. Abdulrahman Al Motrefi**	**10:00 - 11:00am** Pharmacology of drugs used in hypothyroidism **(Pharmacology)** **Prof. Abdulrahman Al Motrefi**
11:00- 12:00 pm Anatomy & embryology of the thyroid and para-thyroid glands **(Anatomy)** **Prof. Saeed Abuel Makarem**	**11:00 - 12:00 pm** Hyper and hypo thyroidism **(Physiology)** **Prof. Abdulmajeed Aldrees**	**11:00-12:00pm** **Self- Directed Learning**	**11:00- 12:00pm** The immune system and endocrine disorders **(Immunology)** **Prof. Adel Almogren**	**11:00 – 12:00pm** Thyroid nodules and thyroid neoplasm **(Pathology)** **Dr. Mohammed Alswayyed**
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 – 2:00pm** **Self- Directed Learning**	**1:00 - 3:00 pm** **Salam**	**1:00 – 2:00pm** **Self- Directed Learning**	**1:00 – 3:00pm** Why applying human factors is important for Patient Safety? **(Patient safety)** **Dr. Ahmed Aboshaiqah**
2:00 – 3:00pm **Self- Directed Learning**	**2:00 – 3:00pm** **Self- Directed Learning**		**2:00 – 3:00pm** **Self-Directed Learning**	

WEEK 3 – BLOCK ENDOCRINE (ENDO 225) (Male Group-A)

Week (3) Starting: 04/02/2018 to 08/02/2018

PARATHYROID GLAND AND CALCIUM METABOLISM

CHAIR PERSON : Dr.Riad Al-Sulimani

CO-CHAIR: Dr.Usman Ghani

Sunday 04 February 2018	Monday 05 February 2018	Tuesday 06 February 2018	Wednesday 07 February 2018	Thursday 08 February 2018
8:00 - 9:00am **Self- Directed Learning**	**8:00 - 9:00 am** Pharmacology of drugs used in calcium and vitamin D disorders **(Pharmacology)** **Dr. Ishfaq Bukhari**	**8:00 - 9:00am** **To examine the normal thyroid gland** **A1** **(Clinical Skills)**	**8:00 - 9:00 am** **Self- Directed Learning**	**8:00 - 9:00 am** **Self- Directed Learning**
9:00 – 10:00 am Calcium homeostasis **(Physiology)** **Dr. Khalid Akregaiey**	**9:00 – 10:00 am** **Self- Directed Learning**	**9:00 - 10:00am** **To examine the normal thyroid gland** **A2** **(Clinical Skills)**	**9:00 – 10:00 am** **Self- Directed Learning**	**9:00 - 10:00 am** **Self- Directed Learning**
10:00 - 11:00am Hypo- and hyper-parathyroidism **(Physiology)** **Dr. Khalid Alregaiey**	**10:00 – 11:00am** **Self- Directed Learning**	**10:00 - 11:00am** Vitamin D,and Rickets **(Biochemistry)** **Dr. Zeyad Kurdee**	**10:00 – 12:00am** **Practical** **Pathology of thyroid and parathyroid gland** **(Pathology)** **Dr. Abdullah Basabien** **Dr. Mohammed Al Swayyed**	**10:00-11:00 am** **Introduction to Osteoporosis** **(Medicine)** **Prof. Riad Sulimani**
11:00 – 12:00pm **Self- Directed Learning**	**11:00 – 12:00pm** **Self- Directed Learning**	**11:00- 12:00pm** **Self- Directed Learning**		**11:00- 12:00pm** Treatment of osteoporosis **(Pharmacology)** **Dr. Ishfaq Bukhari**
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 – 2:00pm **Self- Directed Learning**	**1:00 - 2:00pm** **Self- Directed Learning**	**1:00 – 3:00 pm** **Salam**	**1:00 – 2:00pm** **Self- Directed** **Learning**	**1:00 – 3:00pm** Understanding systems and effect of complexity of patient care **(Patient safety)** **Dr. Hussein Saad**
2:00 – 3:00pm **Self- Directed Learning**	**2:00 – 3:00pm** **Self- Directed Learning**		**2:00 – 3:00pm** **Self- Directed** **Learning**	

WEEK 4 – BLOCK ENDOCRINE (ENDO 225) (Male Group-A)

Week (4) Starting: 11/02/2018 to 15/02/2018

ADRENAL GLANDS

CHAIR PERSON : Dr.Riad Al-Sulimani

CO-CHAIR: Dr.Usman Ghani

Sunday 11 February 2018	Monday 12 February 2018	Tuesday 13 February 2018	Wednesday 14 February 2018	Thursday 15 February 2018
8:00 –10:00 am **MIDBLOCK Examination**	**8:00 -10:00 am** **Problem-Based Learning(PBL)** **Case 2 Part 1**	**8:00 – 9:00am** **Self- Directed Learning**	**8:00 - 9:00am** Adrenal hormones (part 2) **(Physiology)** **Dr. Khalid Alregaiey**	**8:00 -10:00 am** **Problem-Based Learning(PBL)** **Case 2 Part 2**
		9:00 – 10:00am **Self- Directed Learning**	**9:00 – 10:00am** **Histology of adrenal Gland** **(Practical)** **(Anatomy/ Prof. Aly Mohamed)**	
10:00 - 11:00am **Feedback on Midterm Exam Male Group A & B** **Prof. Riad Sulimani** **Lecture Theater C**	**10:00- 11:00 am** Adrenal gland hormones (mineralocorticoids) **(Physiology)** **Dr. Khalid Alregaiey**	**10:00 - 11:00am** Biochemistry of cushing syndrome **(Biochemisty)** **Ahmed Mujammami**	**10:00 – 11:00am** Candidiasis **(Microbiology)** **Dr. Ahmed Al Barrag**	**10:00- 11:00 am** Pathology of the adrenal gland **(Pathology)** **Dr. Mohammed Alswayyed**
11:00- 12:00pm Embryology & anatomy of adrenal glands **(Anatomy)** **Dr. Essam Salama**	**11:00- 12:00pm** Adrenal hormones (glucocorticoids and androgens) (part 1) **(Physiology)** **Dr. Khalid Alregaiey**	**11:00- 12:00pm** Epidemiology of obesity **(Family & Community Medicine)** **Dr. Ebraheem Qusadi**	**11:00- 12:00pm** Physiology of adrenal medulla and pheochromocytoma **(Physiology)** **Dr. Khalid Alregaiey**	**11:00- 12:00pm** Pharmacology of corticosteroids **(Pharmacology)** **Dr. Saeed Ahmed**
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 – 2:00pm Histology of adrenal gland **(Anatomy)** **Prof. Aly Mohamed**	**1:00 – 3:00pm** **(Practical)** **Anatomy and radiology of the adrenal glands** **(Anatomy, Dr Essam Salama and Radiology)** **All Staff**	**1:00 - 3:00 pm** **Salam**	**1:00 – 2:00pm** Biochemistry of Addison's disease **(Biochemisty)** **Ahmed Mujammami**	**1:00 – 3:00pm** Understanding and managing clinical risk **(Patient safety)** **Prof. Hamza Abdulghani**
2:00 – 3:00pm **Self- Directed Learning**			**2:00 – 3:00pm** Biochemistry of obesity: role of hormones **(Biochemistry)** **Dr. Usman Ghani**	

WEEK 5 – BLOCK ENDOCRINE (ENDO 225) (Male Group-A)

Week (5) Starting: 18/02/2018 to 22/02/2018

DIABETES MELLITUS

CHAIR PERSON : Dr.Riad Al-Sulimani

CO-CHAIR: Dr.Usman Ghani

Sunday 18 February 2018	Monday 19 February 2018	Tuesday 20 February 2018	Wednesday 21 February 2018	Thursday 22 February 2018
8:00 - 9:00am Histology of pancreas (exocrine and endocrine) **(Anatomy)** **Dr. Mohammed Atteya**	**8:00 - 10:00 am** **Problem-Based Learning(PBL)** **Case 3 Part 1**	**8:00 - 9:00am** **To take a history related to diabetes signs and symptoms** **A1** **(Clinical Skills)**	**8:00 - 9:00 am** Epidemiology of diabetes mellitus **(Family and Community Medicine)** **Dr. Ebraheem Qusadi**	**8:00 – 10:00 am** **Problem-Based Learning(PBL)** **Case 3 Part 2**
9:00 – 10:00 am Coping with diabetes mellitus in adolescence **(Psychiatry)** **Dr. Mohammad Al Jaffer**		**9:00 –10:00am** **To take a history related to diabetes signs and symptoms** **A2** **(Clinical Skills)**	**9:00 – 10:00 am** Oral hypoglycaemic drugs Part 1 **(Pharmacology)** **Prof. Al Humayyd**	
10:00 - 11:00 am Anatomy of the pancreas **(Anatomy)** **Dr. Mohammed Vohra**	**10:00- 11:00 am** Pathology and pathogenesis of type 1 diabetes mellitus **(Pathology)** **Dr. Mohammed Alswayyed**	**10:00- 11:00 am** Metabolic changes in diabetes mellitus **(Biochemisty)** **Dr. Zeyad Kurdee**	**10:00 - 12:00pm** **Practical** **Measurement of glucose in blood and urine** **(Biochemistry)** **Dr. Usman & Dr Ahmed** **All Staff**	**10:00 - 11:00am** Oral hypoglycaemic drugs Part 2 **(Pharmacology)** **Prof. Al Humayyd**
11:00- 12:00pm Physiology of the pancreas **(Physiology)** **Dr. Ahmad Alsabeeh**	**11:00- 12:00pm** Use of insulin in treatment of diabetes **(Pharmacology)** **Prof. Al Humayyd**	**11:00- 12:00pm** Biochemistry of diabetic ketoacidosis **(Biochemisty)** **Dr. Zeyad Kurdee**		**11:00- 12:00pm** **Self- Directed Learning**
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 Physiology of insulin **(Physiology)** **Dr. Ahmad Alsabeeh**	**1:00 – 2:00pm** Biochemistry of metabolic syndrome **(Biochemistry)** **Dr. Usman Ghani**	**1:00 - 3:00 pm** **Salam**	**1:00 - 2:00pm** Management of diabetic ketoacidosis and hypoglycemia **(Pharmacology)** **Prof. Al Humayyd**	**1:00 - 3:00 pm** Using quality-improvement methods to improve care **(Patient safety)** **Dr. Ahmed Aboshaiqah**
2:00 – 3:00pm Common infections in diabetes mellitus (Diabetic foot) **(Microbiology)** **Prof. Ali Somily**	**2:00 – 3:00pm** Pathology and pathogenesis of type 2 diabetes mellitus **(Pathology)** **Dr. Mohammed Alswayyed**		**2:00 – 3:00pm** Glucose Homeostasis **(Biochemistry)** **Dr. Usman Ghani**	

WEEK 6 – ENDOCRINE BLOCK (Male A)				
Week (6) 25/02/2018 to 01/03/2018				
CONSOLIDATION WEEK				
CHAIR PERSON : Dr.Riad Al-Sulimani				
CO-CHAIR: Dr.Usman Ghani				
Sunday **25 February 2018**	**Monday** **26 February 2018**	**Tuesday** **27 February 2018**	**Wednesday** **28 February 2018**	**Thursday** **01 March 2018**
Consolidation	**Consolidation**	**Consolidation** **1:00 - 4:00 pm** **OSCE (GNT& ENDO)**	**Consolidation**	**Consolidation**

***Week 6: Consolidation week; 25 February 2018 to 01 March 2018**
***Week 7; Final MCQ; 04 March 2018**
***Week 7: OSPE and SAQ; 08 March 2018**
*** PROGRESS TEST; 07 March 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 plagiarism wrong?

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 action including the failure of the subject concerned provided that such act has been confirmed with evidence.

Assessment of Students in the Block (year 2)

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
4- OSPE (Objective Structured Practical Examination)

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

- Continuous Assessment (Tutor Assessment and Attendance) : 15%
- Written Examinations (MCQ) : 55%
    - Mid-Block Exam 25%
    - Final Block Exam 30%
- OSPE : 30 %

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 25% : In the form of MCQs, these are prepared mainly from sessions presented to the students in large group. This exam will consist of 50 MCQs that will assess factual knowledge.

   b. Final written exam 30%: 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 MCQs that will assess factual knowledge too.

4. Objective Structured Practical Examination (OSPE):

This contains 30% 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.

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

The list below comprises the key textbooks and learning resources which have been prescribed and recommended for use in the undergraduate medical course at King Saud University. It is expected that you have your own copy of prescribed textbooks and use them as one of your main resources in learning. Before making any purchases, you might carefully examine all other recommended textbooks in an area and chose the text that matches with your needs and your learning style. Although all these texts are available in the Medical Library, you might need to purchase texts that you use frequently in these years as the demand upon library texts is usually high.

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.

KING SAUD UNIVERSITY
College of Medicine
Department of Medical Education

Feedback to Students on PBL Performance

Year 2 (Academic Year 2017-2018)

Student's name:……Group number…….
Tutor's name…… Block: ENDOCRINE 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 2017-2018)

Student's name: ……..…Group number:……..
Tutor's name: ……..Block: ENDOCRINE 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

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)_____