

1. COURSE NAME & CREDIT LOAD

COURSE CODE: NTD 409

COURSE TITLE: Nutritional Biochemistry

NUMBER OF CREDITS: 3 credits/compulsory

COURSE DURATION: 3hrs per week for 15 weeks: 45 hours

Courseware developed by : Clara R. B. OGUNTONA

B. Sc Biochemistry (University of Rosario, Argentina) M. Sc. Human Biology (Loughborough, UK)

Ph D Community Nutrition (Ibadan)

2. LECTURER DETAILS: As above

3. COURSE DETAILS

3.1 Nutritional Biochemistry. The study of biochemistry is important for increasing our understanding of how organisms function. In humans, at the most basic level, properly functioning balances of chemical reactions within the body are responsible for health, while disfunctioning balances of chemical reactions within the body are responsible for diseases. Without functioning biochemical reactions life in any form would not exist.

3.2 Course Content:

Bioenergetics. Metabolism and biochemical inter-relationships of various nutrients in the body. Metabolism of nucleic acids. Metabolism in nutritional diseases. Diabetes, PEM, Gout, Hyperlipidaemias, Inborn errors of Metabolism. Toxins and Detoxification in animal systems.

3.3 Course Justification:

Knowledge generated from nutritional biochemistry research forms the foundation upon which nutrition-based public health interventions are designed and implemented. Many common diseases and disabilities afflicting human populations in both developed and developing countries result from general malnutrition, deficiencies of specific nutrients, or overnutrition. Inadequate diets or poor dietary habits are associated with increased risk for morbidity and mortality, including birth defects, diabetes, cardiovascular disease, obesity and certain cancers. Specific nutrients, food components, or metabolites singularly or in combination, can contribute to risk for disease or, alternatively, can be protective by preventing disease.

3.4 Course objectives:

The general objective of the course is to enable students to acquire the basic knowledge of the biochemical changes that ensure homeostasis and also the underlying causes and manifestations of diseases.

At the end of the course students would be able to understand

- how nutrients are metabolised
- the interrelationships of metabolic pathways
- the genetic basis of inherited metabolic diseases
- the biochemical basis of nutritional diseases such as diabetes, gout, hyperlipidaemias, etc.
- and explain the symptoms of nutritional diseases

3.5 Course Requirements

As stipulated by University regulations, students must attend at least 70 % of all activities stipulated for the course. The continuous assessment will consist of a test carrying 15 marks, an assignment which carries 10 marks. Attendance will carry 5 marks. The final written exam will carry 70 marks

3.6 Methods of Grading

No	ITEM	SCORE
1.	Class Attendance	5
2.	CAT	15
3.	Assignments	10
4.	Exam	70
	Total	100

3.7 Course Delivery Strategies

The course objectives will be achieved by the face to face traditional lecture on the course content. Whenever available IT material will be used to enrich the delivery. Assignment given to the class will cover items from the course content which will be carry out in groups or individually. At the beginning of each lecture 5 – 10 minutes will be devoted to review the previous lecture.

4.0 LECTURE CONTENT

Week 1. The concept of work and energy. Revision of Thermodynamic Laws. Endergonic and Exergonic reactions. The process of production of energy in the cell. Energy carrier molecules in the cell (ATP, UTP, CTP, GTP). Conversion of energy in nutrients to usable energy. Digestible and Metabolizable energy. Basic Metabolic Rate. Concept of free energy change (ΔG). Relationship between ΔG and the constant of equilibrium K. Role of ATP in the cell. Biological oxidation. Redox Potential. Hydrogen carriers.

Study Questions:

What is the role of ATP in the cell?
What is the difference of digestible and metabolizable energy?
How is BMR measured?
Explain the process of biological oxidation

Reading List:

Lehninger, A. L. Biochemistry. The Molecular Basis of Cell Structure and Function. Worth Publishers. Inc. New York. 1977
Murray, R. K., Granner, D. K. Mayes, P. A. and Rodwell, V.W. Harper's Biochemistry. Prentice-Hall International Inc. London, 1990
Van Damme, M. P., Trembath, M.K. and Gould, J. Biochemistry a Metabolic Challenge. University of Monash IT production. 3rd. edition

Week 2.

Revision of energy concepts. The cytochromes. Oxidative phosphorylation. Components of the respiratory chain. Regulation. Mechanism of oxidative phosphorylation. Intermediary metabolism. Anabolic, catabolic and amphibolic pathways. Glycolysis, Krebs's cycle. Overview of fatty acid metabolism. Overview of amino acid metabolism.

Study Questions:

How do the cytochromes function?
How efficient is the oxidative phosphorylation as a producer of energy?
What kind of metabolic pathway is the citric acid cycle?
What are the roles of the different organelles in metabolism?

Assignment: i) Discuss the role of vitamin A in metabolism.
ii) Discuss the role of vitamin B6 in metabolism

Reading List:

The same as above plus

Schneider, E.D. and Sagan, D. (2006) Into the cool: Energy flow, thermodynamics and life (1st ed.) University of Chicago Press.

Week 3

Revision of different metabolic pathways. Metabolism at different levels of organization. Organ, tissue and cells.
Introduction to Nucleic acids. Structure of DNA. Composition of bases. Site of genetic information. Polaridad of the chains. Duplication. Spatial configuration. Semi-conservative character of the duplication.

Study Questions:

Why is DNA so important?

Explain how the genetic information is contained

How does DNA replicate ?

One of the chains is called the coding strand what is the other one called, why?

Reading List:

As above plus

DNA synthesis replication <http://themedicalbiochemistrypage.org/dna.html>

Week 4:

Revision of DNA characteristics. Transcription of the genetic information to RNA. RNA important differences with DNA. Types of RNA. Messenger RNA. Size. Function. Stability. Transfer RNA. Important characteristics. Structure. Ribosomal RNA. Protein synthesis.

Study Questions:

How is the genetic information in the DNA passed to the RNA?

What are the differences between DNA and RNA?

Give an account of the types and characteristics of RNA

Reading List:

As above plus:

RNA structure <http://themedicalbiochemistrypage.org/rna.html>

Week 5:

Revision of RNAs. Diabetes Mellitus. Types. Diabetes type I, type II ,Gestational, other types. Metabolism in uncontrolled diabetes. Dehydration: causes and consequences. Ketonaemia. Ketonuria. Haemoconcentration. Anuria. Explanation of all symptoms. Increased gluconeogenesis. Aminoacidaemia.

Study Questions:

What are the symptoms of diabetes mellitus?

Explain the metabolic mechanisms that lead to dehydration in uncontrolled diabetes

Why is there aminoacidaemia in uncontrolled diabetes?

Reading List:

Diabetes. <http://themedicalbiochemistrypage.org/diabetes.html>

Wyllie-Rosett J. and F. Vinicor 2001. Diabetes Mellitus in Present Knowledge in Nutrition. 8th ed. Bowman and Rusell editors. ILSI Press. Washington, DC.

Week 6:

Revision of metabolism in uncontrolled diabetes. Factors that can increase the risk of developing diabetes. Causes of deficiency of insulin. Gout. Metabolic disorder of purine catabolism. Formation of "tophi". Inflammation reaction. Hyperuricaemia. Gout diagnosis.

Study Questions:

When does Gout occur?
Which are the enzymes involved?
Are there other diseases causing hyperuricaemia?

Reading List:

Rodwell, V. W. Nucleotides in Harper's Biochemistry. Murray et al. edits. Prentice Hall International Inc. New York
<http://themedicalbiochemistrpage.ogr/gout.html>

Week 7:

Revision of gout and its causes. Hyperlipidaemias. Classification. Types of lipids and lipoproteins increased in plasma in each type. Chylomicrons, Very low density lipoproteins (VLDL), Low density lipoproteins (LDL), High density lipoproteins (HDL). Composition. Hypercholesterolemia.

Study Questions :

What are the causes of Hyperlipidaemias?
Dietary implications of hyperlipidaemias
What are the major differences between the different lipoproteins?

Reading List:

Havel, R. J. 1977. Classification of hyperlipidemias. Ann Rev Med 28: 195-209
Thompson, G.R. 2004. Management of dislipidaemias. Heart 90(8) 949-55

Assignment

- 1) Role of ω -3 fatty acids in cardiovascular disease
- 2) Pre and pro-biotics. Importance

Week 8

CAT for 1 hour. Revision of hyperlipidaemias and gout. Introduction to the biochemistry of PEM

Study Questions:

What is PEM?

Describe the symptoms

Explain most important causes of PEM

Reading List:

PEM, [http://whqlibdoc.who.int./monograph_62\(chp2\).pdf](http://whqlibdoc.who.int./monograph_62(chp2).pdf)

Week 9:

Biochemical and metabolic disorders in PEM. Changes in the Blood. Transferrin and prealbumin. Plasma/amino acid ratio. Albumin level. Globulin levels. Branched amino acid concentration in plasma. Electrolyte and Water metabolism. Changes in the urine. Carbohydrate metabolism and endocrine function.

Study Questions:

What are the immediate consequences of a lack of protein in the diet?

How does it affect the process of digestion and absorption?

What concentration of plasma albumin suggests deficiency?

Reading List:

As above plus

Bender A. E. and Bender D. A. Nutrition for Medical Students. John Wiley & Sons. Chichester. 1982. Pp248.

Week 10

Revision of metabolic changes in PEM. Inborn Errors of Metabolism. Definition. Characteristics. Phenylketonuria (PKU). Conversion of Phenylalanine to Acetyl Co A. Phenylalanine 4-monooxygenase. Transmission of the defective gene. Alternative pathway for conversion of Phenylalanine. Consequences of the defect. Concentrations of phenylalanine and tyrosine indicative of PKU. Treatment.

Study Questions

How common is PKU?

How can be prevented?

What are the consequences of PKU if treatment is not forthcoming?

Reading List:

Bender ...as above

Phenylketonuria Genes and Disease. <http://www.ncbi.nlm.nih.gov/books/NBK22253/>

Week 11

Revision of Inborn Errors of Metabolism. Alkaptonuria. Homogentistic 1,2 dioxygenase. Incidence of alkaptonuria. Abnormal pigmentation of connective tissue. Characteristic of the urine of patients deficient in homogentistic 1,2 dioxygenase. Maple Syrup Urine Disease. α ketoisovaleric dehydrogenase deficiency. Consequences of deficiency.

Study Questions:

Why is the disease called Maple Syrup urine disease?

What are the consequences of the disease?

Can it be prevented?

Reading List:

Maple Syrup Urine disease <http://www.nlm.nih.gov/medlineplus/ency/article/000373htm>

Week 12

Revision of alkaptonuria and Maple Syrup urine disease. Galactosemia. Conversion of Galactose to glucose. 1 phosphate uridylyl transferase absent in Galactosemia. Consequences of galactosemia. Lactose intolerance. Introduction to the concept of Xenobiotics.

Study Questions:

What defect causes alkaptonuria?

Lack of what enzyme causes Galactosemia?

What are the health consequences of galactosemia?

Why is lactose intolerance common in Africans?

Reading List

Galactosemia. <http://www.nlm.nih.gov/medlineplus/ency/article/000366.htm>

Lactose Intolerance. <http://www.nlm.nih.gov/medlineplus/ency/article/000276/htm>

Week 13

Xenobiotics of medical importance. Site of metabolism of these compounds. Metabolism of xenobiotics divided in two phases. Phase 1 is Hydroxylation. Enzymes are monooxygenases or Cytochrome P450 species. Characteristics of Cytochrome P450. Reduction. Hydrolysis. Characteristics of reactions of Phase 1. Cytochrome P- 488

Study Questions:

Explain the objective of the phases in the metabolism of xenobiotics

What is cytochromes P450?

What does the name mean?

Reading List

Murray, R. K. Metabolism of Xenobiotics in Harper's Biochemistry. Murray, R. K et al., editors. Prentice-Hall International Inc. New York 22nd edition. Pp 645-649

Week 14

Phase 2: Conjugation of products of phase 1 leading to polar metabolites. Compounds used for conjugation. Phase 2 reactions. Glucuronidation. Sulfation. Conjugation with Glutathione. Acetylation. Methylation. Factors affection xenobiotic enzymes.

Reading List

As above

Week 15

Revision

Students will revise all the concepts learnt during the course.

Study Questions

Why is ATP such an important molecule?

What is the difference of digestible and metabolizable energy?

How does BMR change during the life span?

Explain the process of oxidative phosphorylation

Cytochromes are similar to haemoglobin. Discuss

Discuss the production of ATP through the respiratory chain
How many types of metabolic pathways exist? Give examples
What are the roles of the different organelles in metabolism?
Discuss the structure of DNA
The sequence of bases in the DNA contains the genetic code. Explain
The replication of DNA is called semi conservative. Why?
What are the most important features of mRNA?
What are the differences and similarities between DNA and RNA?
How many tRNAs are there?
How many types of diabetes mellitus exist?
Explain the metabolic mechanisms that lead to dehydration in uncontrolled diabetes
Why do ketoacids increase in uncontrolled diabetes?
What is Gout?
Are there other diseases causing hyperuricaemia?
Are Hyperlipidaemias genetic diseases?
Which is the most common of the inborn errors of amino acid metabolism?
Is PKU common?
What are xenobiotics?
Which are the main phases in the metabolism of xenobiotics?