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OF SKÖVDE

Name:_____

Personal ID number:_____

School of Health sciences

WRITTEN EXAMINATION

Course: Pathophysiology and pharmacology

Examination: Written exam 2

Course code: BM544G

Credits for written examination: 3.5

Date: 2024-11-22

Examination time: 8:15-12:30

Examination responsible: Anna Benrick

Teachers concerned: Cathal O'Hare and Katarina Skogfält

Aid at the exam/appendices: No aids allowed

Other

Instructions

- ☐ Take a new sheet of paper for each teacher.
- ☐ Take a new sheet of paper when starting a new question.
- ☐ Write only on one side of the paper.
- ☒ Write your name and personal ID No. on all pages you hand in.
- ☒ Use page numbering.
- ☒ Don´t use a red pen.
- ☒ Mark answered questions with a cross on the cover sheet.

Grade points E ≥ 60%, D ≥ 68%, C ≥ 76%, B ≥ 84%, A ≥ 92%

Examination results should be made public within 18 working days

Good luck!

Total number of pages

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Pathophysiology

1. Describe the pathophysiology of type 1 diabetes mellitus. The answer should include underlying cause(s), beta cell function, insulin production, insulin receptor and insulin sensitivity. (5p)

2. Name three clinical biomarkers that are useful for diagnosing diabetes (1,5p)

3. Which of the following symptoms are associated with hypoglycemia (low blood sugar)? (0.5)

1. Increased thirst, frequent urination, and blurry vision
2. Dizziness, sweating, confusion, and loss of consciousness
3. Weight gain, high blood pressure, and fatigue
4. Nausea, dry mouth, and muscle cramps

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4. Which of the following factors plays a critical role in the development of vascular complications in diabetes, including atherosclerosis? (0.5p)

1. Chronic elevation of free fatty acids, leading to endothelial dysfunction
2. Hyperglycemia-induced oxidative stress resulting in endothelial damage and increased vascular permeability
3. Overproduction of inflammatory cytokines due to macrophage infiltration into tissues
4. Decreased secretion of nitric oxide from endothelial cells, causing impaired vasodilation

5. Which of the following is the most likely cause of the characteristic "fruity breath" odor seen in patients with diabetic ketoacidosis (DKA)? (0.5p)

1. Acetone, a ketone body, is produced as a byproduct of fatty acid metabolism in the absence of insulin.
2. The accumulation of lactic acid in blood leads to an odor resembling fermenting fruit.
3. Elevated blood glucose causes the breakdown of sugar into ethanol, resulting in a sweet smell.
4. Beta-hydroxybutyrate is produced in excess and gives off a sweet-smelling odor when exhaled.

6. Choose whether the following statements are true or false by putting a cross in the appropriate box. A correct answer gives 0.25p, and an incorrect answer gives -0.25p. The sum cannot be less than 0p. Total 1p

	True	False
In type 1 diabetes, hyperglycemia is primarily due to insulin resistance rather than insulin deficiency.		
In type 2 diabetes, the pancreas stops producing insulin entirely.		
Diabetic retinopathy is a common long-term complication in both type 1 and type 2 diabetes, caused by damage to the small blood vessels in the eyes.		
People with type 2 diabetes are more likely to experience diabetic ketoacidosis (DKA) compared to people with type 1 diabetes.		

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7. Explain the pathophysiology of Graves' disease, specifying whether it results in hypothyroidism or hyperthyroidism, and identify which biomarker you would assess for diagnosis, including the expected levels. (6p)

8. Tachycardia and weight loss are common symptoms of hyperthyroidism. Explain the physiological basis for each. (2p)

Tachycardia:

Weight loss:

9. Name two underlying causes of primary hypothyroidism. (1p)

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Pharmacology

10. State two currently available routes of administration for insulin. 1p

11. Describe the mechanism of action of sulfonylureas. 3p

12. Why is the risk of hypoglycemia with α-glucosidase inhibitors less than that with sulfonylureas? 1p

13. Why is the use of α-glucosidase inhibitors limited in clinical practice? 1,5 p

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14. Describe the mechanism of action of sodium–glucose cotransporter 2 inhibitors (SGLT2) 2p

15. Why should metformin (biguanides) be administered with meals? 2p

16. Describe the proposed mechanism by which glucagon-like peptide receptor agonists (GLP-1 agonists) cause weight loss? 2p

17. Why can certain GLP-1 agonists be dosed once weekly? 1,5p

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18. State a therapeutic use of thioamides. 1p

19. Thiazolidinediones act as agonists at which receptor? 1p

20. Choose whether the following statements are true or false by putting a cross in the appropriate box.
A correct answer gives 0.25p, and an incorrect answer gives -0.25p. The sum cannot be less than 0p. Total 2p

	True	False
Levothyroxine (T4) has a longer half-life than liothyronine (T3)		
Levothyroxine toxicity mirrors the symptoms of hypothyroidism		
In the cell, levothyroxine is enzymatically deiodinated to T3.		
Urinary tract infections are common side effects of sodium–glucose cotransporter 2 inhibitors (SGLT2)		
Dipeptidyl peptidase-4 inhibitors (DPP-4) inhibitors cannot be administered orally as they are degraded in the gastrointestinal tract		
Thiazolidinediones delay the digestion of carbohydrates, resulting in lower postprandial glucose levels		
Weight gain can occur with thiazolidinediones due to increased subcutaneous fat and cause fluid retention		
Modification of the amino acid sequence of human insulin produces insulins with different pharmacokinetic properties		