وزارة التربية والتعليم العالي
المديرية العامة للتربية
دائرة الامتحانات

امتحانات شهادة الثانوية العامة
فرع علوم الحياة

دورة سنة ٢٠٠٧ الاكمالية الاستثنائية

مسابقة في علوم الحياة
المدة: ٣ ساعات

الاسم:
الرقم:

Answer the following exercises.

Exercise 1 (5pts)

To determine the cause of juvenile diabetes in humans, the following experiments were carried out on mutant rats of the same strain, in which diabetes appears in the first few months of their life.

1st experiment: 100 newborn mutant rats were brought, and divided into two lots, lot A and lot B. Lot A was subjected to the ablation of the thymus, the organ where T lymphocytes undergo maturation, and lot B was used as control. A few months later; the number of the rats that presented diabetes was determined, document 1.

	Number of diabetic rats
Lot A	5/50
Lot B	30/50

Document 1

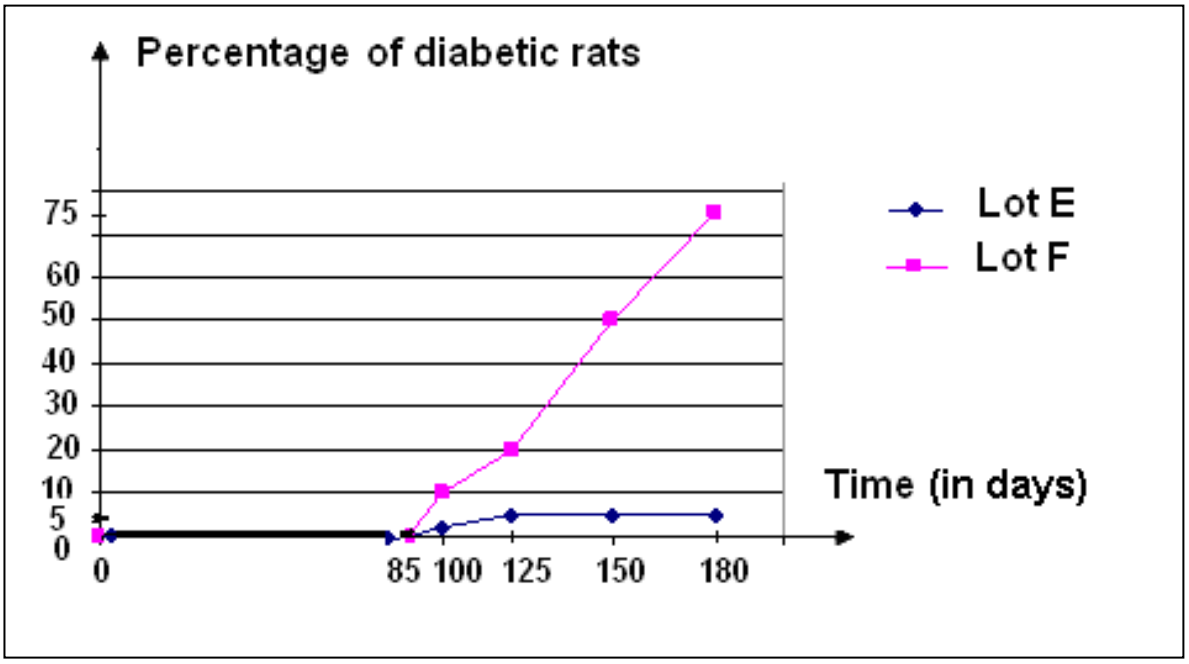
2nd experiment: Two lots of healthy non-mutant rats, lot C and lot D, were brought. The rats of lot C were injected with TL taken from diabetic mutant rats, and the rats of lot D were injected with TL taken from healthy rats. The rats of lot C, only, developed diabetes.

1- Formulate the hypothesis at the origin of these experiments.

2- Interpret each of the carried out experiments. What can one deduce regarding the formulated hypothesis?

3- What name can be attributed to this kind of disease? Justify the answer.

3rd experiment: Two lots of mutant rats, lot E and lot F, were brought. Lot E was treated, from birth, with cyclosporine, an immunosuppressant medicine, and lot F was used as control. Document 2 reveals the percentages of diabetic rats in these two lots of rats.



Document 2

4- Present in a table the different data provided by document 2.

5- Interpret the obtained results. Draw out the mode of action of cyclosporine.

Exercise 2 (5pts)

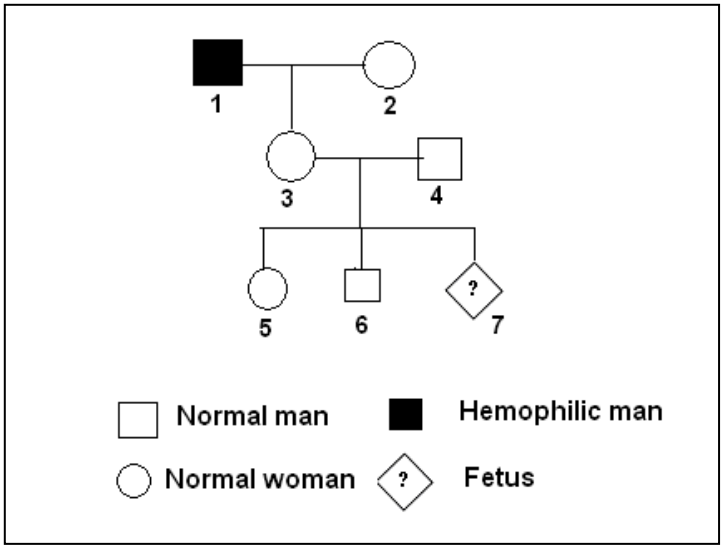
Hemophilia B is characterized by the absence of blood clotting, which may lead to significant hemorrhage. It is linked to the absence of a clotting factor, factor IX, whose synthesis is controlled by a gene located on the non-homologous segment of the X chromosome. This abnormality affects boys and not girls.

1- Explain the absence of this abnormality in girls?

Document 1 shows the pedigree of a family, one member of whom has the abnormality.

2- Show that this disease is recessive.

3- Determine the genetic risk that the fetus will be hemophilic.

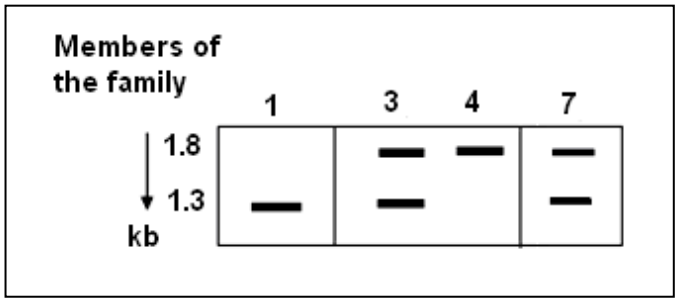


Document 1

Ultrasound scan was done to determine the sex of the fetus. It revealed that it is a boy. The doctor then prescribed analysis of DNA by the method of Southern blotting. The used probe permits to distinguish the mutated and normal forms of the implicated gene. The obtained results appear in document 2.

4- Specify the band that corresponds to the defective allele. Justify the answer.

5- Identify, from the DNA analysis, the problem of the child that will be born.



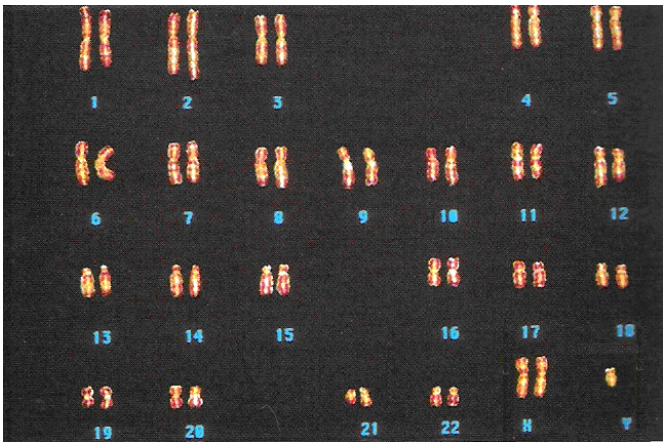
Document 2

The doctor completed the diagnosis by establishing the karyotype of the fetus, document 3.

6- Establish, based on documents 2 and 3, the diagnosis of the fetus.

7- Specify the stage of meiosis at which the abnormality took place. Justify the answer.

8- Schematize the behavior of chromosomes at the origin of this abnormality.



Document 3

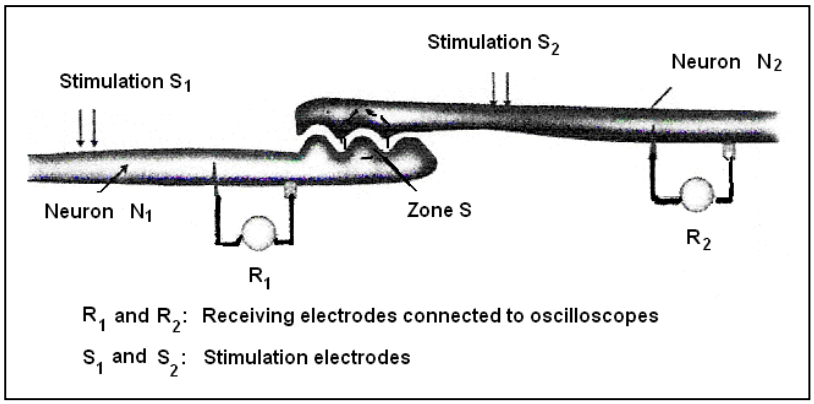
Exercise 3 (5pts)

To understand the mechanism of transmission of the nervous message at the level of a synapse, experiments were carried out on two neurons N_1 and N_2 of a squid, using the setup that appears in document 1.

1st experiment: The nerve fiber of N_1 was stimulated by S_1. An action potential was recorded in R_1 then in R_2.

2nd experiment: The nerve fiber of N_2 was stimulated by S_2. An AP was recorded only in R_2.

3rd experiment: A micro-drop of acetylcholine was deposited at the level of zone S between N_1 and N_2. An AP was recorded only in R_2.



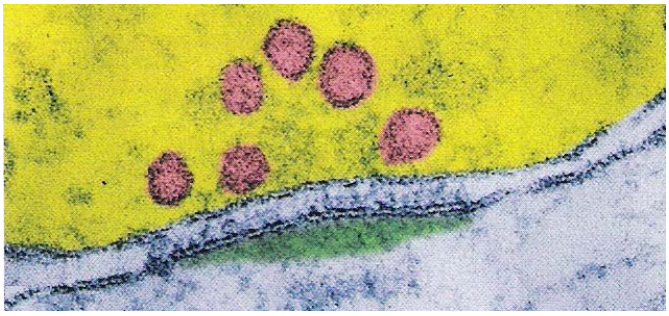
Document 1

4th experiment: A micro-drop of acetylcholine was injected in neuron N_1 and another drop in N_2. No AP was recorded in R_1 or R_2.

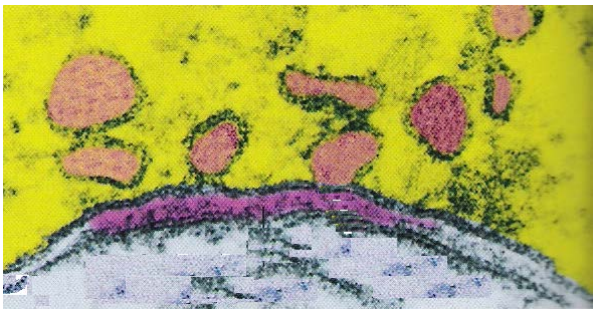
1- Interpret the 1st and 2nd experiments. What can one deduce?

2- Interpret the 3rd and 4th experiments. Deduce the level of the nerve cell at which acetylcholine acts.

Documents 2a and 2b present electronic micrographs of the synapse at the level of zone S at two different moments.



Document 2a



Document 2b

3- Specify the state in which this zone was found when each of the micrographs was taken. Justify the answer.

The study of a synapse made it possible to establish the relation between the frequency of the presynaptic action potentials, the number of vesicles that release their neurotransmitter, and the quantity of acetylcholine liberated into the synaptic cleft. Document 3 shows the results.

Frequency of presynaptic AP (a.u)	**1**	**2**	**4**	**6**
Number of vesicles (in thousands)	**1**	**2**	**4**	**6**
Quantity of acetylcholine (in a.u)	**100**	**200**	**400**	**600**

Document 3

4- Based on the analysis of document 3, determine how the nervous message is coded during synaptic transmission.

5- Based on what preceded, and with reference to acquired knowledge, explain how the nervous message is transmitted at the level of the synapse.

Exercise 4 (5pts)

To determine the reaction of the hepatic and muscular cells to the pancreatic hormones, insulin and glucagon, the following experiments were carried out.

1st experiment: The concentration of hepatic glycogen and the activity of an enzyme implicated in the hydrolysis of this glycogen were measured, following injection of glucagon. The results appear in document 1.

1- Analyze the obtained results. Draw out the mode of action of glucagon.

2nd experiment: A muscle was placed for 10 minutes in a medium containing glucose and insulin, or glucose without insulin. Then the quantity of glucose absorbed by the muscle and the quantity of glycogen stored in the muscle in each used medium were measured. The results are shown in document 2.

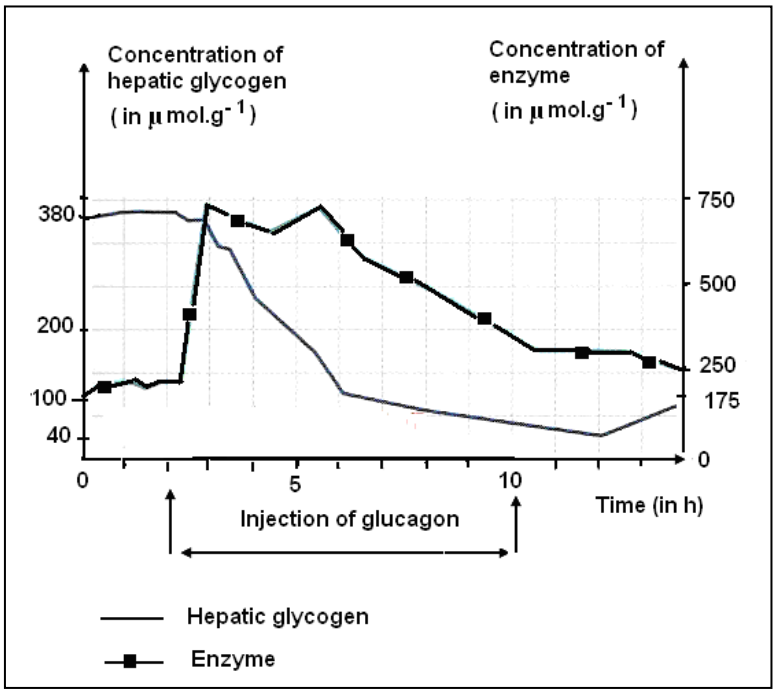
2- Construct a histogram showing the results of the obtained measurements in each of the two media.

3- Compare these results. Draw out the effect of insulin on the muscle.

3rd experiment: The muscles and the liver were perfused with different solutions of insulin having increasing concentrations. Then the quantity of glucose released by the liver and the quantity of glucose used by the muscles were measured, document 3.

4- Interpret the obtained results.

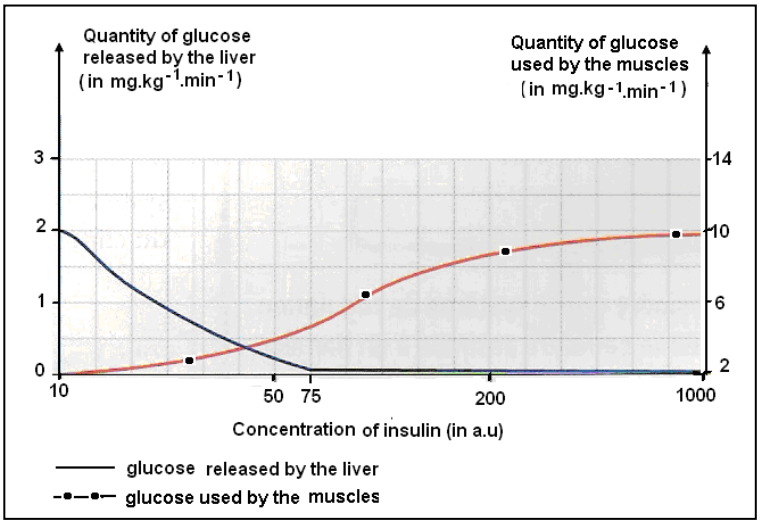
5- Determine, based on what had preceded, the reaction of muscular cells and hepatic cells to the pancreatic hormones.



Document 1

	Medium without insulin	Medium with insulin
Absorbed glucose (in mg/g muscle)	**1.43**	**1.88**
Muscular glycogen (in mg/g muscle)	**2.45**	**2.85**

Document 2



Document 3

مسابقة في علوم الحياة
اسس التصحيح

الاسم:
الرقم:

Exercise 1 (5pts)

1- Hypothesis: T lymphocytes of the mutant rats are at the origin of juvenile diabetes. **(0.5pt)**

2- The number of the rats that have juvenile diabetes is 5/50 in lot A, which had undergone ablation of the thymus. On the contrary, the number of the rats that have juvenile diabetes is larger (30/50) in the control lot B. This indicates that the thymus, the place of maturation of T lymphocytes, is implicated in the appearance of diabetes.
The 2nd experiment revealed that the healthy rats of lot C, injected with TL taken from mutant rats, developed diabetes, whereas the healthy rats of lot D, injected with TL taken from healthy rats, did not develop diabetes. This implies that the appearance of the disease is linked to the presence of the TL of the mutant rats. Thus the formulated hypothesis is valid and they are the TL of the mutant rats that are responsible for the disease. **(1.5pt)**

3- Auto-immune disease. **(0.25pt)**
The T lymphocytes are directed against the self; they recognize it as modified self and attack it. **(0.25pt)**

4- **(1pt)**

Time (in days) / Diabetic rats (in %)	0	85	100	125	150	180
Lot E	0	0	2	5	5	5
Lot F	0	0	10	20	50	75

Variation of the percentages of diabetic rats as a function of time in lots E and F

5- The percentage of diabetic rats was null in the two lots of rats until day 85. At day 100 the percentage increased to become 2% in the rats treated with cyclosporine, lot E, and 10% in the untreated rats, lot F. The percentage continued increasing to become 5% in lot E, and 20% in lot F. This percentage remained stable at 5% in the treated rats from day 125 to day 180, whereas it continued to increase in the untreated rats after day 125 to become 75% at day 180. This implies that the treatment with cyclosporine had prevented the appearance of diabetes in the mutant rats of lot E. Thus cyclosporine is a medicine that inhibits the action of TL responsible for the appearance of diabetes in the mutant rats. **(1.5pts)**

Exercise 2 (5pts)

1- The allele of hemophilia is lethal in the homozygous state. The girl has two X chromosomes. If she is X^hX^h, she dies before birth. **(0.5pt)**

2- The disease is carried by the X chromosome. The sick individual 1 has only one X, which carries the allele responsible for hemophilia, which he will certainly transmit it to his daughter 3. Girl 3 is normal. She carries an X chromosome having the allele without expressing it. Hence the disease is recessive. **(0.5pts)**

3- Fetus 7 has a heterozygous mother. If it is a boy, there is a risk of ½ to have the X chromosome carrying the allele of hemophilia. If it is a girl, the risk is null because her healthy non-hemophilic father cannot give her except one normal X. **(0.5pt)**

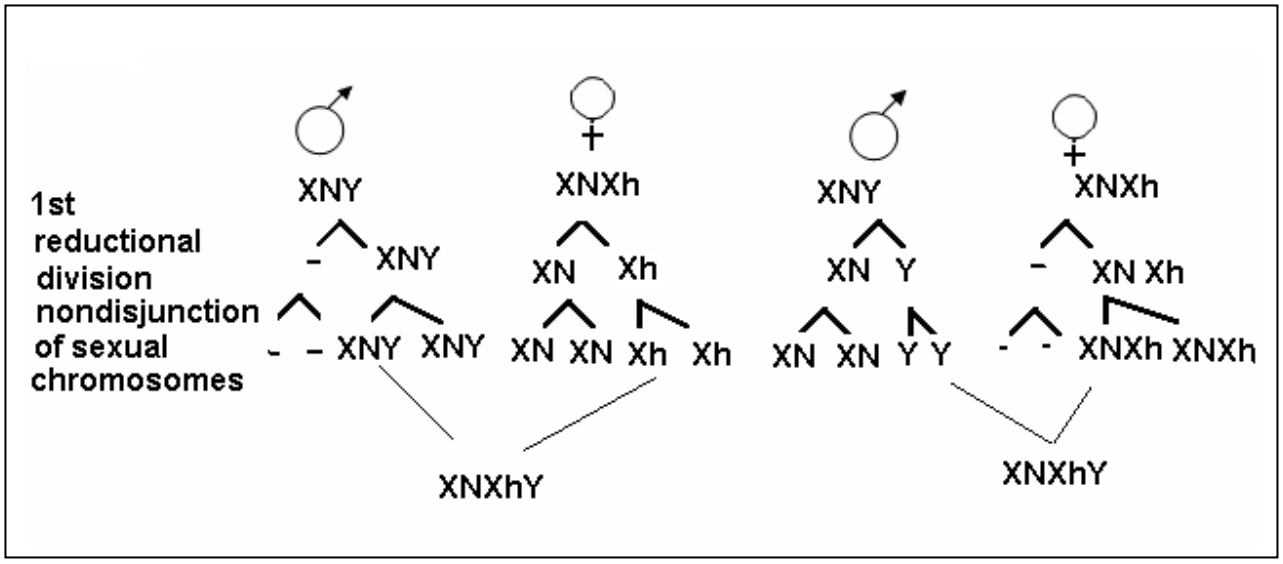
4- The 1.3 kb-band, because document 2 reveals that individual 1, who is a sick man and has only one X, has only one band of 1.3 kb. **(0.5pt)**

5- The fetus is a boy, hence he has only one X chromosome, then he must have only one band of DNA, but according to document 2 he presents two bands. Therefore, it is a boy with 2 X. **(0.5pt)**

6- Fetus 7 is a nonhemophilic boy (doc 2), but he has XXY (doc 3). Thus he will have Kleinfelter syndrome. **(0.5pt**

7- The abnormality of meiosis had taken place during the anaphase of the reductional division by nondisjunction of chromosomes XX or XY, because upon the analysis of DNA there are two different bands that correspond to two X and not to two chromatids of the same X chromosome. In this case the father or the mother could be at the origin of this abnormality. **(1pt)**

8- (1pt)



Exercise 3 (5pts)

1- The stimulation of the nerve fiber by S_1 caused an AP recorded in R_1 then in R_2. On the contrary, the stimulation by S_2 caused the recording of an AP only in R_2. This means that the nervous message passes from N_1 towards N_2, and not in the opposite direction. Thus, the nervous message is unidirectional. **(1pt)**

2- The deposition of the micro-drop of acetylcholine in zone S caused an AP recorded in R_2 that is attached to the postsynaptic neuron, and no recording in R_1 that is attached to the presynaptic neuron. On the contrary, the injection of the micro-drop of acetylcholine in N_1 or N_2 caused no AP in R_1 or R_2. This indicates that acetylcholine acts on the postsynaptic neuron and does not act on the presynaptic neuron; and this action takes place only when the acetylcholine is deposited in the synaptic cleft. **(1.5pt)**

3- Synapse at rest (document 2a); synapse in action (document 2b). Because the synapse in action reveals vesicles that liberate, by exocytosis, their neurotransmitter into the synaptic cleft. This appears only in document 2b. **(1pt)**

4- For a frequency of presynaptic AP equal to 1 a.u, the number of vesicles that release their neurotransmitter is 1 thousand, and the quantity of released acetylcholine is 100 a.u. When the frequency of presynaptic AP increases reaching 6 a.u, the number of vesicles that release their neurotransmitter increases also and reaches 6 thousands, and the quantity of neurotransmitter increases also and reaches 600 a.u. This permits us to say that the quantity of released acetylcholine into the synaptic cleft increases with the frequency of the presynaptic AP, and that the nervous message at the level of the synapse is coded by modulation of the concentration of neurotransmitter. **(1pt)**

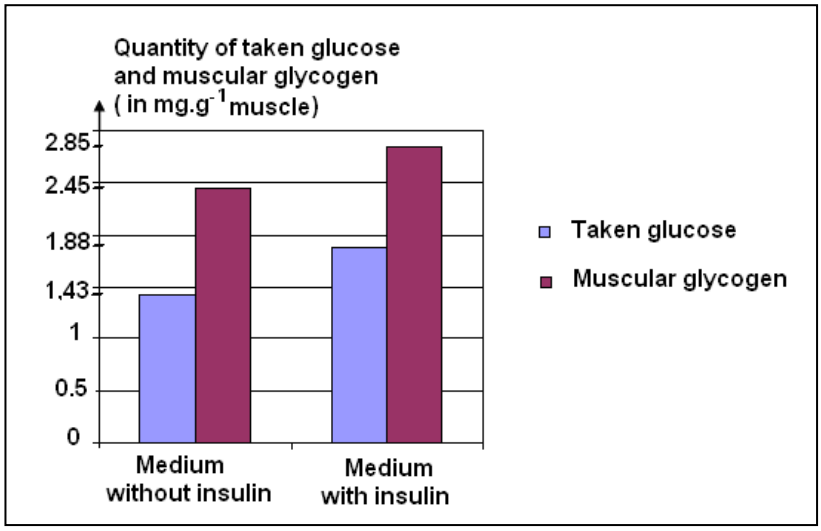
5- Following the arrival of the nervous message at a presynaptic neuron, the voltage-dependent calcium channels of the presynaptic membrane open and allow the entrance of calcium ions. This leads to the fusion of the synaptic vesicles with the presynaptic membrane. The synaptic vesicles pour by exocytosis their content, a neurotransmitter, which is acetylcholine in this case, into the synaptic cleft. This neurotransmitter binds to specific receptors located in the postsynaptic membrane and causes the opening of chemical-dependent sodium channels in the postsynaptic membrane, which causes depolarization at the level of this membrane that will be at the origin of a postsynaptic potential. **(1pt)**

Exercise 4 (5pts)

1- Before the injection of glucagon, the concentration of hepatic glycogen was 380 $\mu mol.g^{-1}$, and the concentration of the enzyme 175 $\mu mol.g^{-1}$. One hour after the injection of glucagon, the concentration of the enzyme increased quickly to become 750 $\mu mol.g^{-1}$ and was followed by a decrease in the concentration of hepatic glycogen that reached 100 $\mu mol.g^{-1}$ after 3h. Then this concentration of glycogen continued to decrease reaching 40 $\mu mol.g^{-1}$. At the same time the concentration of the enzyme decreased also to reach 250 $\mu mol.g^{-1}$, and remained the same for 2h after the injection of glucagon. This implies that glucagon increases the concentration of the enzyme that hydrolyzes glycogen leading to decrease in its concentration in the liver. **(1.5pt)**

2- (1pt)

Histogram showing the variation of the quantity of glucose taken by the muscle and the quantity of muscular glycogen in a medium with insulin or without insulin



3- The quantity of glucose taken by the muscle in a medium without insulin (1.43 $mg.g^{-1}$ of muscle) is smaller than that taken by the muscle in a medium with insulin (1.88 $mg.g^{-1}$ of muscle). The muscular quantity of glycogen (2.45 $mg.g^{-1}$ of muscle) is smaller in a medium without insulin than in a medium with insulin, (2.85 $mg.g^{-1}$of muscle). This means that insulin favors the absorption of glucose by the muscle and increases its storage in the form of muscular glycogen. **(1pt)**

4- The quantity of glucose released by the liver decreases with the increase in the concentration of insulin, from 2 $mg.kg^{-1}.min^{-1}$ at10 a.u concentration of insulin to become almost null when the insulin concentration reaches 75 a.u. On the contrary, the quantity of glucose used by the muscles, which was almost null at an insulin concentration of 10 a.u, increased with the increase in the insulin concentration to become 3 $mg.kg^{-1}.min^{-1}$ when the insulin concentration became 75 a.u. The quantity of glucose released by the liver remains constant, null, from 75 a.u insulin concentration on. On the contrary the quantity of glucose used by the muscle continues to increase to become 10 $mg.kg^{-1}.min^{-1}$ at insulin concentration equal to 1000 a.u. This means that insulin stops the release of glucose by the liver and favors its use by the muscles. **(1pt)**

5- The hepatic cells and muscular cells store glucose in the form of glycogen under the action of insulin. Under the action of glucagons, the hepatic cells hydrolyze the glycogen and release it in the form of glucose into the blood. **(0.5pt)**