وزارة التربية والتعليم العالي
المديرية العامة للتربية
دائرة الامتحانات

امتحانات الشهادة الثانوية العامة
فرع علوم الحياة

دورة سنة ٢٠٠٨ العادية

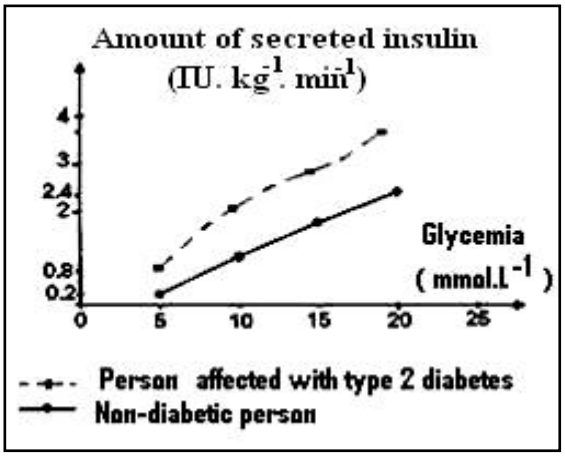
مسابقة في مادة "علوم الحياة"
المدة : ثلاث ساعات

الاسم :
الرقم :

Answer the following exercises.

Exercise 1 (5pts)

In order to determine the disturbances in glycemia regulation, and that are at the origin of type 2 diabetes (non-insulin dependent diabetes), experiments were carried out and summarized in the next documents.

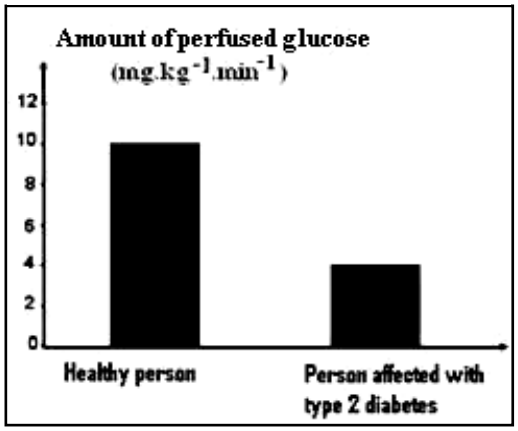


Document 1

1st experiment: A healthy person and another person recently affected with type 2 diabetes are perfused with glucose, in order to gradually increase their glycemia. Meanwhile, the amount of secreted insulin (a hypoglycemic hormone) is measured in both persons (document 1).

1- Interpret the obtained results.

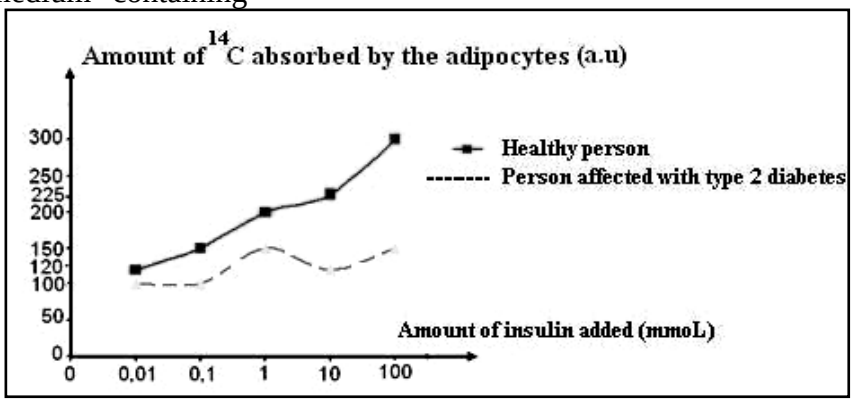
2nd experiment: A healthy person and another person affected with type 2 diabetes are perfused with a constant input of insulin, and at the same time with glucose, in order to maintain their glycemia at $1g.L^{-1}$. The amount of perfused glucose needed for each person is then measured. The results are shown in document 2.



Document 2

2- Compare the obtained results. What can be deduced regarding the efficiency of insulin?

3rd experiment: Adipose cells (adipocytes) are extracted from a healthy person and from another person affected with type 2 diabetes. These cells are cultured in a medium containing glucose labeled ^{14}C. Insulin is added to this medium and the amount of ^{14}C contained in the adipocytes is measured. The results are shown in document 3.



Document 3

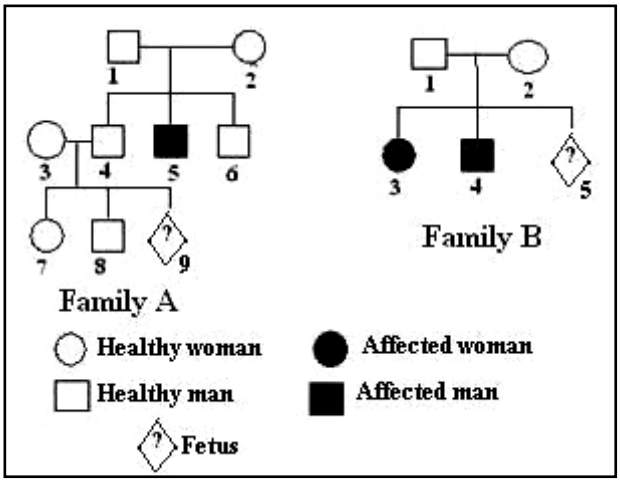
3- Represent, in a table, the variations of the amount of ^{14}C absorbed by the adipocytes versus the quantity of insulin added, in both persons.

4- Interpret the obtained results.

5- Based on what precedes, indicate the disturbances in glycemia regulation that led to type 2 diabetes.

Exercise 2 (5pts)

Phenylketonuria is a disease caused by a deficit in a hepatic enzyme – PAH – responsible for the transformation of an amino acid, phenylalanine, into another one called tyrosine. In Europe the risk of being heterozygous is 1/50.
Document 1 shows the pedigrees of two families A and B which some members are affected with this disease. Couples (3, 4) of family A and (1, 2) of family B ask for a prenatal diagnosis.



Document 1

1- Through a rigorous analysis of the pedigree of family B, determine:
- whether the allele responsible for the disease is dominant or recessive.
- the location of the gene responsible for the disease.

2- Determine the genetic risk for each fetus to be affected with this disease.

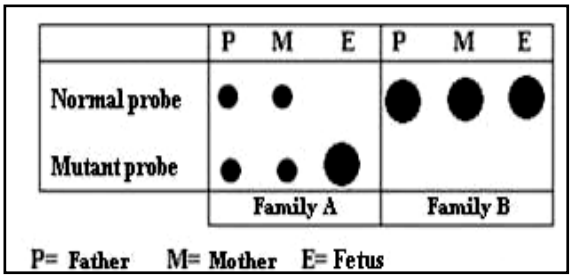
Three mutations were determined to be at the origin of phenylketonuria. Document 2 shows a part of the codon sequences that correspond to three regions X, Y, and Z of the normal allele, and of the three mutant alleles that are responsible for this disease.

Codon / RNA	278……...282…. (Region X)	310……..…….314…. (Region Y)	406…….……410 (Region Z)
Normal allele	**ACC CCC GAA CCU GAC...**	**UCU CUG GGU GCA CCU …**	**AUA CCU CGG CCC UUC**
Mutant 1	**ACC CCC AAA CCU GAC…**	**UCU CUG GGU GCA CCU…**	**AUA CCU CGG CCC UUC**
Mutant 2	**ACC CCC GAA CCU GAC...**	**UCU CCG GGU GCA CCU…**	**AUA CCU CGG CCC UUC**
Mutant 3	**ACC CCC GAA CCU GAC...**	**UCU CUG GGU GCA CCU …**	**AUA CCU UGG CCC UUC**

Document 2

3- For each allele responsible for the disease, locate the mutation and indicate its type.

In order to diagnose the fetuses, the following DNA tests were carried out in both families.
1st test: DNA is extracted from parental and fetal cells and is subjected to restriction enzymes. Hybridization technique is then carried out using two radioactive DNA probes that are complementary to a specific "region X". One of the probes is specific for the normal allele; the other is specific for a mutant allele. The results are shown in document 3.

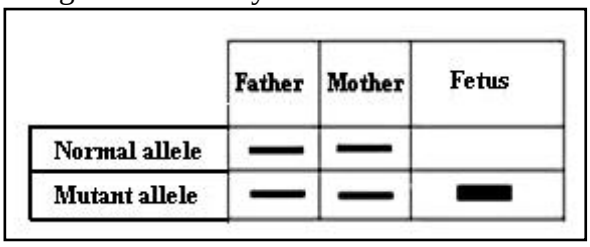


Document 3

4- Draw out the genotypes of the individuals of family A in document 3.

5- Justify that the test performed is not sufficient to establish the diagnosis of family B.

2nd test: Family B is subjected to a second DNA test yet using other restriction enzymes. This method reveals a restriction site (cleavage site) at the level of region Z, while regions X and Y remain intact. The results of this test are shown in document 4.



Document 4

6- Show the importance of the second test in order to obtain an exact diagnosis concerning the fetus of family B.

Exercise 3 (5pts)

In order to study one of the characteristics of the immune response, the experiment shown in document 1 were carried out.

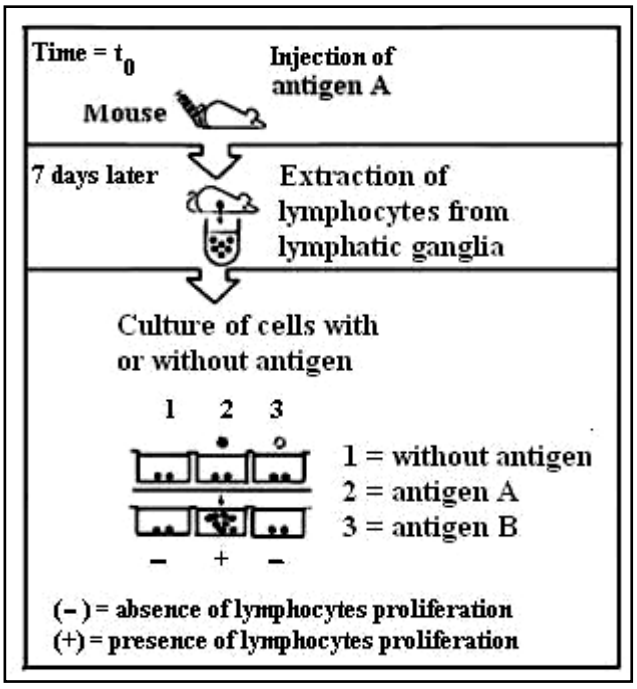
1- Write a short text describing the experiment carried out as well as the results obtained.

2- Interpret the obtained results. Draw out the characteristic of the studied immune response.

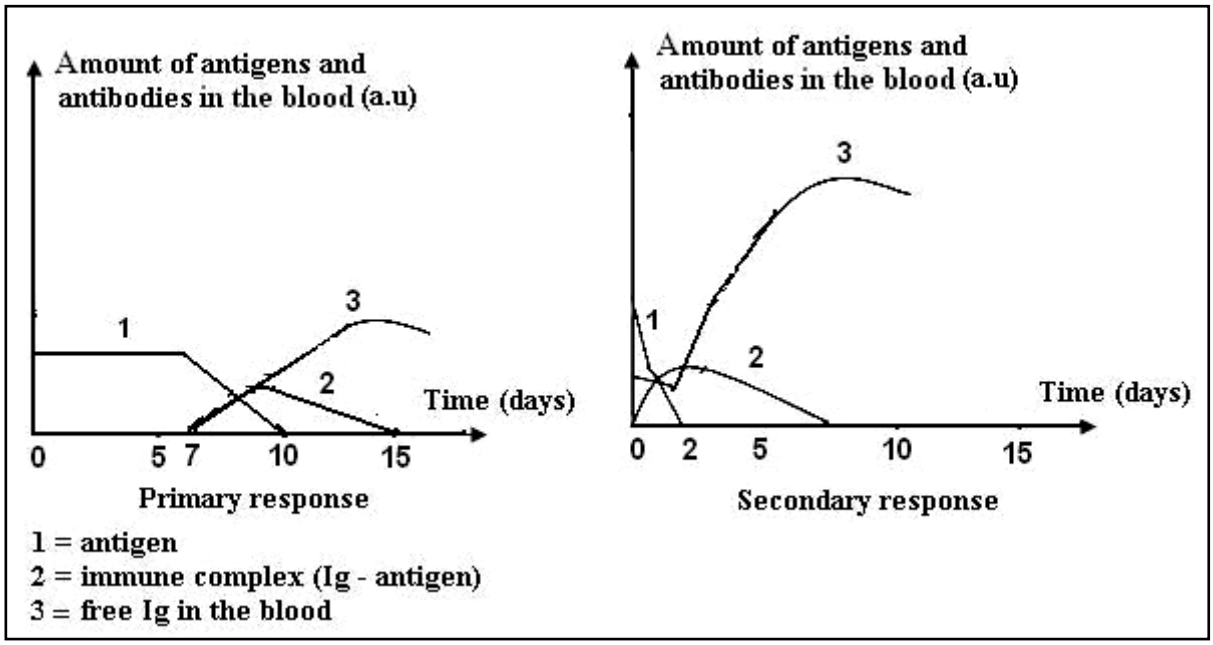
In a second experiment, the same steps are repeated without a seven days time delay. The cells of the lymphatic ganglia are directly extracted after the mouse immunization against antigen A. The results do not show any proliferation of lymphocytes.

3- Explain the necessity of the seven days time delay for the lymphocytes proliferation.

In a third experiment, we estimate the variations in the amounts of antigens and the produced antibodies (Ig) during two separate injections of the same antigen to an individual. The results are shown in document 2.



Document 1



Document 2

4- Compare the variations in the amounts of antigens then in the amounts of antibodies during both contacts. Deduce the characteristics of the immune memory.

5- Explain the appearance then the disappearance of the immune complexes following the antigen's injection.

Exercise 4 (5 pts)

In the framework of studying some aspects of the control mechanism of muscle activity during dancing, studies were carried out and summarized in document 1.

The movements of a dancer are performed in sequences, which are not always predictable, since each of these movements is triggered by an intention: the body is then used as a means of expression.
However, any body's movement is hindered by a force – gravity – which attracts it towards the ground. To control body movement and to reach equilibrium, the dancer uses muscles that block some joint movements and prevent falling down. The posture is thus maintained thanks to a constant adjustment of the muscle: for instance, every time a muscle is stretched, it contracts.

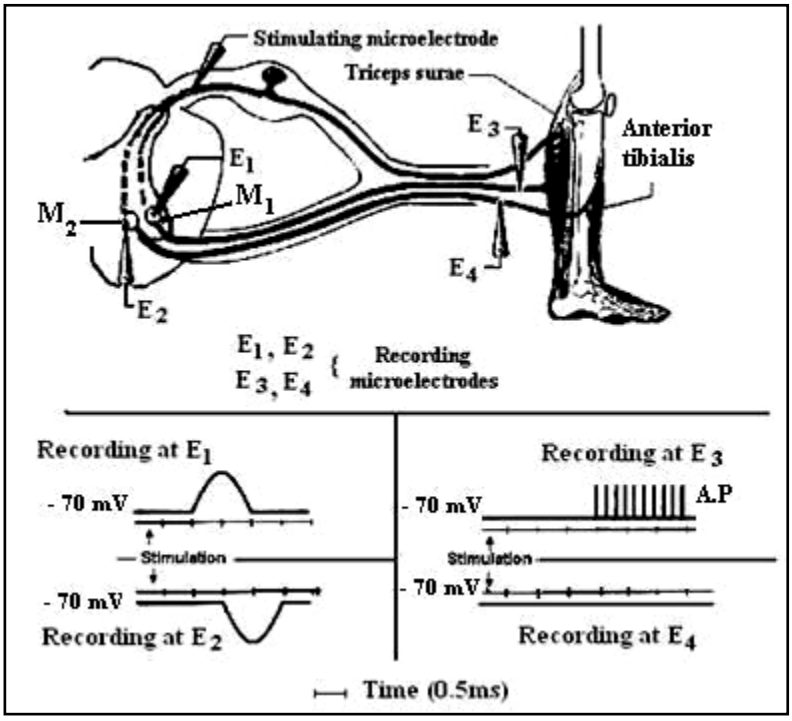
Document 1

1- Pick up from document 1 a statement that justifies the presence of a myotatic reflex, and another one that justifies the presence of a voluntary motor activity.

2- Specify the nerve center responsible for each of these activities.

In order to understand the functioning of the neurons' circuits implied in maintaining posture during dancing, and to know how muscles interfere in maintaining the body's equilibrium the following experiments are performed.

1st experiment: A nerve fiber issued from a neuromuscular spindle of an extensor, the triceps surae is stimulated. This stimulation leads to modifications of the electric status of two motor neurons, M_1 and M_2, located at the level of the grey substance of the spinal cord. One of these motor neurons innervates the extensor while the other innervates the flexor: the anterior tibialis. Document 2 reveals the experimental set up and the results of the recordings.



Document 2

3- Analyze the obtained recordings, and then draw out the effect of the activity of the motor neurons on the concerned muscles.

4- Referring to the recordings E_1 and E_2, determine the number of synapses implied in each of the concerned neurons' circuits knowing that the transmission of a nerve message at the level of a synapse needs 0.5 ms.

2nd experiment: Experiment 1 is repeated and at the same time we stimulate a nerve fiber issued from the superior nerve centers, related to motor neuron M_2 that is linked to anterior tibialis. Many action potentials were recorded at E_4 and no recording was obtained at E_3.

5- Based on the obtained results, specify the effect of this stimulation on both muscles. Justify the answer.

الاسم :
الرقم :

مسابقة في مادة "علوم الحياة"
أسس التصحيح

Exercise 1 (5pts)

1- For a glycemia of 5mmol.L^{-1}, insulin secretion is higher in the diabetic person (0.8 IU $kg^{-1}.min^{-1}$) than in the healthy one (0.2 IU.$kg^{-1}.min^{-1}$). This secretion increases in both persons with the increase in glycemia, however, it is kept higher in the diabetic person, 3.5 IU.$kg^{-1}.min^{-1}$ to 2 IU.$kg^{-1}.min^{-1}$ for a glycemia of 20 mmol. L^{-1}. This implies that insulin is secreted in high amount in both persons and it increases with the increase in glycemia however it is higher in the person recently affected with type 2 diabetes than in the healthy person. **(1pt)**

2- In order to maintain a glycemia of 1g.L^{-1} by the constant input of insulin, the amount of glucose perfused into the healthy person (10 mg.$kg^{-1}.min^{-1}$), is higher than that perfused into the person affected with type 2 diabetes (4 mg.$kg^{-1}.min^{-1}$). This means that insulin considerably decreased glycemia in the healthy individual, and slightly decreased it in the affected person. Therefore, insulin is more efficient in the healthy person than in the affected oneperson. **(1pt)**

3- (1.5 pt)

Quantity of insulin added (mmol)		**0.01**	**0.1**	**1**	**10**	**100**
Quantity of ^{14}C absorbed by the adipocytes (a.u)	**Healthy person**	**120**	**150**	**200**	**225**	**300**
	Person affected with type 2 diabetes	**100**	**100**	**150**	**120**	**150**

Table showing the amount of ^{14}C absorbed by the adipocytes versus the quantity of insulin added.

4- The quantity of ^{14}C absorbed by the adipocytes of a healthy person increases from 120 a.u to 300 a.u when the quantity of insulin added increases from 0.01 mmol to 100 mmol. On the contrary, the quantity of ^{14}C absorbed by a person affected with type 2 diabetes slightly increases and fluctuates between 100 and 150 a.u. This means that insulin favors the absorption of glucose by the adipocytes but this absorption is higher in a non-diabetic person than in a diabetic one. **(1pt)**

5- A person recently affected with type 2 diabetes has a high insulin secretion, but this insulin becomes ineffective on the target cells. Adipose cells store less the excess of glucose. This latter remains in the blood, glycemia is not regulated anymore, it becomes higher and type 2 diabetes is exhibited. **(0.5pt)**

Exercise 2

1- The pedigree of family B reveals that normal parents have a daughter and a boy both affected. This means that the allele responsible for the disease is recessive **(0.25pt)**.
The allele is not transmitted by sex chromosomes because if it was Y-linked on the non-homologous segment of Y the daughters could not be affected, while the father would be; this is not the case **(0.25pt)**..
If the allele was X-linked on the non-homologous segment of X, the daughter would have inherited from the father the X chromosome carrying the allele responsible for the disease; this is not the case. **(0.25pt)**.
If it was linked on the homologous segment of X and Y, then the father should have been affected in order to give an X and a Y, both carrying the affected allele, to his daughter and son respectively; this is not the case. **(0.25pt)**.
Therefore, the allele responsible for this disease is autosomal. **(0.25pt)**

2- The risk for family A: Mother 3 is healthy with no family history of phenylketonuria, then the probability to be heterozygous is 1/50 and in this case, half of the gametes carry the mutant allele. Father 4 is healthy but has an affected brother, then the probability to be healthy and heterozygous is 2/3 and to be healthy homozygous is1/3. If the father is healthy homozygous, the risk is nil since he can only transmit the normal allele to his descendance. However, if he is healthy and heterozygous, half of his gametes carry the mutant allele. Then the risk will be:
2/3 x1/2x1/2 x1/50= 1/300. **(0.5pt)**
The risk of family B: Parents are necessarily heterozygous, then the half of the gametes carry the allele of the disease and the probability of having an affected child is ¼. Then the risk is ¼.

3- Mutant allele 1: Mutation at the level of region X of the gene; 1st nucleotide of codon 280 where G is replaced by A. The nature of this mutation is substitution.
Mutant allele 2: Region Y of the gene; 2nd nucleotide of codon 311 where T is replaced by C. Mutation by substitution.
Mutant allele 3: Region Z of the gene; 1st nucleotide of codon 408 where C is replaced by T. Mutation by substitution. **(1pt)**

4- In family A, the parents carry a normal allele and an allele that has a mutation at the level of region X; they are heterozygous. The fetus has a mutation at the level of region X on both alleles. Therefore, the fetus will be homozygous and affected. **0. 5pt)**

5- Test 1 shows that the individuals of family B are all normal and homozygous. However, the pedigree shows that the parents are normal and heterozygous. Moreover, this test was performed only at the level of region X, while the mutation can affect regions Y or Z. **(0.5pt)**

6- The 2nd test allows detecting the presence of a morbid allele in family B at the level of region Z. If it was only referred to the 1st test, the diagnosis of the fetus would have been “healthy” which is not the case. **(0.5pt)**

Exercise 3 (5pts)

1- Antigen A is injected into a mouse. 7 days later, cells of the lymphatic ganglia are extracted and put in 3 culture mediums: without antigens in medium (1), with antigen A in medium (2), and with antigen B in medium (3). We observe the absence of lymphocytes proliferation in the 1st and 3rd culture mediums and the proliferation of lymphocytes occurs in the second medium. **(1pt)**

2- A high proliferation of lymphocytes extracted from the mouse immunized against antigen A was observed when they are put in culture with this antigen. On the contrary, no proliferation was observed when they are alone or in contact with antigen B. This implies that the proliferation of the lymphocytes, selected after the first contact with antigen A, cannot occur unless the lymphocytes are put again in contact with the same antigen. Thus the immune response is specific. **(1pt)**

3- "7 days time delay" is necessary to induce the immune response. Macrophages phagocyte the antigens and become APC that migrate towards the lymphatic ganglia. APC bind to the lymphocytes via their specific receptors and activate them. These selected lymphocytes rapidly multiply and proliferate upon a second contact with the same antigen. **(1pt)**

4- During the first contact with the injected antigen (primary response), the antigen's amount in the blood decreases starting day 7, to disappear within 10 days. However, during the secondary response, the antigen's amount in the blood decreases and disappears after 2 days, more quickly than at the time of the primary response.
At the time of 1st contact with the injected antigen, the amounts of circulating antibodies in the blood are null and does not appear until the 7th day. They increase to reach a maximum at day 13. On the contrary, during the 2nd contact, these antibodies are present as of day 0, they start to increase at day 2, and reach a maximum at day 7, greater and faster than in the 1st contact. Beyond this day, the amount of antibodies in both cases decreases however remains higher in the 2nd contact.
This shows that during the 2nd contact the antibodies are produced earlier and in greater amount and the elimination of the antigens is faster. Thus the immune memory favors a faster, stronger, and more lasting response. **(1.5pt)**

5- The appearance of immune complexes is due to the neutralization of the antigen by the antibodies secreted by plasmocytes. The disappearance of these complexes is due to the opsonisation and phagocytosis carried out by macrophages. **(0.5pt)**

Exercise 4 (5pts)

1- Myotatic reflex: Each time the muscle is stretched it contracts.
Voluntary motor activity: Certain movements follow an intention. **(0.5pt)**

2- Myotatic reflex: The nerve center is the spinal cord.
Voluntary motor activity: The nerve center is the cerebrum. **(0.5pt)**

3- The stimulation of a nerve fiber issued from the neuromuscular spindle of the triceps surae allowed the recording of a hypopolarization at E_1 placed at the level of motor neuron M_1. This excitatory message leads to the appearance of many AP recorded at E_3 placed at the level of the efferent fiber of this motor neuron linked to the extensor (triceps surae). On the contrary, at the level of E_2, placed at the level of motor neuron M_2, a hyperpolarization is recorded and no recording was obtained on E_4, placed at the level of the efferent fiber linked to the anterior tibialis. Since M_1 sent an excitatory nerve message to the triceps surae, this latter contracted; since M_2 did not send any message to the anterior tibialis, it then remained relaxed. Therefore, the variable activity of the two motor neurons leads to the contraction of the extensor and the relaxation of the flexor. **(2pt)**

4- The nerve message transmitted by the afferent nerve fiber took approximately 0.7ms to reach motor neuron M_1. Since the time needed to cross a synapse is 0.5ms, then one synapse exists along this pathway. The neuron circuit of M_1 is monosynaptic.
The nerve message took 1.2ms to reach M_2. This delay noticed at the level of M_2 versus M_1 is equivalent to the time needed to cross an additional synapse. Therefore, on pathway M_2 we have two synapses. The neuron's circuit of M_2 is then polysynaptic. **(1pt)**

5- Anterior tibialis contracts while triceps surae relaxes. The message coming from the superior nerve centers inhibited the nerve message arriving to the triceps surae, since no recording was noticed on E_3, however a nerve message at E_4, located at the level of the efferent fiber linked to the anterior tibialis, was observed. **(1pt)**