# RAK Medical and Health Sciences University

Ras Al Khaimah, UAE

RAK COLLEGE OF
NURSING (AY 2018-2019)

## NHB 203 HUMAN BIOCHEMISTRY ASSIGNMENT

**A. Description.** One of the requirements of this course is to complete **two (2)** assignments and this assignment is the second. Please keep the following expectations in mind:

1. Each assignment is worth 100 points.
2. Read instructions carefully and be sure to complete all aspects of the assignments.
3. Be thorough and complete in your work.
4. The assignment was developed to correspond to chapters/topics that we will be studying throughout the term. You will need to turn in your work by the deadlines designated in your syllabus and on the assignment sheet. No late assignments will be accepted.
5. All written assignments must be typed and double-spaced.
6. Please use APA rules of citation.
7. The deadline of submission is on May 1, 2018.
8. Hard/printed copy must be submitted. Please print an extra so that I can sign it as a proof that you submitted your assignment on time.

**B. Purpose*:*** This assignment is related to your class in Biochemistry

**C. Type/Length of activity:** Academic Article Reading and Review

**D. Instruction**.
***Refer to the attached Biochemistry Diseases.*** Prepare a 1500 – 3000 words essay discussing the discussing and if possible make illustration of the pathophysiology and discuss its biochemistry. The assignment must contain the four components identified the assignment format.

**E. ASSIGNMENT FORMAT:**

1. A paper at least 4 pages long, not including any reference section
2. The paper must contain the following sections:
   - Introduction
   - Discussion
   - Conclusion
   - References

3. Typed and double spaced, with Arial (10-point font) or Times New Roman (11 or 12-point font) and 1-inch margins
4. Proofread for spelling and grammatical errors
5. Provide in-text citations and reference any of your sources using APA format

**F. <u>HOW WILL YOUR ASSIGNMENTS BE GRADED</u>.** Please find the marking rubric below:

| PARTICULARS | INSTRUCTION | MAX.MARKS |
|---|---|---|
| **Originality** | *The assignment must be original. It must pass through a plagiarism check. The acceptable standard is 30% similarity index* | 30 |
| **Assignment Title** | *Select a topic for your discussion* | 5 |
| **Introduction** | *It must contain why you selected the topic and its relevance. Word count must be 200-500.* | 10 |
| **Discussion** | *Discuss main points. This part must contain 1500 – 2500 words* | 40 |
| **Conclusion** | *It must contain summary and recommendations. This part must contain 100-200 words.* | 10 |
| **References** | *A minimum of three references is required.* | 5 |
| Total | | 100 |

## Assignment Checklist

Before you turn in any written work, be sure you ask yourself the following:

- ❒ When is it due? _____
- ❒ Is it typed and double-spaced?
- ❒ Did I answer all of the questions?
- ❒ Are my answers complete and thorough?
- ❒ Did I explain answers in my own words and avoid copying text from my textbook or other sources?
- ❒ If I needed to take information directly from another source, did I use quotation marks and cite my source (including the textbook), by indicating the author, publication date, and page number?
- ❒ When I provide my opinion, belief, or idea about something, did I also explain why and support my answer?
- ❒ Is my work proofread and free from grammatical errors?
- ❒ Do I understand what I wrote?
- ❒ If someone who did not know much about the topic I am writing about, understand it better after reading my paper?
- ❒ Have I printed extra copy?
- ❒ Do I have a backup copy saved?

| ID No. | BIOCHEMISTRY DISEASES |
|---|---|
| 16904013 | • Pertussis Toxin – Ribosylation of cys-residue on $\alpha_I$ subunit that prevents the inhibition of adenylyl cyclase via $G_I$ and leads to overproduction of cAMP. *B. pertussis* generates its toxin in the lungs due to elevated temperatures. |
| 17904001 | • Vitamin A Deficiency – causes developmental defects of the CNS, craniofacial and cardiac structures. Night vision is defective, and its loss may be first sign of the diseases. Loss of keratinzation of epithelia and reduction in mucous secretions. Chronic eye dryness and lung and GI diseases more prominent. |
| 17904002 | • Hypervitaminosis A – Excess vitamin A increases unbound circulating retinol, which is toxic. Retinol's elevation increases retinoic acid via mass action that may cause teratogenic effects in pregnancy, dermatitis and organomegaly. |
| 17904004 | • Type I Diabetes Mellitus (Insulin Dependent DM) – autoimmune destruction of pancreatic β-cells leads to a lack of insulin secretion. A viral infection in early childhood may increase the risk of DMI. Patients require insulin replacement therapy to prevent DKA, coma, and death. DM symptoms require 85% of β-cells to be lost. |
| 17904005 | • Type II Diabetes Mellitus (Non-insulin Dependent DM) – peripheral resistance to insulin and suppression of glucose stimulated release occurs as a result. Insulin resistance may cause β-cells to be overtaxed by producing insulin continuously, resulting in sluggish insulin release in response to glucose. Insulin resistance due to: |
| 17904006 | • Nephrogenic Diabetes Insipidus (NI) – Renal system does not respond to ADH/Vasopressin due to $V_2$ receptor defect or post-receptor defects (aquaporin II Channel mutation). Dehydration, increases in plasma osmolarity. |
| 17904007 | • Neurogenic Diabestes Insipidus (NI) – Decreased secretion of ADH/Vasopressin from posterior pituitary due to mutations in Pre-pro-vasopressin or a block in axonal transport. Dehydration, increases in plasma osmolarity. |
| 17904008 | • Panhypopituitarism – complete deficiency of all anterior pituitary hormones. Leads to dwarfism, adrenal insufficiency, hypothyroid, hypoglycemia. Treat with GH, cortisone, synthetic thyroid hormone. |
| 17904010 | • Isolated GH Deficiency – selective lack of GH. Hypoglycemia does not lead to GH secretion, as expected in normal individual. Leads to dwarfism. Treat with human GH. |
| 17904012 | • Laron-Type Dwarfism – Defect in GH receptor or signal factors that results in IGF-1 deficiency. Patients are resistant to hGH therapy. Defective GH receptors leads to hypoglycemia, elevated TAGs in adipose tissue. Treat with synthetic IGF-1. |
| 17904013 | • African Pigmy Dwarfism – Defect in post-receptor components of IGF-receptor, therefore no treatment with IGF-1. |
| 17904014 | • Giantism – Excess GH release prior to epiphyseal closure that leads to tall stature, accelerated long bone growth. |
| 17904015 | • Acromegaly – Excess GH release after epiphyseal closure that leads to overgrowth of the extremities and short bones of the face. May be due to tumor, or inactive GTPase of $\alpha_s$ that leads to GH synthesis and release constantly. |
| 17904017 | • Hyperthyroidism (Thyrotoxicosis) – Overproduction of $T_3/T_4$. Most commonly caused by Graves Disease. |
| 17904019 | • Graves Disease – autoimmune production of thyroid stimulating IgG (TSI). TSI activates the TSH receptor that causes uncontrolled production of $T_3/T_4$. Leads to exophthalmos, optic nerve degradation, and periodic paralysis. Treat by blocking hormone production with anti-thyroid drug or ablate thyroid with radioactive Iodine. |
| 17904020 | • Hypothyroidism – Insufficient amounts of free $T_3/T_4$ due to thyroid failure or disease of the pituitary or hypothalamus. Basal metabolic rate is decreased and leads to obesity, bradycardia, myxedema. |
| 17904022 | • Cretinism – intrauterine or neonatal hypothyroidism that leads to congential defects and mental retardation. Treat with synthetic thyroid hormone. |
| 17904023 | • Goiter – enlargement of the thyroid gland. Causes include: |

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|---|---|
| 17904024 | • Humoral Hypercalcemia of Malignancy (HHM) – non-bone metastasis of cancer causes release of PTH related protein that is embryologically active, but in CA, it binds PTH receptor and elicits PTH effect without feedback control of calcium. Leads to hypercalcemia and hypophosphatemia. |
| 17904025 | • Pseudohypoparathyroidism (PsHP) – defective $G_s$ protein causes target cell resistenc to PTH and other $G_s$ coupled protein responses (TSH, $T_4$, GH). Leads to hypocalcemia and hyperphosphatemia. PTH infusion does not elicit a response of elevated phosphate in urine and no cAMP in urine, no elevation of serum calcium. |
| 17904027 | • Nutritional Rickets / Osteomalacia – Dietary deficiency of vitamin D along with insufficient exposure to sunlight, thus reduced endogenous vit. D synthesis. Reduced calcium phosphate levels lead to weak, rubbery bones. Rickets in children, osteomalacia. |
| 17904028 | • Renal Rickets / Osteodystrophy – rickets due to destruction of renal tissue that leads to insufficient $1,25(OH)_2D_3$. Due to loss of renal 1-Hydroxylase reaction. Elevated 25-OH $D_3$ serum levels. |
| 17904029 | • Hereditary Hypocalcemic Vitamin D resistant Rickets (HVDRR) |
| 17904031 | • Hypocalcemic Vitamin D resistant Rickets (HVDRR) |