وزارة التربية والتعليم العالي
المديرية العامة للتربية
دائرة الامتحانات

امتحانات الشهادة الثانوية العامة
فرع علوم الحياة

دورة سنة 2009 العادية

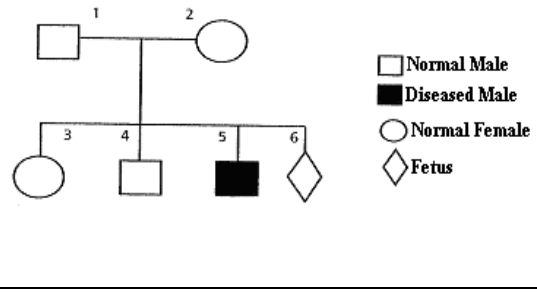
مسابقة في مادة علوم الحياة
المدة: ثلاث ساعات

الاسم:
الرقم:

Answer the following questions

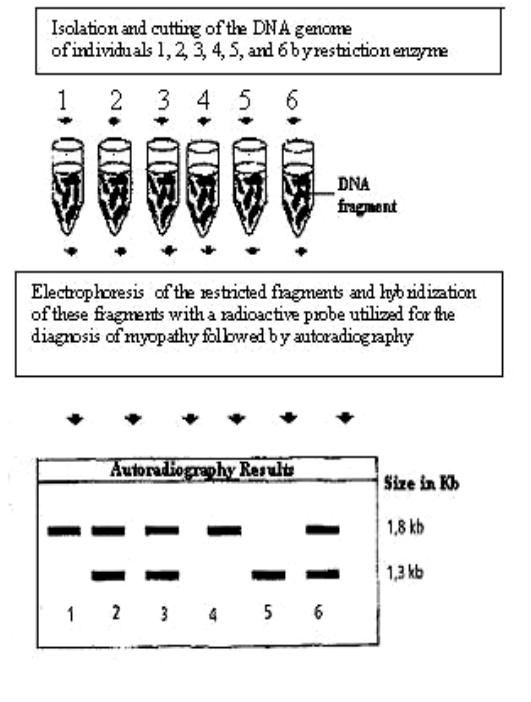
Exercise 1 (5 pts)

Duchene Myopathy is a degenerative disease of muscle fibers which is due to a gene carried on the non- homologous segment of chromosome X.
Boys affected with myopathy do not synthesize the muscle protein, dystrophin, or synthesize an inactive form of dystrophin.
Document 1 represents the pedigree of a family having one member of its family affected with the disease.



Document 1

1-Determine, by referring to the pedigree, whether the allele responsible for the disease is dominant or recessive.
2-Indicate the genotypes of the parents. Justify the answer.
3-Determine the probability of the fetus to be affected.
Parents (1&2) who are expecting a baby want to know whether their fetus is at risk of developing the disease. They consult a doctor who proposes a prenatal diagnostic test by applying Southern Blot technique. The results are shown in document 2.
4-Identify, by referring to document 1 and the autoradiography of document 2, the allele causing the disease. Justify the answer.
5-Specify the sex and the phenotype of the fetus. Justify the answer.



Document 2

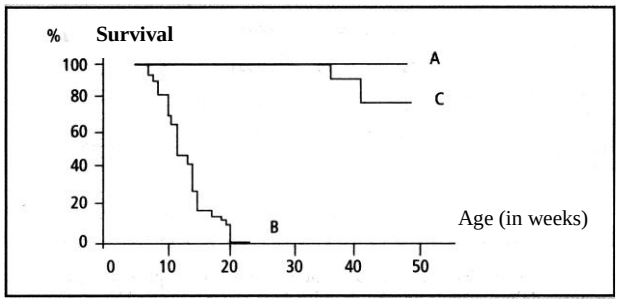
A gene therapy is applied for the first time on mice attaining myopathy similar to Duchene myopathy in humans. This technique consists of injecting the dystrophin gene into a diseased organism by means of a virus vector which is harmless to mice and human species. After this treatment, transversal sections are taken from the diaphragm muscle (respiratory muscle) of 3 groups of mice (A, B and C); then incubated with anti-dystrophin fluorescent antibodies and observed under a fluorescent microscope. The results obtained within 16-18 weeks are shown in document 3.

Mice	Results
A. Normal	Presence of fluorescence
B. Myopathic, non-treated	Absence of fluorescence
C. Myopathic, treated by injecting the dystrophin gene through a virus vector	Presence of fluorescence

Document 3

Document 4 reveals the percentage of survival of the three groups of mice in function of time.

6- Interpret the results obtained in each of documents 3 and 4. What can be deduced about the efficiency of the used gene therapy?



Document 4

Exercise 2 (5 pts)

In an attempt to understand how the HIV that causes AIDS infects selectively T4 cells, we perform the following experiments on many lots of T4 cells (Lymphocytes characterized by the presence of CD4 proteins on their membranes) and T8 cells (lymphocytes characterized by the presence of CD8 proteins on their membranes).

Document 1 presents the experimental procedure and the obtained results.

1-Interpret the obtained results.

Studies and knowledge of the immune system and the immune reactions of persons infected by HIV allow for the preparation of an anti-HIV vaccine. We test the efficiency of this vaccine on Rhesus monkeys.

	Experimental Procedure	**Results**
Lot 1	T4 and T8 cells are placed directly in the presence of HIV	Infection of T4 cells, but no infection of T8 cells
Lot 2	T4 cells are incubated for 20 minutes with several types of antibodies* that do not bind to the membrane protein CD4, then placed with HIV	Infection of T4 cells only
Lot 3	T4 cells are incubated for 20 minutes with antibodies* that bind to the membrane protein CD4, then placed with HIV	No infection of T4 cells
	*Anti-bodies block the biological activity of the molecules to which they bind	

Document 1

Document 2 reveals the variation of the proportion of T8 cells specific to HIV during infection time in vaccinated and non-vaccinated monkeys.

	Time (in weeks)	0	1	2	4	6	8	10	12
Proportion of T8 specific for the HIV (in a.u)	**Lot 1 : Vaccinated monkeys**	0.1	7	6.5	6	4	3	2	2
	Lot 2 : Non-vaccinated monkeys	0	0	0.5	2	1.5	1	1.3	1.5

Exposure to HIV

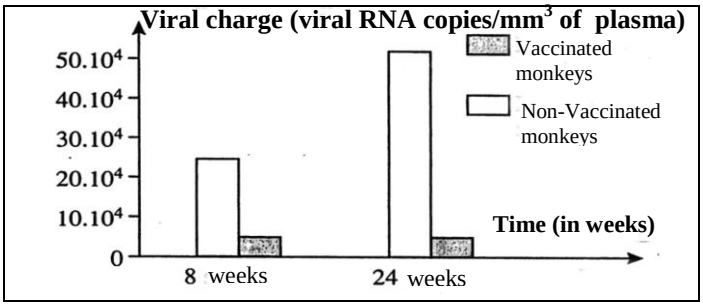
Document 2

2-Draw, on the same graph, the curves obtained from the tabulated data.

3-The immune response in the vaccinated monkeys is rapid and amplified. Refer to the results of document 2 to justify this affirmation.

We measure the viral charge (the number of viral RNA copies /mm3 of plasma that is an indicator of the concentration of the virus in blood) in the vaccinated and non-vaccinated monkeys after 8 and 24 weeks of exposure to the virus. The results are shown in document 3.

4-Compare the obtained results and draw out a relation concerning the effect of the studied vaccine.



Document 3

Exercice 3 (5 pts)

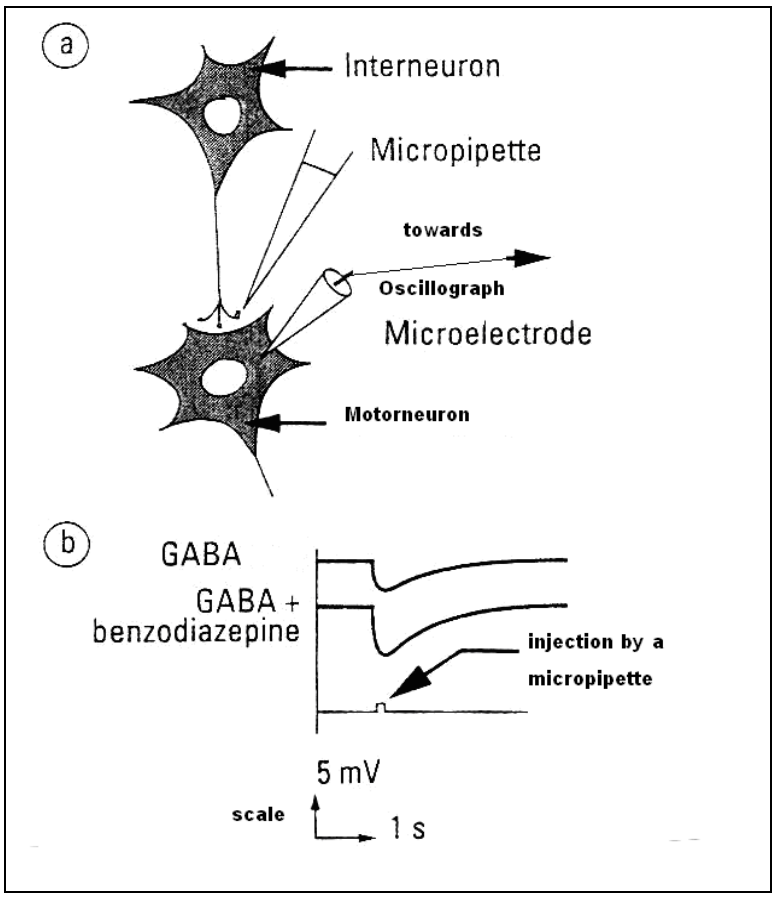
Nowadays, certain molecules that belong to the benzodiazepines family are used in treating anxiety.

In order to study the action of these molecules on the muscular activity, we record by means of a microelectrode the electric activity of the postsynaptic motor neuron following the injection of GABA and/or benzodiazepine into the synaptic cleft using a micropipette. The experimental set up **(a)** and the results **(b)** are represented in document 1.

1- Determine, in reference to document 1, the nature of the studied synapse.

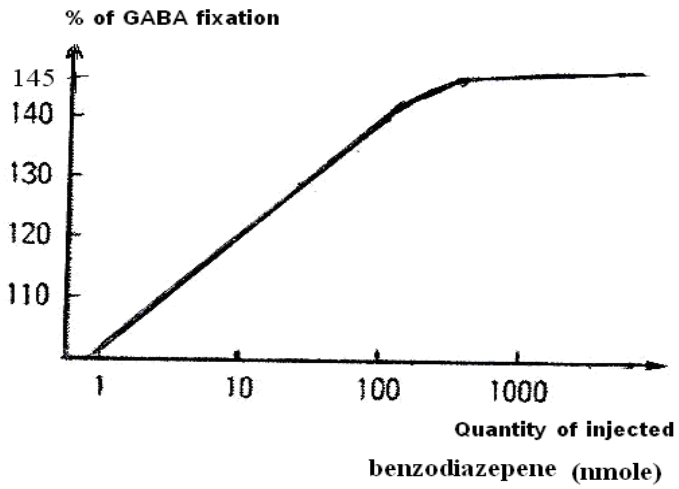
Researchers found that rats administered a chemical substance, called picrotoxine, exhibit involuntary muscular contractions accompanied by signs of anxiety.

2- Specify the effect of picrotoxine and benzodiazepene at the level of this synapse. Justify your answer.

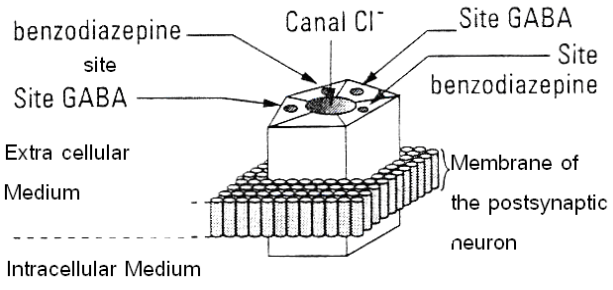


Document 1

In order to understand thoroughly the mode of action of benzodiazepene, we measure the percentage of fixation of GABA on its receptors in function of the quantity of benzodiazepene injected into the synaptic cleft. The results are shown in document 2. Document 3 reveals the organization of the membrane receptor of a motor neuron in the spinal cord.



Document 2



Document 3

3- Represent, in a table, the data shown in document 2.

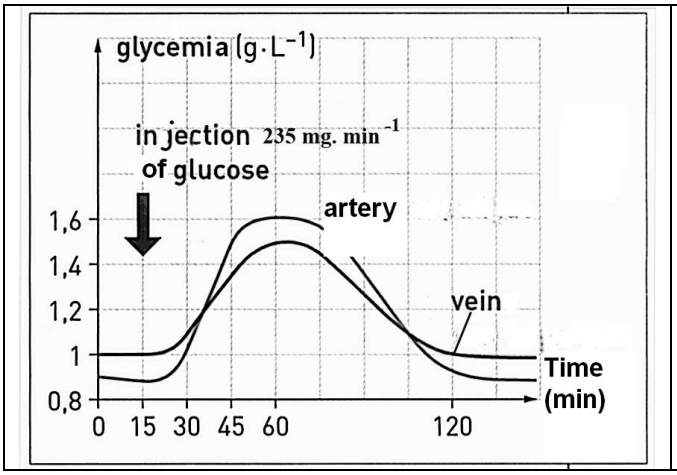
4- What conclusions can be derived from the analysis of document 2?

5- Based on the information derived from all documents, explain the mode of action of benzodiazepene on the muscular activity.

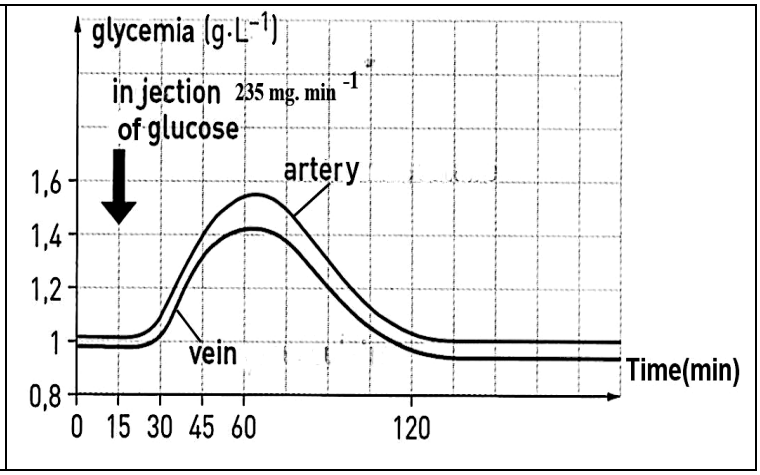
Exercice 4 (5 pts)

We realize a series of experiments to study the role played by certain body organs in response to glucose, insulin and glucagon.

Experiment 1: We measure the variation of glycemia in the arteries and veins of a muscle and liver upon the injection of 235mg/min of glucose. The results are shown in documents 1 and 2.



Document 1



Document 2

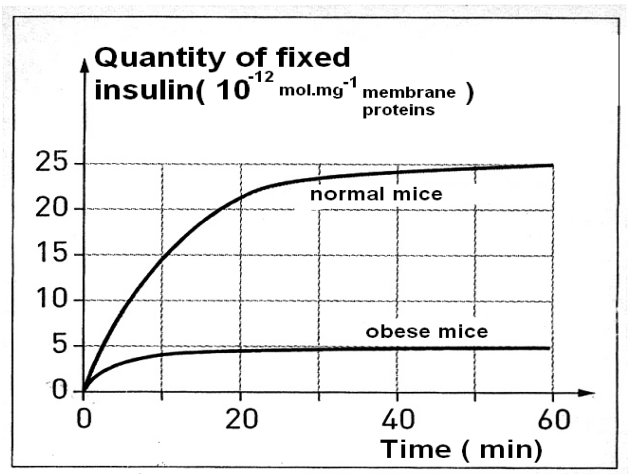
N.B: An artery brings blood to an organ and a vein takes blood away from an organ.

1-Interpret each of documents 1 and 2.

2- Use the acquired knowledge to indicate the document which corresponds to the activity of the liver. Justify the answer.

Experiment 2: We inject labeled glucagon by a radioactive isotope into a normal mouse. The autoradiography of the hepatic cells of this mouse reveals the fixation of glucagon at certain points of the plasma membrane. However, the injection of labeled insulin by a radioactive isotope leads to a similar autoradiography but with different points of fixation of insulin on the plasma membrane.

Experiment 3: Mutant mice show the following characteristics: obesity, chronic hyperglycemia and relative insensitivity to insulin injection that lowers glycemia very slightly.
We remove hepatic cells from normal and obese (mutant) mice and place them, separately, in a medium containing radioactive insulin. Then, we purify the plasma membranes of these cells and measure the quantity of fixed insulin at the level of these membranes. The results are shown in document 3.



Document 3

3-Analyze the results shown in document 3.

4- Based on the information provided by experiments 2 and 3 and the acquired knowledge, explain the origin of diabetes in the mutant mice.

Exercise 1 (5 pts)

1- Couple 1 and 2 who are normal have a child No.5 affected by the disease. This means that the allele causing the disease(m) is recessive masked by the dominant normal allele (N) found in the parents. **(½ pt).**

2- Father 1 is healthy and possesses the normal allele N on chromosome X. Thus, his genotype is X^NY **(½ pt).** Mother 2 is also normal and possesses the allele m on one of her chromosome X since she had a diseased boy. Thus, she is heterozygous with genotype X^NX^m **(1/2pt).**

3- The father produces two gametes of equal probabilities ½ X and ½ Y. The mother produces two gametes of equal probabilities ½ X^N and 1/2 X^m. All the girls would inherit an X^N from their father and would be normal Therefore, the probability to have affected girls is Zero. (¼ **pt**). The boys inherit Y chromosome from their father and either an X^N or X^m from their mother. This means that the probability to have affected boys is ½ or 50% from all boys or ¼ or 25% of all children. (¼ **pt). Or students can do a factorial analysis to provide an answer.**

4- The autoradiography of boy 5, who is affected (from pedigree)shows one band at the level of fragment 1.3Kb. Thus, this fragment corresponds to allele m.**(½ pt).**

5- The autoradiography of fetus 6 reveals two fragments, one fragment at the level of 1.3 KB which corresponds to the mutant allele and another fragment at the level of 1.8Kb which corresponds to the normal allele. This means that the fetus possesses 2 X chromosomes and would be a girl with normal phenotype since X^N dominates X^m. **(1 pt).**

6- **Document 3** reveals the absence of fluorescence in the non treated myopathic mice(group B) and its presence in both normal mice(group A) and diseased mice treated with dystrophin gene(group C). This means that dystrophin is absent in the affected non treated mice and present in the normal and the treated mice **(½ pt).**

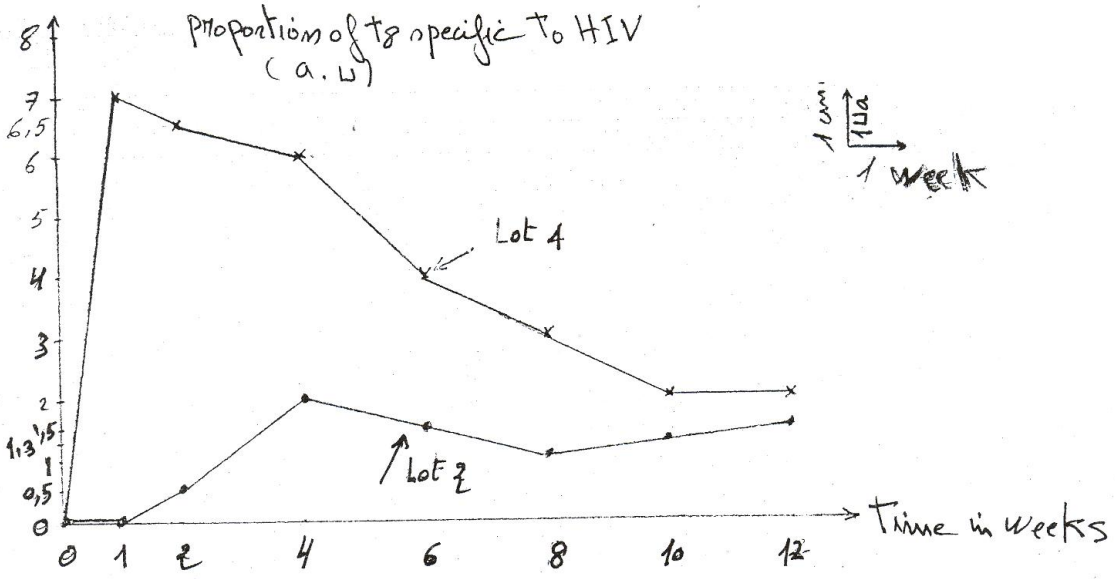
Document 4 shows that the % of survival in groups A and C is constant at 100% from week zero to week 37 . This % of survival remains constant at 100% in group A until week 50 , but decreases to about 75% in mice C. On the other hand, the % of survival in the diseased non treated mice which was 100%, similar to mice A and C at week 10, decreases sharply to reach Zero at week 20. **(½ pt)**This means that the gene treatment improves the survival of the myopathic mice **(½ pt)** . Therefore, treatment by the introduction of the dystrophin gene has allowed for the synthesis of dystrophin in the muscle cells of the diaphragm and has improved the survival of the myopathic mice and thus this treatment is efficient. **(½ pt).**

Exercise 2 (5 pts)

1- The results of lot 1 show that T4 and not T8 cells get infected by HIV. This means that T4 are the target cells of HIV. Moreover, results show that infection of T4

cells occurs in lot 1 and lot 2 where CD4 proteins are free, while no infection of T4 cells occurs in lot 3 where the CD4 proteins are fixed to antibodies. This means that HIV attacks only T4 cells possessing free CD4 on their membrane.

2- Graph showing the variation of the proportion of T8 specific to HIV in function of time in vaccinated and non vaccinated monkeys.**(2 pts)**



3- Document 2 reveals that in the vaccinated monkeys, the proportion of T8 specific to HIV start increasing from the time of viral exposure to reach 7 a. after one week while, in the non vaccinated monkeys, the proportion of T8 starts increasing after a longer period of time (two weeks) following infection. This explains why the immune response is rapid in the vaccinated monkeys. Also, document 2 shows that the proportion of T8 in the vaccinated monkeys reach a value 7 a.u which is higher than the value attained in the non vaccinated monkeys which is 2 a.u. so, the immune response in the vaccinated monkeys is amplified.
(1 pt)

4- In the non vaccinated monkeys, the viral charge is around 25.10^4copies/mm3 of plasma after 8 weeks following viral infection and increases to reach 50.10^4 copies in mm3 of plasma after 24 weeks. These values are always higher than the viral charge in the vaccinated monkeys which remains constant at 5.10^4 copies/mm3 of plasma at weeks 8 and 24. **(1 pt)**

Exercise 3 (5 points)

1- Document 1 reveals that the injection of GABA alone provokes hyperpolarization (Ipsp), of an amplitude ~ 5mv that deviate the membrane potential away from the threshold of depolarization. This means that the synapse is inhibitory. (1 pt)
2- Benzodiazepine enhances the action of GABA, while picrotoxine inhibits its activity, because the injection of GABA and benzodiazepine provokes hyperpolarization of a higher amplitude (~6 mv) compared to that produced after the injection of GABA alone.
picrotoxine increases the muscular contraction and the signs of anxiety, it favors the transmission of the nervous message and consequently it is excitatory. (1 pt)
3- Variation of the percentage of the fixation of GABA on its receptors as a function of the quantity of benzodiazepine injected in the synaptic cleft. (1 pt)

Quantity of benzodiazepine in the synapse (nanomoles)	1	10	100	1000
Fixation of GABA (% in presence of benzodiazepine)	0	120	140	145

4- The percentage of GABA fixation increase from 0 to 145% as the quantity of benzodiazepine increases from 1 to 1000 nanomoles. This indicates that the concentration of benzodiazepine favors the fixation of GABA and increase it's effect. (1 pt)
5- Document 3 that schematizes the structure of the postsynaptic receptor, reveals that the sites of fixation of GABA and benzodiazepine are close to each other and these sites are located on the same membrane structure: Cl^- channel. The presence of benzodiazepine decreases the quantity of GABA indispensible for the opening of Cl^- channel / or favors the opening of great number of Cl^- channels that increases the entering of Cl^- and consequently the inhibition of the nervous message, therefore the muscular contractions decrease.

Exercise 4 (5 pts)

1- Document 1 reveals that before glucose injection at time 15 min, glycemia in the venous blood (1 g/L) is higher than glycemia in the arterial blood (0,9 g/L). Thus, the organ releases more glucose than it stores.) which means that the organ liberate glucose. However, glucose injection causes an increase in glycemia in both blood vessels but this increase is higher in the artery which reachs a value 1.6 g/L after 45 min following injection compared to 1,5g/L in the venous blood. This means the organ stores glucose in case of hyper glycemia (1 pt).

Document 2 reveals that before and after glucose injection , glycemia is always slightly higher in the arterial blood (~ 1.01 g/L) than in the venous blood (~ o.99 g/L). This means the organ only stores glucose. (1 pt)

2- Document 1 corresponds to the liver activity, because it is the only organ capable of storing and releasing glucose. The liberation of glucose happens before the 15^{th} min and after the 105^{th} min where glycemia is low and storage takes place between 35^{th} min and 105^{th} min when glycemia is high. (1 pt)

3- In normal mice, the quantity of fixed insulin on the plasma membrane increases from 0 to reach 25.10^{-12} mol/mg of membrane proteins at 50 min. while, in obese mice the fixed quantity of insulin increases slightly to reach a maximum of 5. 10^{-12} mol/mg at 30 min. Beyond these values the fixed amount of insulin remains constant for the 2 mice but still higher in the normal mice than in obese mice.

4- Insulin can't have a hypoglycemic effect unless it is fixed on its specific receptors (exp. 2). In the obese mice, the quantity of insulin fixed on receptor is low (exp.3), this means that the origin of diabetes in obese mice is the low number of insulin receptors which reduce the hypoglycemic effect of insulin. Glucose will not be absorbed and stored by the hepatic cells, glucose remains in blood leading to chronic hyperglycemia. (1 pt)