مسابقة في مادة علوم الحياة
المدة: ثلاث ساعات

الاسم:
الرقم:

Answer the following questions:

Question I (3 pts)

A- Hepatic cells, like muscle cells, are capable of storing glucose in the form of glycogen. In case of need, glycogen is hydrolyzed into glucose 6 phosphate, but only hepatic cells are capable of liberating glucose into the blood. Document 1 illustrates these reactions.

Glucose + Phosphate group ⟷ **Glucose 6 phosphate (G6P)** ⟷ **Glycogen**

Document 1

a- Name the process of storage of glucose in the form of glycogen, and that of hydrolysis of glycogen into glucose.

Document 2 reveals certain hepatic and muscle cell enzymes that intervene in the reactions of these processes.

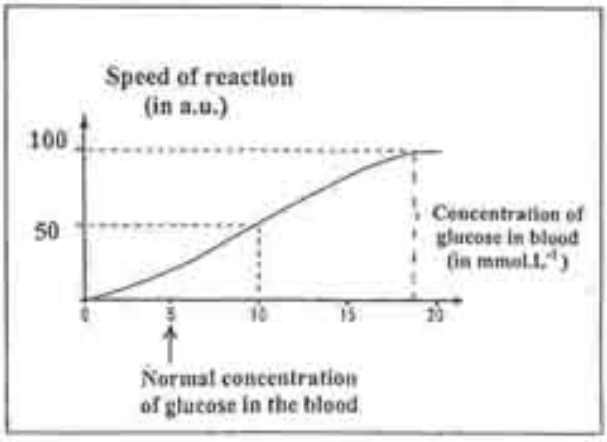
Name of the enzyme	Function	Presence of enzyme	
		Muscle cells	Hepatic cells
Glycogen phosphorylase	Transforms glycogen into G6P	Yes	Yes
Glucose 6 phosphatase	Transforms G6P into glucose	No	Yes

Document 2

b- Justify, by referring to documents 1 and 2, why hepatic cells only are capable of liberating glucose from glycogen into the blood.

B- Certain types of diabetes that are non-insulin dependent, called MODY diabetes, affect some young individuals. The genes responsible for this rare diabetes are known. One of them codes for an enzyme found in the hepatic cells called glucokinase, which transforms glucose into glucose 6 phosphate. Document 3 indicates the speed of the reaction of this enzyme as a function of the concentration of glucose in the medium. When mutations affect this gene, the activity of the synthesized glucokinase becomes nil.

c- Explain, by referring to the acquired knowledge and document 3, how the mutated gene of glucokinase can be responsible for the appearance of non-insulin dependent diabetes.



Document 3

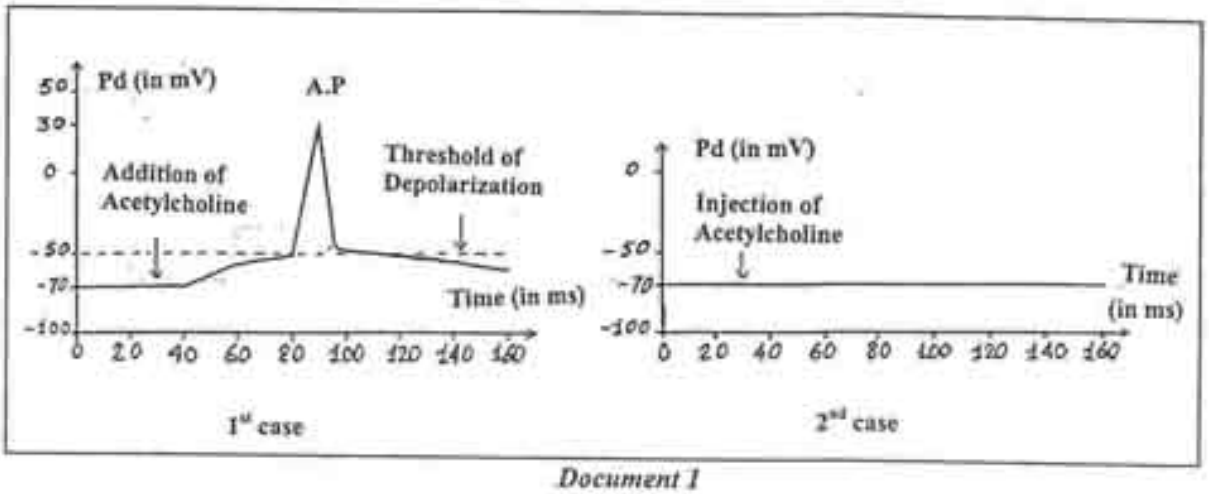
Question II (4 pts)

To understand the intervention of acetylcholine in the functioning of a neuro-muscular synapse, we depend on the results of the following experiments:

1st Experiment: We isolate some muscle fibers and we record the variations of membrane potentials of these fibers under the action of acetylcholine in two different cases:

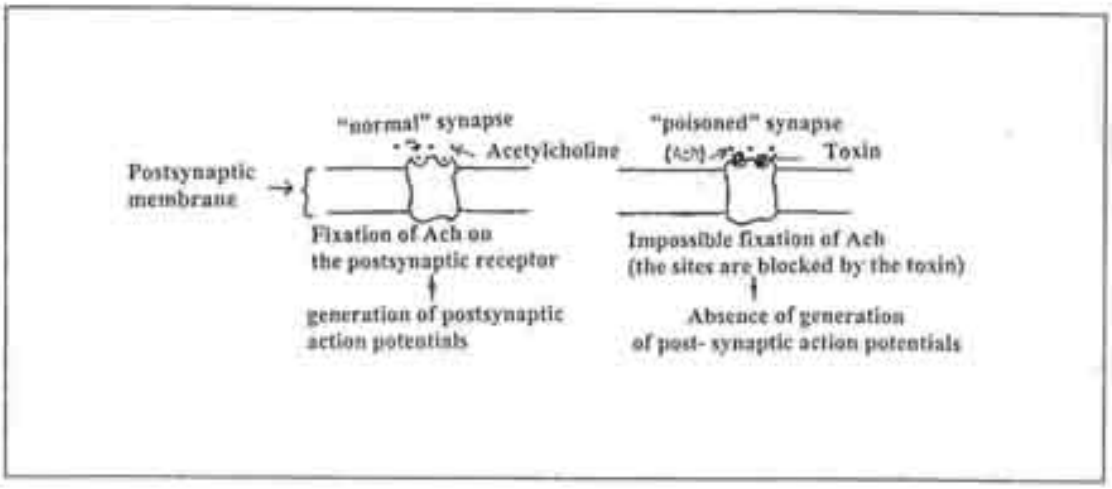
- **Case 1:** We add a sufficient quantity of acetylcholine into the synaptic cleft.
- **Case 2:** We inject the same quantity of acetylcholine inside the muscle fibers.

The results of the recordings are shown in document 1.



Document 1

2nd Experiment: We add into this synapse α-bungarotoxine, a poison found in the venom of the snake, then we add acetylcholine into the synapse, document 2. No muscular contraction is recorded.



Document 2

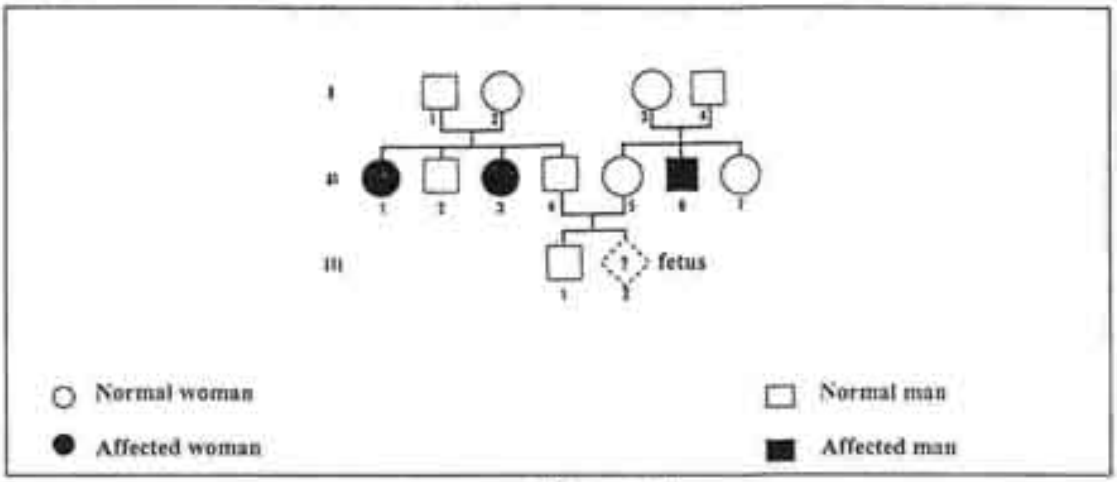
a- Interpret the experimental results of each of the two documents 1 and 2. What can you deduce concerning the intervention of acetylcholine in the muscle activity?

b- Explain, by referring to the acquired knowledge, the steps of the transmission of the nervous message at the level of a neuro-muscular synapse.

Question III (5 pts)

Document 1 represents the pedigree of a family of whom some members, figured in black, are affected by a disease called cystic fibrosis, a hereditary disease manifested by respiratory and digestive troubles. This disease is determined by a mutant allele of a gene called CF. This gene is located on chromosome 7, and very close to a non-coding region that has restriction sites recognized by the restriction enzyme Taq 1.

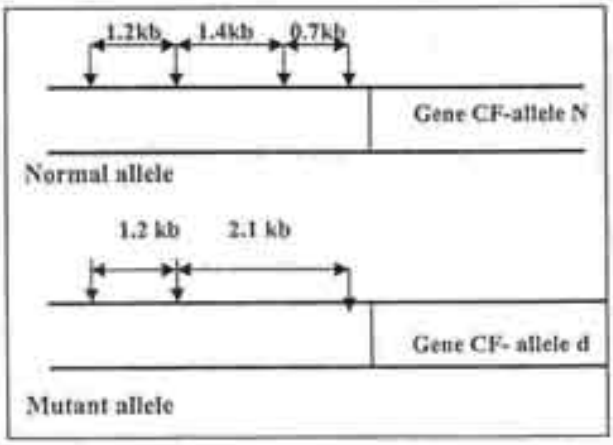
The non-coding region close to the functional dominant allele N has four restriction sites for enzyme Taq 1, while the non-coding region close to the mutated recessive allele d has three restriction sites. The length of the restriction fragments is expressed in kilobase (kb), document 2.



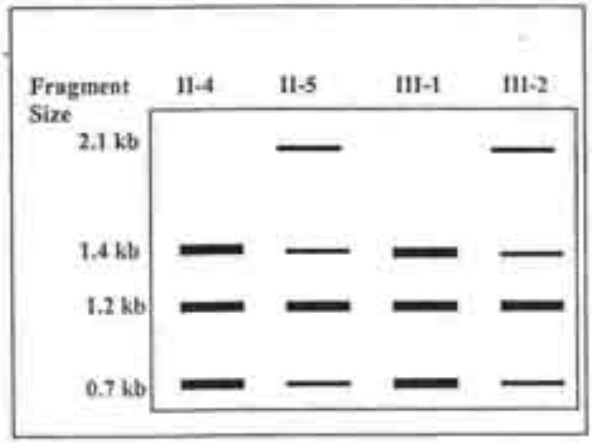
Document 1

a- Indicate the possible genotypes of individuals II-4 and II-5. Justify the answer.

b- Determine the genetic risk of couple II-4 and II-5 to have a sick child.



Document 2



Document 3

c- Specify the site at which mutation took place, document 2. Justify the answer.

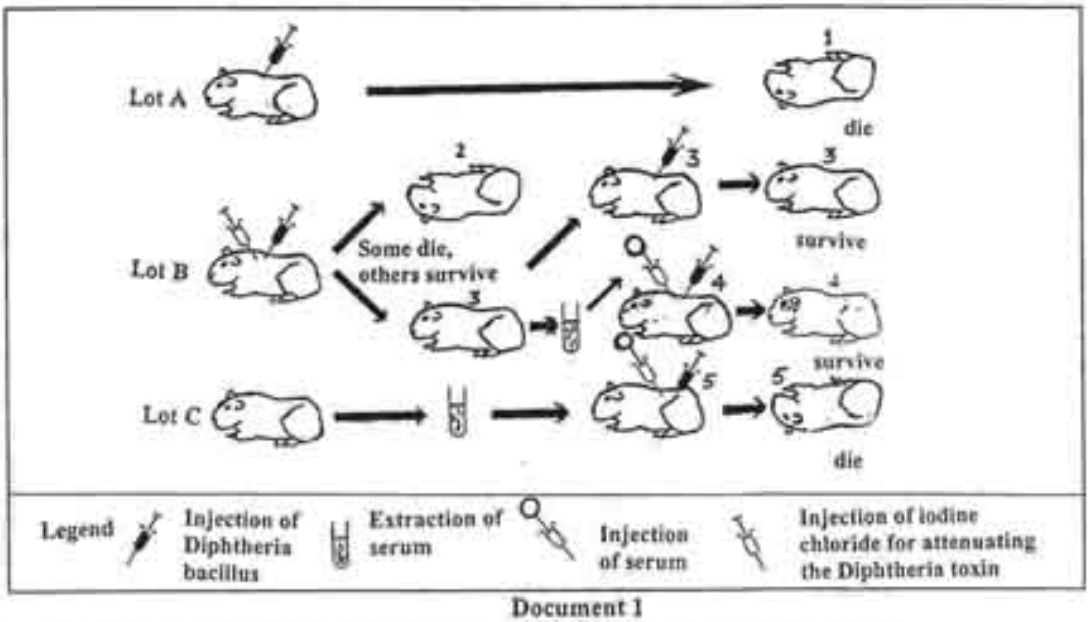
Document 3 shows the results of electrophoresis of the restriction fragments obtained by Southern Blot technique for individuals II-4, II-5, and their children.

d- After analyzing the obtained results, indicate the real genotype of each of individuals II-4, II-5, and the fetus.

e- Based on the above analysis, is this couple in risk of having affected children? Justify the answer.

Question IV (8 pts)

A- In the framework of studying the transmission of immunity against diphtheria, a human disease caused by a bacillus that secretes a deadly protein toxin, the following experiments are conducted on Guinea pigs. Document 1 shows the experimental procedure used and the results obtained.



Document 1

a- Describe, in a short text, each experiment performed and its obtained result.

b- Interpret these experiments. What can you deduce?

c- Indicate the two medical applications that you can draw out from these experiments? Justify the answer.

B- To understand why some infectious diseases can infect an organism only one time during life, even when the same organism is confronted with the same pathogenic microorganism again, we perform the following experiment.

We inject a Guinea pig with an attenuated antigen X, and we measure the amount of plasma anti-X antibodies. After 50 days, when the amount of anti-X antibodies in the plasma becomes nearly nil, we inject the same Guinea pig again with antigen X and another antigen: antigen Y. We measure the amount of plasma anti-X and anti-Y antibodies. The results are shown in document 2.

Time (in days)	0	8	15	30	50	60	75	85	100
Amount of anti-X antibodies (in a.u)	0	0	1	0.8	0.2	1.5	3	2.8	2.6
Amount of anti-Y antibodies (in a.u)	-	-	-	-	0	0	1	0.5	0

1st contact with antigen X

2nd contact with antigen X and the 1st contact with antigen Y

Document 2

d- Draw, on the same graph the curves showing the variation of plasma anti-X and anti-Y antibodies as a function of time, specifying on the graph the contacts with the antigens.

e- Analyze the variations of the amount of anti-X antibodies, document 2. Draw out the characteristics of the secondary immune response.

f- What do the results of antigen-Y injection confirm?

أسس تصحيح مادة علوم الحياة

Question I (3 pts)

a- Glycogenesis (¼ pt) Glycogenolysis (¼ pt)

b- The liberation of glucose into the blood necessitates the transformation of glycogen into G6P, then into glucose under the action of specific enzymes. Document 2 reveals that only hepatic cells contain an enzyme called glucose 6 phosphatase, capable to transform G6P into free glucose, which is liberated in the blood. **(1 pt)**

c- Glucokinase transforms glucose into G6P in the hepatic cells. The speed of the reaction of the enzyme is a function of the concentration of glucose in the medium and becomes maximal when the concentration of glucose increases to a value of 20 $mmol.L^{-1}$. The action of this enzyme increases during hyperglycemia, which leads to more storage of blood glucose and consequently glycemia returns to normal.
When a mutation affects the gene of glucokinase, the synthesized enzyme loses its activity and the hepatic cells can not store excess of glucose, no matter how much the insulin concentration is. Hyperglycemia occurs and this diabetes is not insulin dependent.

(1 ½ pts)

Question II (4 pts)

a- In the 1st experiment, the addition of acetylcholine at the level of the synaptic cleft causes an action potential of an amplitude that reaches +30 mV. While the injection of acetylcholine into the muscle fiber produces no variation of potential. Since the quantity of acetylcholine added or injected is the same, therefore, acetylcholine acts only at the level of the synaptic cleft. **(1 ½ pt)**

Document 2, reveals that in a normal synapse, acetylcholine fixes to the postsynaptic receptors provoking the generation of post synaptic action potential. On the other hand, in the case of the poisoned synapse, acetylcholine does not fix at the postsynaptic receptors, since the toxin blocks them and no post synaptic action potential is generated. This means that the fixation of acetylcholine on the postsynaptic membrane receptors is indispensable for the generation of an action potential. **(1 pt)**
Hence, acetylcholine liberated into the synapse fixes on the postsynaptic membrane receptors and provokes the generation of action potentials that leads to the contraction of the muscle. **(½ pt)**

b- The arrival of action potential at the presynaptic nerve endings permits the opening of the membrane calcium ion channels and the penetration of Ca^{++} into the terminal buds. This leads to the liberation of the neurotransmitter by exocytosis into the synaptic cleft. The liberated neurotransmitters fix to their specific receptors on the postsynaptic membrane, which modifies its potential generating a PSP. **(1 pt)**

Question III (5 pts)

a- II-4 and II-5: NN or Nd. Since they are phenotypically normal, each should have a dominant allele N and another allele that can be either N or d. **(1 pt)**

b- II-4 and II-5 present the normal phenotypes. The risk for each of the two parents to be heterozygous is 2/3 and the risk for two heterozygous couple to have an affected child is 1/4, therefore, the risk of having a sick child is 2/3 x 2/3 x 1/4 = 1/9 **(1pt)**

c- Mutation has occurred at the site between 1.4 kb and 0.7 kb, because, the mutant allele, shows one fragment of 2.1 kb instead of two fragments 1.4 kb and 0.7 kb. **(1 pt)**

d- II-4 has two of each fragment 1.4 kb, 1.2 kb, and 0.7 kb. These fragments correspond to the normal allele. Thus he is homozygous normal of genotype NN. **(½ pt)**
II-5 has one fragment 2.1 kb and 1.2 kb, which corresponds to the mutant allele, and fragments 1.4 kb, 1.2 kb, and 0.7 kb., which correspond to the normal allele. Thus, he is heterozygous normal of genotype Nd. **(½ pt)**
Fetus III-2 has a fragment 2.1 kb, which implies that he has received the mutant gene from his mother. He also has fragments 1.4 kb, 1.2kb, and 0.7 kb, which correspond to the normal allele that he received from his father. Thus, he will be normal heterozygous of genotype Nd. **(½ pt)**

e- No, because the two parents are not heterozygous and the father II-4 who is homozygous gives only one type of gamete N. **(½ pt)**

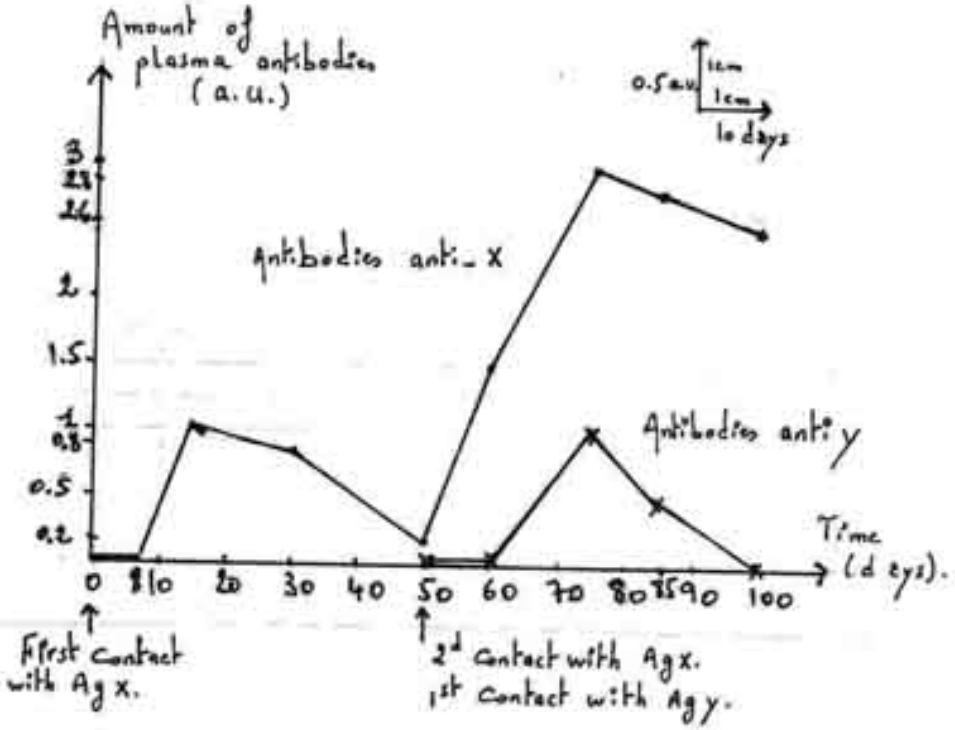
Question IV (8 pts)

a- We inject Guinea pigs of lot A with diphtheria bacillus, they die (1). We inject Guinea pigs of lot B with attenuated diphtheria toxin (iodine chloride + diphtheria bacillus), some Guinea pigs die (2) while others survive (3). We inject the Guinea pigs who survived (3) with diphtheria bacillus again, they survive. We extract serum from the surviving Guinea pigs (3) and we inject it into other Guinea pigs (4) together with an injection of diphtheria bacillus, they survive.
We extract serum from Guinea pigs of lot C and we inject them with diphtheria bacillus into other Guinea pigs (5), they die. **(1 ½ pts)**

b- The injection of diphtheria bacillus (D.B), into Guinea pigs of lot A causes their death, while the injection of iodine chloride and D.B (attenuated diphtheria toxin) into Guinea pigs of lot B does not kill all the Guinea pigs, and those who survive (3) do not die even when they are injected with the D.B (3). Therefore, the attenuated toxin is not deadly, it causes immunity against diphtheria bacillus.
The injection of the serum of immunized Guinea pigs (3) into guinea pigs (4), not immunized, protects them from D.B, while the injection of serum from non-immunized Guinea pigs of lot C could not protect guinea pigs (5) against diphtheria bacillus. This means that the serum obtained from immunized Guinea pigs contains molecules that provides immunity against D.B. Thus, attenuated Diphtheria toxin provides immunity against diphtheria bacillus transferred by the serum.**(2 pts)**

c- Vaccination and serotherapy. Because in the case of vaccination, we give an attenuated toxin, which allows the body to launch an immune response upon contact with the concerned antigen (Guinea pigs 3). In the case of serotherapy, we give the serum which contains anti-bodies, that are against the concerned antigens (guinea pig 4). **(1pt)**

d- (1 1/2 pts)



Variation of the concentration of the plasma anti-X and anti-Y antibodies

e- The amount of plasma anti-X antibodies is nil at the beginning and starts to increase 8 days after the first contact with antigen X. It reached a maximum of 1 a.u on day 15 then it decreased progressively to reach 0.2 a.u on day 50. The second contact with the antigen X on day 50, causes a rapid increase in the amount of anti-X antibodies to reach a maximum of 3 a.u on day 75, then it decreases slowly to become 2.6 a.u on day 100.
Since the second contact with the same antigen causes the production of anti-bodies in larger quantity with less latent period and which persists longer, hence the secondary immune response is characterized by being more rapid, amplified and more persistent. **(1 ½ pt)**

f. The results of antigen Y injection confirm that the immune response is specific, and at the first contact it is always slow, less amplified, and not persistent. **(½ pt)**