

Course Description Template

University Name: *university of warith AL-Anbiyaa*

Faculty/Institute: *College of medicine*

Scientific Department: *Biochemistry*

Academic or Professional Program Name: *Biochemistry*

Final Certificate Name: *M.B.C.h.B.*

Academic System: *Courses*

Description Preparation Date: *20/7/2025*

File Completion Date: *11/9/2025*

Signature:

Head of Branch:

Dr. Riadh Hnewa

Date:

C-00/9/11

Signature:

Vice Dean for Scientific

Affairs: *Dr. Laith M. Al-Kun*

Date: *11/9/2024*

The file is checked by:

Department of Quality Assurance and University Performance

Director of the Quality Assurance and University Performance

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Date: *31.8.2025*

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علي مكي الموسوي
Dean's approval
عميد كلية الطب

Curriculum Skills Map

Please tick the boxes corresponding to the individual learning outcomes of the programme being assessed.

Required learning outcomes of the program

| General and transferable skills (other skills related to employability and personal development) | Emotional and value-based goals | | | | Program Skill Objectives | | | | Cognitive objectives | | | | Essential or Optional | Course name | Course code | Year/Level | | | | |
|--|---------------------------------|---|---|---|--------------------------|---|---|---|----------------------|---|---|---|-----------------------|-------------|-------------|------------|-----------|---------------------|---|---|
| | ١ | ٢ | ٣ | ٤ | ٤ | ٣ | ٢ | ١ | ٤ | ٣ | ٢ | ١ | | | | | ٤ | ٣ | ٢ | ١ |
| ٣ | ٢ | ١ | ٤ | ٤ | ٤ | ٢ | ١ | ٤ | ٤ | ٣ | ٢ | ١ | ٤ | ٣ | ٢ | ١ | essential | Medicinal Chemistry | | |
| ٤ | ٢ | ١ | ٤ | ٤ | ٤ | ٣ | ٢ | ١ | ٤ | ٣ | ٢ | ١ | ٤ | ٣ | ٢ | ١ | essential | Biochemistry | | |
| ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | | | | |
| ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | | | | |
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| | | <ul style="list-style-type: none"> ○ Glycosidic bond formation ○ Benedict's and Barfoed's tests | |
| <p>4W</p> | <p>Chemistry & metabolism of CHO</p> | <p>Carbohydrate Metabolism – Learning Objectives</p> <p>Outline the major pathways of carbohydrate metabolism:</p> <ul style="list-style-type: none"> ○ Glycolysis ○ Gluconeogenesis ○ Glycogenolysis and Glycogenesis ○ Pentose Phosphate Pathway (PPP) <p>Describe the steps of glycolysis, including:</p> <ul style="list-style-type: none"> ○ Key enzymes ○ Energy yield (ATP and NADH) ○ Regulatory points <p>Explain the role of the TCA cycle and its integration with glycolysis.</p> <p>Discuss the regulation of blood glucose:</p> <ul style="list-style-type: none"> • Hormonal control (insulin and glucagon) • Fed vs. fasting state <p>Understand the Cori cycle and its significance in anaerobic metabolism.</p> <p>Describe glycogen metabolism, including:</p> <ul style="list-style-type: none"> • Enzymes involved • Regulation by hormones | <p>LEC 7&8</p> |
| <p>5W</p> | <p>Chemistry & metabolism of CHO</p> | <p>Explain the role of the TCA cycle and its integration with glycolysis.</p> <p>Discuss the regulation of blood glucose:</p> <ul style="list-style-type: none"> • Hormonal control (insulin and glucagon) • Fed vs. fasting state <p>Understand the Cori cycle and its significance in anaerobic metabolism.</p> <p>Describe glycogen metabolism, including:</p> <ul style="list-style-type: none"> • Enzymes involved • Regulation by hormones | <p>LEC 9&10</p> |
| <p>6W</p> | <p>Chemistry & metabolism of CHO</p> | | <p>LEC 11&12</p> |

8W

Chemistry & metabolism of Proteins

Amino Acid Metabolism – Learning Objectives

LEC 15&16

Explain the digestion and absorption of dietary proteins:

- Enzymes involved (pepsin, trypsin, etc.)
- Amino acid transport systems

Describe amino acid catabolism, including:

- Transamination
- Deamination
- Ammonia formation and detoxification

Understand the urea cycle:

- Key steps and enzymes
- Role in nitrogen excretion
- Clinical relevance (e.g. hyperammonemia)

Outline the fate of carbon skeletons of amino acids:

- Entry into TCA cycle, gluconeogenesis, or ketogenesis

Describe the biosynthesis of non-essential amino acids


Identify common inborn errors of amino acid metabolism:

- Phenylketonuria (PKU)
- Alkaptonuria
- Maple syrup urine disease
- Homocystinuria

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| 10W | Chemistry of Enzymes (II) | <p>Discuss enzyme inhibition:</p> <ul style="list-style-type: none"> ○ Competitive vs non-competitive inhibition ○ Reversible and irreversible inhibition ○ Clinical relevance (e.g., drug action) <p>Describe enzyme regulation mechanisms:</p> <ul style="list-style-type: none"> ○ Allosteric regulation ○ Feedback inhibition ○ Covalent modification (e.g., phosphorylation) <p>Understand the concept of Isoenzymes (isozymes):</p> <ul style="list-style-type: none"> ○ Clinical significance (e.g., LDH, CK in myocardial infarction) <p>Enzymes in Clinical Diagnosis</p> <p>Describe the diagnostic importance of liver enzymes:</p> <ul style="list-style-type: none"> ○ ALT (SGPT), AST (SGOT): hepatocellular damage ○ ALP, GGT: cholestasis, biliary obstruction <p>Understand the significance of pancreatic enzymes:</p> <ul style="list-style-type: none"> ○ Amylase and Lipase in acute pancreatitis <p>Describe the cardiac enzymes and biomarkers:</p> <ul style="list-style-type: none"> ○ CK-MB, LDH isoenzymes, Troponins (I, T) for myocardial infarction | LEC 19&20 |
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| <p>13W</p> | <p>Chemistry & metabolism of Lipids (I)</p> | <p>Lipid Chemistry – Learning Objectives</p> <p>Define lipids and describe their general characteristics:</p> <ul style="list-style-type: none"> ○ Hydrophobic or amphipathic molecules ○ Solubility in organic solvents <p>Classify lipids into major groups:</p> <ul style="list-style-type: none"> ○ Simple lipids (e.g., triglycerides) ○ Compound lipids (e.g., phospholipids, glycolipids) ○ Derived lipids (e.g., cholesterol, fatty acids) <p>Describe the structure and function of:</p> <ul style="list-style-type: none"> ○ Fatty acids (saturated vs unsaturated) ○ Triglycerides ○ Phospholipids and glycolipids ○ Cholesterol and its derivatives | <p>LEC 25&26</p> |
| <p>14W</p> | <p>Chemistry & metabolism of Lipids (II)</p> | <ul style="list-style-type: none"> ● Understand essential fatty acids: <ul style="list-style-type: none"> ● Linoleic acid, α-linolenic acid ● Their role in membrane structure and eicosanoid synthesis ● Explain the physical and chemical properties of lipids: <ul style="list-style-type: none"> ● Saponification ● Hydrogenation | <p>LEC 27&28</p> |

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| 16W | Chemistry & metabolism of Lipids (II) | <ul style="list-style-type: none"> ○ Hormonal regulation (insulin, glucagon, epinephrine) ● Understand the biosynthesis and function of ketone bodies: <ul style="list-style-type: none"> • Ketogenesis and ketolysis • Conditions where ketones are produced (fasting, diabetes) ● Explain cholesterol metabolism: <ul style="list-style-type: none"> • Biosynthesis pathway (HMG-CoA reductase) • Regulation and feedback inhibition • Role in steroid hormone and bile acid synthesis ● Describe the structure and function of lipoproteins: <ul style="list-style-type: none"> • Chylomicrons, VLDL, LDL, HDL • Role in lipid transport • Clinical importance of LDL and HDL ● Recognize lipid-related disorders: <ul style="list-style-type: none"> • Hyperlipidemia • Atherosclerosis • Fatty liver disease • Inborn errors (e.g., Tay-Sachs, Gaucher's) ● Relate lipid metabolism to clinical conditions: <ul style="list-style-type: none"> • Obesity • Diabetes mellitus • Cardiovascular diseases | LEC 31&32 |
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| <p>18W</p> | <p>Chemistry of Vitamins and Coenzymes trace elements</p> | <p>Water-Soluble Vitamins</p> <p>Describe the chemistry, sources, functions, coenzyme forms, and deficiency states of:</p> <ul style="list-style-type: none"> ○ Vitamin B1 (Thiamine) – carbohydrate metabolism; deficiency: Beriberi, Wernicke's encephalopathy ○ Vitamin B2 (Riboflavin) – FAD/FMN coenzymes ○ Vitamin B3 (Niacin) – NAD/NADP coenzymes; deficiency: Pellagra ○ Vitamin B5 (Pantothenic acid) – CoA formation ○ Vitamin B6 (Pyridoxine) – amino acid metabolism ○ Vitamin B7 (Biotin) – carboxylation reactions ○ Vitamin B9 (Folate) – DNA synthesis; deficiency: megaloblastic anemia ○ Vitamin B12 (Cobalamin) – DNA & nerve function; deficiency: pernicious anemia ○ Vitamin C (Ascorbic acid) – collagen synthesis, antioxidant; deficiency: Scurvy <p>Understand vitamin absorption, transport, and storage (especially B12 requiring intrinsic factor).</p> <p>Relate vitamin deficiencies to clinical conditions and recognize signs for diagnosis.</p> | <p>LEC 35&36</p> |
| <p> Trace Elements (Micronutrients) – Learning Objectives</p> <p>◆ General Concepts</p> <p>Define trace elements and distinguish them from macro minerals.</p> | | | |

19W

LEC 37&38

Chemistry of Nucleotides & metabolism (I)

Nucleotide Chemistry – Learning Objectives

Define nucleotides and nucleosides:

- Structure and difference between them
- Components: nitrogenous base, sugar, phosphate

Classify nitrogenous bases:

- Purines: adenine, guanine
- Pyrimidines: cytosine, thymine, uracil

Describe the structure and functions of nucleotides:

- Role in DNA/RNA synthesis
- Energy carriers (ATP, GTP)
- Second messengers (cAMP, cGMP)
- Coenzymes (NAD⁺, FAD, CoA)

Understand base pairing and tautomerism in purines and pyrimidines.

Explain nucleic acid structure (briefly, as part of context):

- DNA vs RNA
- 5' to 3' orientation

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| | | <p>◆ Pyrimidine Metabolism</p> <p>Describe the de novo synthesis of pyrimidines:</p> <ul style="list-style-type: none"> • Role of carbamoyl phosphate synthetase II (CPS II) • Formation of UMP, UTP, CTP <p>Understand the synthesis of thymidine nucleotides:</p> <ul style="list-style-type: none"> • Role of thymidylate synthase • Folate dependence <p>Describe pyrimidine degradation:</p> <ul style="list-style-type: none"> • Simpler than purine degradation • Produces β-alanine, β-aminoisobutyrate <p>Recognize clinical disorders of pyrimidine metabolism:</p> <ul style="list-style-type: none"> • Orotic aciduria (UMP synthase deficiency) | |
| <p>21W</p> | <p>Molecular biology</p> | <p>Molecular Biology – Learning Objectives</p> <p>DNA Structure and Function</p> <p>Describe the structure of DNA:</p> <ul style="list-style-type: none"> ○ Double helix, base pairing, antiparallel strands | <p>LEC 41&42</p> |



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| 23W | <p>Transcription (DNA → RNA)</p> <p>Describe the process of transcription:</p> <ul style="list-style-type: none"> ○ RNA polymerase, promoter regions, transcription factors <p>Differentiate between prokaryotic and eukaryotic transcription</p> <p>Explain RNA processing in eukaryotes:</p> <ul style="list-style-type: none"> ○ Capping, polyadenylation, splicing (removal of introns) | LEC 45&46 |
| 24W | <p>Translation (RNA → Protein)</p> <p>Explain the genetic code:</p> <ul style="list-style-type: none"> • Codons, start and stop codons, redundancy <p>Describe the process of translation:</p> <ul style="list-style-type: none"> • Initiation, elongation, termination • Role of ribosomes, tRNA, and aminoacyl-tRNA synthetase <p>Understand post-translational modifications:</p> <ul style="list-style-type: none"> • Phosphorylation, glycosylation, cleavage, etc. <p>Molecular biology</p> | LEC 47&48 |

Second Stage

Unit 7 Musculoskeletal unit (7 hours)

1- Calcium homeostasis 2h

OUTCOME:

Knowing how to investigate abnormal serum calcium homeostasis: what are the actions of calcium and which factors that regulate serum calcium

- Calcium balance: explain calcium balance
- Biological function of calcium: actions of calcium in different tissues
- Control of calcium metabolism: explain calcium levels regulators
- The components of calcium in plasma: explain the free and bound calcium
- Calcium homeostasis: the main hormonal responses to a fall in plasma Ca^{Y+} , and the places where the negative feedback mechanism operates if plasma Ca^{Y+} becomes high. The effect of PTH on the renal tubules
- Parathyroid hormone (PTH) (action and diagnostic importance)
- 1,25-Dihydroxycholecalciferol (action and diagnostic importance)
- Calcitonin (action and diagnostic importance) Investigation of abnormal calcium metabolism: how to use the following tests to assess the calcium status
- Serum calcium
- Effects of serum albumin
- Effects of plasma H^+
- Serum phosphate
- Alkaline phosphatase (ALP)

2- Hypercalcemia 1h

- Hyperuricaemia: explain the effects of the following factors on urate levels
 - Dietary factors
 - Endogenous overproduction of urate
 - Defective elimination of urate
- Gout: • Characteristics of gout attacks
 - Features of primary gout
 - Primary gout (explain how to Diagnose and its pathogenesis)
 - Secondary of hyperuricaemia and gout (explain the causes and their mechanisms to cause hyperuricaemia)

Unit 4 Hematology Unit (9 hours)

1- Metabolic pathways of RBC 1h

- Metabolism: glycolytic pathway, Hexose monophosphate (pentose phosphate) pathway,
- The impact of G¹PD enzyme in protecting the RBC from oxidative damage.

γ- Inflammation 7h

OUTCOME: Knowing the changes in serum proteins in response to diseases

- Explain the plasma proteins commonly measured for the diagnosis and monitoring of specific diseases

- The acute-phase response: what are changes that characterize the body response to infection, inflammation, or trauma - Plasma proteins that change during the acute-phase response: explain their importance and changes that are occurring in following proteins during diseases

- C-reactive protein + ESR
- α¹-Antitrypsin + Clinical consequences of the genetic polymorphism of AAT
- Caeruloplasmin
- α¹-Acid glycoprotein
- Fibrinogen
- Ferritin

- Biochemical tests in myocardial infarction and ischemia: explain which tests could be measured
- Time-course of changes: explain the changes in the levels of cardiac markers according to the duration of MI
- Optimal times for blood sampling: explain the appropriate times to take blood samples for cardiac markers measurement
- Troponin I and troponin T role in diagnosis of MI
- enzymes, such as creatine kinase (CK), CK-MB, aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) role in diagnosis of MI
- myoglobin role in diagnosis of MI
- Elevations of cardiac troponin due to myocardial injury but not due to MI

2- The diagnosis of heart failure and thromboembolic disease 1h

- OUTCOME: knowing which test could be useful in diagnosis of HF and TED
- B-type natriuretic peptide (BNP) (importance and clinical use)
 - D-dimers (importance and clinical use)

3- Investigation of plasma lipid abnormalities 3h

- Plasma total cholesterol: explain the effecting factors and its importance
- Plasma triglycerides: explain the causes of variation
- Plasma LDL: explain its clinical importance
- Plasma non-HDL cholesterol meaning and importance
- Specimen collection: what are the requirements for good sampling for lipid profile?
- Routine investigations: what are the tests that should be requested in patients suspected to be at increased risk of ischaemic heart disease or of a lipid disorder?
- The primary hyperlipoproteinaemias: explain the causes of this disorder, the changes in lipids concentrations in plasma in each one, and Lipoproteins mainly affected:
 - Familial hypercholesterolaemia
 - Familial hypertriglyceridaemia
 - Familial combined hyperlipidaemia
 - Remnant hyperlipoproteinaemia
 - Lipoprotein lipase deficiency (or apoC-II deficiency)

Third Stage

Unit V Gastrointestinal tract disease (4 hours)

1-Stomach 1h

OUTCOME:

Knowing which laboratory tests are useful to diagnose and monitor PU and ZE syndrome Peptic ulcer

- Causes of Peptic ulcer
- Tests for H. pylori infection:
- Urea breath test (method and interpretation)
- Serological tests (uses and indications)
- Faecal antigen testing (importance)
- Gastrin (causes of abnormal results and indication of use) Zollinger–Ellison syndrome
- Definition
- Laboratory diagnosis (use of serum gastrin and provocative test)

2-Acute pancreatitis 1h

OUTCOME:

Knowing which laboratory tests are useful to diagnose acute pancreatitis and pancreatic insufficiency - Serum amylase activities (variation in different clinical conditions)

- Macro-amylasaemia
- Definition and Cause
- Laboratory diagnosis (how can be differentiated from increased serum amylase) Chronic pancreatitis
- Presentation
- Diagnosis of pancreatic insufficiency by Faecal elastase

3-Small intestine and colon (Tests of absorptive function) 1h

OUTCOME:

- Knowing which laboratory tests are useful to diagnose coeliac disease and IBD Coeliac disease
- Serological tests for coeliac disease (the use of Anti-tTG and anti-endomysium IgA)

- γ -glutamyltransferase role to detect hepatobiliary damage
- Hepatic protein synthesis: The measurement of plasma proteins as an index of the liver's ability to synthesize protein
 - Albumin
 - Coagulation factors
 - Immunoglobulins
- Overview of the Serological tests (general indications)
- Overview of the Markers of fibrosis
- Disordered metabolism in the liver diseases (changes in RFTs, glucose and lipids)

6- Jaundiced 1h

OUTCOME:

Knowing how to differentiate between different types of jaundice

- Definition of jaundice
- Types and causes of hyperbilirubinaemia
- Bilirubin and urobilinogen measurements (examples of results in various conditions)
- The investigation of jaundice (to differentiate between different causes of increase serum bilirubin)

Causes of Pre-hepatic hyperbilirubinaemia

Causes of Hepatocellular hyperbilirubinaemia

Causes of Cholestatic hyperbilirubinaemia

The congenital hyperbilirubinaemias

- Biochemical base of Gilbert's syndrome
- Biochemical base of Crigler-Najjar syndrome
- Biochemical base of Dubin-Johnson syndrome and Rotor syndrome

7- Laboratory investigations of abdominal pain 1h

OUTCOME:

knowing which laboratory tests are needed in patients presented with acute abdomen

- What biochemical disorders can be presented as acute abdomen and which tests should be ordered?

- Types and causes of renal stones:
 - Hypercalciuria
 - Oxalate, cystine and xanthine
- Chemical investigations on patients with renal stones to detect the cause and type of renal stone Prostatic specific antigen
- Uses of PSA - Types of PSA tests (ordinary vs high sensitivity test)
- Interpretation of PSA levels to detect the type of prostatic disorders

7- Acid-base balance 3h

OUTCOME:

- 1- knowing how to diagnose and classify acid base disorders
 - 2- Which tests are required to accurately estimate arterial O₂ content
 - Maintenance of hydrogen ion concentration of ECF
 - Transport of carbon dioxide
 - Renal mechanisms for HCO₃⁻ reabsorption and H⁺ excretion
 - Buffering of hydrogen ions - Investigating acid-base balance:
 - Collection and transport of specimens
- Disturbances of acid-base status: causes and ABG analysis findings of:
- Respiratory acidosis
 - Respiratory alkalosis
 - Metabolic acidosis
 - Metabolic alkalosis
- Interpretation of results of acid-base assessment:
- Plasma H⁺ is increased
 - Plasma H⁺ is decreased
 - Plasma H⁺ is normal
 - Mixed acid-base disturbances
- Other investigations in acid-base assessment:
- Total CO₂
 - Anion gap

- Diagnostic criteria
- Types and causes
- Investigations

2-Disorders of the hypothalamus and pituitary gland 1h

OUTCOME:

Knowing how to detect hyperprolactinemia

- Factors that regulate the release of anterior pituitary hormones
 - Prolactin:
 - Secretion and function
 - Macroprolactin concept
 - Hyperprolactinemia
 - Causes of hyperprolactinemia
 - Diagnosis (which causes should be excluded first and how detect persistent elevation of prolactin)
 - Degrees of hyperprolactinemia in various clinical scenarios

3-Thyroid gland 3h

OUTCOME:

Knowing how to use TFTs to detect different types of thyroid disorders

- Thyroid hormone
 - Synthesis and metabolism
 - Plasma transport and cellular action
- Regulation of thyroid function - Investigations to determine thyroid status: what are the changes in the following tests in different types of thyroid disorders?
 - Thyrotrophin (causes of high and low)
 - Free T₄ and free T₃
 - Total T₄ and total T₃
- Interpretation of thyroid function tests in patients being investigated for T_4 suspected thyroid disease
- Categories of patients who should have thyroid function tests performed
 - Hyperthyroidism

OUTCOME:

knowing how to use the laboratory tests to diagnose adrenocortical hyperfunction

- Hyperfunction of the adrenal cortex concept
 - Cushing's syndrome definition
 - Causes of glucocorticoid excess (ACTH dependent vs ACTH independent)
 - Tests used to establish if a clinical diagnosis of Cushing's syndrome is likely: how to do and interpret the following laboratory tests a- Initial screening tests:
 - Low-dose dexamethasone suppression test
 - Urinary free cortisol (UFC)
 - Late night salivary cortisol
 - Interpretation of screening tests
 - b- Confirmatory tests:
 - Loss of diurnal rhythm
 - c- Determining the cause of Cushing's syndrome:
 - Plasma ACTH
 - High-dose dexamethasone suppression test
 - CRH stimulation test
 - d- Other biochemical tests (k⁺, GTT): how the adrenocortical hyperfunction could affect the different metabolites
- ↳ **Investigation of suspected adrenocortical insufficiency 1h**
- OUTCOME: knowing how to use the laboratory tests to diagnose adrenocortical insufficiency
- Classification, causes, and presentation patterns (primary vs secondary causes)
 - Diagnosis of primary adrenal insufficiency (Addison's disease): how to use the following tests to diagnose adrenocortical insufficiency:
 - Cortisol and ACTH measurements
 - Short tetracosactrin (Synacthen) test
 - Further testing in confirmed adrenocortical insufficiency

Fourth stage

Unit 1 \ Biochemical changes of the pregnancy \ hour

OUTCOME:

Understand how different biochemical parameters are affected by pregnancy

- Explain the concept of foetoplacental unit
- Review the use of HCG in detecting pregnancy
- Explain the changes in the steroids hormones
- Explain the Effect of pregnancy on following biochemical tests:
 - Reproductive hormones
 - Cortisol
 - Thyroid function tests
 - Plasma volume and renal function
 - Serum lipids and proteins
 - Liver function tests
 - Iron and ferritin

Unit 2 \ Metabolic complications in pregnancy \ hours

OUTCOME:

Knowing how different metabolic complication of the pregnancy can be diagnosed

- How to monitor DM during pregnancy
- How to diagnose, monitor, and follow up GDM
- Thyroid disorders:
 - What are the categories of patient should have TSH and FT₂ measured

