

# Advanced clinical nutrition

Family



Running Head: ADVANCED CLINICAL NUTRITION FINAL EXAMINATION:

ADVANCED CLINICAL NUTRITION Why are T2D and CVD co-morbidities of obesity? (30 points total). To answer this question, be sure to discuss the parts below in detail (including tissues involved, key factors that play a role (where they come from, mechanisms by which they contribute, etc). Please do NOT provide information directly from any one source. You may use sources (and reference them appropriately- relevant literature, review slides, etc.) but then SUMMARIZE the information in your own words. a) Discuss the pathophysiology of obesity related to T2D (i. e. how does obesity increase risk for T2D? Please be specific) (10 points). Obesity occurs when there is an abnormality in the energy balance equation. However, it is said that the pathogenesis of obesity is far more complex than the simple model of an imbalance between energy intake and energy output. It is far more than the mere result of eating too much and/or moving less. Two major factors that affect the development of obesity are genetics and environmental factors. (Uwaifo, 2011) According to the National Center for Chronic Diseases (2011) “ the epidemics of obesity and the low level of physical activity in young people, as well as exposure to diabetes in utero, may be major contributors to the increase in type 2 diabetes during childhood and adolescence.” Low levels of physical activity and improper diet (particular increased caloric intakes) encourages weight gain. Weight gain is correlated with an increase in insulin concentration, requiring more insulin secretion from the pancreas. (Bray, 1992) This increased effort on the part of the pancreas may lead to pancreatic failure and diabetes mellitus in people who are prone to pancreatic islet insufficiency. (Bray, 1992) Absence or insufficiency of insulin may be important in the sodium retention and hypertension associated with <https://assignbuster.com/advanced-clinical-nutrition/>

obesity. In simpler terms, when there is too much fat in the body (obesity), insulin resistance may occur because fat interferes with the body's utilization of insulin. (Eckman and Zieve, 2010) b) Discuss the pathophysiology of obesity related to CVD (i. e. how does obesity increase risk for CVD? Be specific) (10 points). CVDs, which are the number one cause of death globally, are a group of disorders of the heart and blood vessels and include: coronary heart disease, cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease, and deep vein thrombosis and pulmonary embolism. (WHO, 2011) Obesity is directly related to these disorders, as well as, increased mortality, increased risk of diabetes mellitus, and increased risk of gall bladder disease.(Bray, 1992) The consequences of excess weight can be presented in two ways: series of consequences related with an increase in caloric intake and/or decreased physical activity and the second is the consequences of excess weight gain on a variety of organ systems. The first consequence explains that when there is an increase in body weight, the size of fat cells also increases. And when the maximum size is reached, additional fat cells must be recruited to accommodate for triacylglycerol. And when the maximum size is reached, additional fat cells must be recruited to accommodate for triacylglycerol. Excess BMI (35 kg/m<sup>2</sup>) or 75% above the normal weight means an individual have the hypercellular form of obesity; consequently encouraging the increase in lipoprotein lipase. The accumulation of tryacylglycerol in subcutaneous or visceral depots is influenced by a number of factors such as corticosteroids and sympathetic activity. Increased lower body or fat in the butt is promoted by low concentrations of cortisol and high concentrations of estradiol relative to testosterone. The increase of fat stores is correlated with a linear increase in

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the production of cholesterol. Consequently, the increase cholesterol production is associated with increased cholesterol secretion in bile, and an increased risk of gallstone formation and the development of gall bladder disease. (Bray, 1992) Bile is critical in the absorption of fats and fat soluble vitamins in the small intestines. Bile is made by the liver cells and stored by the gall bladder. When food is ingested, especially fats, cholecystokinin is released, signals the bile duct to let bile enter the intestine, and also signals the contraction of the gallbladder to release concentrated bile into the small intestine where fats are broken down or emulsified. (Torsoli, et al, 1990)

Decreased concentrations of high density lipoprotein (HDL) are associated with increased levels of circulating triacylglycerol in obesity. In other words, the decrease of HDL cholesterol and the increased effort of the heart to supply blood to peripheral organs account for the increase risk for cardiovascular disease and heart attack in obese patients. For example, the disorder called atherosclerosis is a condition that occurs when fatty material, which thickens and hardens (due to calcium deposits) overtime, accumulates along the walls of the arteries and if untreated, may eventually block the arteries. (Dugdale, 2010) One of the symptoms is high blood pressure (HBP). HBP does not mean excessive emotional tension (Cunhal, 2010) but chronic HBPs may lead to damage rendering the organs weak and dysfunctional and therefore making atherosclerosis more likely to happen. Atherosclerosis occurs when fat, cholesterol, and other substances build up in the arterial walls and form hard structures called plaques. (Dugdale, 2010) c) Explain the following figure. Be sure to discuss the meaning of the data (what does this relationship show?). Also discuss the relevance of this data in terms of obesity and metabolic consequences (10 points). The scatter plot shows the <https://assignbuster.com/advanced-clinical-nutrition/>

indirect relationship glucose deposit with the amount of intra-abdominal fat/total adipose tissue. This is especially significant in diabetic individuals. In this particular discussion, insulin resistance, hyperglycemia and obesity will be taken into consideration. The plot will be explained by a certain situation. When a person (with diabetes) eats, for example a high calorie meal, the food will be digested and metabolized to be used up by the body for energy. Since the person with diabetes has insulin resistance, glucose (the simplest sugar produced by the catalysis of food) will not be used up efficiently. And since glucose, the source of energy, is not utilized, this leaves the person hungry still (as if he did not consumed any food) inducing the person to eat more. The unused glucose, then, will just stay in the blood or will be stored in small amounts. One of the clinical manifestations of diabetes is increased appetite, and so the process is repeated. Unused glucose is increased in the blood (hyperglycemia), stored fat is also increased.

2. Diabetes (30 points).

a) Compare/contrast Type 1 to Type 2 diabetes (prevalence, population, metabolic characteristics, development). (8 points)

Type 1 DM	Type 2 DM
Prevalence 1. 18 million (13%) (American Diabetes Association, 2010)	22. 42 million (86%) (American Diabetes Association, 2010)
Population 1. 18 million (American Diabetes Association, 2010)	22. 42 million (American Diabetes Association, 2010)
Metabolic Characteristics (Crandall, 2007) Not Associated with obesity	Have propensity to ketoacidosis thus requiring insulin treatment for control
Extremely low to undetectable plasma levels of endogenous insulin	Associated with specific HLA-D antigens
Presence of islet cell antibodies at diagnoses	Pathology of islets is that there is insulinitis and there is also selective loss of $\beta$ cells
This type is prone to develop diabetes complications such as retinopathy, nephropathy, neuropathy, atherosclerotic	

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cardiovascular disease. Hyperglycemia does not respond to oral anti-hyperglycemia drugs Associated with obesity No propensity to ketoacidosis Plasma levels of endogenous insulin may be variable or may be low, normal or elevated depending on degree of insulin resistance and insulin secretory defect. Not associated with specific HLA-D antigens Absence of islet cell antibodies at diagnoses Islets here are smaller, normal-appearing and deposition of amyloid is common. This type is also prone to develop diabetes complications such as retinopathy, nephropathy, neuropathy, atherosclerotic cardiovascular disease. Hyperglycemia initially respond to oral anti-hyperglycemia drugs in many patients. Development (Crandall, 2007) 30 years of age DM – diabetes mellitus b) Discuss the role of diet in BOTH prevention and treatment of diabetes. (6 points). As discussed in the early part of this paper about the relationship of obesity with diabetes especially the development of insulin resistance, one of the risk factors of having this condition is when there is too much fat in the body. To decrease the amount of fat, diet must be carefully designed such that the diet is composed mainly of nutrient dense foods or those rich in vitamins, minerals and fibre and not fat. c) How would exercise metabolically affect Type 1 vs. Type 2 diabetic individuals? (4 points). Type 1 Type 2 Without insulin, these individuals cannot utilize glucose absorption in the adipose and muscular tissues, thereby further depleting the available glucose levels, thereby producing no additional benefit. With insulin supplementation, exercise will increase the glucose uptake of the muscles thereby lowering the circulating blood glucose. Unlike type 1 diabetes, they still have insulin (but just desensitized), so exercise will increase the glucose uptake of the muscular tissues thereby will help in lowering the circulating blood glucose. d) Compare/contrast the <https://assignbuster.com/advanced-clinical-nutrition/>

significance of an abnormal (high) HbA1c % vs. a HOMA score. What does each of these represent? Why is each one important? (6 points). The blood stream contains red blood cells, of which are made up of a molecule called hemoglobin. Glucose, the simplest sugar, sticks to the hemoglobin in order to form a 'glycosylated hemoglobin' molecule, called hemoglobin A1C or HbA1C. This will mean then that the more glucose in the blood, the more hemoglobin A1C or HbA1C will be present in the blood. (diabeticretinopathy.org. uk, n. d.) HbA1c is a test done to measure the amount of glycated hemoglobin in the blood. (Zeive and Eltz, 2010) The HOMA or Homeostatic Model Assessment is used to estimate a person's insulin sensitivity or resistance. (Condy, 2010). e) Explain the following figure. Identify which data (dotted vs. straight line) applies to the lean individuals vs. the obese. Explain the patterns in blood glucose and insulin levels (6 points).

## 2. Hypertension (HTN) (30 points)

a) Briefly discuss the normal regulation of blood pressure (5 points). Blood pressure (BP) is regulated by cardiac output (CO) and total peripheral resistance (TPR), and both are directly proportional to it. Cardiac output is regulated by a variety of mechanisms such as the aortic baroreceptors, alpha and beta adrenergic receptors and the renin-angiotensin-aldosterone mechanism of the kidney. The total peripheral resistance is regulated by sympathetic and parasympathetic stimulation, while the microcirculation also contributes to its regulation according to the specific needs (for example, during exercise, the blood supply on the extremities are greater than the blood supply on the digestive system). Blood pressure is maintained by three mechanisms: the pumping action of the heart, the size of the blood vessels and the circulatory volume (Black & Hawks, 2005). b) Discuss the pathophysiology of hypertension (Definition? <https://assignbuster.com/advanced-clinical-nutrition/>

Contributing factors? Physiological issues?) (10 points). Hypertension is defined as the elevation of blood pressure above 140/100 mmHg according to the American Heart Association. The contributing factors are age, occurrence of prehypertension, diabetes overweight or obesity, abnormal serum albumin (Wang et al, 2006). The main physiological issue in hypertension is that obese people are the ones who are immediately suspected of developing hypertension due to their increased risks (Mokdad et al, 2003). c) You are asked to develop a miracle food for hypertensive individuals. Describe the nutritional composition of the food. You must fully justify each component, including the mechanism by which it would be anticipated to have efficacy in this condition and it will be expected that you provide evidence from the literature to support why each component of the food is included. Last, please compare your food to a commonly used pharmaceutical to treat HTN (in terms of mechanism of action and expected efficacy) (15 points). An example of a miracle food for hypertensive individuals is the meal plan presented by Nutribase. com (<http://www.nutribase.com/mp/Hypertension%20Meal%20Plan%20-%201200%20calories.pdf>). The sample meal plan consists of oatmeal, skim milk, apple, carrots, chicken salad, raisins, broccoli, catfish, brown rice and non-fat yogurt. The food has a total of 1207 kilocalories, 79g protein, 184g carbohydrates, and 5g fat and 839mg sodium. The low fat content is compensated by a higher carbohydrate and moderate protein which will sustain the body's energy needs without over-consumption of fat. Lower fat content, but not non-fat, lowers the incidence of hypertension (Alonso et al, 2005) while restriction of carbohydrates are not recommended (Samaha et al, 2003). Since sodium increases blood pressure by increasing water in the

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circulation leading to increase in cardiac output, a lower sodium content in this food will significantly decrease blood pressure (Sacks et al, 2001). Low sodium content is comparable to the effects of furosemide wherein its action is to excrete water from the circulation while low sodium diet prevents additional water in the intravascular space. d) 4. Gastrointestinal Diseases

(30 points) a) Compare the pathophysiology of cholestatic liver disease to IBD. (10 points). In order to answer this question, it would be most helpful to briefly discuss “ normal” GI function and then proceed to discuss the processes that are “ off” in each condition. Are there similarities between them? It appears that aside from both being gastrointestinal conditions and unknown aetiology, there are little or similarities in terms of function, organ/s affected, and manifestations. Fatigue in IBD is not always occurring until severe. Cholestatic liver disease Inflammatory bowel disease (IBD) Cause Unknown Unknown Normal function Bile normally leaves the liver via bile ducts Crohn’s Disease: No lesions, granular spots, enlarged lymph nodes and fissures in the bowel. Ulcerative Colitis: no Inflammation, thickening, edema and lacerations, and no bleeding. “ Off” condition Bile stagnation in liver or bile ducts Crohn’s Disease: Lesions, granular spots, enlarged lymph nodes and fissures in the bowel. Ulcerative Colitis: Inflammation, thickening, edema and lacerations. Manifestations Jaundice Pruritus Fatigue Anorexia related to Abdominal pain Diarrhea Fluid imbalance Weight loss Fever Decreased serum hemoglobin and hematocrit levels Rectal bleeding b) Identify nutritional complications that exist in each of these conditions, and briefly explain (10 points) Cholestatic liver disease: Decreased absorption of fat-soluble vitamins (A, D, E, K). IBD: Malabsorption of fat carbohydrates, protein, vitamins, iron, calcium and folic acid. c) What nutritional approaches

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would be appropriate to implement? Be sure to justify each nutritional recommendation (10 points). Cholestatic liver disease: Increased intake of fat-soluble vitamins (A, D, E, K) compensate for the nutritional deficiencies caused by decreased biliary action in fat utilization. IBD: Diet high in protein, carbohydrates, vitamins and minerals but low in fat will compensate for the nutritional deficiencies caused by increased rate of bowel elimination. Chocolates, juices, carbonated drinks, alcohol and nuts will further irritate the bowel so these must be avoided. References: American Diabetes Association (2004) Nutrition Principles and Recommendations in Diabetes. Diabetes Journals Online. Retrieved 14 March 2011 from [http://care.diabetesjournals.org/content/27/suppl\\_1/s36.full.pdf+html](http://care.diabetesjournals.org/content/27/suppl_1/s36.full.pdf+html) Alonso, A. et al (2003). Low-fat dairy consumption and reduced risk of hypertension: the Seguimiento Universidad de Navarra (SUN) cohort. *New England Journal of Medicine*, 348, 2074-81. American Diabetes Association (2010) Type 1. Diabetes Basics. Retrieved 14 March 2011 from <http://www.diabetes.org/diabetes-basics/type-1/>. Black, J. & Hawks, J. H. (2005). *Medical-surgical nursing: clinical management for positive outcomes*. 7th edition. Singapore: Elsevier. Bowen, R. (2001) Secretion of Bile and the Role of Bile Acids in Digestion. Vivo. colostate. edu. Retrieved 14 March 2011 from <http://www.vivo.colostate.edu/hbooks/pathphys/digestion/liver/bile.html> Bray, George A. (1992) Pathophysiology of obesity. *American Journal of Clinical Nutrition*. 55: 488S-494S. Retrieved 14 March 2011 from <http://www.ajcn.org/content/55/2/488S.full.pdf>. Centers for Disease Control and Prevention. (2011) National Diabetes Fact Sheet: national estimates and general information on diabetes and prediabetes in the United States. U. S. Department of Health and Human Services, Centers for Disease Control and <https://assignbuster.com/advanced-clinical-nutrition/>

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