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These files have been meticulously arranged by the
'Together We Can' team,
as we wish you the best of luck on your academic journey,
filled with happiness and success

Join us in creating a better tomorrow,
hand in hand!



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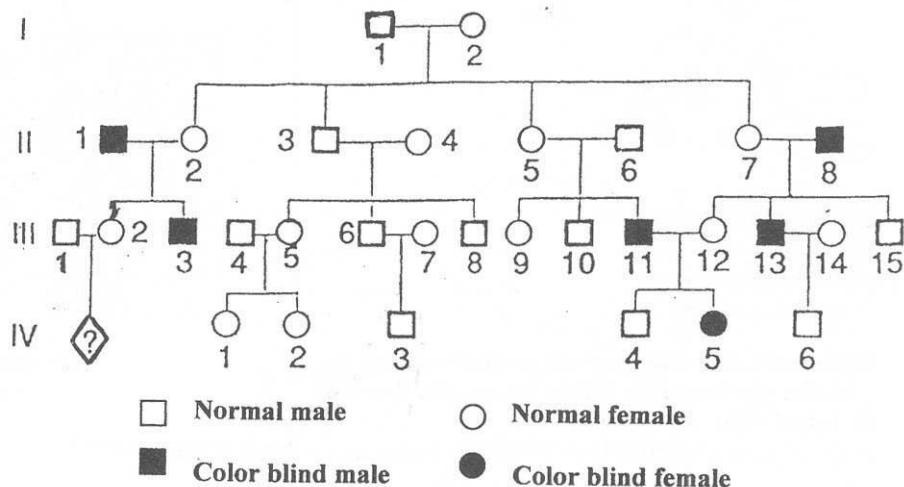
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الاسم : مسابقة في علوم الحياة
الرقم : المدة : ثلاثة ساعات

Answer the following questions.**Question I (6 pts)**

A- Color blindness (daltonism) is a defect in vision of colors. This defect is due to a gene localized on the non-homologous segment of chromosome X. Document 1 represents the pedigree of a family, whose certain members are affected.

a- Is the allele responsible for color blindness dominant or recessive ? Justify the answer.

**Document 1**

b- Write the genotypes of the individual (III-2), her parents, and her husband. Justify the answer for each genotype.

c- Individual (III-2) is expecting a child and is worried if her child will be color blinded.

Make a table of the cross to find the probability of the child to be color blinded.

B- In this family, medical analyses are performed to individual (III-8), who presents physical and sexual troubles. Equally, medical analyses were done to his parents, his sister (III-5) and his brother (III-6).

Among the medical analyses done, was testing for glucose-6-phosphate-dehydrogenase (G6PD), an enzyme whose synthesis depends only on a gene localized on the non-homologous segment of the X chromosome. On this gene locus two alleles may be found; allele A or allele B, that code for the synthesis of G6PD of form A and G6PD of form B respectively. We can distinguish the two forms by electrophoresis.

Document 2 shows the electrophoregrams

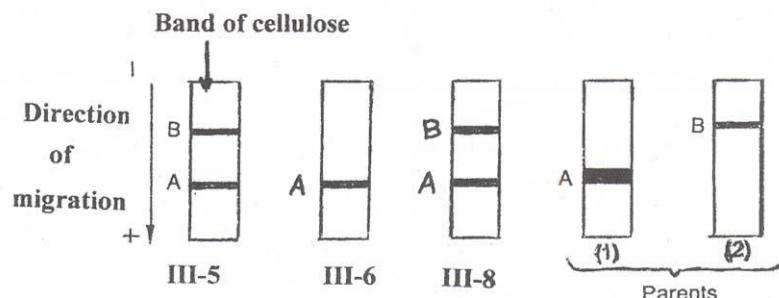
obtained for individuals III-5, III-6, and III-8, and for their parents.

d- Compare the electrophoregrams of the two individuals III-5 and III-6.

e- Indicate, to which of the two parents of the individual III-8, the electrophoregrams 1 and 2 correspond? Justify the answer.

f- Determine the possible disorder that caused the troubles in individual III-8. Which one of his parents is responsible for that? Justify the answer. Name the phase of meiosis during which the disorder took place.

A study was done on the **male descendants** originated from women, whose fathers are color blinded and have a G6PD of form B. These women are of normal color vision like individual III-2 but their

**Document 2**

electrophoregram is like that of individual III-5. The husbands of these women are of normal vision and have a G6PD of form A.

g- Specify the genotype of these women and the genotype of their husbands.

The descendants produced are the following.

75 Males with normal color vision and G6PD of form A.

71 Males with color blindness and G6PD of form B.

4 Males with normal color vision and G6PD of form B.

4 Males with color blindness and G6PD of form A.

h- How can you explain the obtained results? (a table of cross is not required).

i- Calculate the distance between the gene of color blindness and that of G6PD.

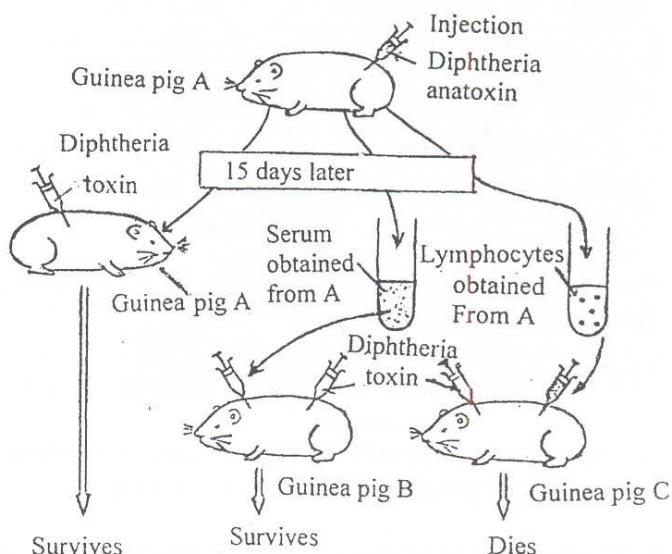
Question II (3 ½ pts)

Diphtheria is a fatal disease due to the action of diphtheria toxin, which spreads through out the body. This toxin is liberated by a bacillus that remains in the throat.

In order to understand the consequences of the injection of diphtheria anatoxin (attenuated toxin, non-virulent), we performed several experiments on guinea pigs. The results are represented in the adjacent document.

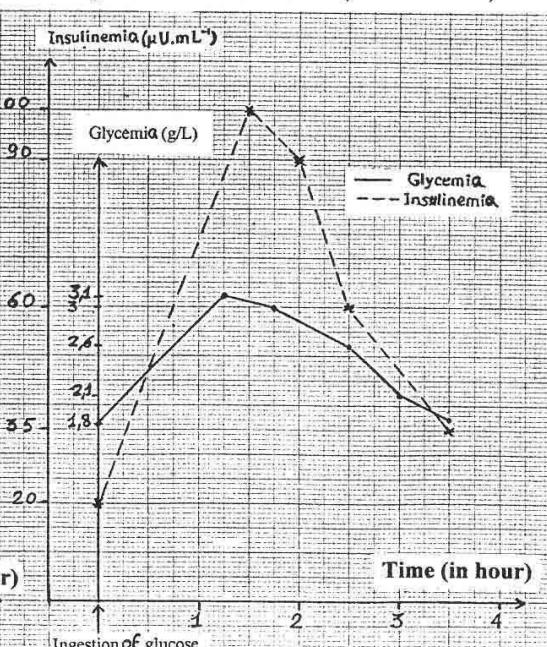
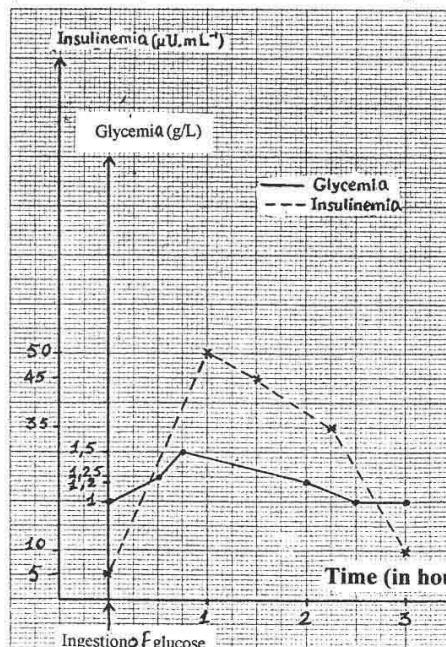
a- Describe, in a few lines, each experiment shown in this document.

b- Interpret each of these experiments. What can you deduce concerning the nature of the immune response?



Question III (5 pts)

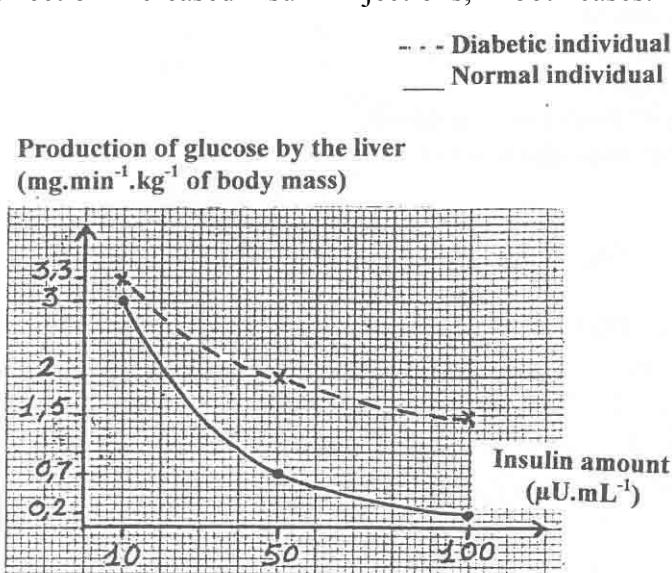
A- A test of provoked hyperglycemia was performed on a normal individual and on a diabetic one. We measure the amount of plasma glucose (glycemia) and the amount of insulin secreted by the pancreas (insulinemia) of a normal individual (document 1a) and of a diabetic one (document 1b).



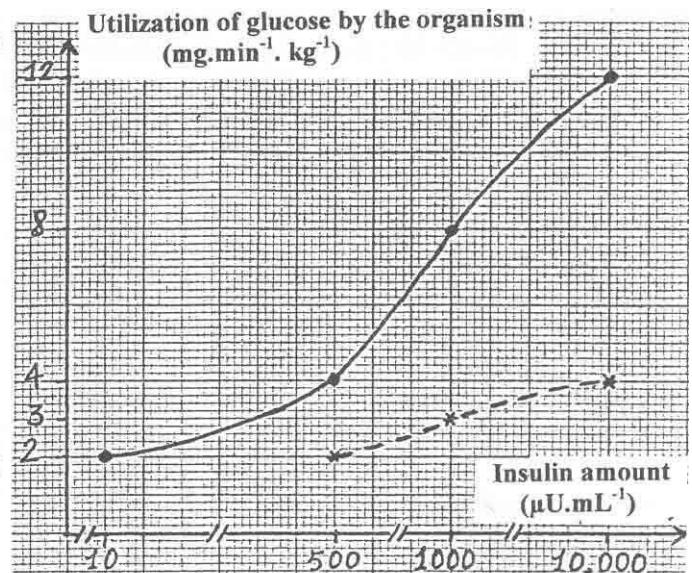
a- Compare the variation of glycemia in the two individuals, then the variation of insulinemia. What can you deduce?

b- Suppose that the molecules of insulin of these two individuals are normal, formulate a hypothesis about the origin of this diabetes.

To understand the results of the provoked hyperglycemia test of document 1, we measure, the release of glucose by the liver (document 2a) and its utilization by the cells of an organism (document 2b), under the effect of increased insulin injections, in both cases.



Document 2a.



Document 2b.

c- Interpret the obtained results. What can you deduce concerning the mode of action of insulin in the regulation of glycemia in the two individuals?

B- The seriousness of the problems of diabetes varies from one patient to another or, in one patient during the different stages of the disease.

Certain studies compare patients at different stages of the disease S₁, S₂, and S₃ with that of a control individual. For this purpose, we measure during a provoked hyperglycemia test, the insulinemia, the glycemia and the utilization of glucose by the organs in these individuals. The results are represented in document 3.

	Control	S ₁	S ₂	S ₃
Insulinemia (in μU·mL⁻¹)	60	100	150	50
Utilization of glucose (in mg·m²·min⁻¹)	300	175	125	100
Average glycemia (in g·L⁻¹)	1.2	1.3	1.5	3.5

Document 3

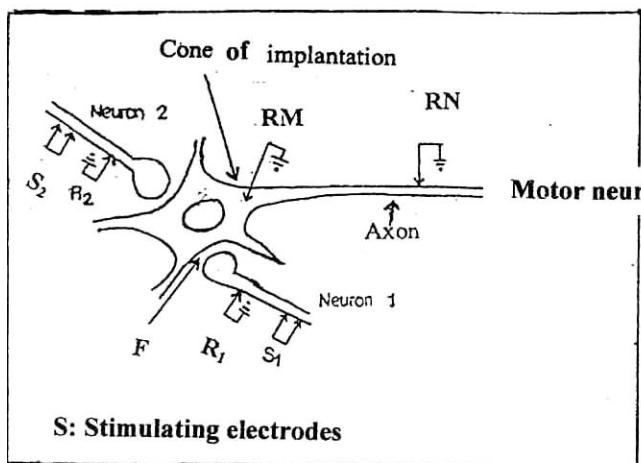
d- Construct two graphs one representing the variation of insulinemia and the utilization of glucose in function of the control and the different stages, and another graph representing the variation of glycemia in function of the control and the different stages.

(Consider the interval between the control and S₁, S₁ and S₂, S₂ and S₃ is 1.5 cm.)

e- Analyze the results of document 3. What can you deduce concerning the activity of the pancreas during each of the two stages S₂ and S₃?

Question IV (5 ½ pts)

In the framework of studying the transmission of a nervous message, we perform the experimental set-up represented in document 1. The axon terminal of neuron 2 has vesicles that contain acetylcholine. The axon terminal of neuron 1 has vesicles that contain GABA. If we effectively stimulate neuron 2 at S₂, we obtain the recordings presented in document 2.



S: Stimulating electrodes

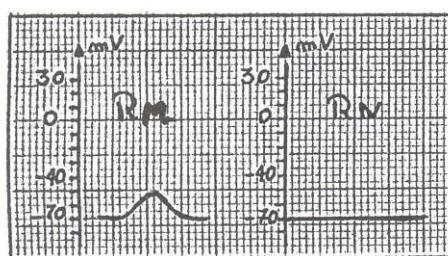
R: Internal receptor
Microelectrode

Document 1. Experimental set-up performed at the level of a motor-neuron (M) in the anterior horn of the spinal cord of a mammal.

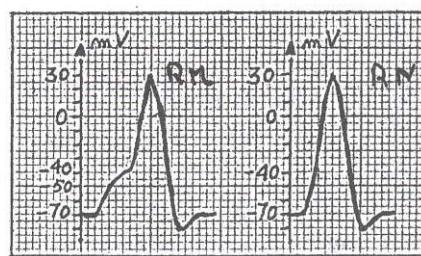
a- What can you conclude after the analysis of these recordings?

We stimulate at S_1 and S_2 simultaneously; we obtain the recordings of document 3.

When we stimulate simultaneously at S_1 and S_2 , followed immediately by the stimulation at S_2 , we obtain the recordings of document 4.



Document 3



Document 4

b- Explain the obtained results in documents 3 and 4 in order to specify the property of the motor neuron revealed by these results.

With the interest of specifying the effect of the drug, barbiturate, on the chemical synapse involving GABA, we perform the following experiments:

1st experiment: we inject into F, at the same time, GABA and barbiturates. The recorded activity at RM reveals a hyperpolarization, which is more significant than when GABA was injected alone.

2nd experiment: if radioactive barbiturates were used in experiment 1, we notice, after the injection, that radioactivity is found to be localized at the level of the postsynaptic membrane.

c- Based on the results of the above two experiments, find out the role of barbiturates in this synapse and at what level of the synapse barbiturates act. Justify the answer.

d- There exists, in the central nervous system, numerous neuro-neuronic synapses where GABA is involved (document 1). By referring to the acquired knowledge, explain the effect of barbiturates on the activity of the muscles of an organism.

Question I (6 pts)

A-

- a- The allele responsible for the abnormality is recessive. The couple II-5 and II-6 are normal and have a boy III-11 who is color blinded. This indicates that the allele (d) is masked in the mother and transmits it to her son III-11. Consider (N) to be the normal allele and (d) the allele for color blindness. (pt)

- b- The genotype of III-2: $X^N X^d$, heterozygote. She receives X^d from her father and X^N from her mother.

The genotype of the mother: $X^N X^d$ since her son III-3 is color blinded.

Since the phenotype of the male is revealed by his genotype:

The genotype of the father: $X^d Y$ since he is color blinded.

The genotype of her husband: $X^N Y$ since he is normal. (pt)

- c- P $X^N Y \times X^N X^d$
γ P: $\frac{1}{2} X^N \frac{1}{2} Y \quad \frac{1}{2} X^N \frac{1}{2} X^d$

Children: $X^N X^N \frac{1}{4}$, $X^N X^d \frac{1}{4}$, $X^N Y \frac{1}{4}$, $X^d Y \frac{1}{4}$.

The probability is $\frac{1}{4}$ of the descendants or is $\frac{1}{2}$ of the boys. (pt)

B-

- d- The electrophoregram of III-5 presents two distinct bands of form A and B, thus he possesses the allele A and the allele B, on the contrary that of III-6 presents only one band A and has allele A.

- e- The electrophoregram 1 corresponds to the mother because she possesses one thick band that corresponds to two alleles A carried by two chromosomes X. The electrophoregram 2 corresponds to the father since he has one band B that corresponds to the allele B carried only by the X chromosome of the father. ($\frac{1}{2}$ pt)

- f- The electrophoregram of III-8 reveals the presence of two alleles A and B which indicates that he possesses two bands that correspond to two alleles A and B. This indicates that he possesses two X chromosomes, thus, trisomy XXY. The father is at the origin of the trouble in III-8 since his mother can only give him X^A , thus his father, who is supposed to give him only chromosome Y, has given him also the chromosome X^B which he carries. The abnormality is produced during anaphase I of meiosis.

- g- Genotype of women that are color blinded like III-2: $X^{dB} X^{NA}$

Genotype of husbands: $X^{NA} Y$ (pt)

- h- Since the gene of color blindness and of the G6PD are localized on chromosome X, then the male descendants should be distributed into two phenotypes instead of four. Therefore, the four obtained phenotypes originate from 4 types of female gametes as a result of crossing over during meiosis. (pt)

- i- Frequency of crossing over = $\frac{(4+4) \times 100}{75 + 71 + 4 + 4} = 5.2\%$

The frequency of crossing over indicates that the distance between the studied genes is 5.2 CM. (pt)

Question II (3 ½ pts)

- a- We inject diphtheria anatoxin into guinea pig A. 15 days later:
- We inject into guinea pig A diphtheria toxin, it dies. (pt)
 - We obtain serum from guinea pig A and we inject it into guinea pig B then we inject diphtheria toxin, it survives. (pt)
 - We obtain lymphocytes from guinea pig A and we inject it into guinea pig C then we inject the diphtheria toxin, it dies. (pt)

b- The injection of diphtheria toxin into guinea pig A after 15 days from being injected with diphtheria anatoxin, does not provoke its death. This implies that diphtheria anatoxin provided immunity to this guinea pig against this toxin. (pt).

The death of guinea pig C, who received lymphocytes from guinea pig A together with diphtheria toxin, shows that the lymphocytes of guinea pig A did not ensure protection against this toxin. (pt)

On the contrary, the survival of guinea pig B, after the injection of the serum of guinea pig A and with diphtheria toxin shows that this serum contains the immune elements, which ensure protection against this toxin. (pt)

Therefore, the immune response is humoral mediated. (pt)

Question III (5 pts)

A-

- a- The ingestion of glucose by a normal individual is followed by an increase of glycemia from 1 to 1.5 g/L after 45 minutes, while in diabetic individuals, glycemia, was very high at the beginning 1.8 g/L, increases until 3.1 g/L with a time difference of 30 minutes.

Similarly, the ingestion of glucose by a normal individual leads to an increase of insulinemia from 5 to 50 $\mu\text{U.L}^{-1}$ after 1 hour while in the diabetic, insulinemia, which was very high at the beginning 20 $\mu\text{U.L}^{-1}$ increases to 100 $\mu\text{U.L}^{-1}$ with a time difference of 30 minutes. Eventually, glycemia and insulinemia return to their initial values after 3 hours in the normal individual but very slowly in the diabetic 3 ½ hours.

The comparison of the variation of glycemia and insulinemia in the normal and the diabetic individuals shows that the pancreas responds to hyperglycemia by a secretion that lowers glycemia. This secretion is more abundant and is of low effect in a diabetic. Hence insulin is slightly effective in diabetics. (pt)

- b- Hypothesis: The receptors of the target cells are abnormal.

Or

The receptors of the target cells are not numerous. (pt)

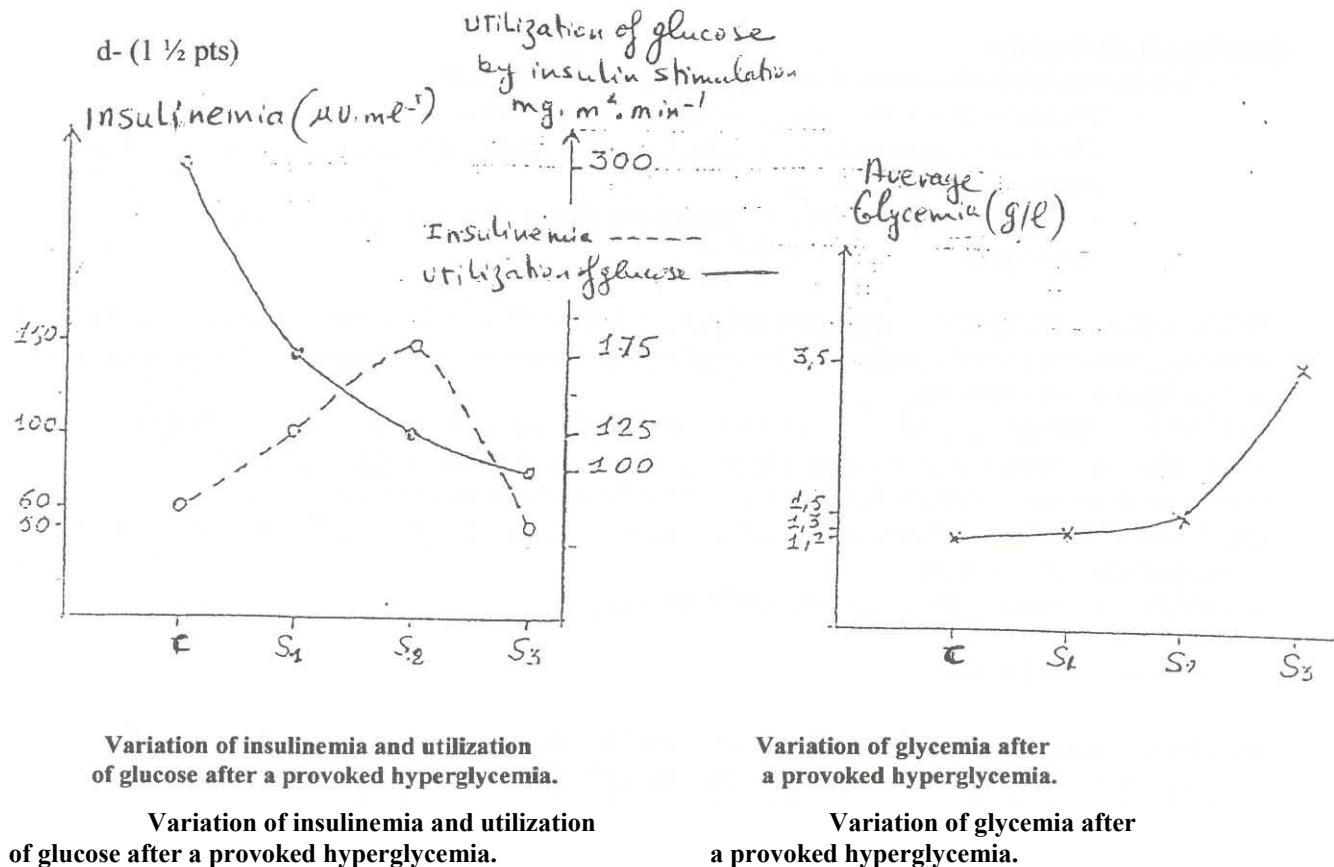
- c- In the normal individual, the production of glucose by the liver cells decreases very rapidly from $3 \text{ mg} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ when insulin increases from 10 to 100 $\mu\text{U.mL}^{-1}$. Similarly, the production decreases in diabetics from 3 to $15 \text{ mg} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ but very low compared to the normal. Hence, insulin acts by decreasing the production of glucose, but the liver cells of the diabetic are less sensitive to the action of insulin. In the normal individual, the use of glucose by the body cells increases from 2 to $12 \text{ mg} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$; as insulin increases from 10 to 10,000 $\mu\text{U.mL}^{-1}$.

In the diabetic individual, the utilization of glucose by the cells, begins to be observed when the concentration of insulin is 500 $\mu\text{U.mL}^{-1}$ and increases slightly from 2 to $4 \text{ mg} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ when insulin increases from 500 to 10,000 $\mu\text{U.mL}^{-1}$.

This shows that insulin acts by increasing the utilization of glucose by the cells of the body while the cells of a diabetic is less sensitive to its action.

Hence, insulin can decrease glycemia by inhibiting the production of glucose by the cells of the liver and accelerates its utilization by the cells of the body. But in a diabetic it is less effective. (0)

d-



B-

e- As we pass from the normal state to the second stage of the disease, we observe an increase in insulinemia from 60 to $150 \mu\text{U} \cdot \text{mL}^{-1}$. and a slight increase of glycemia from 1.2 to $1.5 \text{ g} \cdot \text{L}^{-1}$ on the contrary, there is a decrease in the utilization of glucose from 300 to $125 \text{ mg} \cdot \text{m}^2 \cdot \text{min}^{-1}$.

At the third stage, we observe a strong increase of glycemia that reaches $3.5 \text{ g} \cdot \text{L}^{-1}$. On the contrary, there is a strong decrease of insulinemia that reaches $50 \mu\text{U} \cdot \text{mL}^{-1}$ and a decrease of glucose utilization of $100 \text{ mg} \cdot \text{m}^2 \cdot \text{min}^{-1}$. (pt)

This shows that the pancreas has an elevated activity during the first two stages of the disease to compensate the weak utilization of glucose and the maintenance of the constant amount of glycemia.

Stage S_3 , the activity of the pancreas decreases that provokes an increased glycemia. Thus, the pancreas is hyperactive during stage S_2 and decreases its activity at stage S_3 . (pts)

Question IV (5 ½ pts)

- a- The stimulation at S_2 of neuron 2 produces an AP of 100mv amplitude recorded at R_2 and propagates through the synapse to reach the motoneuron M where an AP of 100mv is recorded at R_N . Since the synapse (Neuron 2- M) has provoked an AP, we can say that it is an excitatory synapse whose neurotransmitter is acetylcholine. ()
- b- When we simultaneously stimulate S_1 and S_2 , we observe an EPSP at R_M of low amplitude and nothing at R_N . This signifies that the synapse (neuron 1-M) is inhibitory and produces an IPSP. This IPSP generated by S_1 and the EPSP generated by S_2 are added, a spatial summation and give an EPSP having low amplitude which is incapable to generate an AP that can propagate to the motor neuron. When we stimulate S_1 and S_2 then S_2 again, we observe a spatial summation for (S_1 and S_2) then temporal summation (S_2, S_2) this will increase the EPSP enough to trigger an AP that can propagate. Since the neuron is capable to make a summation of the PSP, this indicates that the property of the motor neuron is integration. ()

- c- Experiment 1 shows that barbiturates promotes the action of GABA, we observe an increased hyperpolarization upon injecting GABA together with barbiturates.
Experiment 2, barbiturates is found on the postsynaptic membrane. This indicates that barbiturates act at the level of the postsynaptic membrane. ()
- d- The GABA produces an IPSP at the level of the postsynaptic membrane, preventing the formation of AP in the motor neuron. These AP are responsible for motor functions by provoking the contraction of the muscles. The GABA prevents this motor function. The barbiturates, which promotes the effect of GABA, favoring muscular relaxation (relaxant, tranquilizers).
()

الاسم :
الرقم :مسابقة في "علوم الحياة"
المدة : ثلاثة ساعات**Answer the following questions****Question I (5 ½ pts)**

Document 1 represents the pedigree of a family with some of its members, shown in black, having a rare hereditary disease that occurs mainly in males and very rarely in females.

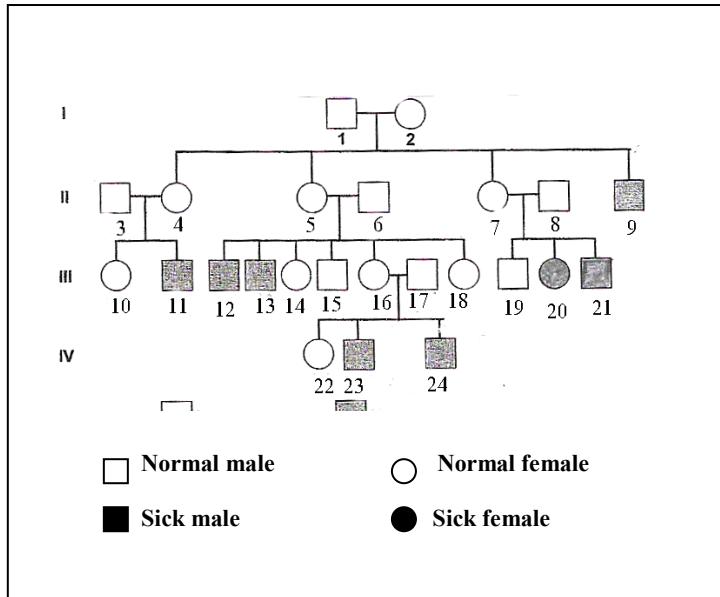
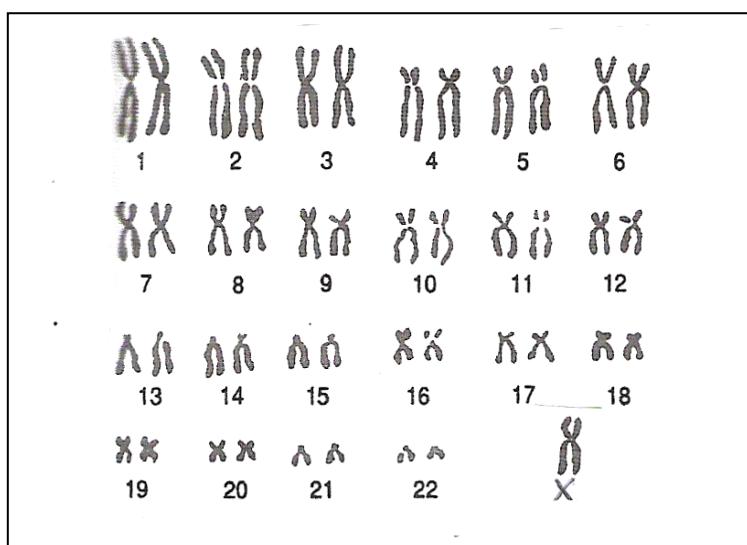
- a- Is the allele responsible for the disease dominant or recessive? Justify the answer.
 b- Discuss logically the chromosomal localization of the gene responsible for this disease (**without considering female 20**).

- c- Illustrate, chromosomally, the genotype of each of the individuals 13 and 16. Justify the answer.

Female 20 presents, besides her disease, an abnormality, which is manifested by the absence of menstruation, absence of the development of mammary glands... To identify this abnormality we perform the karyotype of female 20, document 2.

- d- Write the chromosomal formula of this female. Give the name of the abnormality revealed by the karyotype.

- e- Based on the karyotype, how can you explain the appearance of the disease in female 20?
 f- Knowing that this chromosomal abnormality results from an error in meiosis during spermatogenesis, schematize the chromosomal behavior of the concerned chromosomes only (consider one case only).

**Document 1. Pedigree of the transmission of the disease****Document 2. Karyotype of female 20****Question II (4 pts.)**

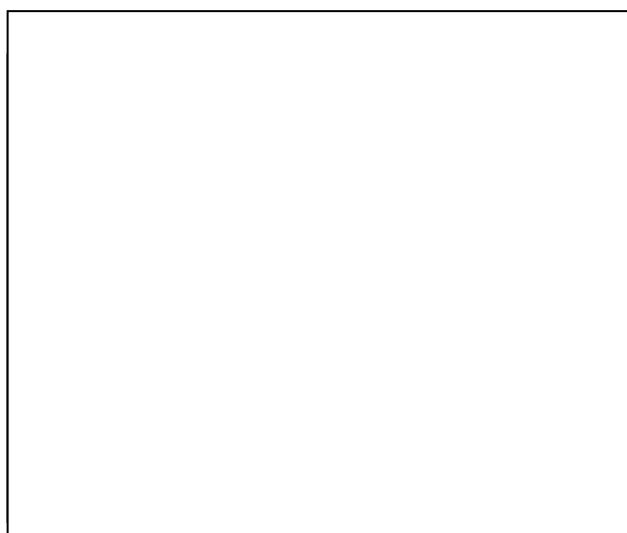
In the framework of studying the mode of action of a chemical contraceptive, we follow up the variation of the ovarian and pituitary hormonal secretion over time in two women having normal cycles, in two different situations: woman A, who does not take a contraceptive pill, and woman B, who takes an estro-progesterone contraceptive pill. The results are presented in documents 1 and 2 for woman A, and 3 and 4 for woman B.



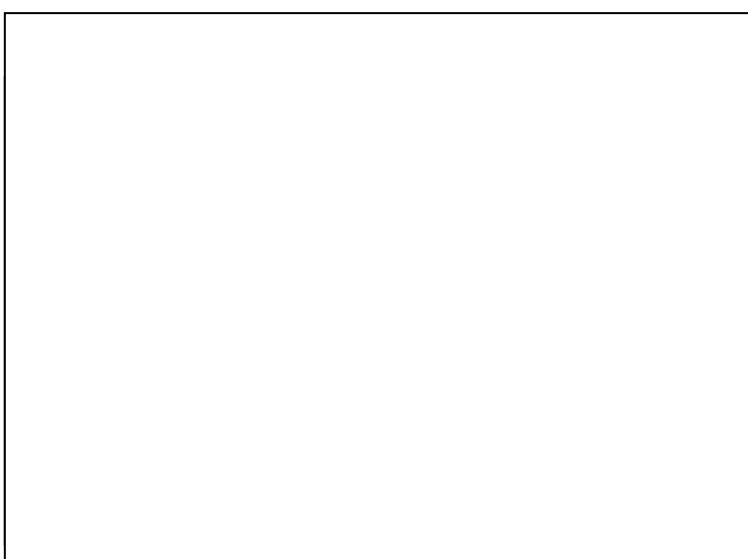
Document 1. Variation of the concentration of the secreted pituitary hormone



Document 2. Variation of the concentration of the secreted ovarian hormones



Document 3. Variation of the concentration of the secreted pituitary hormone



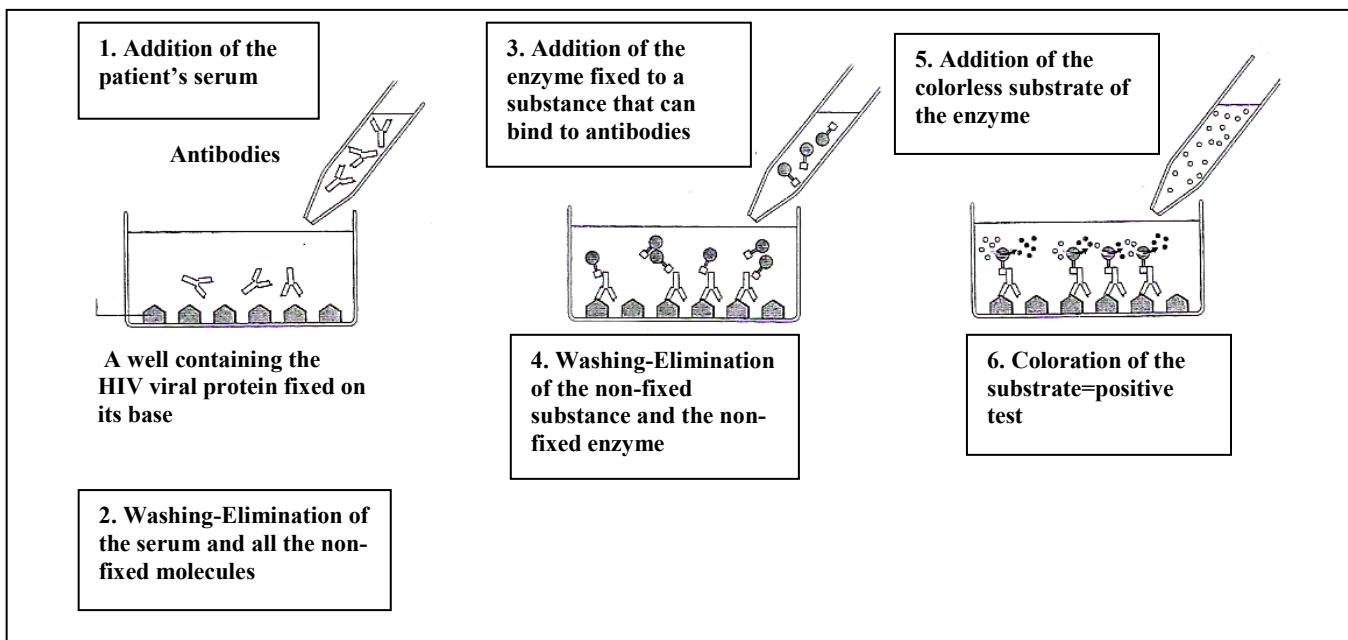
Document 4. Variation of the concentration of the secreted ovarian hormones

- a- Compare the variations of the concentration of estradiol on one hand, and the variation of the concentration of progesterone on the other hand, in the two women. Point out the effect of the pill on the ovaries.
- b- In reference to the documents and to the acquired knowledge, explain the observed differences between the given two situations.

Question III (6 pts.)

AIDS, or Acquired Immunodeficiency Syndrome is a disease due to a virus called HIV, or Human Immunodeficiency Virus. This disease affects the immune system and develops through many years, more or less rapidly depending on the individual.

Individual A is suspected to be infected by the virus. He consulted a doctor who prescribed blood analysis and a test called ELISA test. Document 1 reveals the different steps and the obtained results of this test.



Document 1. ELISA test: steps and result

- a- Write a short text describing document 1.
- b- What does the obtained result indicate? How can you explain this result?

Document 2 reveals the amount of T4 lymphocytes, measured over time, of a patient **B** who presents severe signs of infection.

Duration in months	3	6	12	18	30	40	50	70
Amount of T4 lymphocytes/mm ³ of blood	550	750	800	500	450	300	200	50

Document 2. Variation of the amount of T4 lymphocytes in function of time

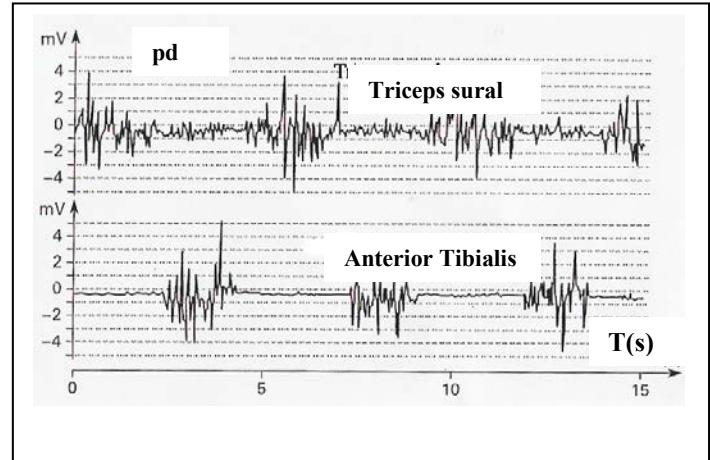
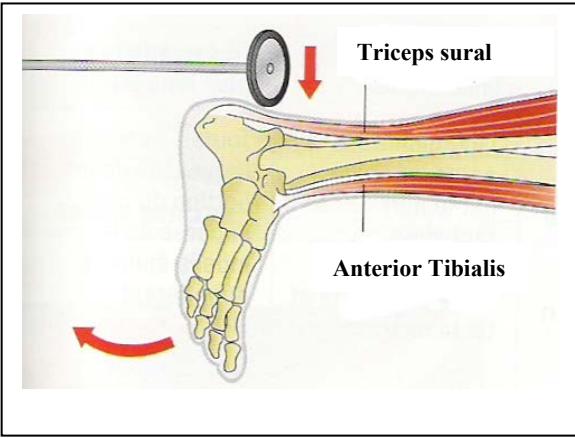
- c- Draw the curve of the variation of the amount of T4 lymphocytes in function of time.
- d- Analyze the curve, then find out the cause of the observed immune deficiency starting from the 40th month.
- e- Knowing that the blood analysis done for patient **A** shows that the amount of T4 lymphocytes is equal to 800/mm³ of blood, and in reference to document 2, find out the duration of infection in patient **A**.

Question IV (4 ½ pts)

To understand the activity of the two muscles of the lower leg, the anterior tibialis and the triceps sural, during a reflex act and during their voluntary movement, the following experiments are done and the results are presented in following documents.

1st experiment: Stretching of the triceps sural by hitting the Achillian tendon, connected to the muscle, immediate extension of the foot and the contraction of the mentioned muscle is provoked, document 1.

2nd experiment: We place electrodes on the skin of a person at the level of the triceps sural and the anterior tibialis, and we ask this person to perform alternating movements of his foot: extension followed by flexion. The obtained recordings are presented in document 2.



Document 1

- a- What type of reflex is revealed in the first experiment? Justify the answer.
 b- Interpret the results of the second experiment. What can you deduce concerning the role of each of these two muscles?

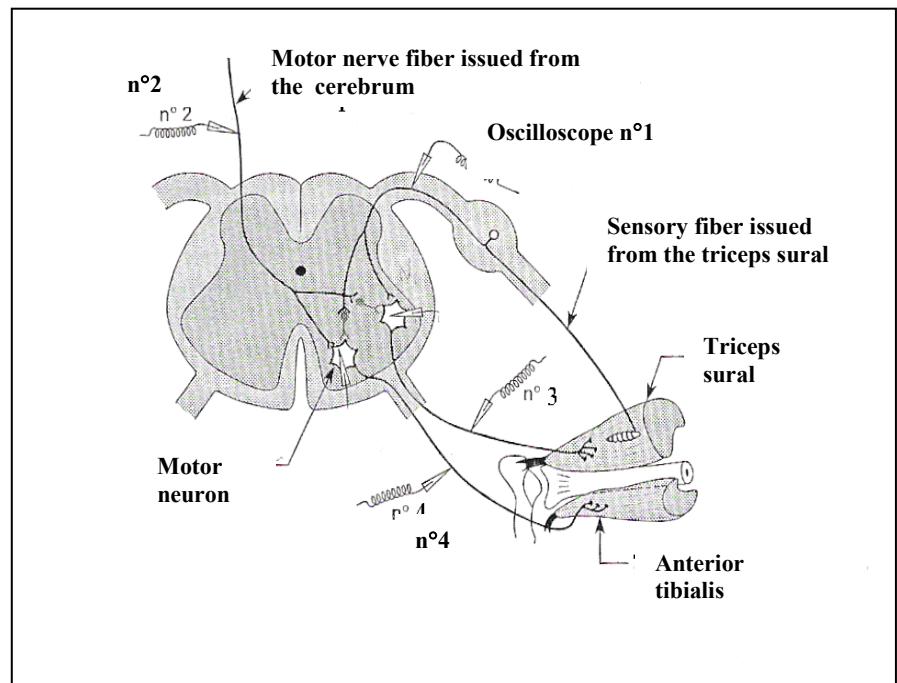
In order to know if a person is capable to control an Achillian reflex we perform the experimental set-up shown in document 3. We record the electric activity at the level of the triceps sural, the anterior tibialis, and the corresponding network of neurons, in the following two cases:

Case A: Hitting the Achillian tendon.

Case B: Hitting the Achillian tendon during a strong voluntary contraction of the anterior tibialis.

The results are presented in document 4.

Document 2



Document 3

	Obtained recordings at the level of the oscilloscopes				Muscular activity	
	n° 1	n° 2	n° 3	n° 4	Triceps sural	Anterior tibialis
Case A	+	-	+	-	Contraction	Relaxation
Case B	+	+	-	+	Relaxation	Contraction

(+) presence of action potential

(-) absence of action potential

Document 4

- c- Compare the obtained results. Deduce the role of the cerebrum in this activity.

Document 4

Question I (5 ½ pts.)

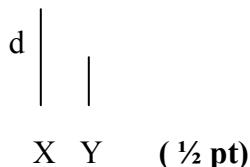
- a- The allele responsible for the disease is recessive. Individual 9, sick, his parents are healthy (couple 1-2). This individual receives the allele responsible for the disease from his parents who have the allele which is masked. (½ pt)

Let "N" be the symbol of the normal allele, and "d" the symbol of the allele of the disease.

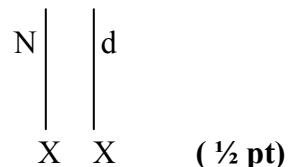
- b- Localization of the gene:

- Since the disease affects mainly the males and very rarely the females, hence the most probable hypothesis is that the gene is sex linked.
- If the gene is carried on the part of Y that has no homologue on X, the transmission should be from fathers to sons. In this case all the boys should have sick fathers. Thus, male 9 who is sick has a normal father 1, which is not the case. Hence the allele responsible for the disease is carried on the X that has no homologue on Y. (1 pt)

c- 13



16



13: Since he is a sick boy, has one chromosome X, carrying the allele d. (½ pt)

16: Since she is a healthy girl and has two sick boys 23 and 24, she must be heterozygote carrying the sick allele. Thus this female must be $X^N X^d$. (½ pt)

- d- Chromosomal formula = $44 + X$

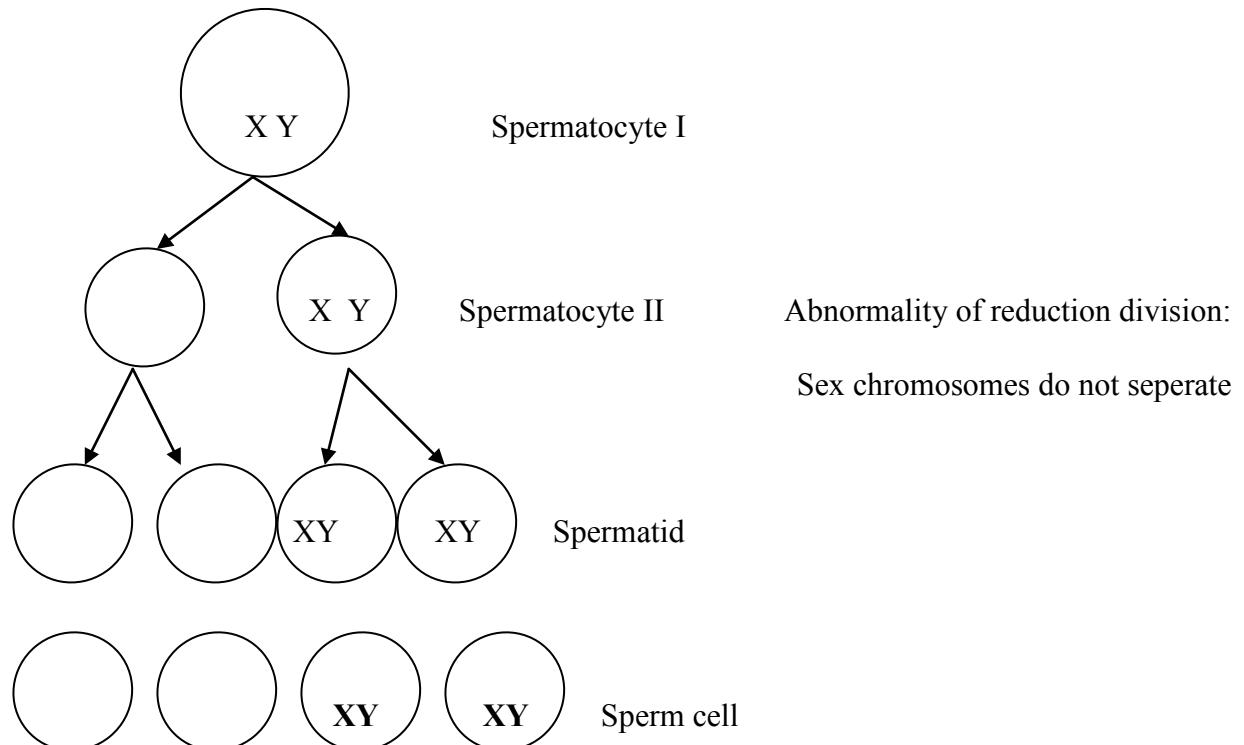
Turner syndrome

(¼ pt)

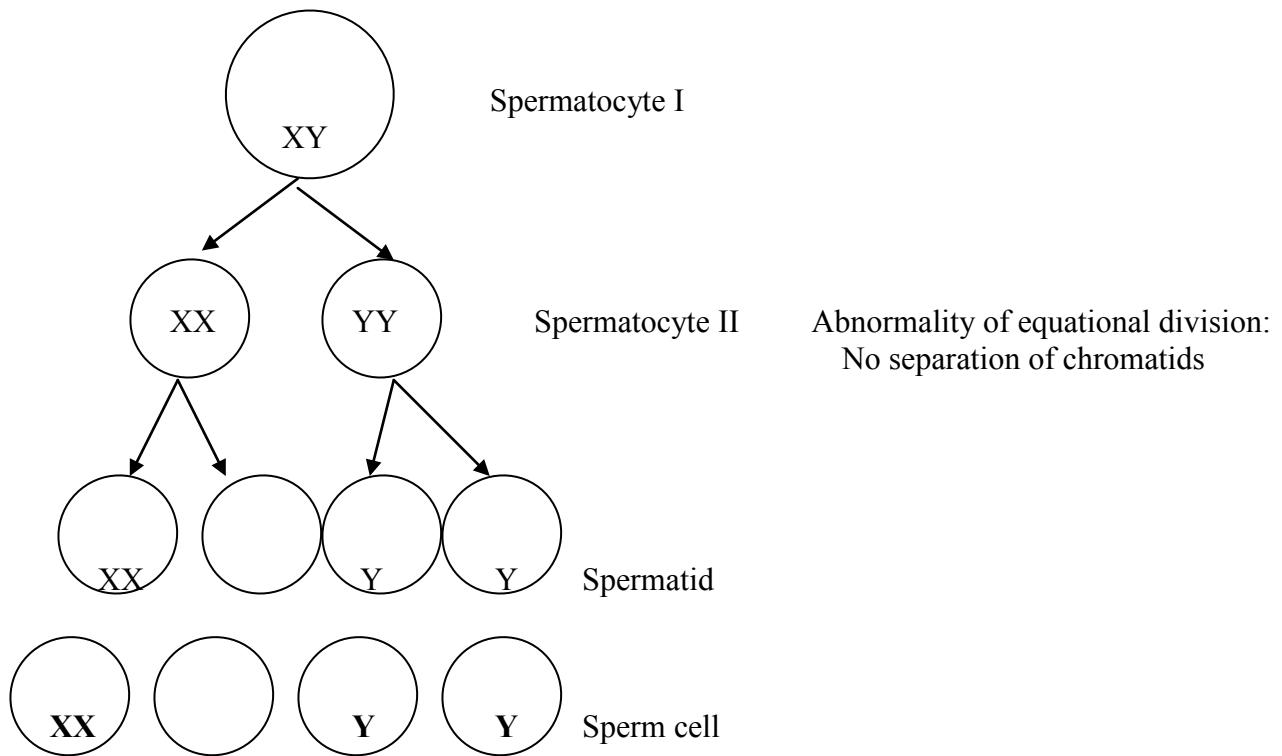
(¼ pt)

- e- This karyotype shows one chromosome X having the sick allele, and since it does not have an allele on a homologue, the disease is expressed in this female. (½ pt)

f-



Or



(1 pt.)

Question II (4pts,)

- a- The secretion of estradiol fluctuates between 5 pg/mL and 8 pg /mL all through the cycle in woman B similarly in woman A from day 0 to day 12, who shows two estradiol peak, of 20 pg/mL on day 13 and another of 15 pg/mL on day 20.

In woman A, the secretion of progesterone by the ovaries is null from day 0 to day 14. It starts increasing from day 14 to day 21 to reach 170 ng/mL then it decreases gradually until it becomes null on day 28.

Moreover, in woman B, the concentration of progesterone fluctuates around a null value through the cycle. **(2 ½ pts.)**

- b- In woman A, not taking the pill, A peak of estradiol is observed on day 13, which is followed by an LH peak on day 14. There is a correlation between the LH peak and the secretion of estradiol: the estradiol peak triggers, by positive feedback the peaking of LH that provokes ovulation. This is not the case in woman B, who lacks an estradiol peak, which does not provoke LH peaking. The contraceptive pill inhibits the secretion of FSH and LH that inhibits the development of the follicles and decreases the secretion of estradiol. The absence of LH peak prevents ovulation. **(1 ½ pt.)**

Question III (6 pts)

- a- We add the serum of the patient to a well containing the viral protein of HIV fixed on its base. Then we wash the well to eliminate the serum and all the non-fixed molecules. We then add an enzyme fixed to a substance capable to bind to antibodies. We wash again the well to eliminate the non-fixed substance and the enzyme. We finally add to the well a colorless substrate of the enzyme, a coloration appears indicating that the test is positive. **(1 ½ pt)**
- b- Patient A is seropositive. The positive test indicates that the serum of patient A contains anti-HIV antibodies. This means that individual A is infected and his immune system reacts to synthesize the specific antibodies. **(½ pt.)**
- c- **(1 ½ pt.)**

**Amount of T4 lymphocytes
/mm³ of blood**

T4 L/mm³ of blood

months

**Duration
(in months)**

Variation of the amount of T4 lymphocytes in function of time

- d- During the first 12 months, the amount of T4 increased from $550 / \text{mm}^3$ of blood to a maximum of $800/\text{mm}^3$ of blood. Starting from the 12th month, the amount of T4 decreased to become $50/\text{mm}^3$ of blood after 70 months. **(1 pt)**
The total immune deficiency observed, takes place starting from the 40th month, is due to the absence of T4 (destruction). **(½ pt)**
- e- Since the number of lymphocytes is $800/\text{mm}^3$ of blood, we can say that the duration of the infection is almost 12 months. **(1 pt)**

Question IV (4 ½ pts.)

- a- Myotatic reflex. **(¼ pt)**
because the muscle responds to its own stretching by contraction. **(¼ pt)**
- b- The recordings of document 2 during extension reveal that when the triceps sural is in action, the anterior tibialis is at rest (0 to 2.5 seconds) and during flexion the triceps sural is at rest, and the anterior tibialis is in action (2.5 to 5 seconds). This implies that the two muscles are antagonistic, thus, the triceps sural is responsible for extension and the anterior tibialis is responsible for flexion.Hence the triceps sural in an extensor muscle while the anterior tibialis is a flexor muscle. **(2 pts.)**
- c- In the Achillian reflex (Case A), the recordings reveal the action potentials by oscilloscopes 1 and 3 and no recordings by oscilloscopes 2 and 4, which leads to the contraction of triceps sural and the relaxation of anterior tibialis. On the other hand, when we ask for the voluntary activity for the contraction of the anterior tibialis while stretching of the triceps, the recordings reveal action potential in 1, 2, and 4, and no recording in 3, which leads to the relaxation of the triceps sural that should have contracted and to the contraction of the anterior tibialis that should have relaxed. This implies that when neuron 2 of the cerebrum is active, it modifies the activity of motor neurons 3 and 4, which stops the reflex act. Thus, the cerebrum controls the reflex activity. **(2 pts)**

الاسم:
الرقم:

مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات

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.



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.

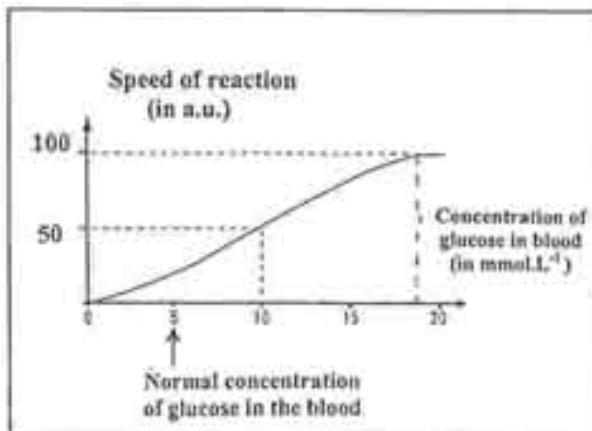
Name of the enzyme	Function	Presence of enzyme	
		Muscle cells	Hepatic cells
Glycogen phosphorylase	Transforms glycogen into G6P	Yes	Yes
Glucose 6 phosphatase	Transforms G6P into glucose	No	Yes

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

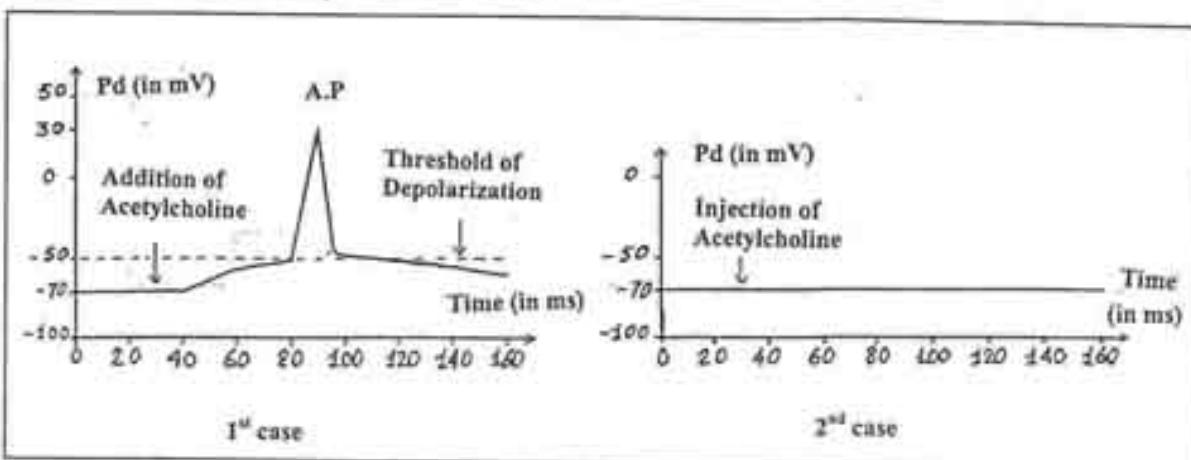
Question II (4 pts)

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:

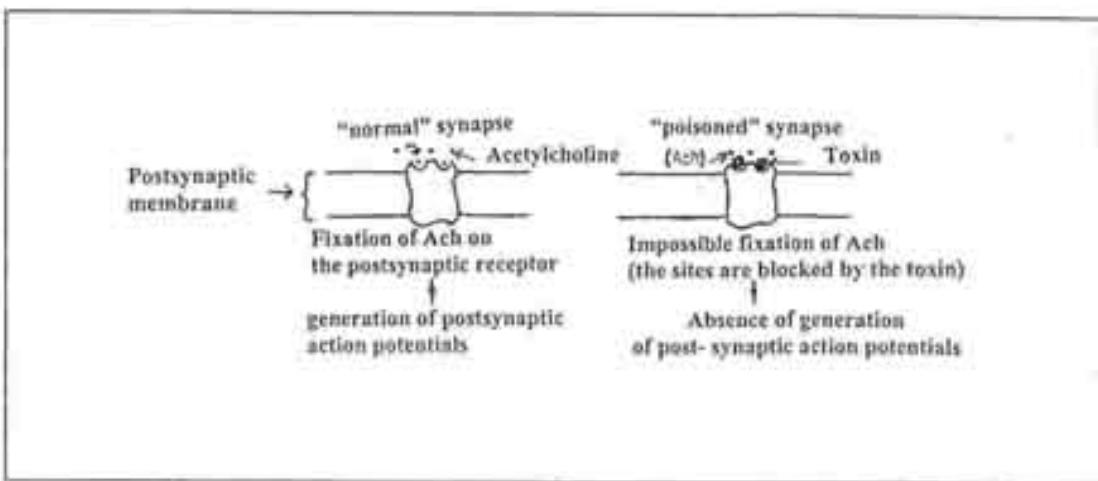
- Case 1: We add a sufficient quantity of acetylcholine into the synaptic cleft.
- Case 2: We inject the same quantity of acetylcholine inside the muscle fibers.

The results of the recordings are shown in document 1.



Document 1

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



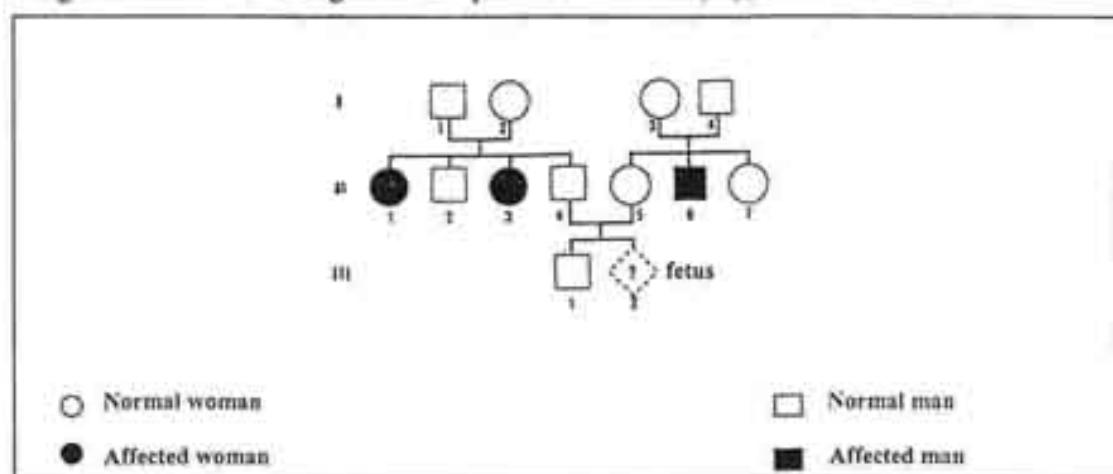
Document 2

- 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?
- 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)

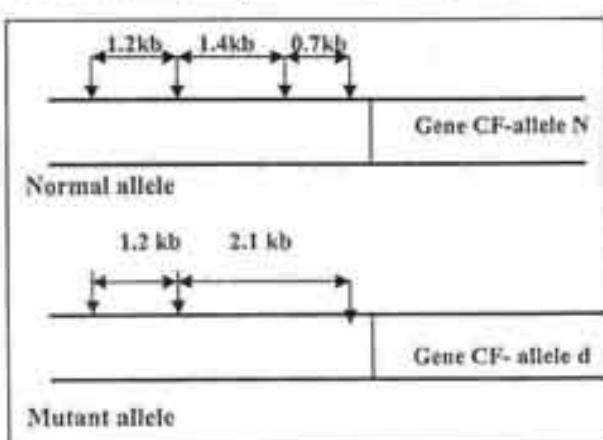
Document 1 represents the pedigree of a family of whom some members, figured in black, are affected by a disease called cystic fibrosis, a hereditary disease manifested by respiratory and digestive troubles. This disease is determined by a mutant allele of a gene called CF. This gene is located on chromosome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

The non-coding region close to the functional dominant allele N has four restriction sites for enzyme Taq I, 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.

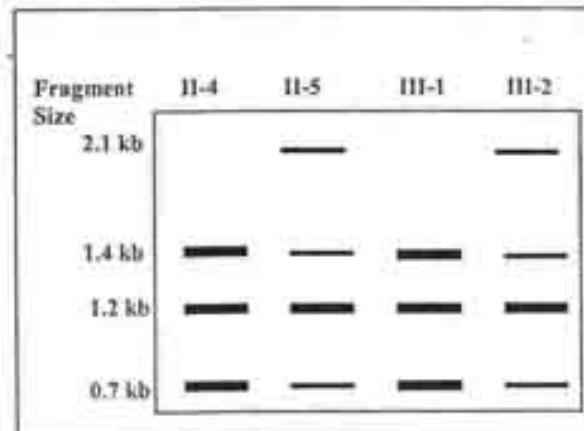


Document 1

- Indicate the possible genotypes of individuals II-4 and II-5. Justify the answer.
- Determine the genetic risk of couple II-4 and II-5 to have a sick child.



Document 2



Document 3

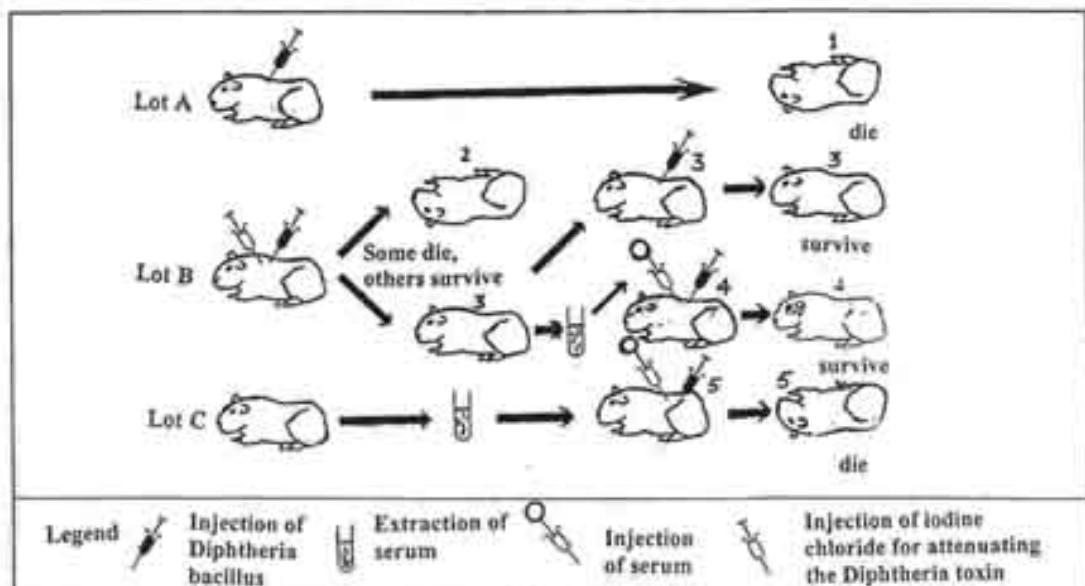
- Specify the site at which mutation took place, document 2. Justify the answer.

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.

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

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?

Question 1 (3 pts)

(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)

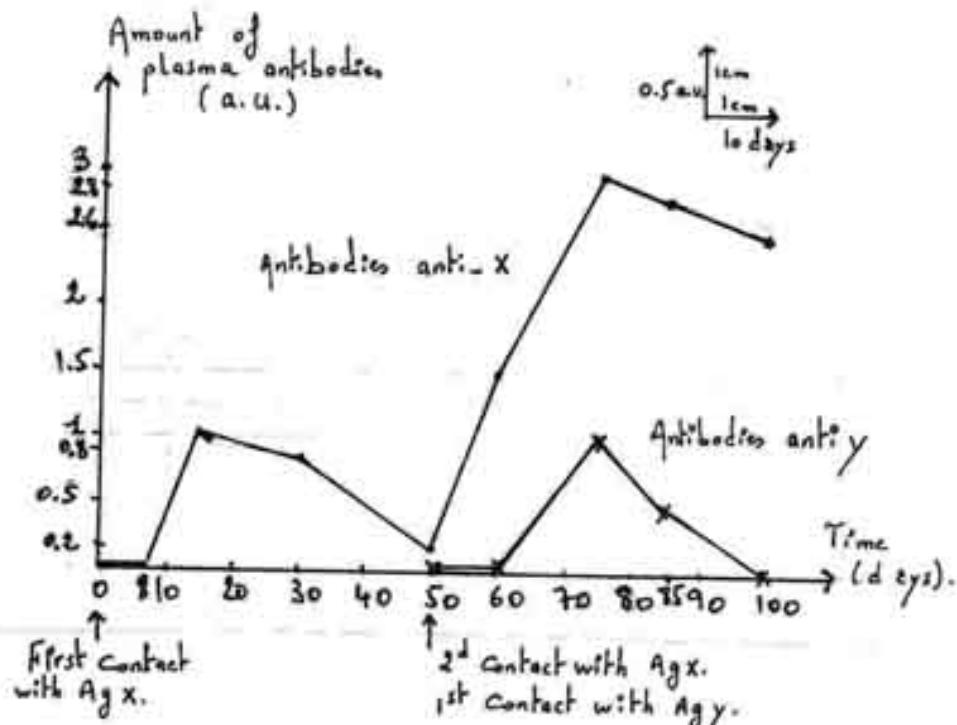
Question III (5 pts)

- 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 $2/3 \times 2/3 \times 1/4 = 1/9$ (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 antibodies, 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

- c- 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)

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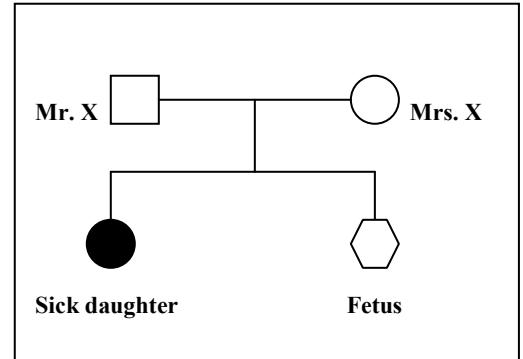
مسابقة في "علوم الحياة"
المدة : ثلاثة ساعات

Answer the following questions.

Question I (5 pts)

Mr. and Mrs. X have a daughter suffering from sickle cell anemia, document 1. This hereditary sickness, whose mode of transmission is autosomal recessive, is characterized by an abnormality in the β -globin molecule which leads to the deformation of the red blood cells. Mrs. X is pregnant and the couple demand prenatal diagnosis to know if their second child will be affected by sickle cell anemia.

- a- Indicate the genotype of Mr. and Mrs. X and that of their daughter. Justify the answer.
- b- Based on logical reasoning, find the probability for this couple to have an affected child.



Document 1

Document 2 reveals the sequences of parts of the non-transcribed strands of the β -globin alleles: HbA is the normal allele while HbS is the mutant allele of the β -globin gene responsible for sickle cell anemia. A direct diagnostic method by radioactive probe is done for this family. Many copies of the parts of the β -globin gene can be obtained from the DNA of each person by this technique. These copies are separated in two lots, and each lot is placed in the presence of a different radioactive probe, document 3; each probe is capable to bind with either allele HbA or HbS. The results of autoradiography are shown in document 4.

Position of the nucleotide	1	10	20
HbA	CTCCTGAGGAGAAGTCTGCC		
HbS	CTCCTGTGGAGAAGTCTGCC		

Document 2

Probe n°1	GAGGACACCTCTTCAGACGG
Probe n°2	GAGGACTCCTCTTCAGACGG

Document 3

	Mr. X	Mrs. X	Daughter	Fetus
Probe n°1				
Probe n°2	[redacted]	[redacted]	[redacted]	

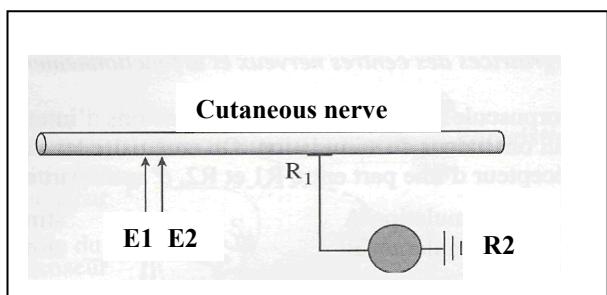
Document 4

- c- Specify, based on document 2, the location of the mutation and its type. Justify the answer.
- d- Determine, in reference to documents 2 and 3, which allele corresponds to each probe used.
- e- Do the results of document 4 confirm the genotypes you have indicated in question a? Justify the answer. Draw out the genotype and the phenotype of the fetus.
- f- Justify why prenatal diagnosis is more accurate than a pedigree in detecting a genetic disease.

Question II (3 ½ pts)

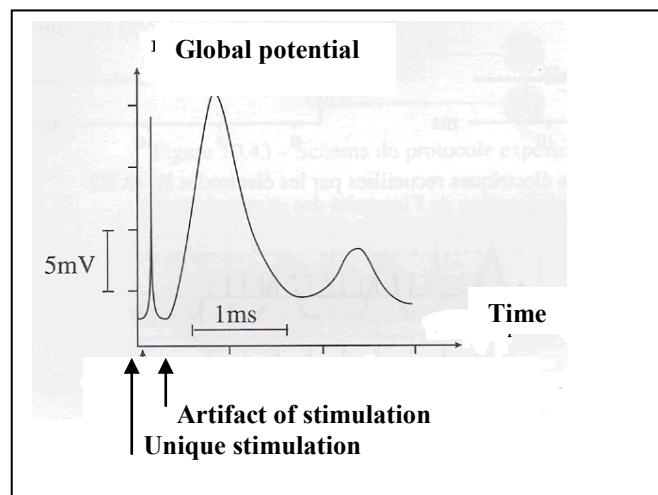
In the framework of studying the electric activity of a mammalian nerve, we establish the experimental set-up shown in document 1. E1 and E2 are stimulating electrodes while R1 and R2, which are placed far from E1 and E2, are recording electrodes. R1 is placed on the surface of the nerve and R2 is connected to a fixed potential.

By the help of E1 and E2, we apply a unique stimulus on the nerve, of intensity above threshold. The response of the nerve to this stimulus is shown in document 2, where the curve shows two successive global potentials instead of one global potential.



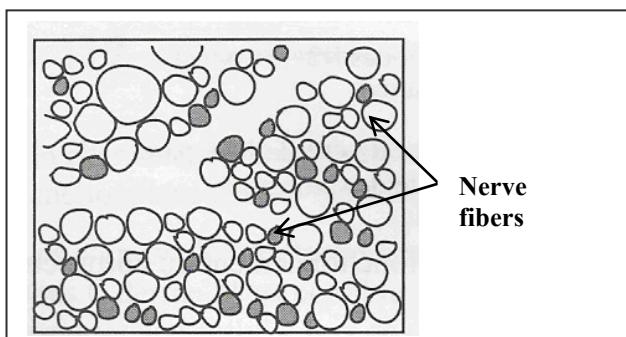
Document 1

- Draw out the problem, which arises in this study.
- Formulate a hypothesis that explains the obtained recording.

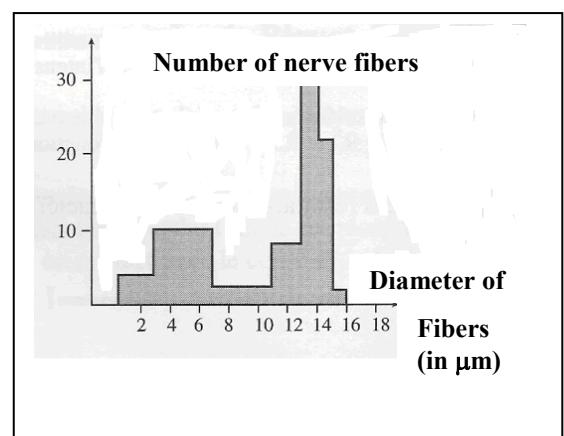


Document 2

To verify the formulated hypothesis, studies are done on this nerve whose results are shown in documents 3, 4, and 5.



Document 3: Transverse section done at the level of the nerve



Document 4: Distribution of the nerve fibers according to their diameters

The speed of propagation of the action potential is 50 meters per second in the nerve fibers having a diameter around 14 μm ; and is 10 meters per second in the nerve fibers having a diameter around 4 μm .

Document 5

- Is the hypothesis that you have formulated validated? Justify the answer in reference to documents 3, 4, and 5.
- To what can you attribute the difference in the amplitude between the two global potentials obtained?

Question III (4 pts)

For determining the relation between the T4 lymphocytes and the T8 lymphocytes, also called cytotoxic T lymphocytes (Tc), we perform the following experiments:

- We remove from the spleen of a mouse, immune cells and we culture them in different mediums, document 1. We add to the culture mediums infected cells taken from an infected mouse of the same species. We detect cytotoxicity from the infected cells that are destroyed by the immune cells present in the mediums, document 2.

Medium 1	Immune cells in serum
Medium 2	Immune cells in a medium that leads to the elimination of T4 lymphocytes
Medium 3	Immune cells in a medium that leads to the elimination of T8 lymphocytes

Document 1

- a- Interpret the experiments done. What can you deduce concerning the condition for the appearance of cytotoxicity in a medium?

Experiment 1	Immune cells removed from medium 1 + infected cells from a mouse of the same species	Presence of cytotoxicity
Experiment 2	Immune cells removed from medium 2 + infected cells of a mouse of the same species	Absence of cytotoxicity
Experiment 3	Immune cells removed from medium 3 + infected cells from a mouse of the same species	Absence of cytotoxicity
Experiment 4	Immune cells removed from mediums 2 and 3 + infected cells from a mouse of the same species	Presence of cytotoxicity

Document 2

- The following microscopic observations reveal the mode of action of Tc lymphocytes in the presence of infected cells.

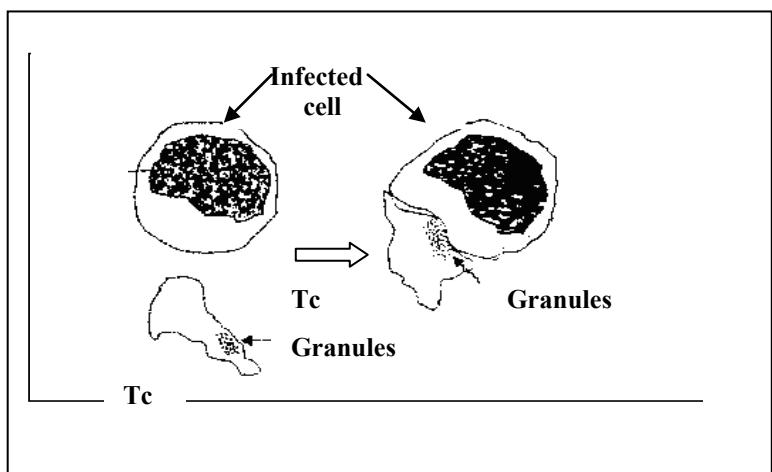
1st observation: In the presence of infected cells, the Tc lymphocytes that are rich in granules containing perforin, come in contact with these cells, document 3.

2nd observation: In the presence of non-infected cells, the Tc lymphocytes do not reveal granules containing perforin in their cytoplasm, and do not come in contact with these cells.

3rd observation: The membrane of the infected cells shows many pores in the region of contact with the Tc lymphocytes.

4th observation: In some mutant mice the Tc lymphocytes present a deficiency in perforin. No pores are observed at the level of the membrane of the infected cells in the region of contact with Tc lymphocytes, and the consequence is the non-destruction of the infected cells.

- b- Draw out from the analysis of the microscopic observations the role of perforin in the destruction of infected cells.
- c- From what has been preceded and based on the acquired knowledge, explain how the T8 lymphocytes become active cytotoxic T lymphocytes and how do they destroy the target cells.

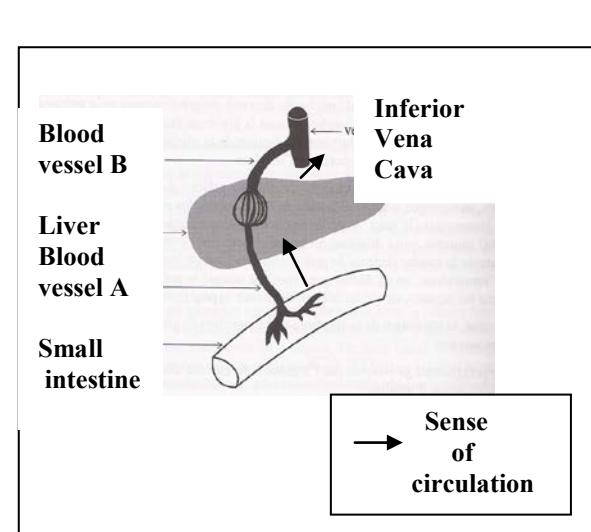


Document 3

Question IV (7 ½ pts)

In the framework of studying the regulation of glycemia in the human body, we measure the amount of glucose in the blood of two blood vessels A and B before and after a meal given at 12 O'clock. The results are shown in document 1.

Document 2 reveals the connection between the small intestine and the liver. The two blood vessels A and B, and the sense of blood circulation at this level are also shown in the document.



Document 2

Document 1

- a- Construct one table that includes the different values shown in document 1.
- b- Interpret the obtained results. Deduce the role of the liver.
- c- Name, each of the two blood vessels A and B.

We measure the variation of glycemia and the concentrations of pancreatic hormones: insulin and glucagon in the blood of 10 persons during fasting for 3 days. We start measuring the amounts one day before fasting. The results are shown in document 3.

	24 hrs. before fasting	Beginning of fasting	24 hrs.	48 hrs.	72 hrs.
Glycemia (in mg.dL^{-1})	89	86	78	72	70
Glucagon (in mU.mL^{-1})	126	126	157	189	190
Insulin (in pg.mL^{-1})	9	10	5	4	3

Document 3

- d- Construct, on the same graph, the two curves that represent the variations of the concentrations of glucagon and insulin secreted as a function of time.
- e- Interpret the results shown in document 3. What can you deduce concerning the hormonal secretion of the pancreas?
- f- From what has been preceded, explain how glycemia regulation takes place after intestinal absorption of nutrients and during fasting.

الاسم :
الرقم :مسابقة في "علوم الحياة"
أسس التصحيح**Question I (5 pts)**

- a- N is the symbol of Normal and s is the symbol of the sickled.
 Mr, and Mrs. X: N//s ($\frac{1}{4}$ pt). They have normal phenotype but have a sick child, the parents carry the allele of the sickness which is masked. ($\frac{1}{4}$ pt)
 The daughter: ss ($\frac{1}{4}$ pt) having sickle cell anemia, a recessive sickness cannot appear unless when it is pure. ($\frac{1}{4}$ pt)
- b- Both parents are heterozygotes, since half of their gametes have the sick allele s
 The probability for this couple to have sick children is $1/2 \times 1/2 = 1/4$ ($\frac{1}{2}$ pt)
- c- The mutation is located on position 7. It is a mutation by substitution because the two alleles of the β -globin gene have the same sequence of nucleotides but differ at position 7 where adenine is replaced by thymine. (1 pt)
- d- The radioactive probe binds to the part of the alleles by complementing with the nitrogenous bases.

Probe n°1: GAGGACACCTCTTCAGACGG
Complementary DNA : CTCCTGTGGAGAAGTCTGCC

This DNA is that of HbS, thus, probe 1 permits visualizing the mutant allele while probe 2 permits visualizing the normal allele (1 pt)

- e- Yes, in the two parents the two probes are visualized, which confirms that the parents are heterozygotes of a genotype Ns ($\frac{1}{4}$ pt). With respect to the daughter we visualize only probe 1 that corresponds to the mutant allele, which confirms that she has the genotype ss ($\frac{1}{4}$ pt). The DNA of the fetus does not permit to visualize except probe 2, which corresponds to the allele N, thus, the fetus has the genotype NN and he has a normal phenotype ($\frac{1}{2}$ pt).
- f- Prenatal diagnosis is more accurate because it depends on the gene itself and gives the real genotype of the concerned person. On the other hand, the pedigree permits to detect the phenotype and the possible genotype ($\frac{1}{2}$ pt)

Question II (3 ½ pts)

- a- Why does the recording present two global potentials in response to a unique stimulus? (½ pt)
- b- Hypothesis: The nerve is constituted of two lots of nerve fibers of different diameters. (½ pt)
Or : The nerve is constituted of two lots of nerve fibers of different nature.
- c- Yes (or no). Document 3 reveals that the nerve is constituted of many nerve fibers and that these nerve fibers are of different diameters. Document 4, confirms the information of document 3 and reveals the presence of two lots of nerve fibers in the nerve. Document 5 indicates that nerve fibers of big diameters favor a more rapid propagation of action potential which leads to the appearance of two global potentials spaced by around 1 ms. (1 ½ pt)
- d- The amplitude of the response of the nerve depends on the number of the stimulated nerve fibers, since the number of the nerve fibers of 14 µm in diameter (30) is greater than the number of the nerve fibers of 4 µm in diameter (10), then the first global potential recorded is the result of the activity of all the nerve fibers of diameter 14 µm and the second global potential recorded corresponds to the nerve fibers of 4 µm in diameter. (1pt)

Question III (4 pts)

- a- In experiment 1 where cytotoxicity is observed, all the immune cells are present. On the other hand, cytotoxicity does not appear in absence of T4 (experiment 2) inspite of the presence of T8 and neither in absence of T8 (experiment 3) although T4 is present, which is confirmed by experiment 4 where cytotoxicity is observed where the immune cells taken from mediums 2 and 3 are placed together with the infected cells. This indicates that T4 only or T8 only are incapable to provoke cytotoxicity, thus, the presence of both is obligatory (1 pt). Hence, the appearance of cytotoxicity necessitates the cooperation between T4 and T8. (½ pt)
- b- The microscopic observations reveal that in the presence of infected cells a contact takes place between Tc rich in perforin and these cells (1st observation) while in the presence of non-infected cells, the Tc do not show perforin and are not in contact with these cells (2nd observation). On the other hand, there is the appearance of pores in the region of contact between Tc and the infected cells (3rd observation) and these pores do not appear in the case of a deficiency in perforin (4th observation), Tc are thus, incapable to provoke the destruction of infected cells. From what has preceded, we can say that perforin is necessary when there is a contact between immune cells and infected cells, which is responsible for the formation of pores at the level of the membrane of the infected cells, followed by their destruction. (1 ½ pt)
- c- After recognizing the antigen, the activated T4 multiply and differentiate into cells that secrete interleukins. Interleukin 2 acts on certain T8 lymphocytes provoking their multiplication and their differentiation into effector cells: Tc lymphocytes. Tc binds to infected cells and secretes perforin that provokes the appearance of pores on the membrane of the infected cells. These pores permit the passage of granzymes that attack the DNA of the infected cells leading to their destruction. (1 pt)

Question IV (7 ½ pts)

a- (1pt)

Time (in hours)	11:30	12	12:30	13	13:30	14	14:30	15	15:30	16
Amount of glucose (in g.L ⁻¹)										
Blood vessel A	0.8	0.8	3	1	0.8	0.8	0.8	0.7	0.7	0.6
Blood vessel B	0.9	0.9	1.2	1	0.9	0.9	1	1	1	1

↑
Food intake

Variation of the amount of glucose in blood vessels A and B as a function of time

b- Before food intake, the amount of glucose in the blood vessel A is 0.8 gL^{-1} and in vessel B is 0.9 gL^{-1} . After food intake the amount of glucose in blood vessel A increases rapidly to become 3 gL^{-1} at 12:30, while it increases slightly in blood vessel B to become 1.2 gL^{-1} . 30 minutes later a rapid decrease in the amount of glucose is observed in blood vessel A (1 gL^{-1}) and continues to decrease to become 0.6 gL^{-1} at hour 16. On the other hand, the amount of glucose in blood vessel B remains constant through out the experiment and fluctuates around 1 gL^{-1} . This indicates that the food provokes the great fluctuation in the amount of glucose in blood vessel A that enters the liver but slight fluctuation in blood vessel B coming out of the liver. (1 pt)
Hence, the liver stores the excess of glucose ($\frac{1}{2}$ pt)

c- Blood vessel A: Portal vein ($\frac{1}{4}$)
Blood vessel B: sub hepatic vein ($\frac{1}{4}$ pt)

d- (2 pts)

Variation of the concentration of insulin and glucagon secreted as a function of time

- e- 24 hours before fasting glycemia was 89 mg dL^{-1} , the concentration of glucagon was 126 mU mL^{-1} and the concentration of insulin was 9 pg L^{-1} . This concentration remains constant at the beginning of fasting, but only glycemia decreases slightly (86 mg dL^{-1}). At the beginning of fasting until 72 hours glycemia continues to decrease to reach 70 mg mL^{-1} and also the amount of insulin decreases to reach 3 pg mL^{-1} while the concentration of glucagon increases to become 190 mU L^{-1} . This indicates that fasting decreases the amount of glucose in the blood and as glycemia decreases the secretion of insulin decreases while the secretion of glucagon increases. Therefore, the secretion of pancreatic hormones depends on the amount of glucose in the blood. **(2 pts)**
- f- After intestinal absorption, the amount of glucose in the blood increases and also the concentration of insulin, which lead to the storage of the excess of glucose in the liver. During fasting, glycemia and the concentration of insulin decrease but the secretion of glucagon increases to return glucose to the blood and maintain a normal glycemia. **(½ pt)**

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مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات

Answer the following questions.

Question I (5 ½ pts)

Hemophilia A, is a genetic recessive disease due to an abnormality of a blood coagulation factor: factor VIII. This factor is the expression of a gene located on the non-homologous segment of chromosome X. We designate, by h, the allele responsible for the disease and by N the normal allele.

Document 1 reveals the pedigree of a family that expresses this disease. Woman 6 is pregnant and asks for prenatal diagnosis for her fetus.

- Indicate the genotypes of persons 6 and 7. Justify the choice.
- Show by logical reasoning, that this pedigree does not permit a sure diagnosis concerning the fetus.
- Determine the genetic risk of this child to be hemophiliac.

To clarify the diagnostic problem of hemophilia in the fetus, two tests were done. The first test is a karyotype of the fetus, document 2.

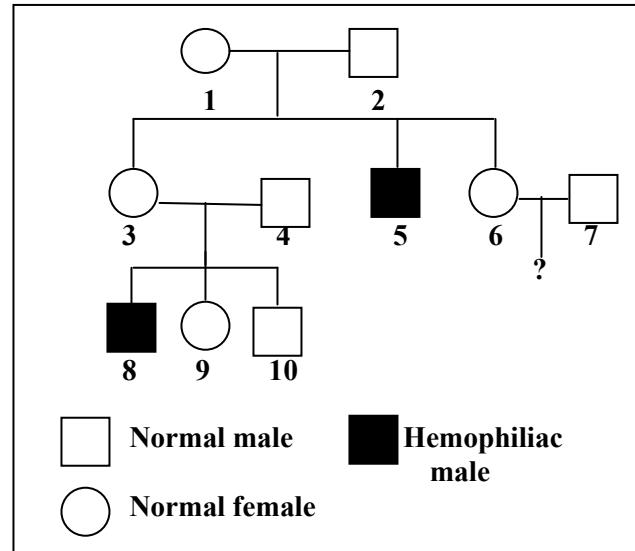
- Does this karyotype solve this problem? Justify the answer.

The second test is the analysis of the DNA of chromosome X. The DNA of the mother, the fetus, and the sick person 8, are subjected to restriction enzymes. The obtained DNA fragments are separated by gel electrophoresis, then hybridized by a probe.

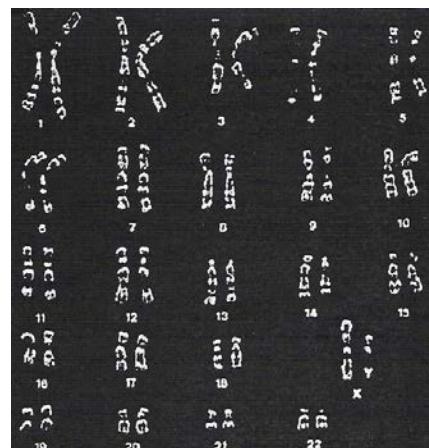
Because we cannot use an intragenic probe to distinguish the hemophilia allele from the normal allele that codes for factor VIII, we use probe ST14 that can mark a polymorphic zone, very close to this gene. This zone has 10 alleles, but only alleles 3 and 5 are present in this family.

An autoradiography is done and the results are shown in document 3.

- Specify, starting from the analysis of the obtained autoradiogram, the real genotype of the mother and the fetus.
- We estimate a 4% recombination between the polymorphic zone and the gene coding for factor VIII. In this case, is the second test reliable for diagnosing hemophilia in the fetus? Justify the answer.



Document 1



Document 2

	Mother	Fetus	Person 8
Allele 3	[Blank]	[Black bar]	[Blank]
Allele 5	[Blank]	[Black bar]	[Blank]

Document 3

Question II (5pts)

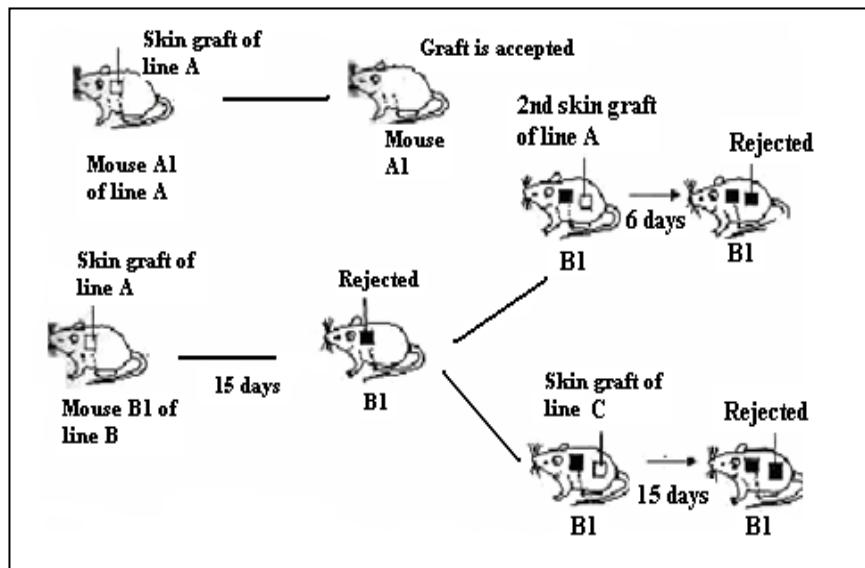
In the framework of studying immune responses, we do the following experiments.

1st Series of experiments: We perform skin graft between mice A, B, and C of different lines, document 1.

- a- Interpret these experiments.
- b- Indicate two characteristics of the immune system revealed by these experiments.

2nd Series of experiments: We graft skin of mouse A into mouse B under different conditions. The experiments done and the obtained results are shown in document 2.

- c- Starting from the analysis of document 2, specify the organs involved in graft rejection.



Document 1

3rd Series of experiments: We remove cells infected by virus X from a mouse of line A. We incubate the cells with radioactive chromium ^{51}Cr . This ^{51}Cr is absorbed and binds to proteins in the cells. After incubation, we wash these cells and culture them with different effector cells obtained from the same mouse A. The supernatants are then collected for measuring the quantity of ^{51}Cr released by the lysed target cells. Document 3a shows the experiments that are carried out, and document 3b shows the obtained results.

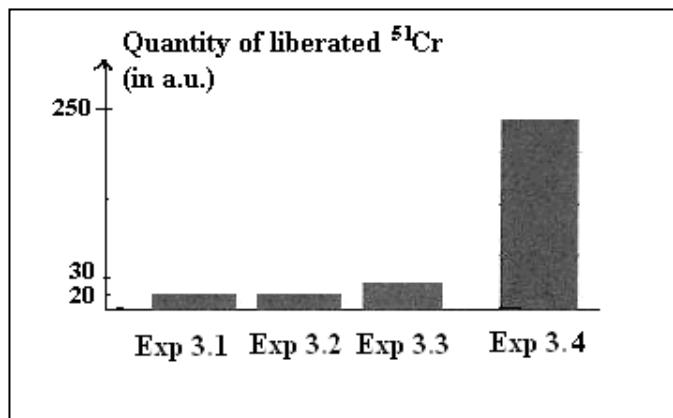
N° of experiment	Experimental conditions	Results
1	Control mouse B	Graft is rejected
2	Mouse B deprived of its thymus	Graft is accepted
3	Irradiated mouse B (destruction of bone marrow)	Graft is accepted

Document 2

N° of experiment	Effecter cells of mouse A
3.1	None
3.2	Macrophages
3.3	LT4 + LT8
3.4	LT4 + LT8 + macrophages

Document 3a

- d- Interpret the obtained results.
- e- By referring to the acquired knowledge, explain how the effector cells of document 3a intervene in the lysis of infected cells.



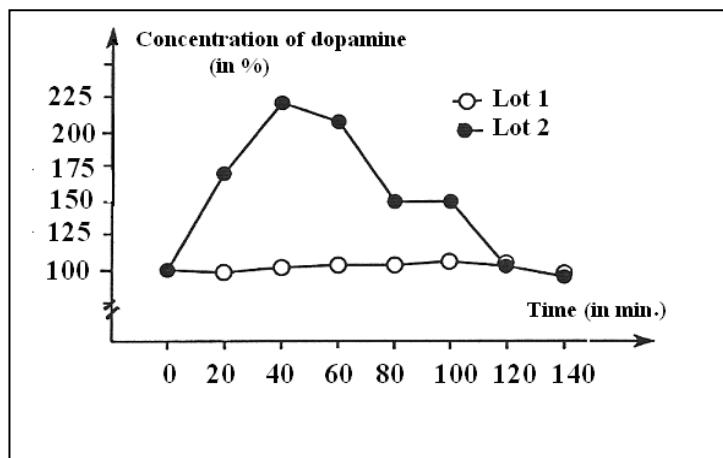
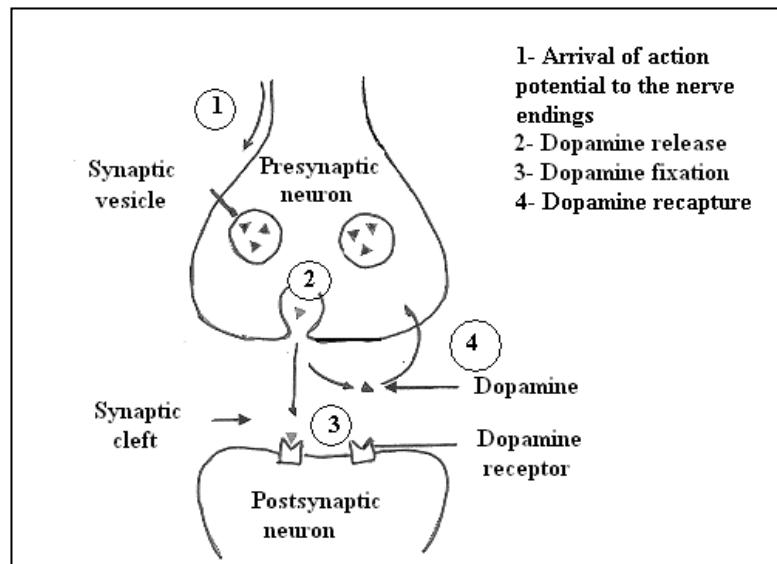
Document 3b

Question III (4 ½ pts)

Studies are done to show the mode of action of cocaine at the level of the dopamine synapse. Dopamine is a cerebral neurotransmitter. Document 1 reveals the functional diagram of the dopamine synapse.

- a- Write a short text describing the mode of action of this synapse.

We measure the concentration of dopamine in the synaptic cleft in two lots of rats. The rats of lot 1 are kept as a control and those of lot 2 received an injection of cocaine at time $t = 0$ minute. The results are shown in document 2.



Document 2

- b- Interpret the obtained results.
c- Based on documents 1 and 2, propose two hypotheses that explain the mode of action of cocaine at the level of this synapse.

Document 3 indicates the effects of cocaine on the nervous system.

- d- Which of the two proposed hypotheses is validated in document 3? Justify the answer.
e- Pick out from the text the statements that indicate that cocaine consumption leads to tolerance.

Document 1

Cocaine disrupts the fragile balance that allows the few billions of neurons of our brain to function... In the brain, the privileged target of cocaine, are the neurons that secrete dopamine. Normally, the neurotransmitter substances are liberated by a neuron and passes into the synaptic cleft to fix on receptors of the next neuron. Some are recaptured by a specific pump to be liberated later when needed. Cocaine blocks this pump of dopamine recapture. As a consequence: the neurotransmitter stimulates the neighboring neurons permanently. Under the repeated effects of cocaine, the neurons adapt to the abnormally elevated concentration of this substance. The brain is thus, forced to maintain an increased production of this neurotransmitter. This production can only be maintained by the frequent consumption of the drug. This leads to the anxious behavior of cocaine addict in constantly searching for the drug.

Document 3

Question IV (5 pts)

In the framework of studying birth control, two women A and B use two different types of pills. Mrs. A uses pill of type X. We measure the concentration of progesterone in this woman before and after taking pill X. The results are presented in documents 1 and 2.

Without pill X

N.B. Progesterone concentration is more than 20 ng/mL during the second half of the cycle, which indicates that ovulation took place

With pill X

Document 1

- a- Construct a table that includes the variations of the concentration of progesterone of Mrs. A, before and after taking pill X.
- b- Compare the variations of the concentration of progesterone, before and after this woman takes pill X. What can you deduce concerning the effect of pill X?

Mrs. B uses another pill Y to interrupt her early pregnancy. To understand the effect of pill Y, we perform experiments on three lots of rabbits that did not reach puberty. Document 3 shows the experimental conditions and the obtained results.

- c- Draw out from document 3 the target organ and the effect of pill Y. Justify the answer.
- d- Name the birth control method that corresponds to each of the two pills used.

Document 2

	Lot 1	Lot 2	Lot 3
Injection of estradiol	+	+	+
Injection of progesterone	-	+	+
Intake of a appropriate dose of pill Y	-	-	+
Results	Thickening of the endometrium, no formation of uterine lace	Thickening of the endometrium, formation of uterine lace	Thickening of the endometrium, no formation of uterine lace

(+) presence

(-) absence

Document 3

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أسس التصحيح

Question I (5 ½ pts)

- a- Woman 6 : Normal woman but having a hemophiliac brother, she can be either homozygous $X^N X^N$ or heterozygous $X^N X^h$. (¾ pt)
Man 7: $X^N Y$; normal man and having only one X, thus he carries the normal allele. (½ pt)
- b- The child to be born can be either a girl or a boy. If it was a girl, this pedigree permits a sure diagnosis; she will be normal because her father can give her only X^N . But if he was a boy, the diagnosis is sure if the mother was homozygous and he will be normal, but if the mother is heterozygous we cannot determine whether the boy is normal or hemophiliac because his mother can give him either X^N or X^h . (1pt)
- c- If this child was a girl, the risk is null.
If this child was a boy, its phenotype depends on the allele provided by his mother. The possibility of the mother of being heterozygote is $\frac{1}{2}$. If she was heterozygous there is a possibility of $\frac{1}{2}$ for giving him X^h and since we do not know the sex of the fetus there is a chance of $\frac{1}{2}$ to be a boy. Hence the genetic risk becomes $\frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} = 1/8$. (1pt)
Or
The probability of the mother to be heterozygous is $\frac{1}{2}$, in this case $\frac{1}{4}$ of her children will be hemophiliac. Hence, the genetic risk = $\frac{1}{2} \times \frac{1}{4} = 1/8$
- d- No, because the karyotype reveals that it is a boy. If it was a girl the problem would have been solved. (½ pt)
- c- Person 8, has only allele 5. Being hemophiliac, we can say that allele 5 is linked with allele h that codes for hemophilia.
Mother 6, who is normal, has the two alleles 3 and 5 each one is on an X chromosome. Since allele 5 is linked with allele h, then allele 3 must be linked with the normal allele N. She is thus, healthy but has the allele h, her genotype is $X^N X^h$. (¾ pt)
The fetus has only allele 3, thus he received X^N from his mother and Y from his father, thus, he will be normal of genotype $X^N Y$. (½ pt)
- f- No, because there is a possibility of crossing over between the polymorphic zone and the gene.
Non-sister chromatids of the two homologous X chromosomes will exchange segments leading to the formation of a chromosome X on which allele 5 is linked with the normal allele N and another chromosome X on which allele 3 is linked with the hemophiliac allele h. Thus, the fetus will be hemophiliac even if his autoradiogram shows the presence of allele 3. (½ pt)

Question II (5pts)

- a- The skin graft of mouse A received by mouse A₁ of the same line is accepted while the skin graft of a mouse A received by mouse B₁ of line B different from A, is rejected after 15 days. This same mouse rejects a second graft of A, 6 days after grafting, on the other hand it takes 15 days to reject the graft received from a mouse of line C.
Therefore, the graft succeeds only between mice of the same line and the rejection of the graft is faster upon second recognition. **(1pt)**
- b- Recognition of the non-self by the immune system, the presence of an immune memory, and specificity of the immune response **(½ pt)**
- c- Skin graft taken from a mouse of line A to mouse B (control) leads to the rejection of the graft. On the other hand , the graft is accepted when it is done on mouse B that is deprived of its thymus (experiment 2) or on mouse B that is subjected to the irradiation of the bone marrow (experiment 3). Therefore, the thymus and the bone marrow are involved in graft rejection. **(1pt)**
- d- The quantity of ⁵¹Cr released by the cells lysed in a medium deprived of effector cells of mouse of line A and in a medium containing macrophages is 20 a.u. This quantity increases to become 30 a.u. in a medium containing LT4 and LT8 and reaches 250 a.u. in a medium containing LT4, LT8 and macrophages.
Hence, graft rejection requires the co-operation between these three types of immune cells. **(1pt)**
- e- The macrophages digest the free virus, recognized as non-self, and transform them into peptides and present them on HLA molecules of class II. These macrophages are thus, antigen presenting cells (APC). These latter migrate towards the lymphatic ganglia where they activate the LT4 (LT_H) that secrete IL-2.
IL-2 activates the LT8 (LT_c), which adheres to the membrane of the target cell and releases perforin and granzymes that perforate the membrane and degrades the DNA of the target cell leading to its lysis. **(1½ Pt)**

Question III (4 ½ pts)

- a- The arrival of action potential to the nerve endings of the presynaptic neuron allows the release of dopamine in the synaptic cleft. Dopamine fixes on its postsynaptic receptors, then it is recaptured by the presynaptic neuron. **(1 ½ pt)**
- b- At the beginning of the experiment, the percentage of dopamine concentration in the two lots of rats is 100%, this percentage remains almost constant in the control rats of lot 1, while after the injection of cocaine at time $t = 0$ min. it increases in the rats of lot 2, to become more than twice (225%) after 40 minutes. This percentage starts to decrease to become normal again 100% after 120 minutes.

Therefore, cocaine permits the increase, for a certain time, the quantity of dopamine in the synaptic cleft. **(1pt)**

- c- Cocaine increases the release of dopamine in the synaptic cleft.
Cocaine prevents or decreases the recapture of dopamine by the presynaptic neuron. **(½ pt)**
- d- The validated hypothesis that cocaine prevents the recapture of dopamine because it blocks the pump that allows its recapture. **(½ pt)**
- e- Under the repeated effects of cocaine, the neurons adapt to the abnormally elevated concentration of this substance. The brain is thus, forced to maintain an increased production of this neurotransmitter. This production can only be maintained by the frequent consumption of the drug. **(½ pt)**

Question IV (5 pts)

a- (1 ½ pts)

Time in days	0	4	12	16	18	20	22	28
Concentration of progesterone without pill X (in ng/mL)	0	0	0	5	25	30	30	0
Concentration of progesterone without pill X (in ng/mL)	0	0	0	0	5	10	5	0

Variations of the concentration of progesterone as a function of time with or without the pill

- b- Mrs. A with or without taking pill X, the concentration of progesterone is the same, almost null (0.2 ng/mL) from day 1 until day 12. Without pill X, this concentration in a cycle begins to increase to reach 30 ng/mL (> 20 ng/mL) on day 20, indicating that ovulation has taken place. On the other hand, in the cycle with the pill X, the concentration of progesterone increases slightly from 0 - 8 ng/mL (< 20 ng/mL) from day 14 to day 24 of the cycle, which indicates that ovulation did not take place. This concentration starts to decrease from day 24 in a cycle without the pill X and from day 20 in a cycle with the pill to become null, in both cases, on day 28. Therefore, in the presence of pill X, the concentration of progesterone does not reach a high concentration, but remains less than the value that induces ovulation and decreases rapidly. Hence, pill X has an effect of preventing ovulation. (1 ½ pts)
- c- The target organ is the uterus and the effect of this pill is to inhibit the development of the uterine lace, because in the presence of injections of estrogen and progesterone (lot 2) there is a thickening of the endometrium and a development of the uterine lace. On the other hand, the injection of estrogen alone (lot 1) there is only a thickening of the endometrium. This indicates that progesterone acts on the development of the uterine lace. When we add to the injections of estrogen and progesterone the intake of pill Y (lot 3), the uterine lace did not develop. This indicates that pill Y has blocked the action of progesterone on the development of the uterine lace. (1 ½ pts)
- d- Pill X corresponds to a contraceptive method
 Pill Y corresponds to a contragestive method. (½ pt)

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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.

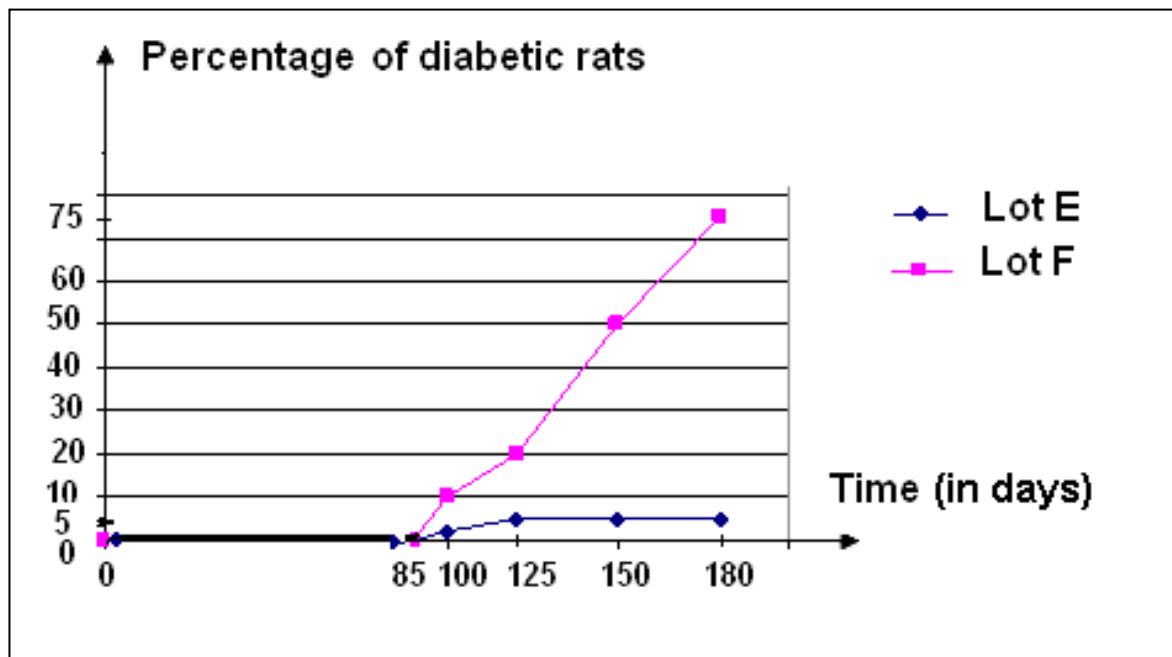
	Number of diabetic rats
Lot A	5/50
Lot B	30/50

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.

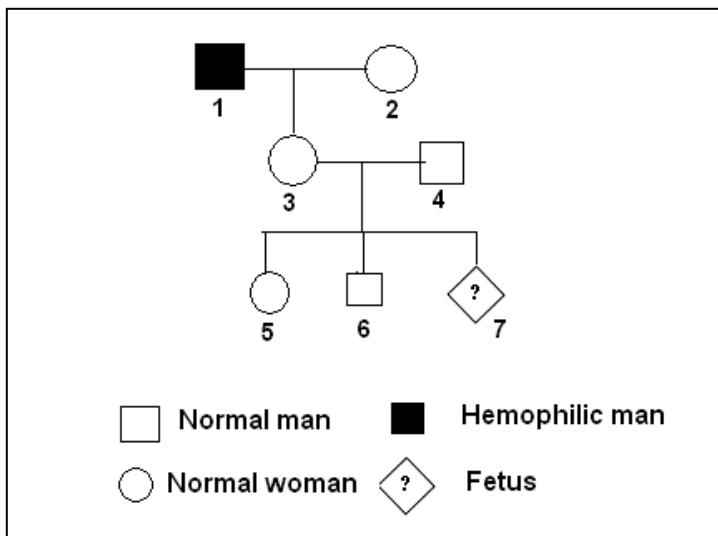
- 1- Explain the absence of this abnormality in girls?
- Document 1 shows the pedigree of a family, one member of whom has the abnormality.
- 2- Show that this disease is recessive.
- 3- Determine the genetic risk that the fetus will be hemophilic.

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.

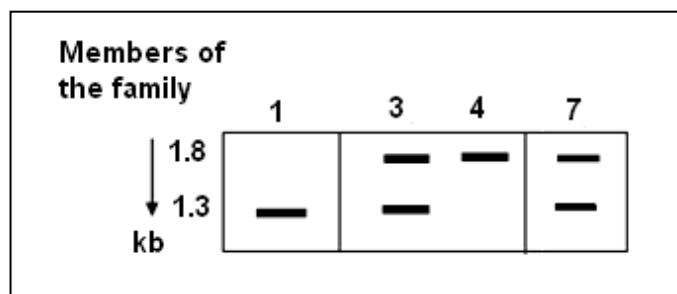
- 4- Specify the band that corresponds to the defective allele. Justify the answer.
- 5- Identify, from the DNA analysis, the problem of the child that will be born.

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

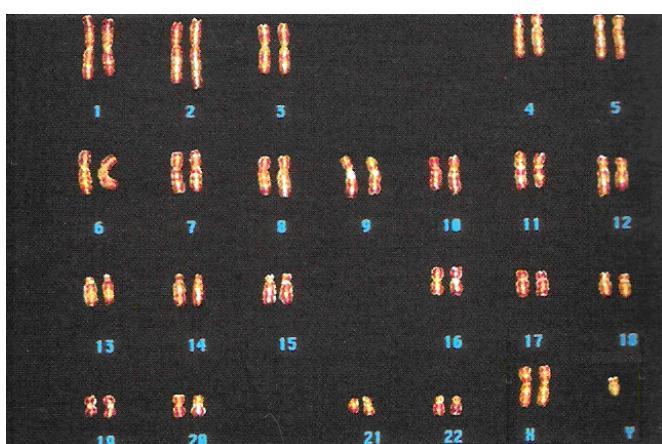
- 6- Establish, based on documents 2 and 3, the diagnosis of the fetus.
- 7- Specify the stage of meiosis at which the abnormality took place. Justify the answer.
- 8- Schematize the behavior of chromosomes at the origin of this abnormality.



Document 1



Document 2



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 .

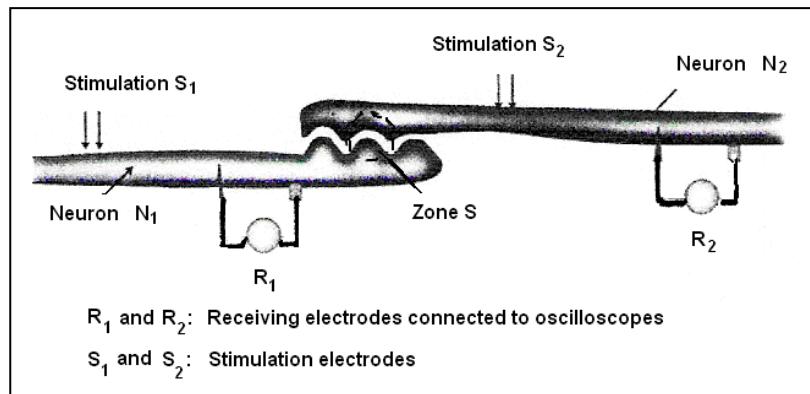
2nd experiment: The nerve fiber of N_2 was stimulated by S_2 . An AP was recorded only in R_2 .

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 .

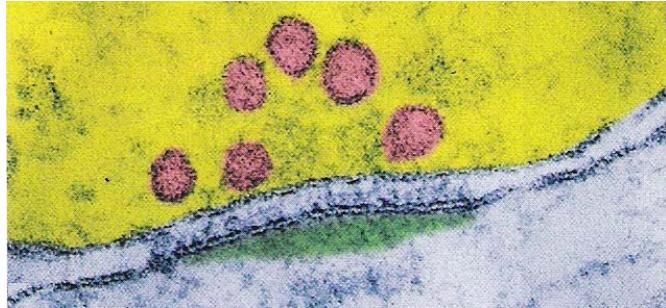
4th experiment: A micro-drop of acetylcholine was injected in neuron N_1 and another drop in N_2 . No AP was recorded in R_1 or R_2 .

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

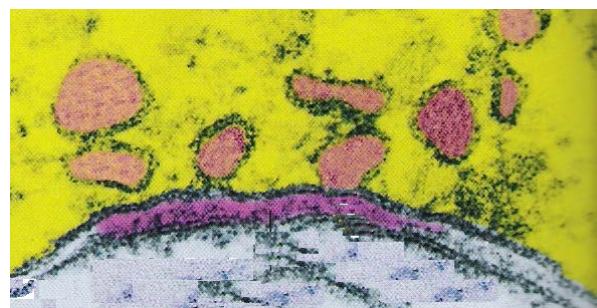
2- Interpret the 3rd and 4th experiments. Deduce the level of the nerve cell at which acetylcholine acts. Documents 2a and 2b present electronic micrographs of the synapse at the level of zone S at two different moments.



Document 1



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.

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.

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

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.

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

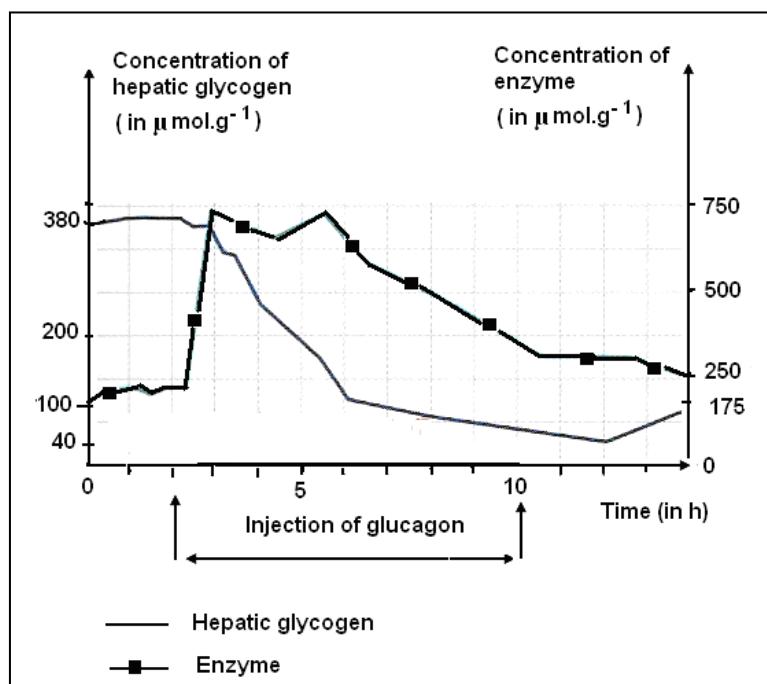
2nd experiment: A muscle was placed for 10 minutes in a medium containing glucose and insulin, or glucose without insulin. Then the quantity of glucose absorbed by the muscle and the quantity of glycogen stored in the muscle in each used medium were measured.

The results are shown in document 2.

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

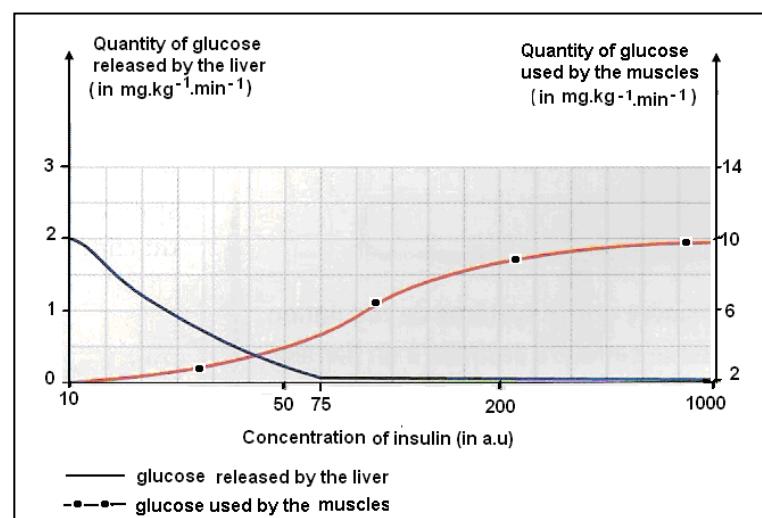
- Interpret the obtained results.
- Determine, based on what had preceded, the reaction of muscular cells and hepatic cells to the pancreatic hormones.



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

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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)

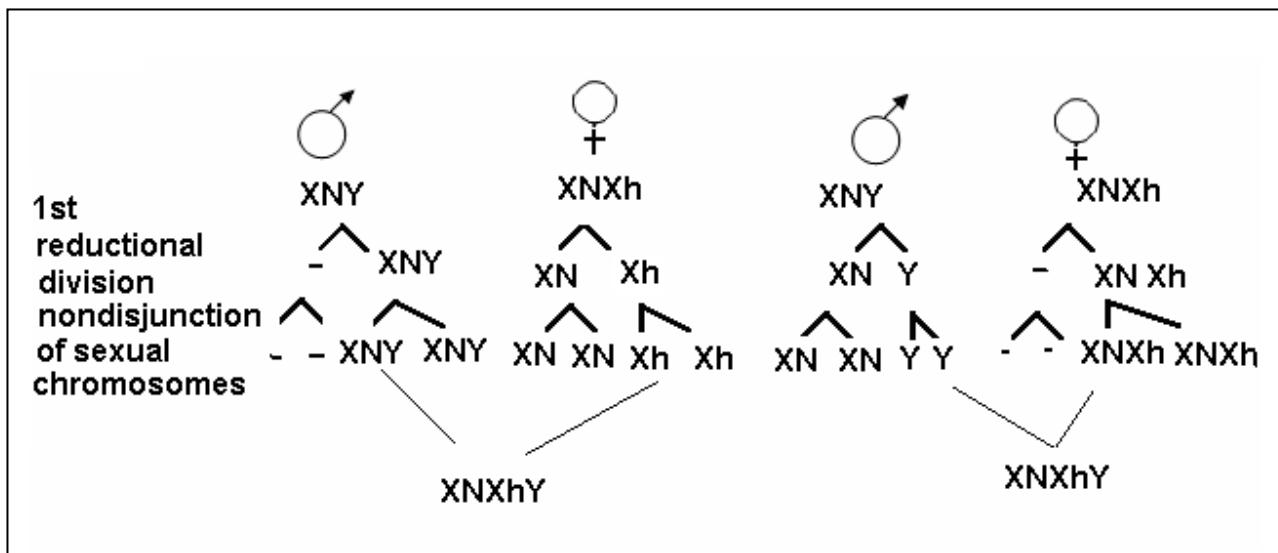
Time (in days)		0	85	100	125	150	180
Diabetic rats (in %)		0	0	2	5	5	5
Lot E		0	0	2	5	5	5
Lot F		0	0	10	20	50	75

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 $\frac{1}{2}$ 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)

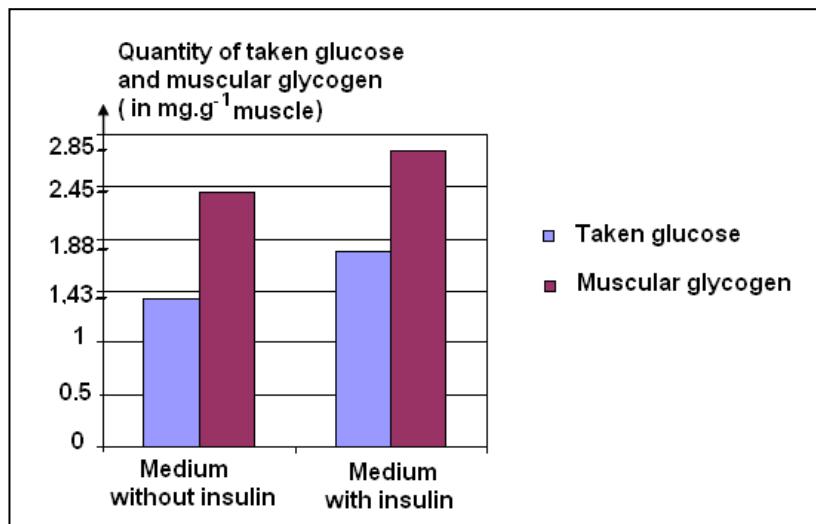
- 1- The stimulation of the nerve fiber by S_1 caused an AP recorded in R_1 then in R_2 . On the contrary, the stimulation by S_2 caused the recording of an AP only in R_2 . This means that the nervous message passes from N_1 towards N_2 , and not in the opposite direction. Thus, the nervous message 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 $380 \text{ } \mu\text{mol.g}^{-1}$, and the concentration of the enzyme $175 \text{ } \mu\text{mol.g}^{-1}$. One hour after the injection of glucagon, the concentration of the enzyme increased quickly to become $750 \text{ } \mu\text{mol.g}^{-1}$ and was followed by a decrease in the concentration of hepatic glycogen that reached $100 \text{ } \mu\text{mol.g}^{-1}$ after 3h. Then this concentration of glycogen continued to decrease reaching $40 \text{ } \mu\text{mol.g}^{-1}$. At the same time the concentration of the enzyme decreased also to reach $250 \text{ } \mu\text{mol.g}^{-1}$, 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 $2 \text{ mg.kg}^{-1}.\text{min}^{-1}$ at 10 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 $3 \text{ mg.kg}^{-1}.\text{min}^{-1}$ 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 $10 \text{ mg.kg}^{-1}.\text{min}^{-1}$ 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)

الاسم :
الرقم :

مسابقة في مادة "علوم الحياة"
المدة : ثلاثة ساعات

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.

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 1 g.L^{-1} . The amount of perfused glucose needed for each person is then measured. The results are shown in document 2.

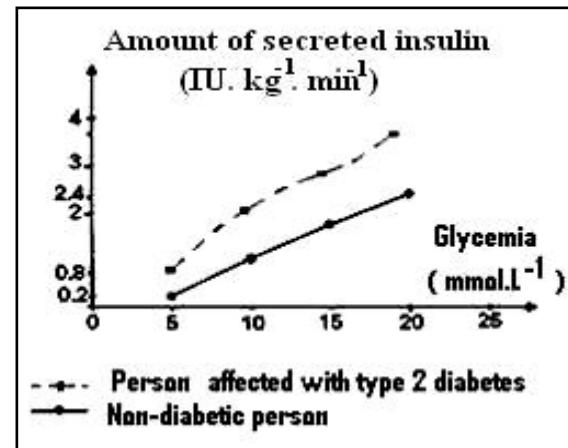
2- Compare the obtained results. What can be deduced regarding the efficiency of insulin?

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.

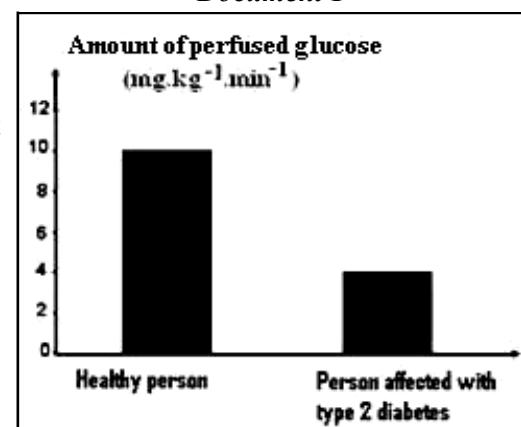
3- Represent, in a table, the variations of the amount of ^{14}C absorbed by the adipocytes versus the quantity of insulin added, in both persons.

4- Interpret the obtained results.

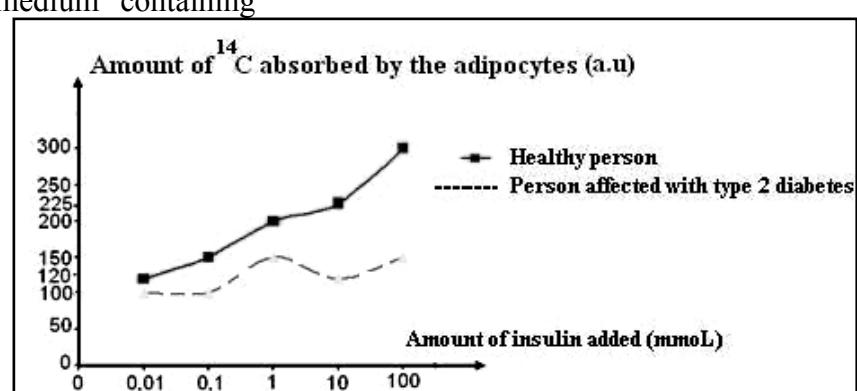
5- Based on what precedes, indicate the disturbances in glycemia regulation that led to type 2 diabetes.



Document 1



Document 2

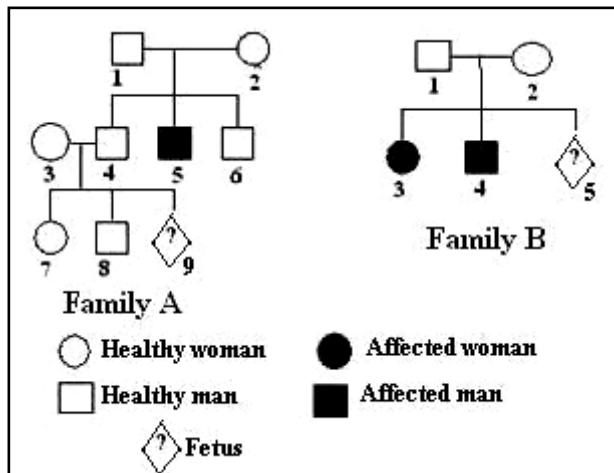


Document 3

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.

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



Document 1

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

- 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.

	P	M	E	P	M	E
Normal probe	●	●		●	●	●
Mutant probe	●	●	●			
Family A						Family B

P= Father M= Mother E= Fetus

Document 3

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

	Father	Mother	Fetus
Normal allele	—	—	
Mutant allele	—	—	—

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

Document 4

Exercise 3 (5pts)

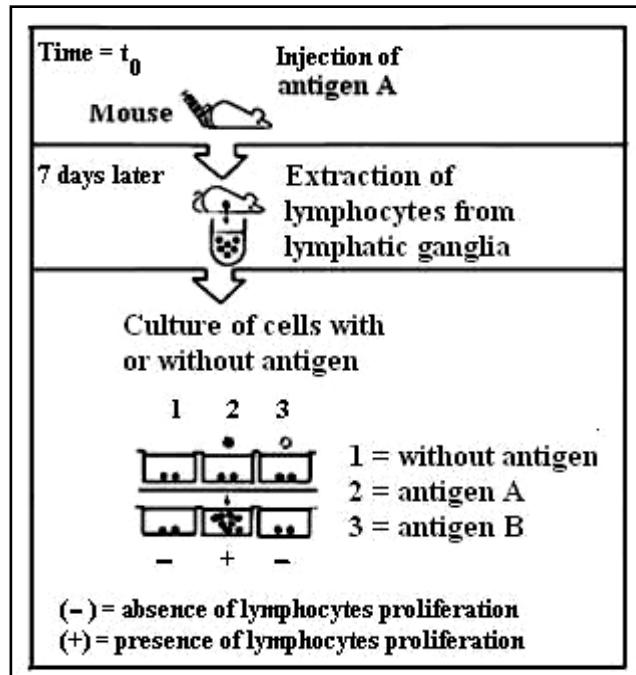
In order to study one of the characteristics of the immune response, the experiment shown in document 1 were carried out.

- 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.

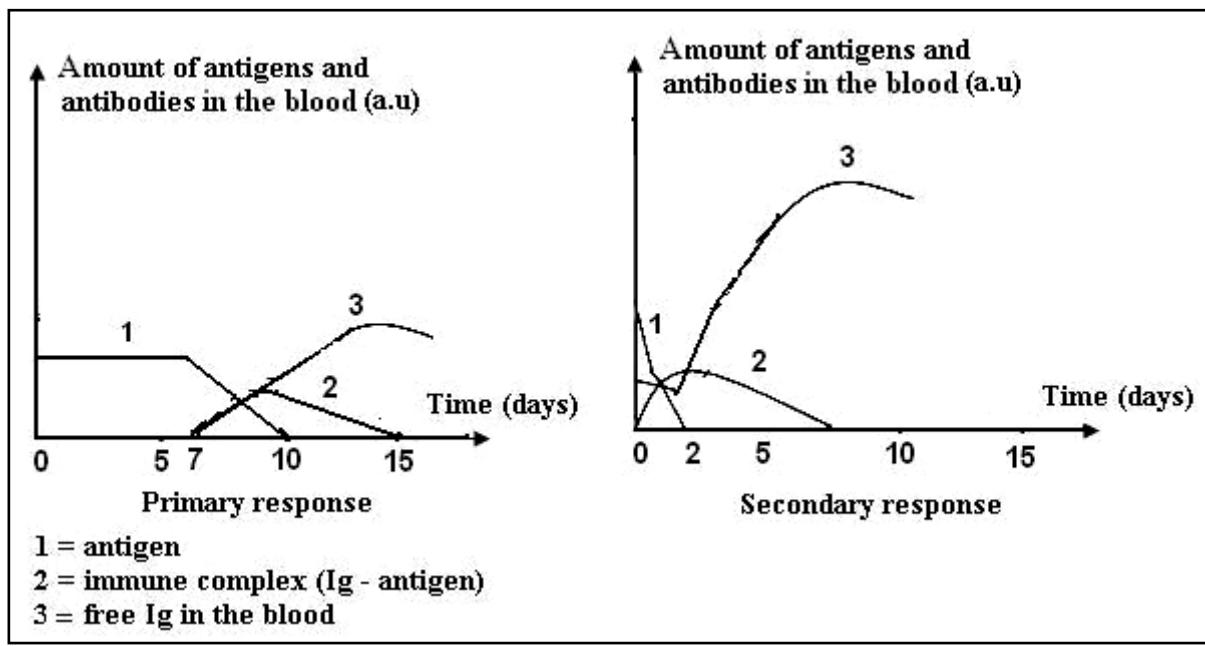
In a second experiment, the same steps are repeated without a seven days time delay. The cells of the lymphatic ganglia are directly extracted after the mouse immunization against antigen A. The results do not show any proliferation of lymphocytes.

- 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.

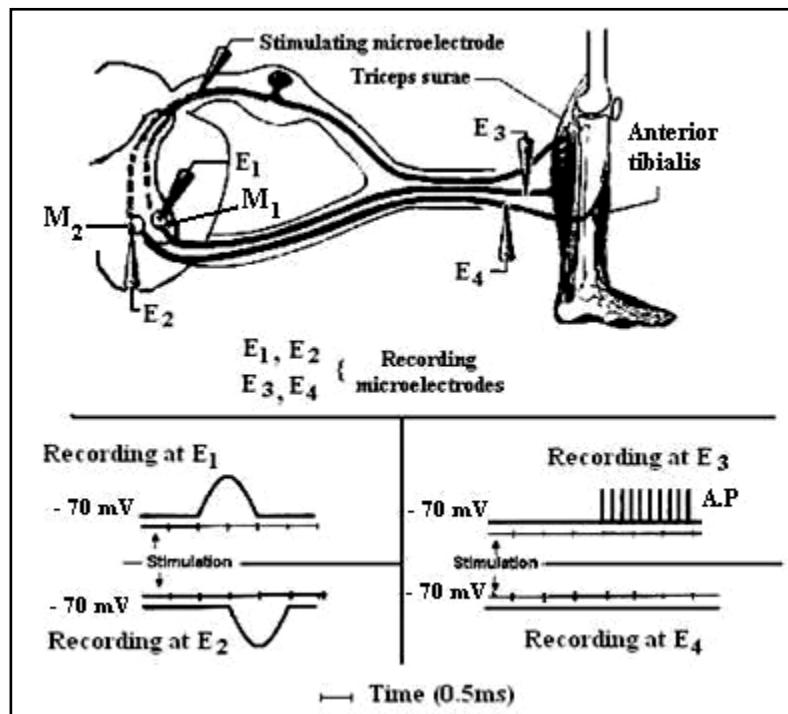
However, any body's movement is hindered by a force – gravity – which attracts it towards the ground. To control body movement and to reach equilibrium, the dancer uses muscles that block some joint movements and prevent falling down. The posture is thus maintained thanks to a constant adjustment of the muscle: for instance, every time a muscle is stretched, it contracts.

Document 1

- 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.
- 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, M₁ and M₂, 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.



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

- Referring to the recordings E₁ and E₂, 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 M₂ that is linked to anterior tibialis. Many action potentials were recorded at E₄ and no recording was obtained at E₃.

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

Document 2

الاسم :
الرقم :

مسابقة في مادة "علوم الحياة"
أسس التصحيح

Exercise 1 (5pts)

- 1- For a glycemia of 5 mmol.L^{-1} , insulin secretion is higher in the diabetic person ($0.8 \text{ IU kg}^{-1}.\text{min}^{-1}$) than in the healthy one ($0.2 \text{ IU.kg}^{-1}.\text{min}^{-1}$). This secretion increases in both persons with the increase in glycemia, however, it is kept higher in the diabetic person, $3.5 \text{ IU.kg}^{-1}.\text{min}^{-1}$ to $2 \text{ IU.kg}^{-1}.\text{min}^{-1}$ 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 1 g.L^{-1} by the constant input of insulin, the amount of glucose perfused into the healthy person ($10 \text{ mg.kg}^{-1}.\text{min}^{-1}$), is higher than that perfused into the person affected with type 2 diabetes ($4 \text{ mg.kg}^{-1}.\text{min}^{-1}$). 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 one person. (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 is 1/3. If the father is healthy homozygous, the risk is nil since he can only transmit the normal allele to his descendants. However, if he is healthy and heterozygous, half of his gametes carry the mutant allele. Then the risk will be:

$$2/3 \times 1/2 \times 1/2 \times 1/50 = 1/300. (\textbf{0.5pt})$$

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 $1/4$. Then the risk is $1/4$.

- 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 phagocytose 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 E₁ placed at the level of motor neuron M₁. This excitatory message leads to the appearance of many AP recorded at E₃ 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 E₂, placed at the level of motor neuron M₂, a hyperpolarization is recorded and no recording was obtained on E₄, placed at the level of the efferent fiber linked to the anterior tibialis. Since M₁ sent an excitatory nerve message to the triceps surae, this latter contracted; since M₂ 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 M₁. Since the time needed to cross a synapse is 0.5ms, then one synapse exists along this pathway. The neuron circuit of M₁ is monosynaptic.
The nerve message took 1.2ms to reach M₂. This delay noticed at the level of M₂ versus M₁ is equivalent to the time needed to cross an additional synapse. Therefore, on pathway M₂ we have two synapses. The neuron's circuit of M₂ 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 E₃, however a nerve message at E₄, located at the level of the efferent fiber linked to the anterior tibialis, was observed. **(1pt)**

الاسم :
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مسابقة في مادة "علوم الحياة"
المدة : ثلاثة ساعات

Answer the following exercises.

Exercise 1 (5pts)

Phenylketonuria is a recessive autosomal disease that affects 1/10,000 of newborns world wide. This disease is related to a deficiency in an enzyme called PAH. In normal conditions, this enzyme metabolizes phenylalanine into tyrosine, in the presence of a co-factor DHBP. This deficiency leads to an increase in the amount of phenylalanine in the blood accompanied with serious troubles.

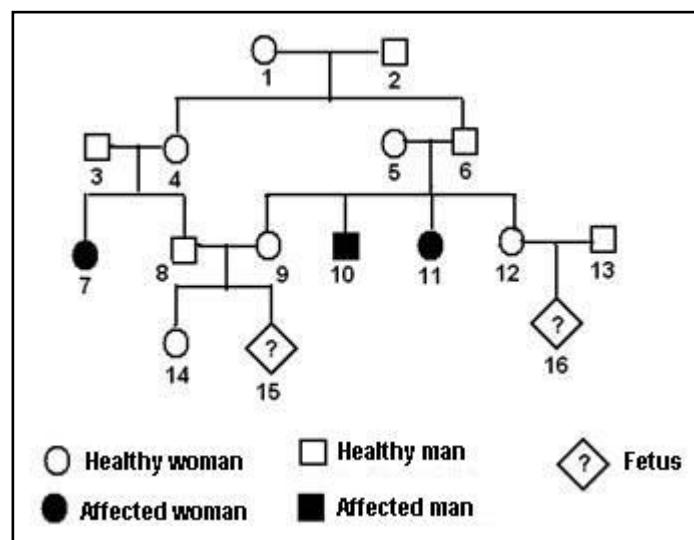
A study performed on 1,200 children selected from an isolated community, showed that 30 children were heterozygous for PAH.

- Calculate the proportion of heterozygous children in this community; and then determine the genetic risk for a child to be affected with phenylketonuria.
- Compare the genetic risk obtained to the world wide risk. Formulate a hypothesis that explains the difference between these two risks.

In order to verify the formulated hypothesis, a study was carried out on a family of this community, which pedigree is shown in the adjacent document.

- By referring to the pedigree, justify that the disease is recessive and autosomal.
- Determine, for each of the fetuses 15 and 16, the risk to be affected.
- Do the obtained results confirm the formulated hypothesis? Justify the answer.

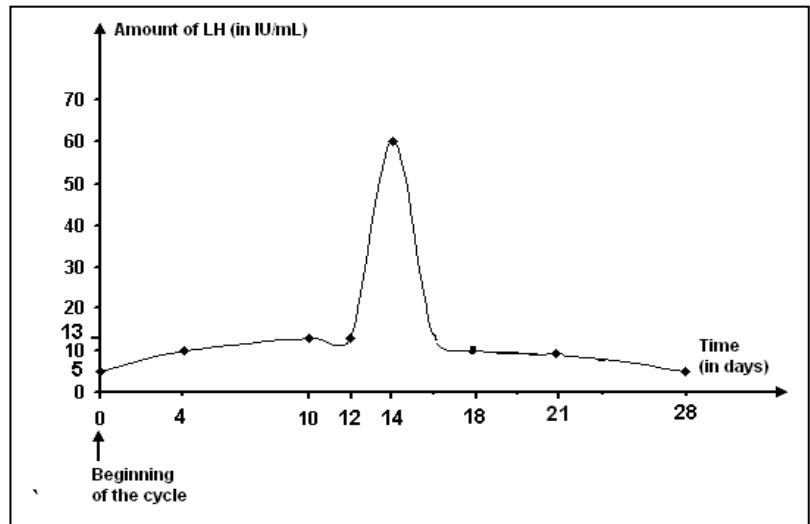
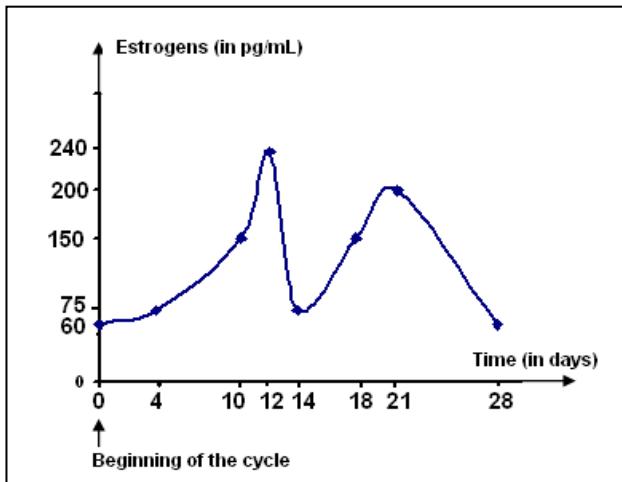
Daughter 7 marries an affected man. Their first child was normal. All the tests performed confirm that the child is legal, and that the husband, unlike his wife, has a normal amount of PAH.



- Determine the probable cause of the disease of the husband of daughter 7.
- Justify, genetically, the birth of a normal child by this couple.

Exercise 2 (5pts)

In order to determine the reason of sterility in a 30 years old woman, a gynecologist prescribed the determination of estrogens and LH hormones levels in the course of a sexual cycle. The obtained results are shown in documents 1 and 2.



Document 1

Document 2

1. Draw up in the same table, the variations of the plasma amount of estrogens and LH in this woman.

Advanced analysis showed a production of normal gametes. The doctor affirms that the results show an absence of disturbances in the functioning of the hypothalamus, pituitary, and ovaries and that the anomaly is mainly in the genital ducts.

2. By referring to documents 1 and 2 and to acquired knowledge, justify the doctor's affirmation.
3. Name the technique that allows treating this woman's sterility.

Document 3 reveals the amount of estrogens and LH in another woman of the same age, who also suffers from sterility.

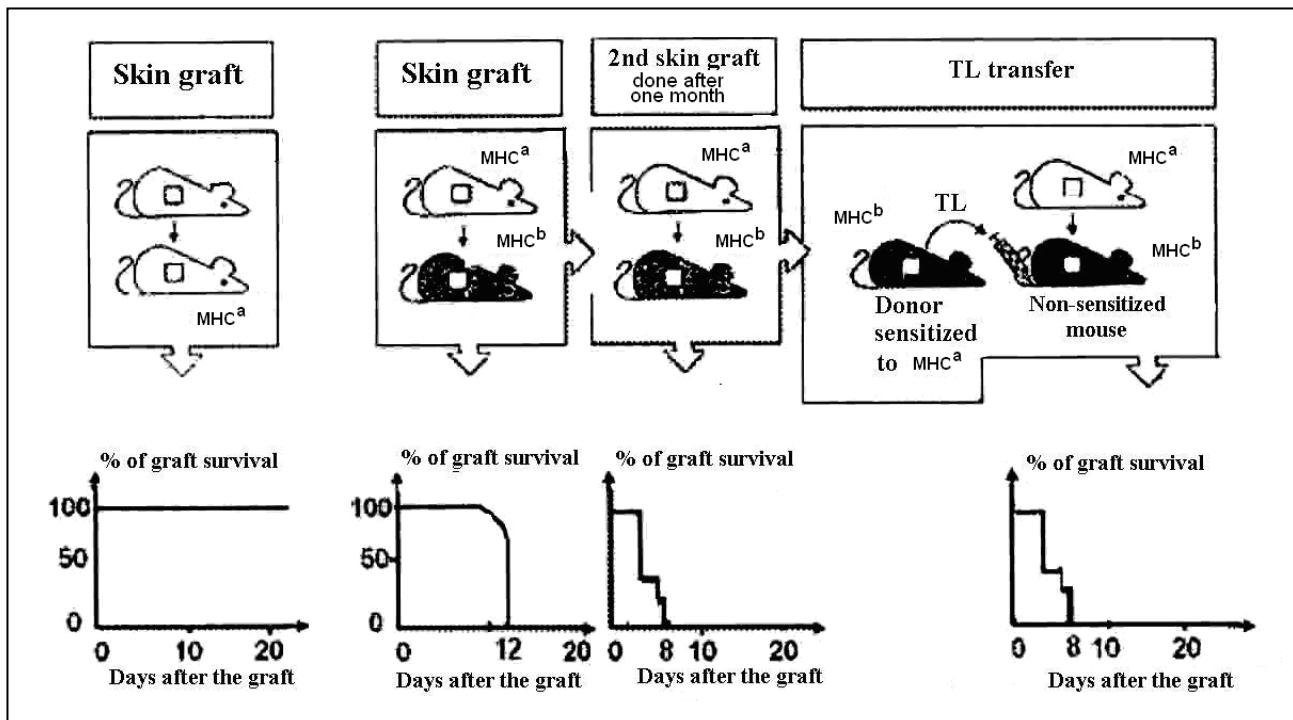
Days after the beginning of menstruation	0	4	10	12	14	18	24	28
Amount of estrogens (pg/mL)	29.1	30	30.4	29.9	29.3	30.3	30	30.3
Amount of LH (mIU/mL)	5.3	6.8	6.3	7	6.2	6	7.3	6.5

Document 3

4. Interpret the obtained results.
5. Explain the probable origin of this woman's sterility.
6. Propose a treatment that may solve the problem of sterility in this woman.

Exercise 3 (5 pts)

In order to know the mechanisms responsible for rejecting or accepting grafts, experiments on mice of the same line or different line are done. The experiments and their results are shown in document 1

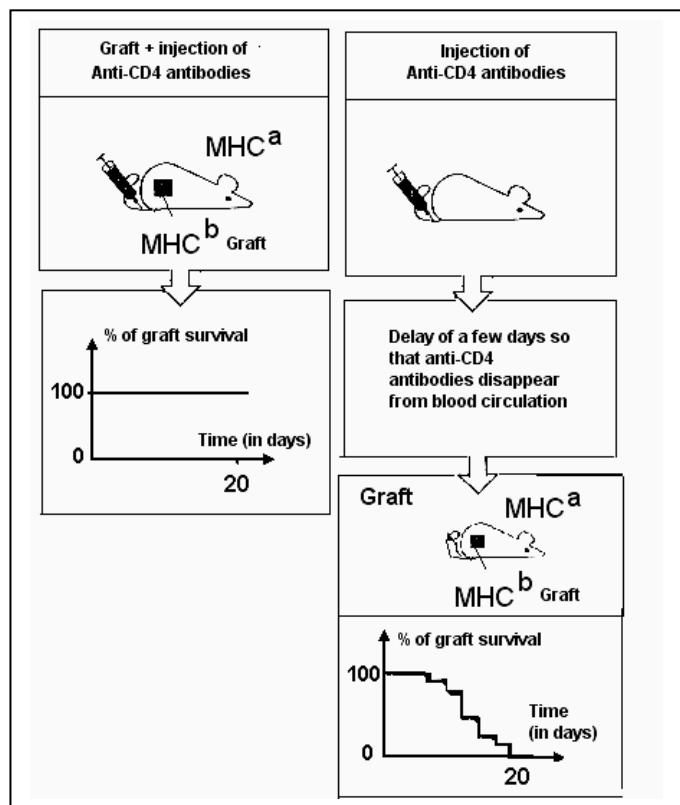


Document 1

1. Interpret these experiments. Draw out the conditions of a graft rejection.

Two types of T-lymphocytes (TL) are recognized: TL4 with a CD4 receptor and TL8 with CD8 receptor. The experiments revealed in document 2 are carried out in order to determine the role of TL involved in graft rejection.

2. Interpret the obtained results. What can be deduced?
3. Justify that these experiments are not sufficient to assure, which of the two types of TL is involved in graft rejection.
Suggest an experiment that allows solving this problem.
4. Explain how the anti-CD4 antibodies intervene in accepting grafts. Draw out a practical medical application.



Document 2

Exercise 4 (5pts)

Myotatic reflex is a muscle response triggered by a stimulus whose receptor is the neuromuscular spindle.

Tapping the Achillean tendon provokes the stretching of the foot's extensor muscle into variable lengths. Simultaneously, we record the nerve message transmitted all along a nerve fiber issued from the neuromuscular spindle of this muscle. The results are shown in document 1.

- Interpret the recordings obtained. What can be deduced?

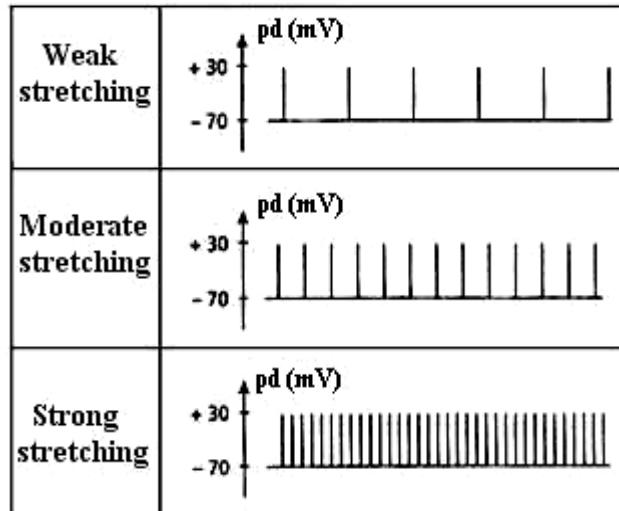
The nerve fibers issued from the neuromuscular spindles are connected, inside the spinal cord either directly or by means of interneurons, to the motor neurons of two muscles: one is an extensor, and the other is a flexor.

The activity of these motor neurons is recorded in response to an afferent message. The results are shown in document 2. For each recording obtained, arrow "1" corresponds to the beginning of the stimulation and arrow "2" corresponds to the end of the stimulation.

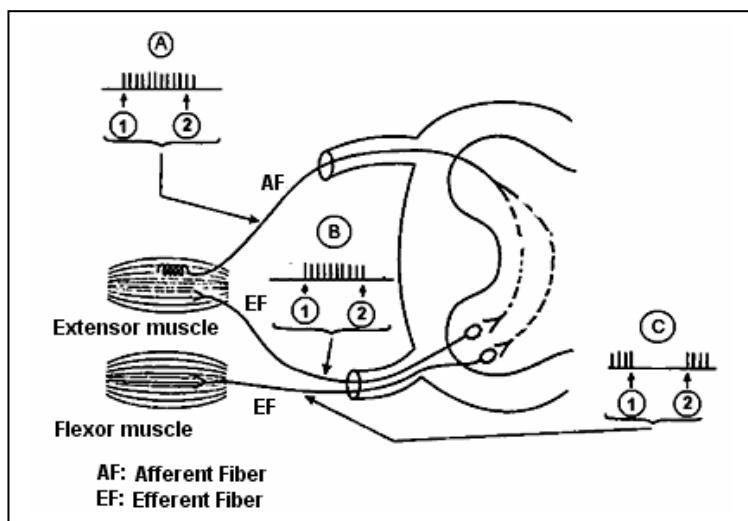
- Compare the obtained recordings.
- Explain the role of the spinal cord in the establishment of this reflex and specify the neuronic circuit implied.

The tensions of the extensor and flexor foot's muscles during this reflex were recorded as shown in document 3.

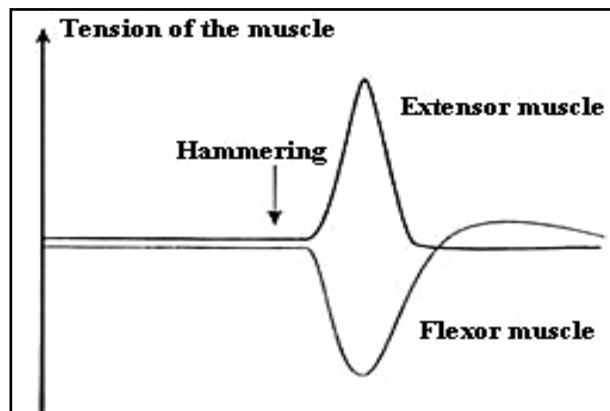
- Specify the movement that was done. Justify your answer based on documents 2 and 3.
- What can these muscles be qualified as? Justify the answer.



Document 1



Document 2



Document 3

الاسم :	مسابقة في مادة "علوم الحياة"
الرقم :	المدة : ثلاثة ساعات
	اسس التصحيح

Exercice 1 (5pts)

- 1- Proportion d'hétérozygotes : $30/1200 = 1/40$. (0.25pt)

Le mode de transmission étant autosomal, pour avoir un enfant atteint à partir des parents sains, il faut que les deux parents soient hétérozygotes. La probabilité pour chacun d'eux d'être hétérozygote est de 1/40. Les parents hétérozygotes ont un risque de $\frac{1}{4}$ d'avoir un enfant atteint. Alors le taux de naissance d'enfants ayant la phénylcétonurie dans cette communauté est de : $1/40 \times 1/40 \times \frac{1}{4} = 1/6400$. (0.5pt)

- 2- Le taux obtenu 1/6400 est supérieur au taux mondial 1/10000. (0.25pt)

Hypothèse : Le mariage consanguin dans cette communauté augmente le risque de phénylcétonurie. (0.25pt)

- 3- Les couples (3-4) et (5-6) normaux ont eu des enfants (7, 10 et 11) atteints par la maladie. Ceci implique que chacun des parents porte l'allèle de la maladie à l'état caché ; alors l'allèle de la maladie est récessif. (m symbole de l'allèle malade). (0.25pt)

La maladie se transmet selon le mode autosomal. Si le mode était gonosomique et le gène porté par la partie propre à Y, tous les individus atteints seraient des garçons et devraient avoir le même phénotype que leur père. L'enfant 10 est malade et son père 6 est sain, alors ce n'est pas le cas.

Si le gène est porté par la partie de X n'ayant pas d'homologue sur Y, les filles 7 et 11 devraient avoir comme génotype Xm /Xm et chacun des parents devrait leur donner un Xm, ce qui n'est pas le cas, car le père de chacune de ces filles est sain.

Si le gène est porté par la partie homologue de X et Y, les enfants atteints 10 et 11 devraient avoir pour génotype, respectivement, Xm /Ym et Xm /Xm. Le père devrait donner un Xm à sa fille et un Ym à son fils et, dans ce cas, il devrait avoir comme génotype Xm /Ym et devrait être malade, ce qui n'est pas le cas. (0.75pt)

- 4- Le fœtus 15 : les parents de ce fœtus sont tous les deux sains mais leurs parents (grands-parents du fœtus) sont hétérozygotes. Alors, la possibilité pour les parents de ce fœtus d'être hétérozygotes est de 2/3 des individus sains. Le risque pour que les deux parents soient hétérozygotes est de $2/3 \times 2/3$ et le risque d'avoir un enfant atteint est de $\frac{1}{4}$. Alors le risque que le fœtus 15 soit atteint est de $2/3 \times 2/3 \times \frac{1}{4} = 1/9$. (0.5pt)

Le fœtus 16 : Sa mère a le même risque que sa sœur 9 d'être hétérozygote : 2/3. Le père fait partie de la communauté et son risque d'être hétérozygote est de 1/40. Si les parents de ce fœtus sont hétérozygotes, il y a possibilité d'avoir $\frac{1}{4}$ des enfants atteint. Alors le risque que le fœtus 16 soit atteint est de : $2/3 \times 1/40 \times 1/4 = 1/240$. (0.5pt)

- 5- Oui. Le fœtus 15 a un risque 1/9 plus élevé d'être atteint par la maladie que le fœtus 16 (1/240). Les parents du fœtus 15 sont des cousins issus d'une famille où la maladie est présente. Par contre, les parents du fœtus 16 ne sont pas issus d'une même famille. Alors, l'hypothèse est valide et le mariage consanguin favorise la maladie.(0.5pt)

- 6- Le mari de la fille 7 est malade mais a un taux sanguin de PAH normal. Alors, on peut dire que la maladie doit avoir une autre origine que la PAH. D'après la donnée, la phénylalanine est métabolisée par la PAH en présence d'un co-facteur le DHBP, ceci nous permet de dire que la cause probable de la maladie chez ce mari est une absence ou un déficit en DHBP. (0.75pt)

- 7- L'enfant a eu l'allèle normal du gène de la PAH de son père et l'allèle normal du gène de DHBP de sa mère, pour cela il est de phénotype normal. (0.5 pt)

Exercice 2 (5pts)

1- (2pts)

Temps (en jours)	0	4	10	12	14	18	21	28
Taux d'œstrogènes (en pg/mL)	60	75	150	240	75	150	200	60
Taux de LH (en mUI/mL)	5	10	13	13	60	10	10	5

Variations des taux de d'œstrogènes et de LH au cours d'un cycle sexuel de la femme

- 2- La femme produit des gamètes normaux ce qui révèle un état normal du contrôle endocrinien et du fonctionnement ovarien. En effet, les résultats obtenus montrent une augmentation du taux d'œstrogènes entre les jours 0 et 10, ce qui indique un développement normal des follicules. Le pic d'œstrogènes au 12^e jour provoque un rétrocontrôle positif sur le complexe hypothalamo-hypophysaire qui se traduit par un pic de LH au 14^e jour provoquant l'ovulation. Alors, le problème réside effectivement au niveau des trompes. (1pt)
- 3- Cette femme peut avoir recours à la technique FIVETE. (0.5pt)
- 4- Pendant 28 jours, les dosages des œstrogènes et de LH chez cette femme montrent un taux presque constant d'œstrogènes qui fluctue entre 29,1 et 30,4 pg/mL et aussi un taux presque constant de LH qui fluctue entre 5,3 et 7,3 mUI /mL tout au long du cycle. Ceci implique qu'il n'y a pas de variations cycliques pour les taux de LH et d'œstrogènes chez cette femme. (0.5pt)
- 5- Les dosages réalisés révèlent une absence de variations cycliques des hormones surtout la décharge des œstrogènes au 12^e jour qui doit déclencher le pic de LH à l'origine de l'ovulation. Alors, la stérilité probable de cette femme est due à un déficit d'œstrogènes bloquant le pic de LH et par suite l'ovulation. Ceci signifie que cette stérilité peut être due à des perturbations soit dans les fonctions de l'hypothalamus, soit de l'hypophyse soit des ovaires. (0.5pt)
- 6- Il faut donner un traitement hormonal à cette femme. Par exemple, on peut lui administrer une forte dose d'œstrogènes qui va stimuler la production d'un pic de LH qui va déclencher l'ovulation. (0.5pt)

Exercice 3 (5pts)

1- La greffe de peau d'une souris CMH^a à une autre de même souche montre une survie de la greffe de 100% qui persiste au delà de 20 jours. Par contre, la greffe de peau effectuée entre deux souris de souches différentes, CMH^a et CMH^b, survit à 100% jusqu'au jour 10, puis le pourcentage de survie diminue et s'annule et la greffe est rejetée au bout de 12 jours. Ce % est encore plus faible quand la souris CMH^b reçoit, un mois plus tard, une deuxième greffe de peau d'une souris CMH^a et la greffe est rejetée au 8^{ème} jour < 12 jours.

De même, l'injection des LT, prélevés d'une souris CMH^b immunisée contre CMH^a, à une souris CMH^b qui reçoit pour la première fois une greffe de peau d'une souris CMH^a, conduit à un rejet de greffe au bout de 8 jours comme dans le cas où la greffe est réalisée pour la deuxième fois.

Ceci implique que la survie de greffe n'est observée qu'entre des individus de même souche. Le rejet de greffe se fait entre des individus de souches différentes et il est plus rapide lors d'un 2^{ème} contact avec le même antigène et les acteurs du rejet de greffe sont les LT.

Donc le rejet de greffe se fait entre deux souches différentes et nécessite la présence des LT. (1.5 pt)

2- La greffe entre des souches différentes, réalisée en même temps que l'injection d'anticorps anti- CD4, réussit et son pourcentage de survie est maximal. Par contre, si cette injection d'anticorps anti- CD4 est réalisée quelques jours avant la greffe, dans un délai pour que ces anticorps disparaissent de la circulation sanguine, la greffe est rejetée. Ceci signifie que les anticorps anti-CD4, quand ils sont présents, empêchent le rejet de greffe. Donc les LT4 ont un rôle dans le rejet de greffe.(1pt)

3- Cette expérience est insuffisante pour trancher lequel des deux types de lymphocytes est impliqué dans le rejet de greffe. La question 2 révèle que les LT4 ont un rôle dans le rejet de greffe mais on ne peut pas trancher s'ils sont les seuls ou bien si les LT8 entrent en jeu aussi. (0.5pt)

Pour cela, il faut reprendre les expériences du document 2, et ajouter deux autres souris. A la 1^{ère} souris, on injecte des anticorps anti- CD8 seulement et à la 2^{ème} souris, des anti-CD4 et anti- CD8, avant la réalisation de la greffe entre des souches différentes et on observe l'évolution de la greffe si elle survit ou si elle est rejetée pour trancher qui est responsable du rejet de greffe : les LT4 ou les LT8 ou bien les deux à la fois. (1pt)

4- Les anticorps spécifiques des CD4 sont injectés en même temps que la greffe, ils se fixent sur les récepteurs des LT4 et les bloquent. Les LT4 bloqués ne s'activent pas, ne prolifèrent pas et ne se différencient pas en cellules sécrétrices d'interleukines 2. Ainsi les LT8 ne sont pas activés et la greffe réussit. (0.75pt)

Les anticorps anti CD4 peuvent être utilisés comme médicaments immunsupresseurs en cas de greffe. (0.25 pt)

Exercice 4 (5pts)

- 1- A un étirement faible du fuseau neuromusculaire, on obtient 6 potentiels d'action (PA) de 100mV d'amplitude chacun. Au fur et à mesure que l'étirement augmente (étirement fort), le nombre de potentiels d'action augmente (34 PA) tandis que l'amplitude de ces PA reste constante. Alors la réponse de la fibre augmente seulement en fréquence avec l'augmentation de l'intensité du stimulus. Donc le message nerveux est codé en fréquence de P.A. (1pt)
- 2- L'enregistrement (A) obtenu sur la fibre afférente du muscle extenseur montre une fréquence de PA de même amplitude entre 1 et 2. Cet enregistrement est identique à celui obtenu en (B) au niveau de la fibre efférente du muscle extenseur. Par contre, l'enregistrement (C) au niveau de la fibre efférente du muscle fléchisseur montre l'absence des PA pendant la période de stimulation. (1pt)
- 3- Au niveau de la moelle épinière, la fibre afférente du muscle extenseur entre en synapse directe avec le motoneurone du muscle extenseur. Cette synapse est excitatrice, elle conduit à un message nerveux dans la fibre efférente du muscle extenseur. De même, cette fibre afférente du muscle extenseur entre en contact, au niveau de la moelle, avec un interneurone par l'intermédiaire d'une synapse excitatrice et, l'interneurone entre en contact avec le motoneurone du muscle fléchisseur par l'intermédiaire d'une synapse inhibitrice, alors le message nerveux est inhibé. La moelle épinière, par l'intermédiaire de ses différentes synapses, est capable de coordonner l'activité des différents motoneurones et des muscles dont ils dépendent. (1.5pt)
- 4- Mouvement d'extension. (0.25pt). La tension du muscle extenseur a augmenté indiquant que le muscle s'est contracté suite à l'arrivée du message nerveux par la fibre efférente. La tension du muscle fléchisseur a diminué indiquant que le muscle s'est relâché car il n'a reçu aucun message nerveux. (0.75pt) A
- 5- Muscles antagonistes (0.25pt). Car l'un se contracte, l'autre se relâche, en même temps, pour effectuer le mouvement d'extension du pied. (0.25pt) A

الاسم:
الرقم:

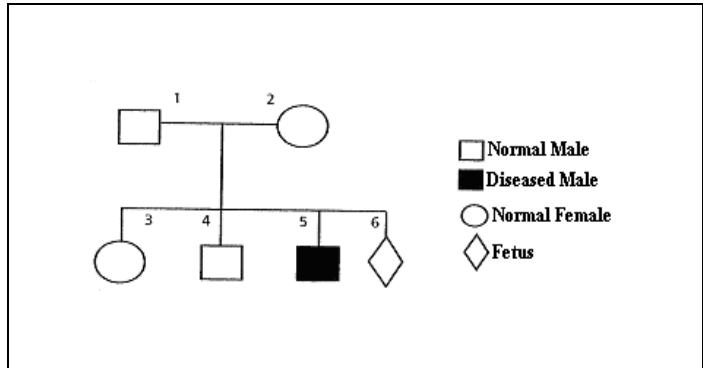
مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات

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.



1-Determine, by referring to the pedigree, whether the allele responsible for the disease is dominant or recessive.

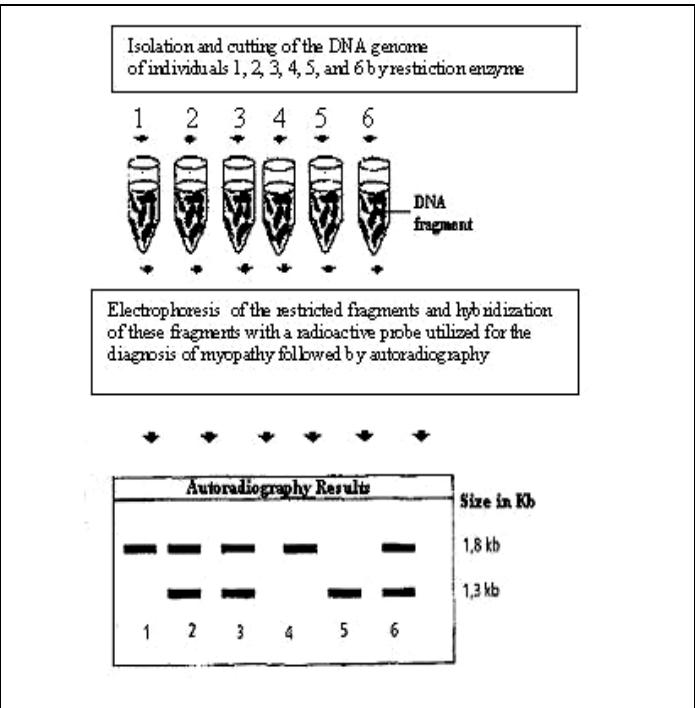
2-Indicate the genotypes of the parents. Justify the answer.

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

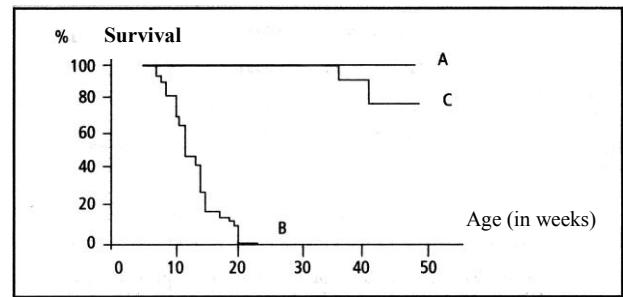
A gene therapy is applied for the first time on mice attaining myopathy similar to Duchene myopathy in humans. This technique consists of injecting the dystrophin gene into a diseased organism by means of a virus vector which is harmless to mice and human species. After this treatment, transversal sections 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 2 reveals the

Document 1

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

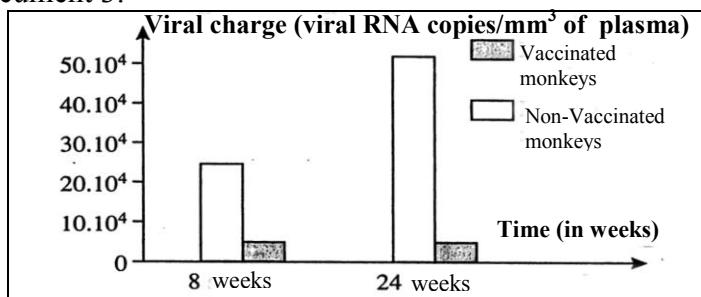
- 2-Draw, on the same graph, the curves obtained from the tabulated data.

- 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)

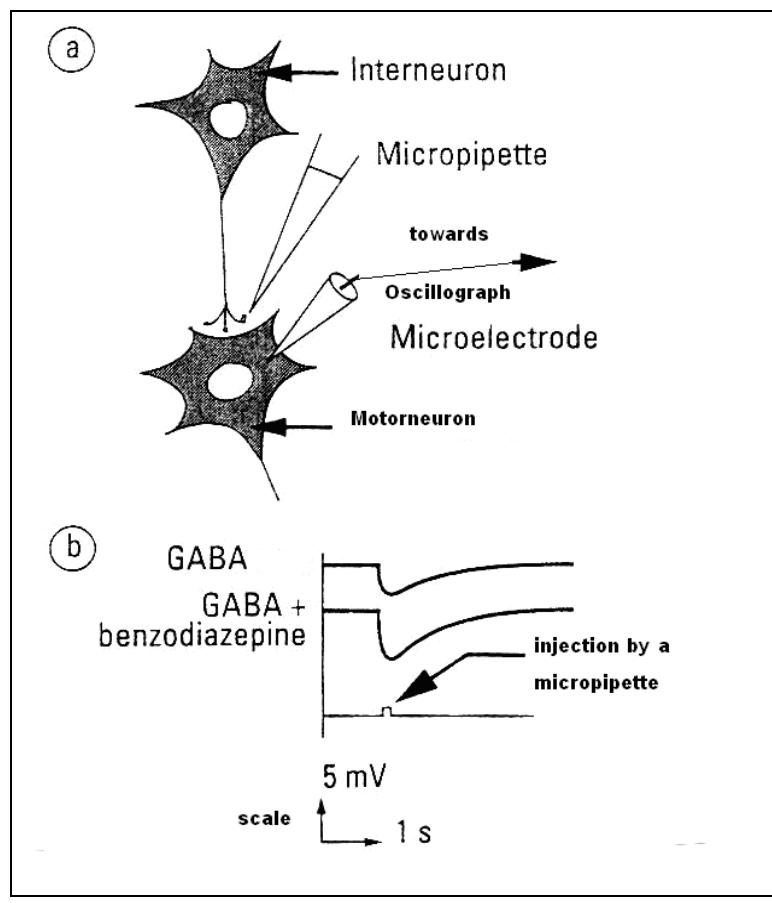
Nowadays, certain molecules that belong to the benzodiazepines family are used in treating anxiety.

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.

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

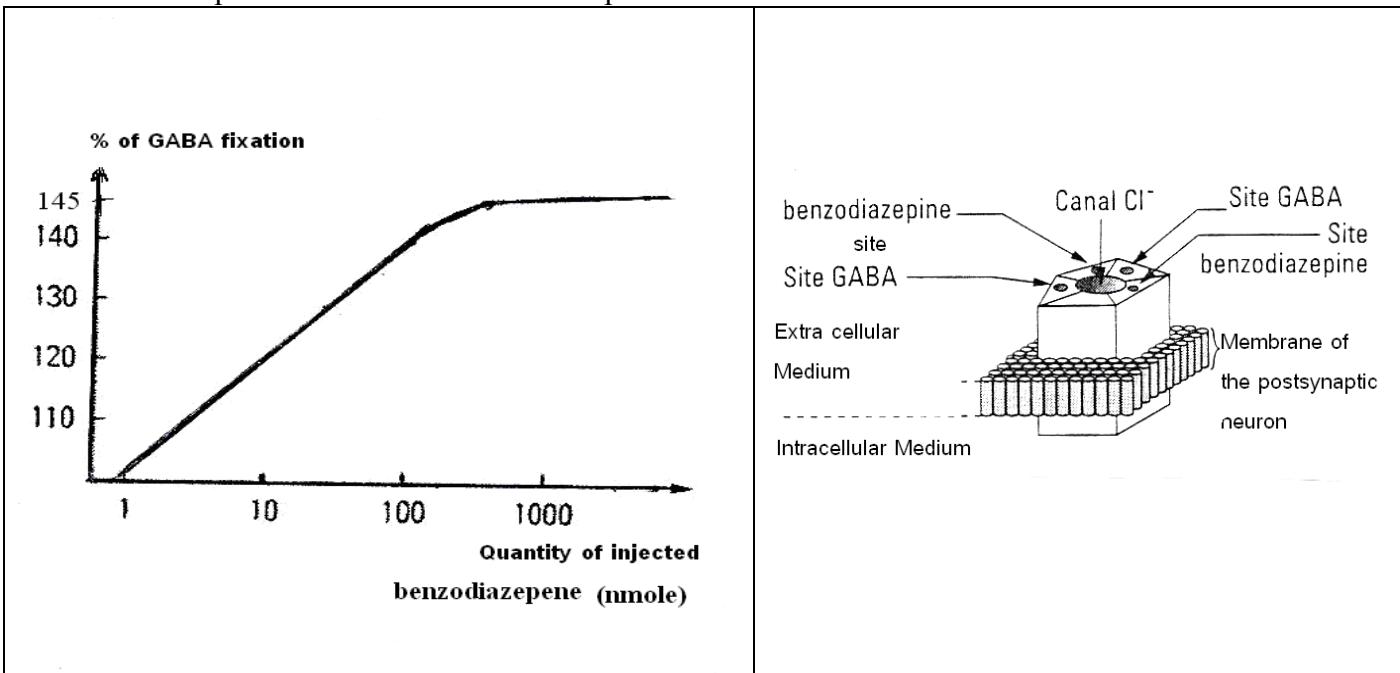
Researchers found that rats administered a chemical substance, called picrotoxin, exhibit involuntary muscular contractions accompanied by signs of anxiety.

- Specify the effect of picrotoxin and benzodiazepine 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

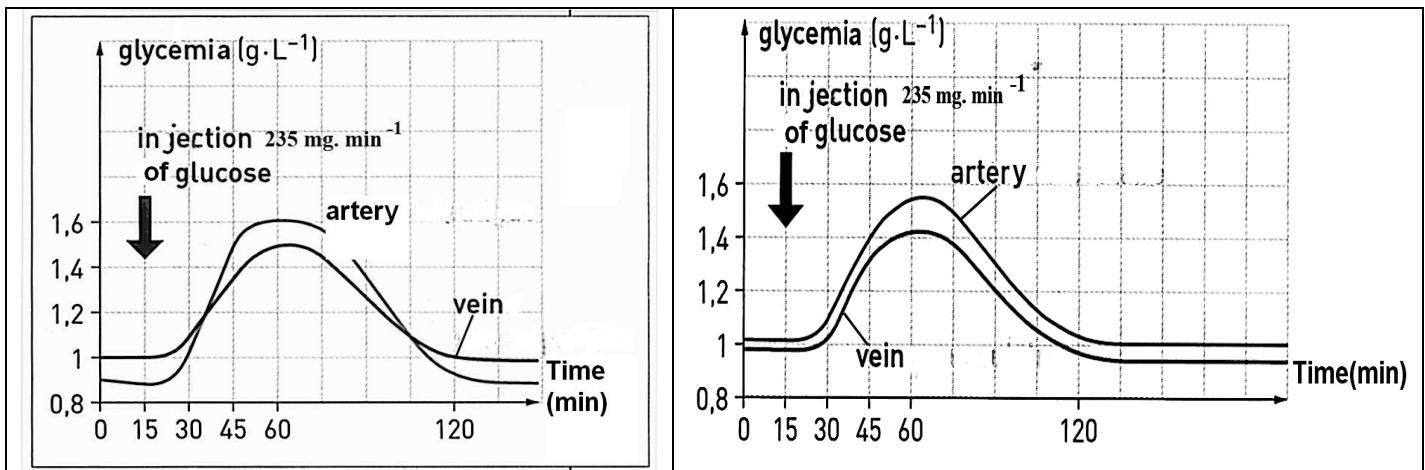
Document 3

- Represent, in a table, the data shown in document 2.
- What conclusions can be derived from the analysis of document 2?
- 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.



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.

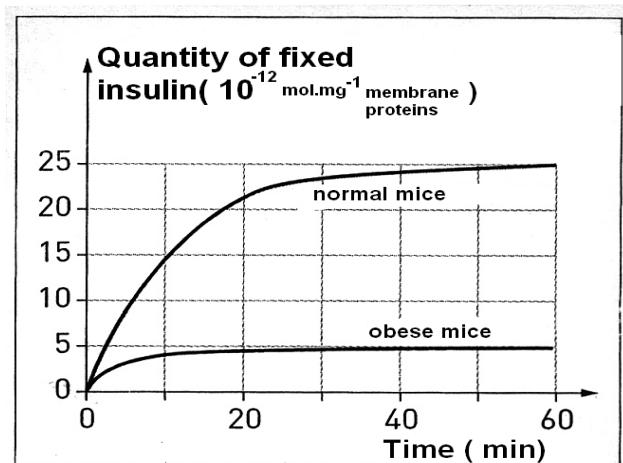
Experiment 2: We inject labeled glucagon by a radioactive isotope into a normal mouse. The autoradiography of the hepatic cells of this mouse reveals the fixation of glucagon at certain points of the plasma membrane. However, the injection of labeled insulin by a radioactive isotope leads to a similar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

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.



Document 3

Exercise 1 (5 pts)

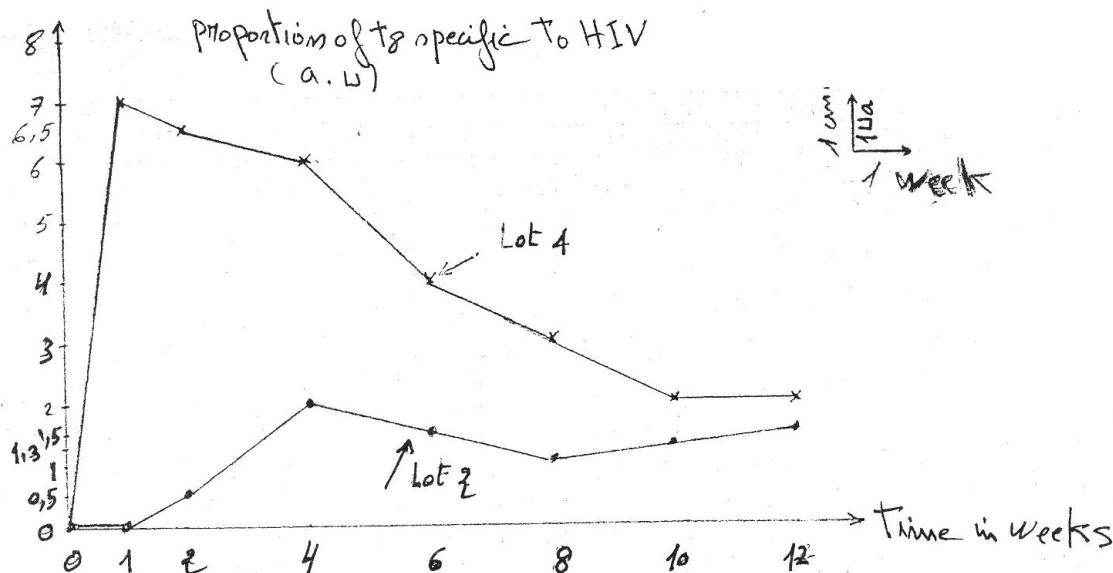
- 1- Couple 1 and 2 who are normal have a child No.5 affected by the disease. This means that the allele causing the disease(m) is recessive masked by the dominant normal allele (N) found in the parents. ($\frac{1}{2}$ pt).
 - 2- Father 1 is healthy and possesses the normal allele N on chromosome X. Thus, his genotype is $X^N Y$ ($\frac{1}{2}$ 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 $X^N X^m$ (1/2pt).
 - 3- The father produces two gametes of equal probabilities $\frac{1}{2} X$ and $\frac{1}{2} Y$. The mother produces two gametes of equal probabilities $\frac{1}{2} X^N$ and $\frac{1}{2} X^m$. All the girls would inherit an X^N from their father and would be normal. Therefore, the probability to have affected girls is Zero. ($\frac{1}{4}$ 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 $\frac{1}{2}$ or 50% from all boys or $\frac{1}{4}$ or 25% of all children. ($\frac{1}{4}$ 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.($\frac{1}{2}$ 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 ($\frac{1}{2}$ 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. ($\frac{1}{2}$ pt)This means that the gene treatment improves the survival of the myopathic mice ($\frac{1}{2}$ 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. ($\frac{1}{2}$ 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.u 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 $25 \cdot 10^4$ copies/mm³ of plasma after 8 weeks following viral infection and increases to reach $50 \cdot 10^4$ 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 $5 \cdot 10^4$ 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 (I_{psp}), 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 picrotoxin 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.
picrotoxin 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 its 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 indispensable 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 reaches 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 (~ 0.99 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 15th min and after the 105th min where glycemia is low and storage takes place between 35th min and 105th 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 $25 \cdot 10^{-12}$ mol/mg of membrane proteins at 50 min. while, in obese mice the fixed quantity of insulin increases slightly to reach a maximum of $5 \cdot 10^{-12}$ 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)

الاسم:

مسابقة في مادة علوم الحياة

الرقم:

المدة ثلاثة ساعات

Answer the following exercises.

Exercise 1 (5 Pts)

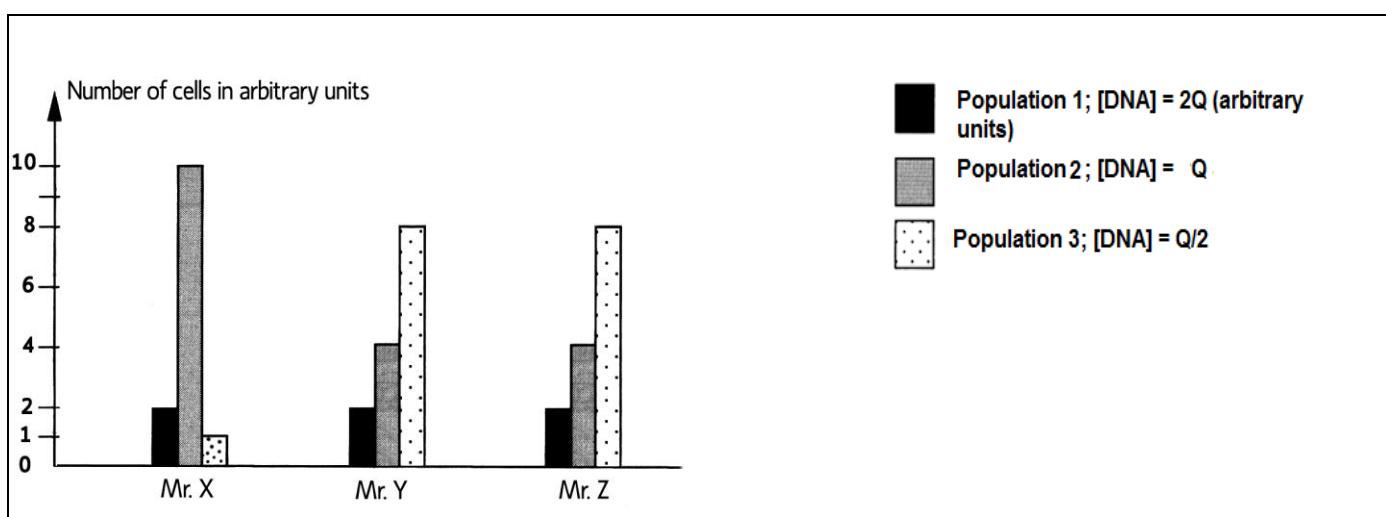
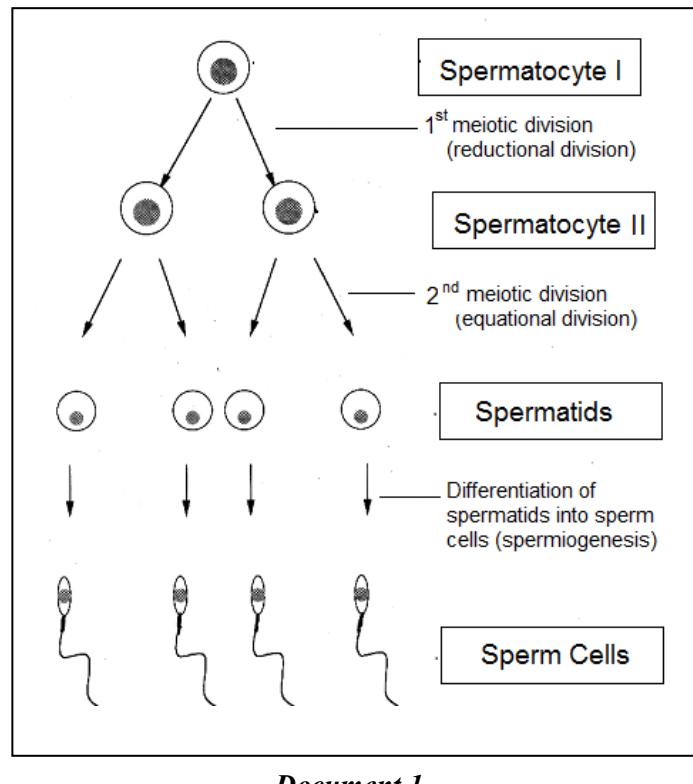
Mr. X and Mr. Y are two adult sterile men. We perform different tests to specify the origin of this defect.

Document 1 shows certain stages of spermatogenesis. The germ cells, whose names are framed in boxes, are found in the wall of the seminiferous tubules.

- 1- Describe the different stages of spermatogenesis represented in document 1.

We perform a quantitative study for the amount of DNA of the germ cells extracted directly, by biopsy, from a fragment of the testicles of these two sterile men and that of a fertile man Mr. Z. Three different populations of germ cells are obtained. The number of each cell population, as well as the amount of DNA in each of them are shown in document 2.

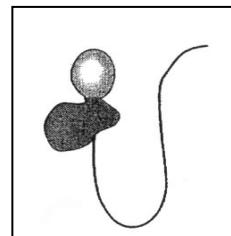
- 2- Indicate the germ cells corresponding to each of the three populations shown in document 2. Justify the answer.



- 3- Explain the variation of the number of germ cells of the three populations in the fertile man Mr. Z.
4- Determine, by referring to document 2, the cause of sterility of Mr. X.

Microscopic observations of the semen of Mr. Y showed sperm cells, where the majority of these cells showed an aspect identical to that schematized in document 3.

- 5- Explain the origin of the sterility of Mr. Y.

**Document 3**

Exercise 2 (5 pts.)

RU 486 (mifepristone) is a molecule that has a contragestive action. It prevents the implantation of the embryo, and terminates early gestation.

Document 1 presents the time of the appearance of menses and the variation of the amount of progesterone in a control group of women and in women having absorbed RU 486.

- Determine, from document 1, the effect of RU 486 on the secretion of progesterone and on the appearance of menses.

We inject three lots of female rats with the same quantity of different molecules labeled with a radioactive element called tritium (^3H). Fifteen minutes following the injection, we remove the uterus of these female rats. Autoradiography was done on thin sections of the uterine mucosa. We count the silver grains that became black by radioactive emission and which reveal the concentration of radioactive molecules present in the nuclei of the uterine mucosa cells.

Document 2 shows the results obtained on 300 uterine mucosa cells.

Lots	1	2	3
Injections done	RU486 labeled with tritium	Progesterone labeled with tritium	Equal quantities of non-labeled RU486 and progesterone labeled with tritium
Average number of silver grains (grains/per cell)	8	8	2

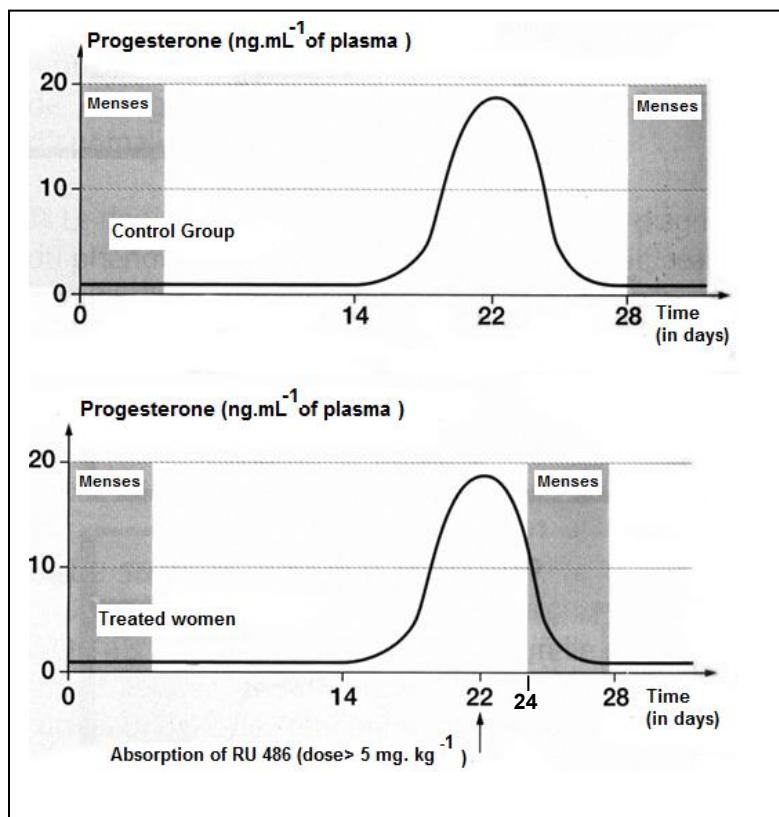
Document 2

- Construct a histogram that translates the data of document 2.

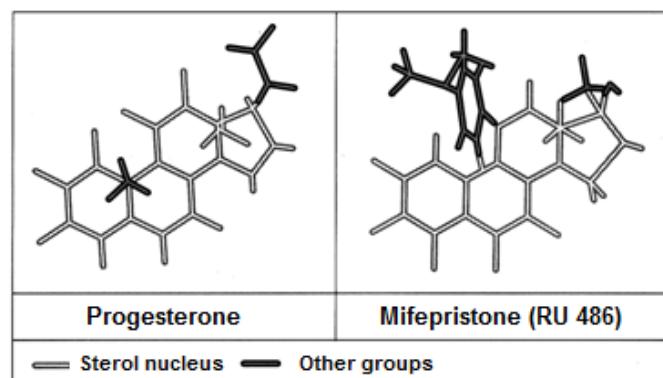
- Interpret document 2 and deduce the mode of action of RU 486.

Document 3 reveals the structure of RU 486 and that of progesterone molecules.

- Explain, by referring to the information derived from documents 2 and 3 and to the acquired knowledge, the results obtained in the treated women (document 1).



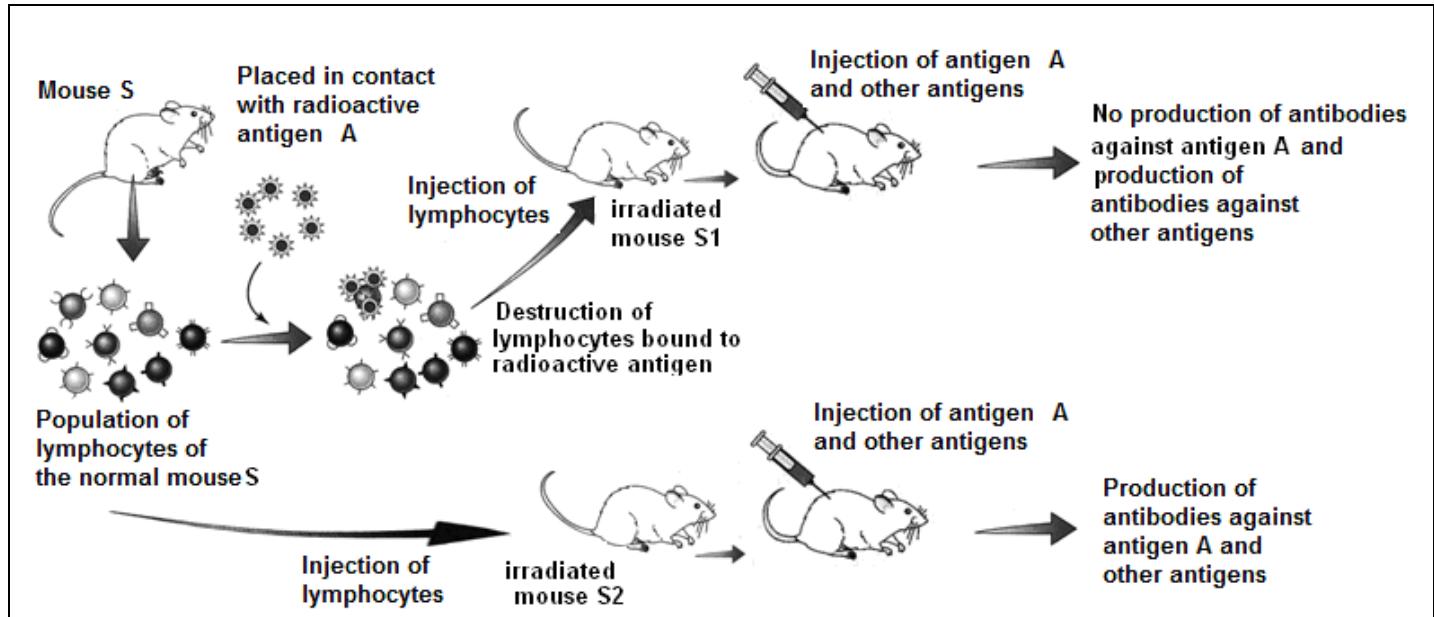
Document 1



Document 3

Exercise 3 (5 pts.)

In the framework of studying clonal selection of B lymphocytes which are at the origin of antibodies, we perform several experiments on mice of line S that are not immunized against an antigen A (document 1). According to the theory of clonal selection, each lymphocyte acquires during its development the ability to react with a specific antigen, even without being previously exposed to it.



Document 1

N.B. irradiation leads to the destruction of immune cells.

1- Refer to document 1 to show that:

- 1-1 B lymphocytes are ready to respond to an antigen before encountering it;
- 1-2 B lymphocytes possess a surface receptor;
- 1-3 The immune response is specific.

To determine the phenomenon responsible for the secretion of the most effective antibodies, researchers performed the following experiment. They injected mice with a chemical substance recognized by the immune system as a foreign antigen. This antigen is characterized by having several antigenic determinants.

At different times following the injection, researchers sacrificed the mice and dissected their lymphatic ganglia to detect the B lymphocytes which recognize the injected antigen. Document 2 shows the results of this experiment.

Time since the injection of the antigen (in days)	Aspect of the lymphatic ganglia	Number of the different detectable B lymphocytes clones	Efficiency of the immune response
5	Beginning of swelling	10	Average
10	Strong swelling	1 or 2	Very high

Document 2

2- Explain the swelling of the lymphatic ganglia mentioned in document 2.

3- To what can we attribute the number of B Lymphocytes clones 5 days following the injection of the antigen?

4- Formulate a hypothesis that explains the decrease in the number of B lymphocytes clones detected 10 days following the injection of the antigen.

Exercise 4 (5 pts.)

Pain sensation necessitates the intervention of several neuronic circuits. Document 1 represents the structures implicated in pain sensation and in its modulation.

We study certain mechanisms which control the transmission of nociceptive message or pain message, in an attempt to show the mode of action of enkephalin and morphine.

In two different experiments 1 and 2, we stimulate at S1 the nociceptors of the skin using the same effective intensity and we record the electric activity of three nerve fibers:

- Sensory nerve fiber by an electrode E1 connected to oscilloscope O1;
- Nociceptive medullary nerve fiber by an electrode E2 connected to oscilloscope O2; and
- Nerve fiber of enkephalin interneuron by an electrode E3 connected to oscilloscope O3.

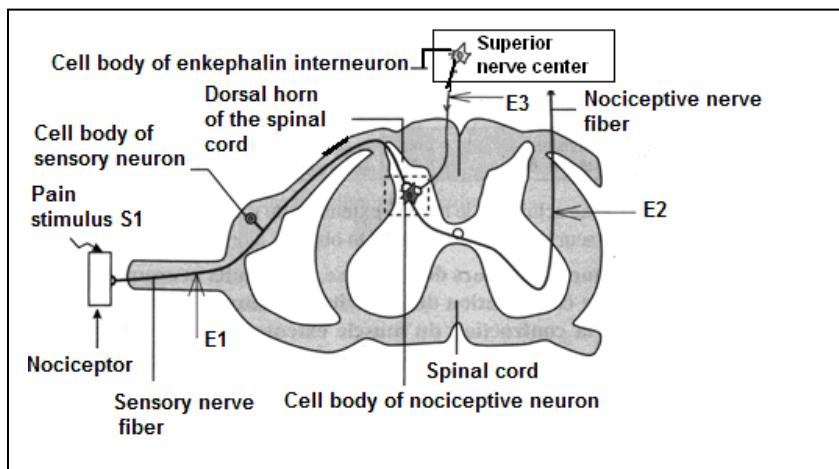
The conditions and the obtained results are shown in document 2.

1- Draw out, in reference to document 2, the role of enkephalin. Justify the answer.

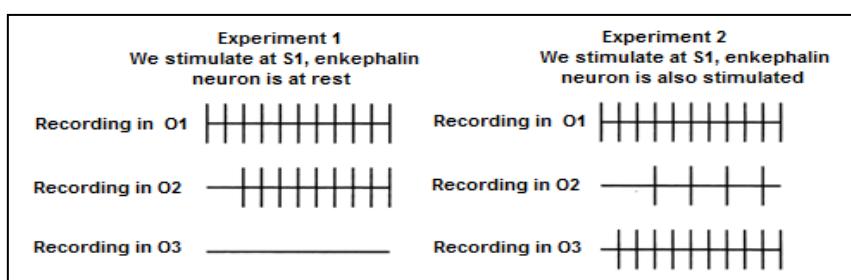
2- Explain how the results recorded by O2 in experiment 2 put in evidence the integrative role of the nociceptive neuron.

In the framework of studying the action of morphine on the medullary nociceptive neuron, we perform experiments 3 and 4.

Experiment 3: By the help of a microelectrode, we record the activity of the medullary nociceptive neuron at the level of the dorsal horn after an intense electric stimulation of the sensory fibers.

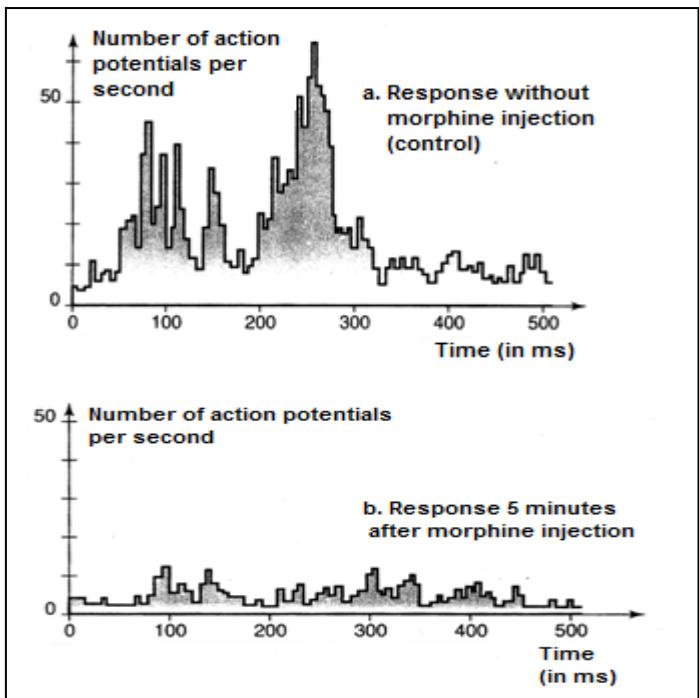


Document 1



Document 2

N.B. Each vertical line corresponds to an action potential (A.P.)



Document 3

Experiment 4: Under the same conditions of experiment 3, we also record the activity of the medullary interneuron after the injection of a morphine dose by a micropipette at the level of the dorsal horn. Document 3 shows the obtained results.

3- Determine, from document 3, the role of morphine.

4- Morphine and enkephaline are agonist substances. Justify this statement.

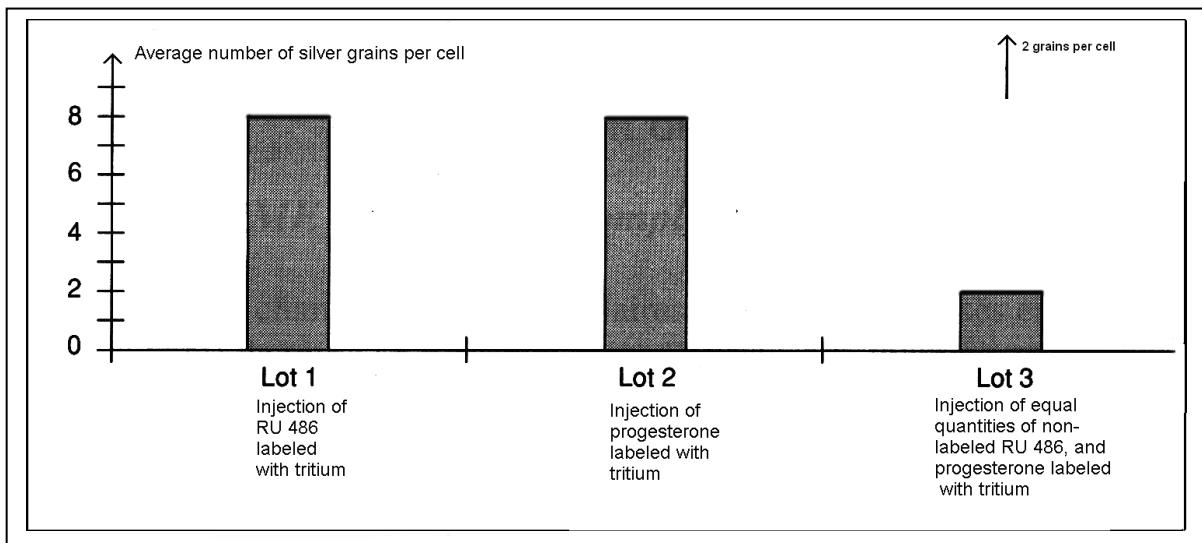
Exercise 1 (5 Pts)

- 1- During the first meiotic division (reductional division), spermatocyte I produces two spermatocytes II that are subjected to the second meiotic division (equational division) , each producing two spermatids. Then, the spermatids differentiate into sperm cells (spermiogenesis). (1/2 pt)
- 2- Population 1 corresponds to spermatocytes I because the quantity Q is duplicated during the S phase of interphase and becomes $2Q$ in spermatocyte I that has $2n$ chromosomes of 2 chromatids each.($\frac{1}{2}$ pt)
Population 2 corresponds to spermatocytes II because after the reductional division of meiosis we obtain spermatocytes II that have n chromosomes each of 2 chromatids corresponding to the quantity Q of DNA.($\frac{1}{2}$ pt)
Population 3 corresponds to spermatids or sperm cells because after the equational division of meiosis, we obtain 4 cells (spermatids) each having n chromosomes of one chromatid each corresponding to the quantity $Q/2$ of DNA. This same quantity remains constant after spermiogenesis that gives sperm cells. ($\frac{1}{2}$ pt.)
- 3- In the fertile man, the number of germ cells is doubled from 2 to 4 then to 8 passing from population 1 to population 3 because the number of cells is doubled after each meiotic division. Each spermatocyte I produces 2 spermatocytes II and each spermatocyte II produces 2 spermatids (1-2-4) (1/2 pt)
- 4- In the sterile man X, the number of spermatocytes I is the same as in the fertile man (2 a.u.), but the number of spermatocytes II in the sterile man is much higher than that in the fertile man ($10 \text{ a.u} > 4 \text{ a.u}$). On the other hand, the number of spermatids or sperm cells in the sterile man is abnormally lower than that in the fertile man ($1 \text{ AU} < 8 \text{ AU}$). Therefore, not all spermatocytes II had divided into spermatids during meiosis. Hence, the cause of sterility in man X is an abnormal meiosis, which is blocked at the stage of spermatocytes II leading to an insufficient number of sperm cells (oligospermia) (1 pt)
- 5- Document 2 reveals that in the sterile man Y, the number of cells of the three populations is the same as in the fertile man Z; this indicates that meiosis took place normally in man Y, that is why he has a normal number of spermatids and sperm cells, therefore, oligospermia did not happen . (1/2 pt) On the other hand, document 3 reveals one type of sperm cell that has a normal flagellum and a normal head, but the middle piece is larger than in the normal sperm cell. This is due to the non elimination of residual cytoplasm .($\frac{1}{2}$ pt) Hence, the origin of sterility of man Y is the abnormal spermiogenesis (1/2 pt)

Exercise 2 (5 Pts)

- 1- RU 486 has no effect on the secretion of progesterone because in the control and treated women we observe an increase in the amount of progesterone from almost null level to about 18 ng/mL of plasma from day 14 till day 22. Then, this amount of progesterone decreases progressively until it reaches the initial value(almost null) on day 26 and remains constant at this value until day 28.($\frac{1}{2}$ pt)
RU 486 leads to the early appearance of menses because we observe the appearance of menses on day 24 in the treated women 4 days before the appearance of menses in the control group which occurs on day 28.($\frac{1}{2}$ pt).

2-



Histogram showing the average number of the silver grains per cell in function of the injections done in 3 lots (2 pts)

- 3- The average number of silver grains per cell is the same (8 grains/ cell) for the two lots 1 and 2 injected by RU 486 labeled with tritium and progesterone labeled with tritium respectively. This signifies that progesterone and RU 486 fix in the same manner at the level of the nucleus of the uterine mucosa cell. On the other hand, this number decreases to 2 grains/cell in lot 3 injected with non-labeled RU 486 and progesterone labeled with tritium. This indicates that RU 486 prevents the fixation of a large quantity of progesterone (75%) in the nucleus of endometrial cells. Therefore, RU 486 is a competitive substance to progesterone. (1 pt)
- 4- The two molecules, progesterone and RU 486, have a similar structure at the level of sterol nucleus (doc .3). This allows RU 486 to fix on the progesterone nuclear receptors, and since RU 486 fixes more efficiently than progesterone by occupying almost 75% of the progesterone receptors , it prevents progesterone from performing its action.(Lot 3, doc. 2). This inhibits protein synthesis leading to the sloughing off of the surface layer of the endometrium and to the early appearance of menstruation (1 pt).

Exercise 3 (5 Pts)

1-1 B lymphocytes are ready to respond to an antigen before encountering it, because we observe anti-A antibodies production in mouse S2 which received all lymphocytes. On the contrary, there is no production of anti-A antibodies in mouse S1 which received all lymphocytes except the lymphocytes that can recognize antigen A (already destroyed by radioactivity after the fixation on the radioactive antigen A). This indicates that the lymphocytes that recognized antigen A were present before any contact with this antigen. (1 pt)

1-2 The experiment shows that the radioactive antigen is fixed on plasma membrane of the B lymphocytes that recognized this antigen. This implies the presence of a membrane receptor capable of the fixation of this antigen. (1 pt)

1-3 The immune response is specific to this antigen because we observe the production of antibodies against all antigens except anti-A antibodies in mouse S1 that received all lymphocytes except the lymphocytes that can recognize antigen A.(1 pt).

- 2-** The swelling started after 5th day and the strong swelling observed on the 10th day correspond to the activation of lymphocytes(T4 and BL) and to their rapid and important proliferation on day 10 leading to the formation B lymphocytes clones that recognize the antigen. (**1 pt**)
- 3-** The 10 clones of BL 5 days following the injection is attributed to the presence of 10 different antigenic determinants at the level of the antigen. (**½ pt**)
- 4-** Hypothesis: There is an important clonal selection of B lymphocytes where only lymphocytes that can recognize the most effective antigenic determinant are kept. (**1/2 pt**)

Or

There is an important clonal selection of B lymphocytes where only lymphocytes that can recognize the most frequent antigenic determinant are kept.

Exercise 4 (5 pts)

- 1-** The role of enkephalin is to decrease pain sensation (**½ pt**), because we observe in O2 a pain message of 4 AP after the stimulation of the sensory nerve fiber and the enkephalin interneuron (experiment 2), which is lower than the message recorded in O2(9 AP) after the stimulation of the sensory nerve fiber only (experiment 1). This shows that the enkephalin liberated after the stimulation of the interneuron inhibited partially the transmission of the pain message. (**1 pt**)
- 2-** The nociceptive neuron has an integrating role. It performs spatial summation for two nerve messages coming from the presynaptic fibers, the first is an EPSP caused by the sensory neuron (11 AP in O1) and the second is an IPSP caused by enkephalin neuron (10 AP in O3). The result of this summation is a message of weaker frequency (4 AP in O2). (**1 1/2 pt**)
- 3-** The frequency of action potentials at the level of the medullary neuron is high, it varies from 5 AP/s till a maximum of 65 AP/s after the stimulation of the sensory nerve fiber in the absence of morphine (doc. 3a). On the other hand, this frequency decreases sharply and fluctuates between 2 and 12 AP/s after the stimulation of the sensory nerve fiber with the injection of morphine (doc. 3b). This indicates that morphine inhibits the activity of the nociceptive neuron by decreasing the frequency of AP that leads to the decrease in pain sensation. (**1 1/2 pt**)
- 4-** They are agonists because morphine and enkephaline have the same effect of decreasing pain sensation at the level of the nociceptive medullary neuron. (**1/2 pt**).

الاسم:
الرقم:مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات**Answer the following Exercises:****Exercise 1 (5 points) Transmission of hereditary characteristics in Drosophilae**

In an attempt to study autosomal heredity in drosophilae, we cross a drosophila of pure race having gray body, red eyes and well-formed wings with another drosophila of pure race having black body, purple eyes and deformed wings.

We obtain in F1 100% drosophilae having gray body, red eyes and well-formed wings.

- 1-** Indicate the dominant allele and recessive allele for each of the studied genes.

We perform, in drosophilae, two other experimental crosses 1 and 2, represented in the adjacent figures.

- 2-** Name the type of the performed crosses.

- 3-** Explain the results obtained in the first cross.

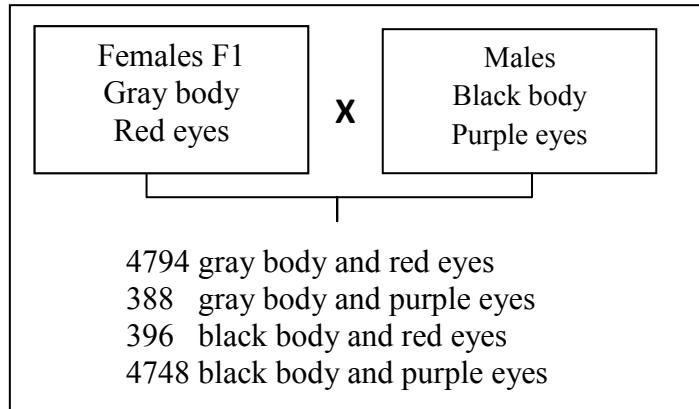
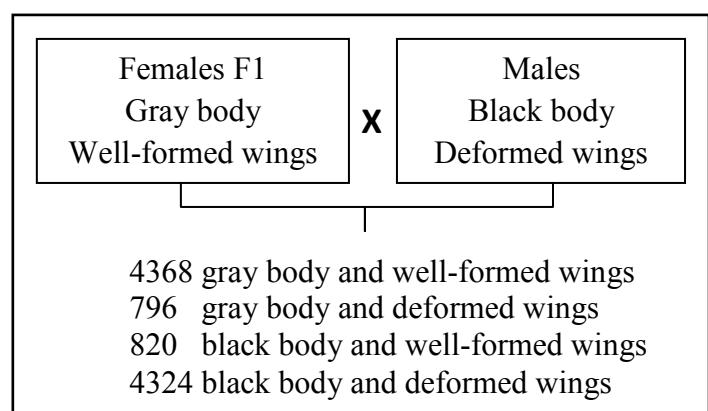
The results of the two crosses put in evidence the existence of a certain type of genetic recombination during meiosis in female drosophilae F1.

- 4-** Name this type of genetic recombination and illustrate by explanatory schematic drawings the behavior of the corresponding chromosomes of the second cross.

- 5-** Determine, by referring to the first and second crosses, whether the genes responsible for eye color and form of wings are linked or independent.

- 6-** Calculate the percentage of recombination between the studied genes in each of the two crosses.

- 7-** Knowing that the percentage of recombination between the genes of eye color and form of wings is 8%, establish a factorial map which reveals the location of the three studied genes on a chromosome.

First Cross**Second Cross**

Exercise 2 (5 points)

Types of diabetes

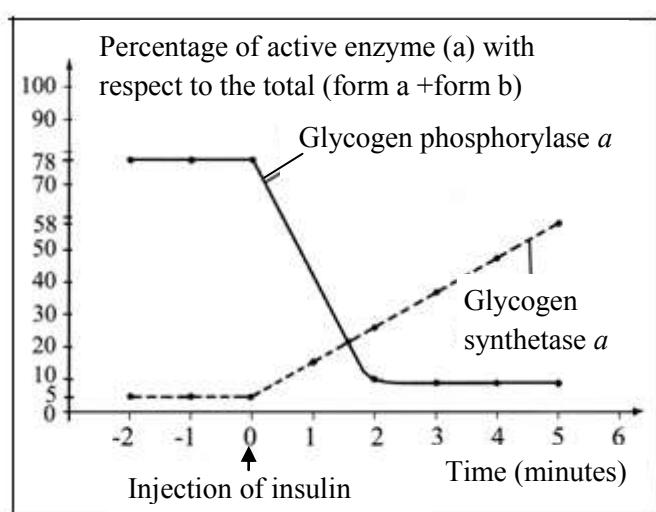
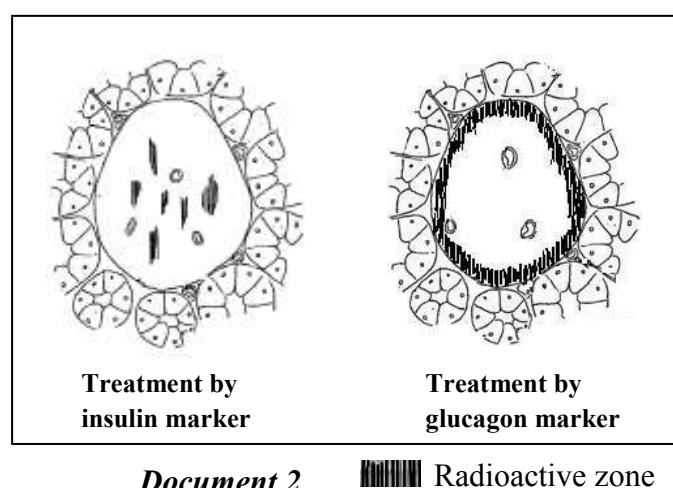
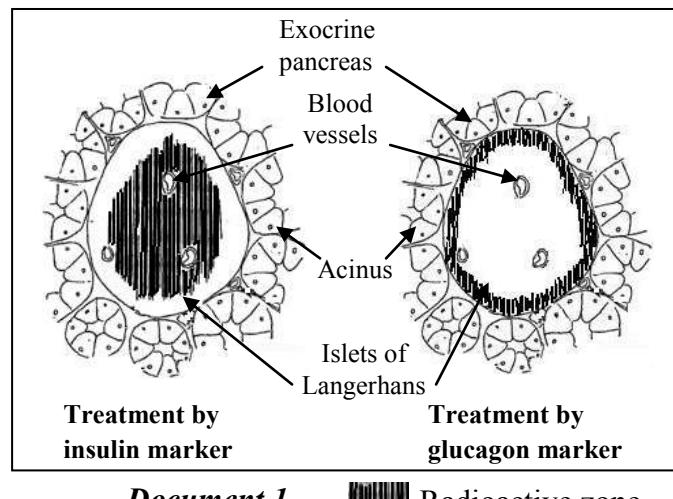
In order to determine the possible origin of diabetes in animals, we treat microscopic cross sections of the pancreas by a radioactive marker of insulin or by a radioactive marker of glucagon. Document 1 reveals schematically the results for animal X having normal glycemia, and document 2 reveals the results for animal Y suffering from severe hyperglycemia.

- Pick out, from document 1, the structure which is at the origin of the secretion of insulin and glucagon, and their locations in the cross sections. Justify the answer.
- Specify the cause of hyperglycemia in animal Y. Justify the answer by comparing documents 1 and 2.

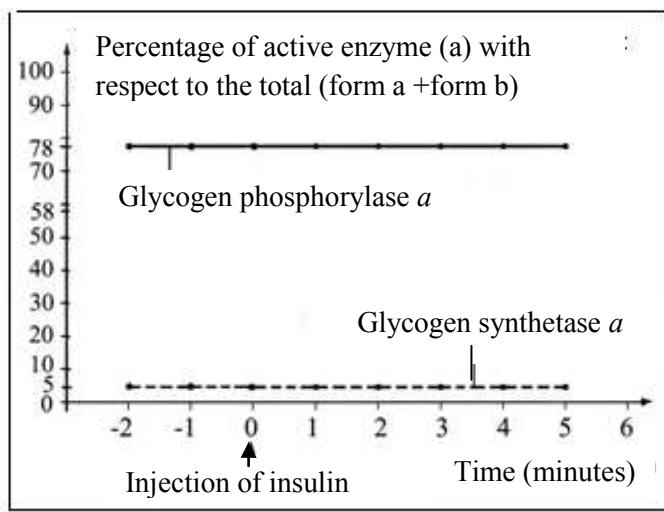
Animal Z, which has another form of diabetes, shows a cross section of the pancreas similar to that shown in document 1.

- Formulate two hypotheses which explain the possible origin of diabetes in animal Z.

Glycogen synthetase and glycogen phosphorylase are two hepatic enzymes implicated in the synthesis of glycogen and in the degradation of glycogen respectively. Each enzyme exists in two forms: an active form a (functional) and an inactive form b (non functional). The documents below reveal the variations in the percentages of these enzymes in function of time in animal X (document 3) and animal Z (document 4).



Document 3



Document 4

- Do the results obtained in documents 3 and 4 validate the hypotheses formulated in question 3? Justify the answer.
- Explain, by referring to document 3 and acquired knowledge, the mode of action of insulin on hepatic cells and its effect on glycemia.

Exercise 3 (5 points) Specificity of Lymphocytes and Antibodies

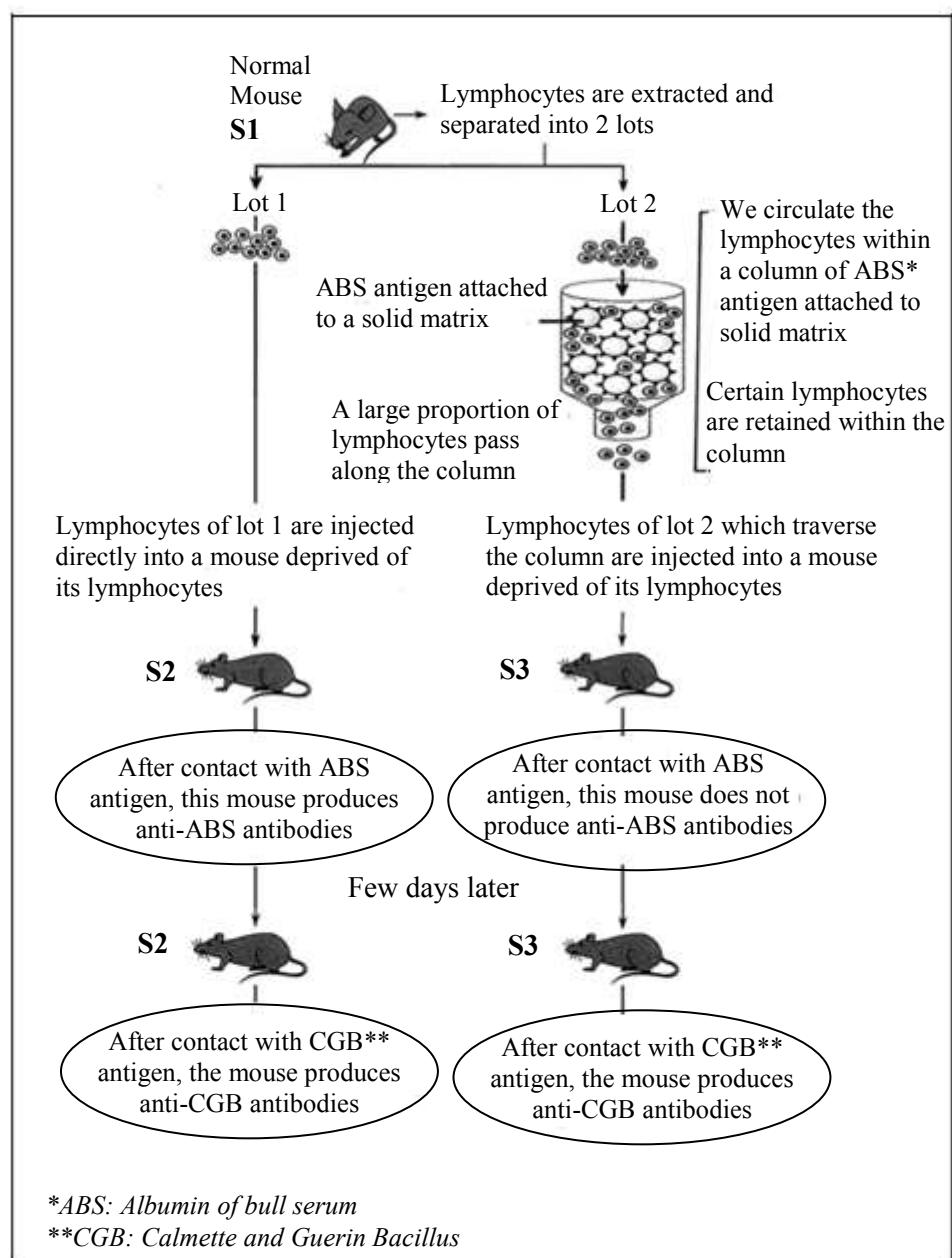
In the midst of the twentieth Century, two hypotheses were proposed to explain the high diversity of antibodies.

First hypothesis: Any lymphocyte encountering any antigen is capable of producing antibodies specific to this antigen.

Second hypothesis: Only some lymphocytes which correspond to an antigen are capable of producing antibodies specific to this antigen

To verify one of these two hypotheses, an experiment was performed on mice of the same strain. The steps and results of this experiment are represented in document 1.

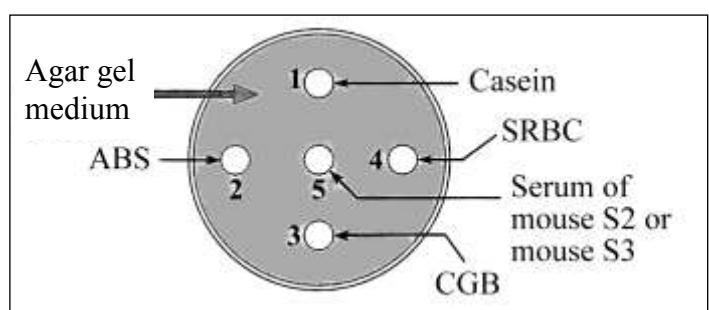
- 1- Write a text which describes the experiment shown in document 1.
- 2- Interpret this experiment to deduce which hypothesis is validated.
- 3- Name the different types of lymphocytes implicated in the immune response revealed by this experiment.



Document 1

Document 2 shows a serological test called, immuno diffusion in gel where antibodies and antigens are deposited in wells in agar gel medium. We deposit an antigenic substance in each of the wells 1, 2, 3 and 4 and deposit either serum taken from mouse S2 or serum taken from mouse S3 in the central well 5.

- 4- Specify where precipitation arc (s) would be formed with each serum after 24 hrs of antigens' deposit. Justify the answer.
- 5- Schematize the mechanism which leads to the formation of the precipitation arcs.



Document 2

Exercise 4 (5 pts) Properties of motor neuron

In order to study the characteristics of a nerve message in an achillian reflex before and after it passes through the spinal cord, we use the experimental set up presented in document 1 and we realize the experiments described below.

The experimental set up in document 1 shows the location of the stimulating electrodes on the afferent fibers and that of recording electrodes on different oscilloscopes. Oscilloscope O1 permits recording the effect of stimulations of one or more afferent fibers; Oscilloscopes O2 and O3 permit recording the electric responses of motor neurons M1 and M2 respectively at the level of the implantation cone. Oscilloscope O4 permits recording the electric activity at the level of the axon of motor neuron M1.

Experiment 1 : We apply two successive effective stimulations on one of the afferent fibers Fa1, and we vary the time between these two stimulations. The results, recorded by O2, are shown in document 2.

1- Interpret the results obtained in document 2.

Experiment 2 : We apply stimulations of increasing intensities on the afferent fibers and we record the results on the four oscilloscopes (document 3).

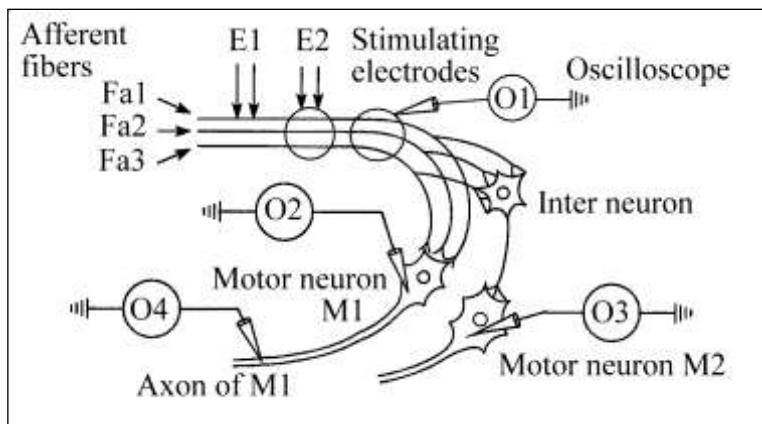
2- Explain the recordings obtained by O1.

3- Compare the recordings obtained by O2 and O4. Draw out the corresponding characteristics of the nerve message.

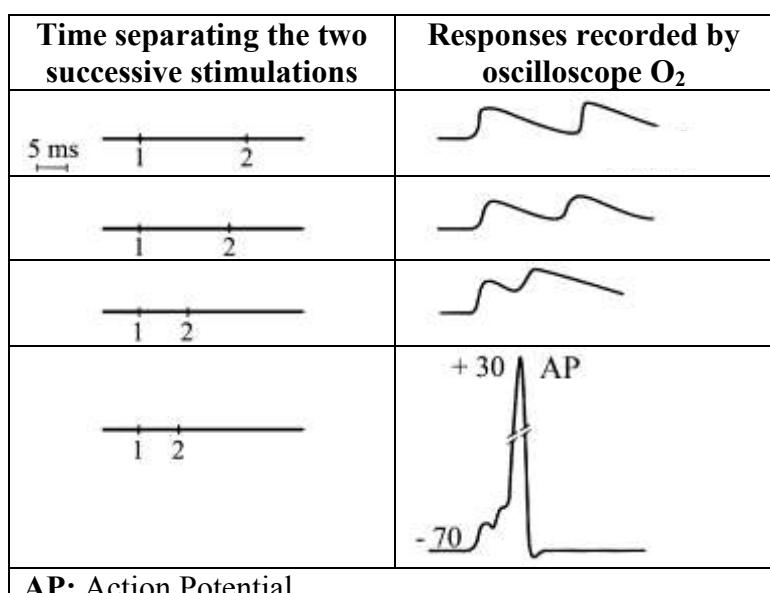
4- Indicate the type of summation which is revealed by experiment 2 at the level of the motor neuron M1. Justify the answer.

Each of the motor neurons M1 and M2, innervates a muscle intervening in the achillian reflex that provokes the extension of the foot.

5- Specify, by referring to documents 1 and 3, and by using the acquired knowledge, the motor neuron innervating the flexor muscle. Justify the answer.



Document 1



AP: Action Potential

Document 2

	Stimulation 1	Stimulation 2	Stimulation 3	Stimulation 4
Recordings obtained by O₁ after stimulation				
Recordings obtained by O₂				
Recordings obtained by O₄				
Recordings obtained by O₃				

As: Artifact of stimulation

GP: Global Potential

Document 3

مسابقة في علوم الحياة
اسس التصحيح

Exercise 1 (5 points)

- 1- The allele gray color (G) is dominant over the allele black color (g) which is recessive ($\frac{1}{4}$ pt), the allele red eye (R) is dominant over the purple eye color (r) which is recessive ($\frac{1}{4}$ pt) and the allele well-formed wing (F) is dominant over the allele deformed wings (f) which is recessive ($\frac{1}{4}$ pt)

- 2- Back cross or test cross ($\frac{1}{4}$ pt)

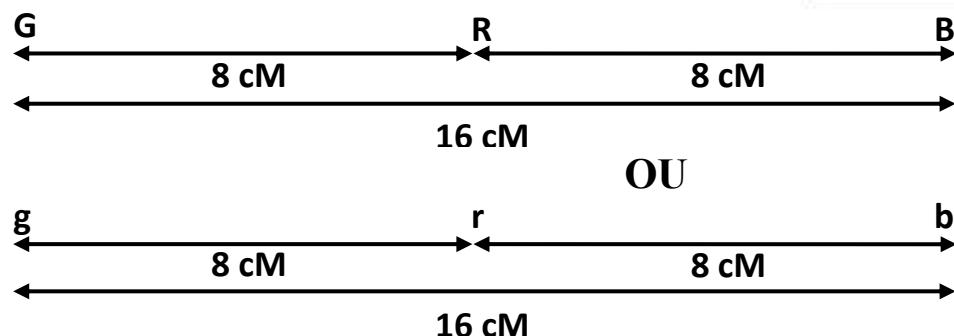
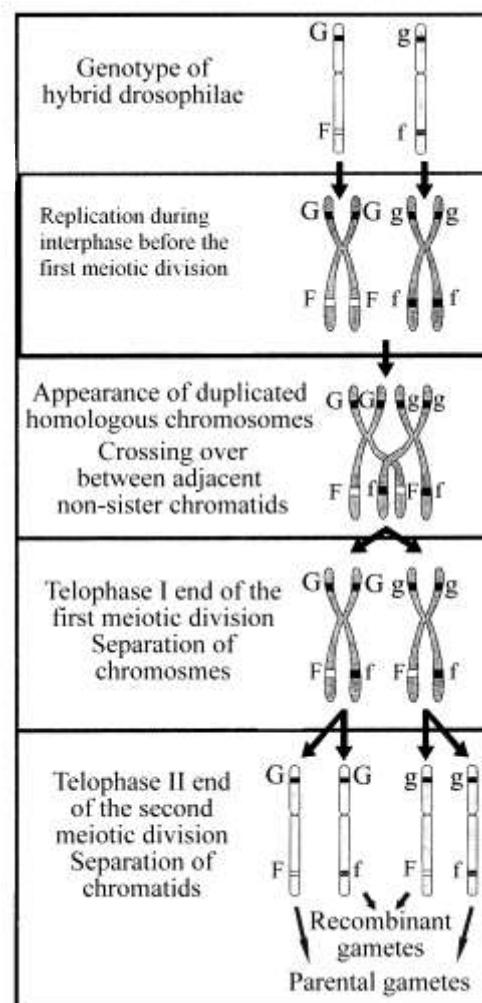
- 3- This back cross is neither the case of independent segregation because it didn't give four equal phenotypes nor the case of absolute linkage because this cross didn't give 2 equal phenotypes. This back cross gives 4 unequal phenotypes, so it's the case of partial linkage followed by crossing over (1pt). Moreover, the genes are linked in Cis, since the parental phenotypes, which occur in higher percentages, show the dominant allele Gray linked with the dominant allele red on one chromosome, and recessive alleles linked on homologous chromosome. ($\frac{1}{4}$ pt)

- 4- Intra-chromosomal recombination or crossing over ($\frac{1}{4}$ pt)
Drawing (1 pt)

- 5- These genes are linked because the gene for body color is linked to the gene for eye color (first cross), and the gene for body color is linked to the gene for wing shape (second cross). Therefore, the gene determining eye color and shape of wings are linked. ($\frac{1}{2}$ pt).

- 6- % of recombination between the genes G and R :
 $784 \times 100 / 10326 = 7.59\% \text{ or } \approx 8\%; (\frac{1}{4} \text{ pt})$
 % of recombination between genes G and F;
 $1616 \times 100 / 10308 = 15.67\% \text{ or } \approx 16\% (\frac{1}{4} \text{ pt})$

- 7- Factorial Map ($\frac{1}{2}$ pt)



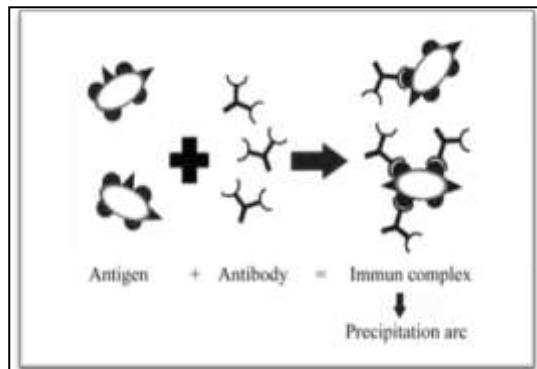
Exercise 2 (5 points)

- 1-** The structural origin of insulin: Islets of Langerhans ($\frac{1}{4}$ pt) . Glucagon is localized at the periphery of the islet of Langerhans ($\frac{1}{4}$ pt) and insulin is localized at the central part of islets of Langerhans ($\frac{1}{4}$ pt) because the radioactive glucagon appears at the periphery and radioactive insulin appears at the center. ($\frac{1}{4}$ pt).
- 2-** Feeble concentration of insulin ($\frac{1}{4}$ pt), because the concentration of radioactivity at the periphery of the cross section of the pancreatic tissue is the same in documents 1 and 2. This means that the secretion of glucagon is normal in animal Y. Whereas, the concentration of radioactivity at the central part of the islets of Langerhans decreased greatly in animal Y compared to that in animal X which is normal. ($\frac{1}{2}$ pt).
- 3-** **Hypothesis 1:** The produced insulin is abnormal and incapable of fixing on its receptors.
Hypothesis 2 : Insulin receptors at the level of target cells are abnormal (or little/ absent) which prevents the action of insulin **OR**
H3: Insulin receptors or insulin are blocked by antibodies that inhibit the action of insulin. ($\frac{1}{2}$ pt for each hypothesis.)
- 4-** The results validate the hypothesis related to receptors : abnormal receptors; absent or blocked by antibodies($\frac{1}{4}$ pt) , because the percentage of glycogen phosphorylase a and glycogen synthetase a in animal Z (doc. 4) remain constant at 78% and 5% respectively before insulin injection (-2 to 0 min) and after injection (0-5 min). On the contrary, in case of animal X (doc.3), the percentage of glycogen phosphorylase a , which is 78% before insulin injection decreases to reach 10% ,2 minutes following insulin injection and remains constant at this value up to 5 minutes. Whereas, the percentage of glycogen synthetase a which is 5% before insulin injection, increases to reach 58% , 5 minutes following insulin injection. This shows that in animal Z, normal insulin didn't act on the target cells to change the concentration of these enzymes. Thus, the origin of diabetes might be due to the absence of insulin receptors or production of auto- reactive antibodies. (1 pt).
- 5-** Insulin is fixed on its receptors located on the membrane of hepatic cells and forms a hormone-receptor complex which changes the function of the target cells ($\frac{1}{2}$ pt): It increases the concentration of glycogen synthetase a and decreases the concentration of glycogen phosphorylase a (doc. 3) which means that glucose is transformed into glycogen, and consequently, glycemia decreases. ($\frac{1}{2}$ pt)

Exercise 3 (5 points)

- 1- We extract lymphocytes from a normal mouse S1 and separate them into two lots. Lymphocytes of lot 1 are directly injected into mouse S2 deprived of its lymphocytes. After contact with ABS antigen (albumin of bull serum), this mouse produces anti- ABS antibodies. We circulated the lymphocytes of lot 2 in a column of solid matrix attached to ABS antigen. Some lymphocytes are retained within this column and a great proportion of these lymphocytes pass along the column. The lymphocytes of lot 2 which traverse the column are injected in mouse S3 deprived of its lymphocytes. After the contact with ABS antigen, mouse S3 doesn't produce anti- ABS antibodies. Several days later, following contact with CGB antigen (Calmette and Guerin Bacillus), mice S2 and S3 produce anti-CGB antibodies. ($1 \frac{1}{2}$ pt).
- 2- Mouse S2, deprived of its lymphocytes, and which receives lymphocytes extracted from a normal mouse S1, produces anti-ABS antibodies upon its contact with ABS antigen. On the contrary, no anti-ABS antibodies are produced by mouse S3 also deprived of its lymphocytes, and which receive lymphocytes that traverse a column of ABS antigens attached to solid matrix, upon its contact with the same antigen. This means that mouse S2 only receives lymphocytes specific to ABS antigen. ($\frac{1}{2}$ pt) However, these two mice S2 and S3 were able to produce anti- CGB antibodies upon their contact with CGB antigen. This means that both mice possess BL specific to CGB antigen. ($\frac{1}{2}$ pt). Thus, the production of specific antibodies, against a certain antigen, necessitates the presence of a category of BL specific to that antigen. **Therefore**, lymphocytes are specific to an antigen and this validates the second hypothesis. ($\frac{1}{2}$ pt).
- 3- B lymphocytes ($\frac{1}{4}$ pt) and T4 Lymphocytes ($\frac{1}{4}$ pt)
- 4- With the serum of mouse S2, a precipitation arc is formed between well 2 and well 5; and between well 3 and well 5 ($\frac{1}{4}$ pt), because mouse S2 produced anti -ABS antibodies and anti-CGB antibodies specific to ABS and CGB antigens respectively. ($\frac{1}{4}$ pt).
With serum of mouse S3, the precipitation arc is formed between well 3 and well 5 ($\frac{1}{4}$ pt), because mouse S3, in the absence of lymphocytes specific to ABS antigen, produces only anti-CGB antibodies which have an antigen binding site specific to CGB antigen. ($\frac{1}{4}$ pt).

5- Drawing ($\frac{1}{2}$ pt)



Exercise 4 (5 points)

- 1-** Two hypopolarizations, of the same amplitude are obtained after two successive stimulations separated approximately with a time difference of 16 ms. These two hypopolarizations get closer and sum up, producing an action potential with an amplitude 100 mV when the time difference between the two stimulations decreases to 6 msec. This means that the motor neuron M1 integrates the successive messages arriving from the same afferent neuron Fa1. (**1½ pt**)
- 2-** The recordings obtained by O1 show a global potential of the nerve fibers with amplitude that increases from 100 mV to 250 mV as intensity of stimulations increases from 1-3. In each stimulation, a certain number of nerve fibers is activated, this explains the increase in the amplitude of potential as intensity increases from 1-3. However, the global potential remains constant at 250 mV following the fourth stimulation which corresponds to the stimulation of all nerve fibers. (**¾ pt**).
- 3-** We observe hypopolarization in O2, on the contrary, we observe no response in O4 following the first stimulation(**1/2pt**). We observe an AP of a constant amplitude of 100 mV in O2 and O4, following stimulations 2, 3 and 4 of increasing intensities. (**1/2 pt**).

The nerve message propagates starting at a certain intensity, (**¼ pt**) with the same amplitude irrespective of the distance. (**1/4 pt**)

- 4-** Spatial summation (**¼ pt**) because as intensity of stimulation increases, more nerve fibers are activated and the motor neuron receives many nerve messages, EPSP, from many fibers simultaneously. The motor neuron integrates (sums up) these messages (**½ pt**)
- 5-** The motor neuron M2 innervates the flexor muscle (**¼ pt**) because during an achillian reflex, the flexor muscle is relaxed. Document 3 shows a hyperpolarization recorded by O3 which is connected to the cell body of the motor neuron M2 innervating the flexor muscle. This hyperpolarization can't propagate so the flexor muscle relaxes. (**½ pt**)

الاسم:
الرقم:مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات**Answer the following exercises:****Exercise 1 (5 points)**

We are interested in studying the events that accompany the sexual reproduction in mammals. These events are studied at cellular and molecular levels.

Female rabbits were mated with sterile males in order to induce ovulation, and then they were inseminated with sperm cells taken from different levels of the genital tract of adult fertile male rabbits. One day following the insemination, the aspect of the cells that were taken from the oviducts was observed under the microscope.

Document 1 presents the percentages of the two main aspects (schema X and Y) observed according to the site where the sperm cells were removed.

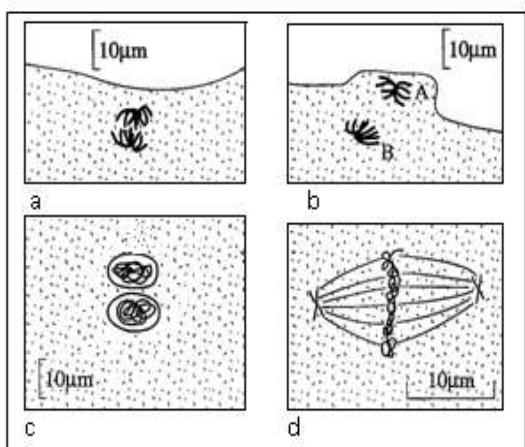
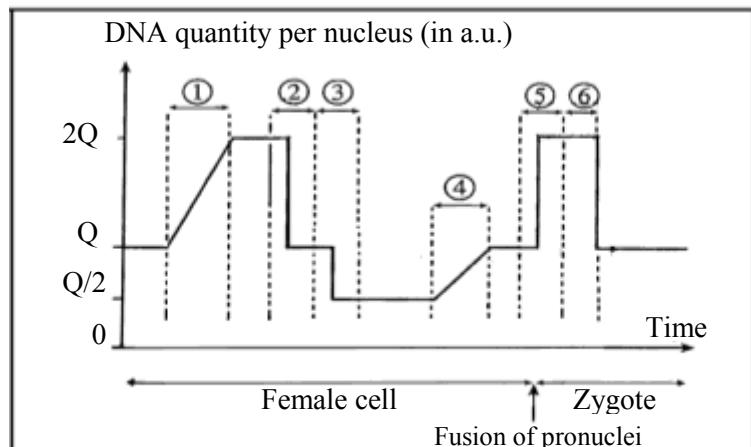
Sexual reproduction in mammals

Aspect of the cells taken from the oviducts one day after the insemination		X	Y
Site from where sperm cells were removed.			
Head of the epididymis	Testicle	100%	0%
	Proximal part of the body of the epididymis	85%	15%
	Distal part of the body of the epididymis	35%	65%
	Tail of the epididymis	8%	92%
Vas deferens			

Document 1

- Explain briefly the structural modifications that take place during the passage of the cell from aspect X to aspect Y.
- Determine, by referring to document 1, the role of the epididymis.

Document 2 reveals, in chronological order, some steps of the evolution of the fertilized oocyte II and that of the zygote. Document 3 represents the evolution of the DNA quantity per nucleus of the female cell and that of the zygote.

**Document 2****Document 3**

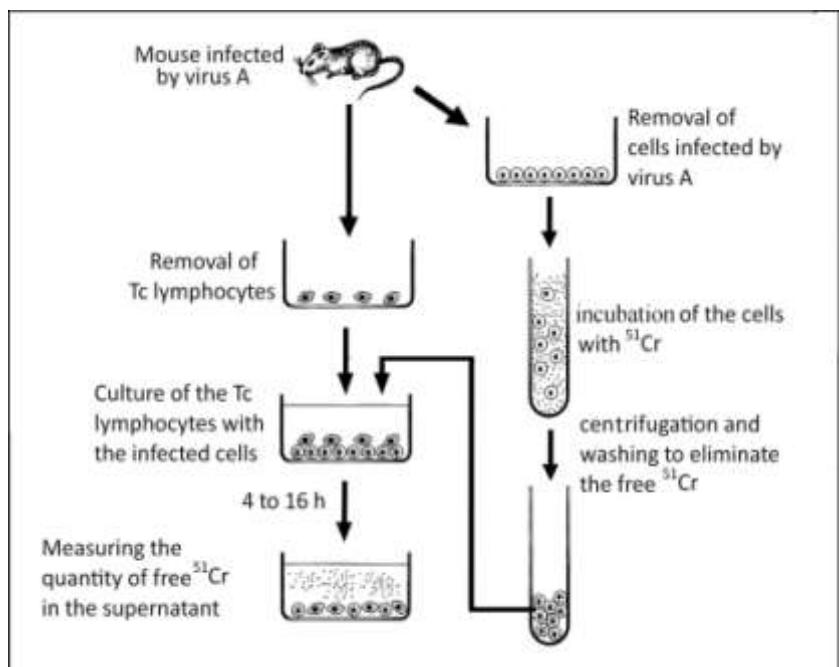
- 3-1- Name the two principal mechanisms of the sexual reproduction in mammals.
3-2- Specify the importance of each of these mechanisms. Justify the answer by referring to document 2.
- Match each of the schema b, c and d of document 2 with a numbered step of the curve of document 3. Justify the answer.

Exercise 2 (5 points)

Cytotoxicity of Tc lymphocytes

The cells that are infected by a virus express on their plasma membranes some antigens of this pathogen. These antigens can be recognized by specific receptors of the cytotoxic lymphocytes (Tc). In the attempt to prove the cytotoxicity of the Tc lymphocytes, we perform the experiment schematized in document 1, where cells infected by virus A are incubated with ^{51}Cr , a substance that binds to intracellular proteins after being absorbed by the cell.

1. Describe the experiment schematized in document 1.
2. Justify, by referring to document 1, how the presence of free ^{51}Cr in the supernatant reveals the cytotoxic role of Tc lymphocytes.
3. Specify the type of the immune response revealed by the experiment of document 1. Justify the answer.



Document 1

Dermal cells of mice of strain X or of strain Y, infected or not by a virus, are cultured in vitro. Tc lymphocytes removed from other mice of strain X, infected or not by a virus, are added to the culture medium. Document 2 presents the experimental conditions and the obtained results.

Origin of the cultured dermal cells Origin of the added Tc lymphocytes	Healthy mice X	Mice X infected by virus A	Mice X infected by virus B	Mice Y infected by virus B
Healthy mice X	No destruction of dermal cells	No destruction of dermal cells	No destruction of dermal cells	No destruction of dermal cells
Mice X infected by virus A		Destruction of the infected dermal cells by the Tc lymphocytes	No destruction of dermal cells	
Mice X infected by virus B		No destruction of dermal cells	Destruction of the infected dermal cells by the Tc lymphocytes	

Document 2

4. Interpret the results of document 2 and deduce the conditions that are indispensable for the functioning of the Tc.
5. Explain the mechanism that leads to the destruction of target cells by Tc lymphocytes.

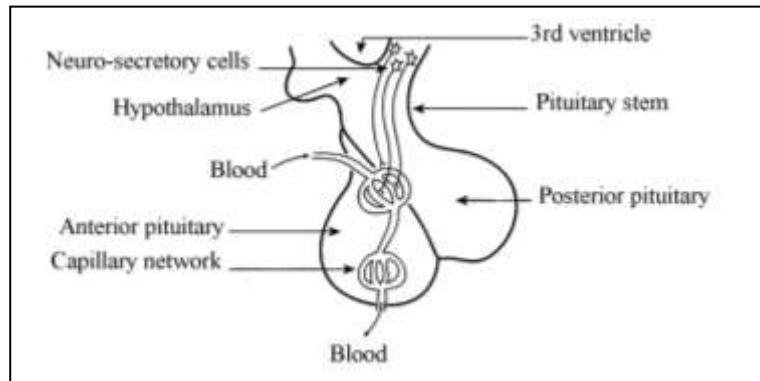
Exercise 3 (5 points)

Regulation of the sexual cycles

We aim to study the ovarian and uterine cycles by performing experiments on adult mammals.

Document 1 illustrates the hypothalamo-pituitary complex implicated in the regulation of these cycles.

Experiment 1: The ablation of the anterior pituitary is followed by the atrophy of both the ovaries and the uterus along with the disappearance of the cycles.



Document 1

Experiment 2: In animals submitted to the ablation of the pituitary gland and receiving regular injections of anterior pituitary extracts, we can observe a redevelopment of the ovaries and sometimes a reestablishment of the ovarian and uterine cycles. However in an ovariectomized animal, injected by anterior pituitary extracts, we never observe a reestablishment of the uterine cycle.

Experiment 3: Lesions of the posterior hypothalamus have the same effect as the ablation of the anterior pituitary.

1. Interpret the results of each of the three experiments.

Experiment 4: Bilateral ovariectomy provokes a hypertrophy of the pituitary gland followed by an abnormal high production of gonadotropin hormones. This experiment allows us to admit the existence of a feedback mechanism exerted by the ovaries on the production of FSH and LH.

In order to determine the types of this feedback, an ovariectomized female monkey receives, for four periods of 15 days each, injections of ovarian hormones with different doses and composition. For each period the average level of FSH and LH production is measured (document 2).

Periods of 15 days	Characteristics of the injections		Plasmatic levels	
	Composition	Plasmatic levels	of FSH in ng/ml	of LH in ng/ml
1	Estrogen	0	> 15	> 50
	Progesterone	0		
2	Estrogen	70 pg/ml	Around 6	Around 4
	Progesterone	0		
3	Estrogen	300 pg/ml	Around 12	Around 40
	Progesterone	0		
4	Estrogen	300 pg/ml	< 4	< 3
	Progesterone	4 pg/ml		

Document 2

2. State the types of the feedback revealed in document 2. Justify the answer.
3. Establish, by referring to the four experiments, a functional diagram showing the relations between the different organs involved in the regulation of the sexual cycles.

Exercise 4 (5 points)

Myasthenia is a neuromuscular disease characterized by a difficulty in performing efficient muscular contractions. In order to determine the cause of this difficulty we performed experiment 1.

Experiment 1

A microelectrode introduced into a muscle fiber of the leg muscle permits the recording of the electrical activity, obtained in the case of a healthy individual (A) and a myasthenic individual (B), following the stimulation of the motor neuron.

Document 1 shows the experimental set-up and the results.

1. Analyze the obtained recordings.
2. Specify, by referring to document 1 and to the acquired knowledge, the physiological consequences that can be observed at the level of the muscles of these two individuals.

Physicians thought that the abnormal functioning of the neuromuscular junction might be at the origin of myasthenia.

Document 2 presents the organization of the neuromuscular junction or motor end plate.

3. Label each of the structures 1, 2, 3 and 4 of document 2.

In order to determine the origin of this disease we performed experiment 2.

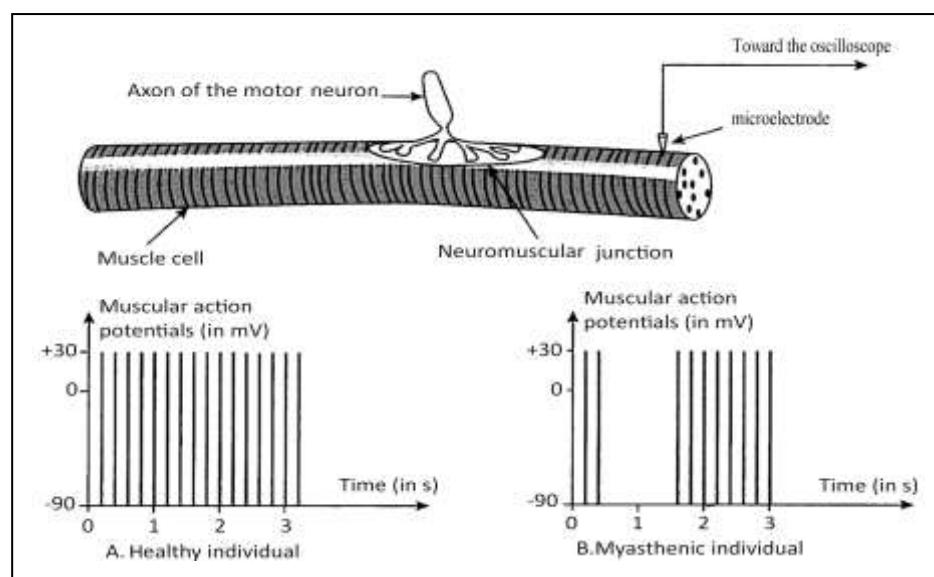
Experiment 2

- α -bungarotoxin, a toxic molecule extracted from some snakes' venom, has a spatial configuration that is similar to that of acetylcholine which is the neurotransmitter of the neuromuscular synapse. α -bungarotoxin has the capacity to bind acetylcholine receptors. Its injection to a healthy mouse induces immediately symptoms that are similar to those of myasthenia.
- Biopsies of muscular tissues are performed to a healthy individual (A) and to a myasthenic individual (B).

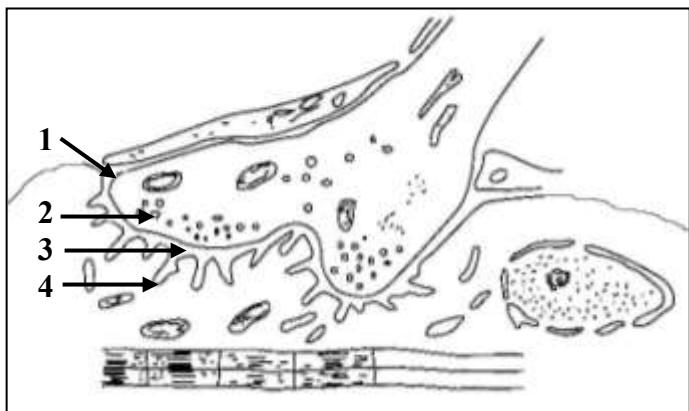
Removed cells are placed in presence of radioactive α -bungarotoxin. This toxin is then localized, by autoradiography, on the membrane of a muscle cell in the form of black grains. Document 3 shows the obtained results.

4. Compare the two autoradiographies A and B of document 3.
5. Determine, referring to experiment 2, the origin of myasthenia.
6. Referring to the information drawn out from documents 1 and 3, write a text that explains the symptoms of this disease.

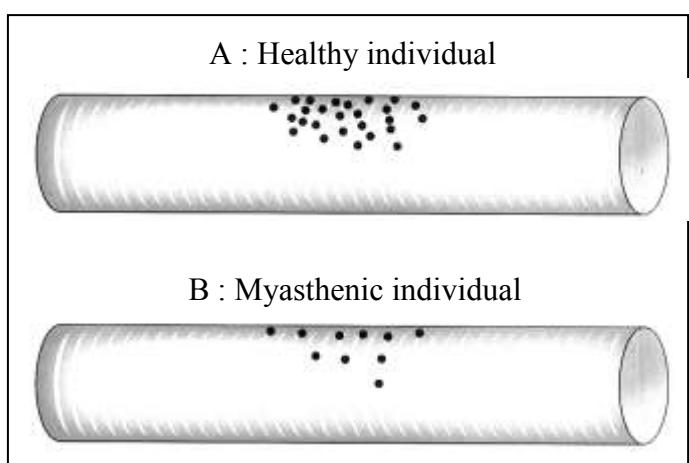
Neuromuscular disease



Document 1



Document 2



Document 3

مسابقة في علوم الحياة**اسس التصحيح****Exercise 1 (5 points)**

1. The scheme X represents an oocyte II blocked at metaphase II after having released the first polar body. Once fertilized by a sperm cell this cell releases the content of its cortical granules forming the fertilization membrane (**1/4pt**) and resumes meiosis II(**1/4pt**) releasing the second polar body(**1/4pt**). The male and female pronuclei are formed(**1/4pt**). This is how the cell passes from aspect X to aspect Y.
2. The percentage of non fertilized cells of scheme X is 100% while that of fertilized cells of scheme Y is 0% when the sperm cells are collected from the testicles; however as we proceed through the epididymis the percentage of scheme X decreases to reach 8% while the percentage of the scheme B increases to reach 92%. This shows that the testicles produce sperm cells with no fertilization capacity and that this fertilization capacity became more and more important as the sperm cells proceed through the epididymis. (**3/4pt**)
Therefore the epididymis is the site where the sperm cells acquire their fertilization capacity. (**1/4pt**)
3. 3-1- Meiosis (**1/4pt**) and fertilization (**1/4pt**).
3-2- meiosis allows the reduction of the chromosomal number to obtain haploid cells. (**1/4pt**) This is revealed in scheme a and/or b that show anaphase II and the separation of chromosomes into two haploid sets. (**1/4pt**)

Fertilization restores the diploid state of the species (**1/4pt**) This is revealed in scheme c that shows the male and female pronuclei before their fusion (**1/4pt**)

4. Scheme b corresponds to step 3 (**1/4pt**) since there is separation of the two haploid lots of chromosomes each with one chromatid. This corresponds to the second meiotic division where the DNA quantity is reduced from Q to Q/2. (**1/4pt**)

Scheme c corresponds to step 4 (**1/4pt**) since it shows the male and female pronuclei just before their fusion. The female pronucleus undergoes replication of its DNA resulting in an increase of its DNA quantity from Q/2 to Q (**1/4pt**)

Scheme d corresponds to step 6 (**1/4pt**) since it shows the metaphase of the first mitotic division of the zygote having 2n chromosomes with 2 chromatids each and that corresponds to a DNA quantity of 2Q(**1/4pt**)

Exercise 2 (5 points)

1. We remove from mouse X infected by virus A cells infected by virus A and Tc lymphocytes. We incubate the infected cells with ^{51}Cr , then we perform centrifugation and wash to eliminate the free ^{51}Cr . After that we culture the removed Tc lymphocytes with the infected cells. After 4 to 16 h we measure the quantity of free ^{51}Cr in the supernatant. **(1 ½ pt)**
2. The disappearance of ^{51}Cr after washing then its reappearance in the supernatant after culturing the infected cells with Tc lymphocytes indicate that these latter have destroyed the infected cells liberating the ^{51}Cr bound to intracellular proteins. **(1pt)**
3. Specific cell mediated immune response **(1/4 pt)**. Since the Tc cells have destroyed the infected cells. **(1/4 pt)**
4. Dermal cells of healthy mice are not destroyed by the Tc regardless from which mice they are removed.

Dermal cells of mice X infected by virus A or by virus B are only destroyed by Tc lymphocytes of mice X infected by the same virus: respectively virus A or virus B. Thus the cells infected by a virus are destroyed only by Tc of mice infected by the same virus. However dermal cells of mice of strain Y infected by virus B are not destroyed by Tc of mice of different strain X even though they are infected by the same virus B; thus the infected cells are destroyed only by Tc of mice of the same strain. **(1 pt)**

Therefore, the Tc cells destroy the infected cells if they belong to the same strain and are infected by the same virus. **(1/2 pt)**

5. The Tc lymphocytes recognize and bind, by its TCR, to target cells expressing the modified self: self MHC carrying a non self peptide of the antigen that is at the origin of their activation. They will then release, by exocytosis, perforine molecules forming hollow polyperforine channels through the cell membrane, and then they release granzymes molecules that penetrate the target cell through these channels leading to its DNA degradation and to the target cell destruction. **(1/2 pt)**

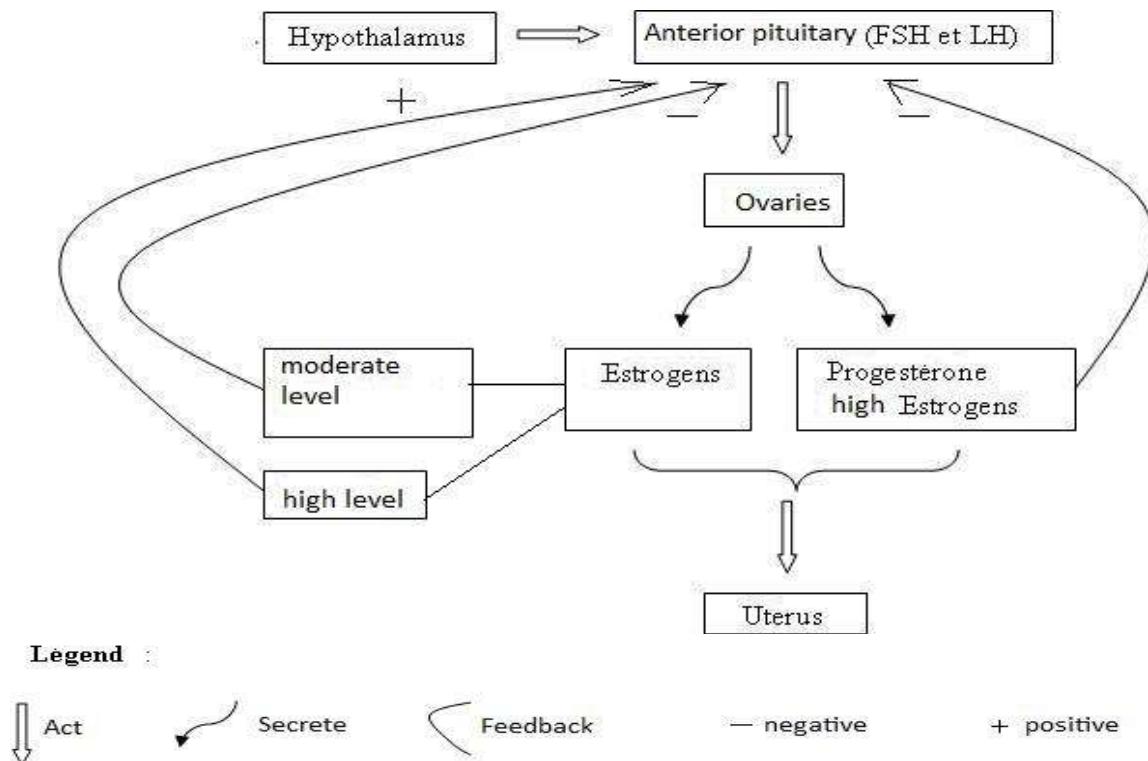
Exercise 3 (5pts)

1. Atrophy of the ovaries and the uterus with a disappearance of the cycles are observed following the ablation of the anterior pituitary. This implies that the anterior pituitary gland is indispensable for the development of the ovaries and the uterus and for their cyclic activities. (1/2pt)

The ovaries redevelop and the ovarian and uterine cycles are sometimes reestablished when anterior pituitary extracts are injected to animals submitted to the ablation of the pituitary gland. However the uterine cycle is never reestablished when the same injections are given to these animals after ablation of their ovaries. This implies that the anterior pituitary gland acts through blood directly on the ovaries and indirectly on the uterus. (1/2pt)

The same effects as those of the ablation of the anterior pituitary are observed following the lesions of the posterior hypothalamus. This implies that the hypothalamus activates the pituitary gland. (1/2pt)

2. Negative feedback: the moderate level of estrogens (70 pg/ml) alone or the high level of estrogens in presence of progesterone decreases the release of FSH and LH by the anterior pituitary gland (1pt).
 Positive feedback: the high level of estrogens alone (300pg/ml) increases the release of FSH and LH by the anterior pituitary gland (1pt).
3. Functional diagram showing the relations between the different organs. (1½ pt)



Exercise 4 (5pts)

1. For the healthy individual the frequency of the action potentials is high: 15 AP of same amplitude (120mV) during 3 seconds, while for the myasthenic individual this frequency is lower: 10 AP with similar amplitude during the 3 seconds, with an absence of AP for certain moments. **(1 pt)**
2. For the healthy individual, the regular presence of AP induces the continuous contraction of the muscle. While for the myasthenic individual, the absence of AP for certain moments induces a decrease in the contraction of the muscle. **(1/2 pt)**
3. 1- Presynaptic membrane **(1/4 pt)**
2- Secretory vesicles containing neurotransmitters **(1/4 pt)**
3- Synaptic cleft **(1/4 pt)**
4- Postsynaptic membrane **(1/4 pt)**
4. The autoradiography of the fiber of the healthy individual reveals an important concentration of black grains; on the other hand, this concentration is less important for the myasthenic individual. **(1/2 pt)**
5. The comparison shows, for individual A, an important fixation of α -bungarotoxin molecules to the acetylcholine receptors and a less important fixation for individual B. The presence of these molecules on the muscle fiber reveals the presence of acetylcholine receptors. Thus for the healthy individual A there is a great number of acetylcholine receptors at the level of the postsynaptic membrane of the neuromuscular junction while this number is less important for the myasthenic individual B.
Therefore the myasthenia is due to a lack in acetylcholine receptors at the level of the motor end plate. **(1 pt)**
6. In the myasthenic individual there is a release of acetylcholine neurotransmitters following the stimulation of the motor neuron. However only few of these neurotransmitters bind to the post synaptic membrane of the muscle fiber due to the lack in post synaptic membrane receptors (doc 3)
This lack in binding inhibits the appearance of a regular train of AP (doc 1) and is thus responsible for the weak muscular activity, the muscular contraction is not maintained.
(1 pt)

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Answer the following exercises:

Exercise 1 (5 points)

Retinitis pigmentosa

Retinitis pigmentosa, a hereditary disease, is the main cause of visual impairment (30% of visual deficiencies). The disease starts by affecting night vision and reducing the visual field. It is caused by progressive degeneration of rod cells, which are photoreceptor cells of the retina containing the protein rhodopsin.

To understand the origin of this disease, we study the structure of proteins encoded by different alleles of the rhodopsin gene.

The rhodopsin gene consisting of 1044 pairs of nucleotides encodes a protein of 348 amino acids.

Document 1 represents a portion of the nucleotide sequences of the alleles of the rhodopsin gene and that of the amino acids sequences of the corresponding proteins in individuals with normal phenotype and individuals with retinitis pigmentosa.

Individual's phenotype	Portion of the nucleotides sequence of the allele		portion of the amino acids sequence of the protein	
normal	391 ↓ ...CTG GCC ATC GAG CGG TAC...	408 ↓ ...CTG GCC ATC GAG CTT TAC...	131 ↓ ...Leu-Ala-Ile-Glu-Arg-Tyr...	136 ↓ ...Leu-Ala-Ile-Glu-Leu-Tyr...
Affected with retinitis pigmentosa	391 ↓ ...CTG GCC ATC GAG CTT TAC...	408 ↓ ...CTG GCC ATC GAG CTT TAC...	131 ↓ ...Leu-Ala-Ile-Glu-Leu-Tyr...	136 ↓ ...Leu-Ala-Ile-Glu-Leu-Tyr...

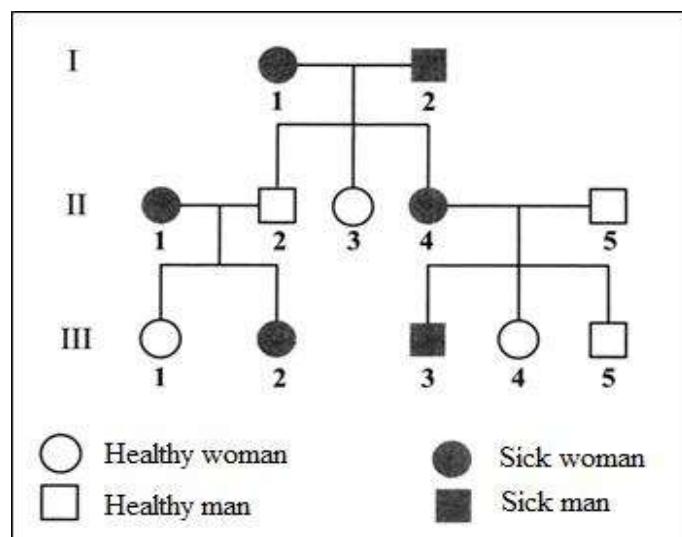
Leu = leucine, Ala = alanine, Ile = isoleucine, Glu = glutamic acid, Arg = arginine, Tyr = tyrosine.

Document 1

- Pick out from the text the cause of retinitis pigmentosa.
- Compare the two nucleotides sequences and the two amino acids sequences presented in document 1. Draw out the origin of this disease.
- Explain how the modifications in the nucleotides sequence of the allele (doc.1) lead to the appearance of the previously mentioned symptoms of retinitis pigmentosa.

Document 2 presents the pedigree of a family having some of its members affected with retinitis pigmentosa.

- Specify if the allele responsible for the disease is dominant or recessive and indicate its chromosomal location. Justify both answers.
- Determine the genotypes of individuals II3 and II4.
- Woman III2 married her cousin III3; determine the risk for this couple to have children with retinitis pigmentosa.



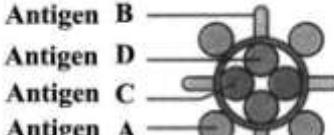
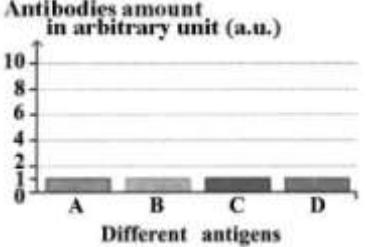
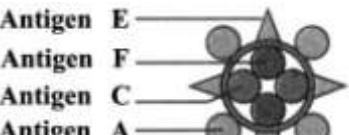
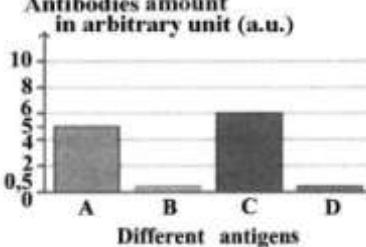
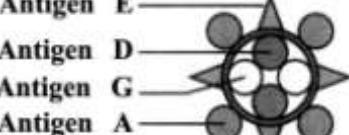
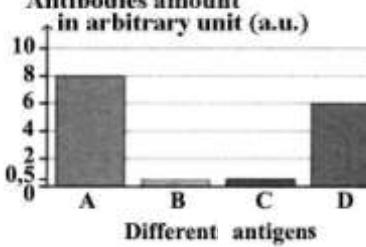
Document 2

Exercise 2 (5 points)

Immunological memory

When an organism encounters the same pathogen more than once during its lifetime, the immune response against this pathogen becomes more and more efficient. The flu virus exists in different variants having different antigens. We study the immune responses triggered by an individual upon contact with the flu virus three times during his life time.

The document below presents the age of this individual at the time of contact with one of the three variants of the flu virus and the evolution in the amount of antibodies specific to the antigens of variant 1.

Age of the individual at the time of infection	Antigens of the variant	Evolution in the amount of antibodies specific to the antigens of variant 1										
 2 years old individual in contact with variant 1 of the flu virus	 Variant 1 of the flu virus	 <table border="1"> <caption>Antibodies amount in arbitrary unit (a.u.)</caption> <thead> <tr> <th>Different antigens</th> <th>Antibodies amount (a.u.)</th> </tr> </thead> <tbody> <tr> <td>A</td> <td>~1.5</td> </tr> <tr> <td>B</td> <td>~1.0</td> </tr> <tr> <td>C</td> <td>~1.0</td> </tr> <tr> <td>D</td> <td>~1.0</td> </tr> </tbody> </table>	Different antigens	Antibodies amount (a.u.)	A	~1.5	B	~1.0	C	~1.0	D	~1.0
Different antigens	Antibodies amount (a.u.)											
A	~1.5											
B	~1.0											
C	~1.0											
D	~1.0											
 The same individual at the age of five years in contact with variant 2 of the flu virus	 Variant 2 of the flu virus	 <table border="1"> <caption>Antibodies amount in arbitrary unit (a.u.)</caption> <thead> <tr> <th>Different antigens</th> <th>Antibodies amount (a.u.)</th> </tr> </thead> <tbody> <tr> <td>A</td> <td>~5.0</td> </tr> <tr> <td>B</td> <td>~0.5</td> </tr> <tr> <td>C</td> <td>~5.5</td> </tr> <tr> <td>D</td> <td>~0.5</td> </tr> </tbody> </table>	Different antigens	Antibodies amount (a.u.)	A	~5.0	B	~0.5	C	~5.5	D	~0.5
Different antigens	Antibodies amount (a.u.)											
A	~5.0											
B	~0.5											
C	~5.5											
D	~0.5											
 The same individual at the age of 20 years in contact with variant 3 of the flu virus	 Variant 3 of the flu virus	 <table border="1"> <caption>Antibodies amount in arbitrary unit (a.u.)</caption> <thead> <tr> <th>Different antigens</th> <th>Antibodies amount (a.u.)</th> </tr> </thead> <tbody> <tr> <td>A</td> <td>~8.0</td> </tr> <tr> <td>B</td> <td>~0.5</td> </tr> <tr> <td>C</td> <td>~0.5</td> </tr> <tr> <td>D</td> <td>~5.0</td> </tr> </tbody> </table>	Different antigens	Antibodies amount (a.u.)	A	~8.0	B	~0.5	C	~0.5	D	~5.0
Different antigens	Antibodies amount (a.u.)											
A	~8.0											
B	~0.5											
C	~0.5											
D	~5.0											

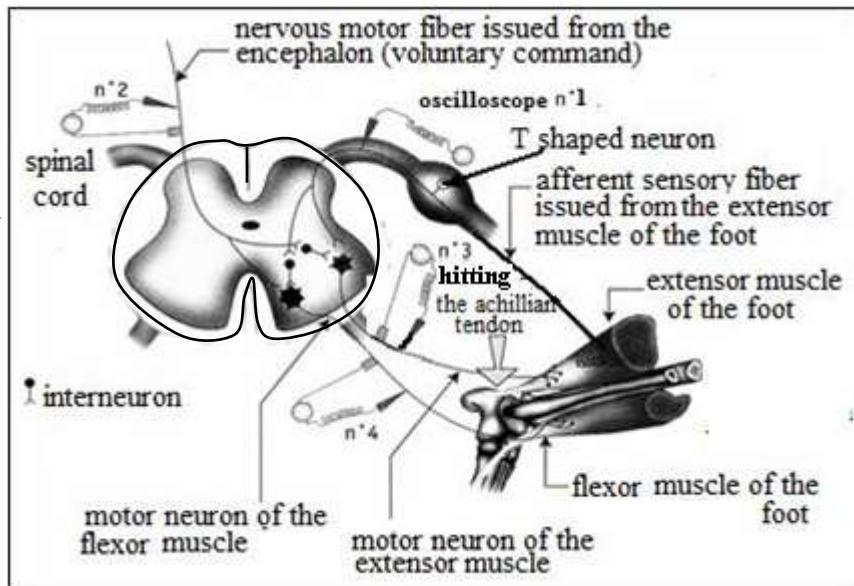
- 1- Name the specific immune response revealed in the above document. Justify the answer.
- 2- Justify the following statements by referring to the document.
 - a- The secondary immune response is more amplified than the primary immune response.
 - b- The secreted antibody is specific to the antigen and not to the variant of the virus.
 - c- The organism keeps memory for an encountered antigen for more than ten years.
- 3- Name two cells implicated in the immune response triggered against variant 1 of the flu virus and specify the role of each cell.
- 4- Explain how the secreted antibodies contribute to the destruction of the flu virus.
- 5- Specify if the revealed immune response is capable alone to eliminate cells infected by the virus. Justify the answer.

Exercise 3 (5 points) Achillian reflex and voluntary movement

An individual can control or inhibit an achillian myotatic reflex by voluntary muscle activity. Several experiments were performed in order to explain the interaction between voluntary activities and reflexes. The experimental set ups and results are presented in documents 1, 2 and 3.

Document 1 presents the structures involved in the achillian reflex.

Document 2 shows the electromyogram of the extensor muscle of the foot upon hitting the Achillian tendon in the absence of voluntary flexion of the foot (curve 1) and during slight voluntary flexion of the foot (curve 2).



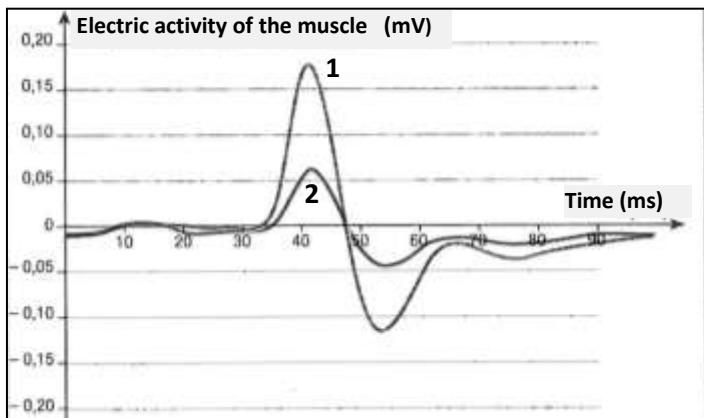
Document 1

- Interpret the results of document 2.

Document 3 presents the recordings of the electric activity of the neuronal network involved in the achillian reflex obtained under the same experimental conditions as those of document 2.

- Match each of the cases A and B in document 3 to its corresponding curve 1 or 2 in document 2. Justify the answer.
- Explain the results obtained at the level of oscilloscope n°3 in document 3 in the cases A and B.

We ask this individual to perform a strong voluntary flexion of his foot before hitting the achillian tendon.



Document 2

- Based on document 3, draw in this case, the recordings obtained at the level of the oscilloscopes n° 1, 2, 3 and 4. Justify the answer for each recording.

Recordings of the activity of the neuronal network	Oscilloscope			
	n° 1	n° 2	n° 3	n° 4
Case A		—		—
Case B				

Document 3

N.B: Each vertical line corresponds to an action potential

Exercise 4 (5 points)

Regulatory system of glycemia

In the frame work of studying the regulation of glycemia, experimental data have been collected from healthy people or animals. Document 1 shows the glucose concentration in the blood entering and that leaving the muscle and the encephalon.

	Glucose concentration (in mg/100 mL of blood)	
	Blood entering	Blood leaving
Muscle at rest	90	87
Encephalon at rest	91	80

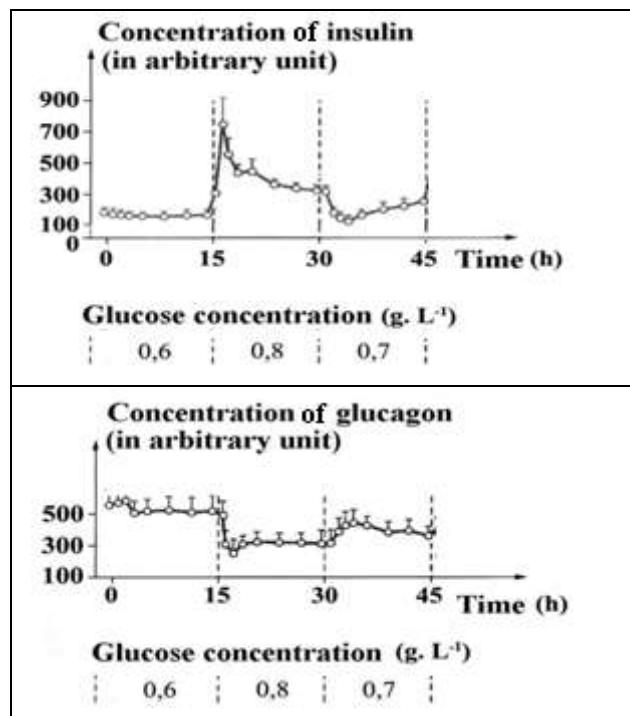
Document 1

- 1- Analyze document 1 and draw out the adequate relation.

Document 2 represents the variations in the concentration of insulin and glucagon secreted by the isolated pancreas of a dog that is perfused with a liquid having different glucose concentrations.

Document 3 shows the effect of the injection of glucagon on glycemia and hepatic glycogen level.

- 2- Construct a table showing the variation of hepatic glycogen concentration in function of time (doc.3).
 3- Interpret the results of each of document 2 and document 3.



Document 2

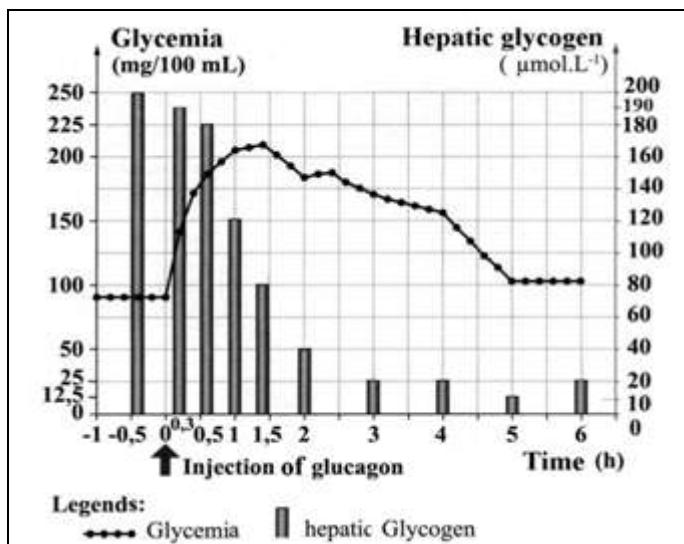
In order to study the role of insulin, researchers carried out the following two experiments:

Experiment 1: They measured the amount of glucose absorbed by muscles and the muscular glycogen reserve in a medium with or without insulin. The obtained results are presented in document 4.

- 4- Compare the results of document 4 and draw out the role of insulin on muscle cells

Experiment 2: They measured the amount of glucose consumed by encephalon cells in a medium with or without insulin. The result showed that this consumption was around 6g/h in both media with and without insulin.

- 5- Determine whether the cells of the encephalon are target cells for insulin.



Document 3

Amount of glucose absorbed by the muscle (in mg/g of muscle) every 10 minutes		Amount of glycogen content in the muscle(in mg/g of muscle) after 10 minutes	
medium without insulin	medium with insulin	medium without insulin	medium with insulin
1.43	1.88	2.45	2.85

Document 4

مسابقة في علوم الحياة
اسس التصحيح

Part of the Ex	Exercise 1 (5 points)	Mark
1	<p>It is caused by progressive degeneration of rod cells which are photoreceptor cells of the retina containing the protein rhodopsin.</p>	0.25
2	<p>The allele of the individual with normal phenotype and that of the affected individual are identical except at their nucleotides 404 and 405: the normal allele has two GG nucleotides, while the allele responsible for retinitis has two nucleotides TT. The two amino acids sequences are identical except at their 135th amino acid: arginine (Arg) in the sequence of the normal individual and leucine (Leu) in the sequence of the affected individual. Thus the modification of the nucleotides sequence of the rhodopsin gene is translated in a modification of the protein which is at the origin of the disease.</p>	1
3	<p>The mutation by substitution of nucleotides 404 and 405 of the DNA was transcribed at the mRNA level by a new codon that results in a new amino acid leucine instead of arginine. This new amino acid sequence affects the three-dimensional structure of the protein rhodopsin, which becomes non-functional. Since this protein exists in the rod cells (photo receptor cells), the change in its function is manifested by impaired night vision in a person with retinitis pigmentosa.</p>	0.75
4	<p>The allele of the disease is dominant with respect to the normal allele since the healthy man II2 has both his parents I1 and I2 affected by retinitis pigmentosa, thus, the parents carry the normal allele which is masked by the allele of the disease. (D = allele of the disease, n = normal allele) The allele of the disease is localized on an autosome. Since: If the allele of the disease is carried by the non –homologous segment of chromosome Y then, it should be transmitted from father to son, however sick father I2 has a healthy son II2. Therefore, the allele is not carried by the non homologous segment of chromosome Y. If the allele of the disease is carried by the non homologous segment of chromosome X, then the sick father I2 should transmit this dominant allele to all his daughters who will be all sick, however daughter II3 is healthy ,thus the allele is not carried by the non homologous segment of X chromosome. If the allele is carried by the homologous segment of chromosome X and Y, then boy II2 who is normal (recessive) should have received Y chromosome carrying the normal allele from his father . Similarly, girl II3 who is normal should have inherited X chromosome carrying the normal allele from her father . Therefore, their father should have the genotype XnYn and would be normal which is not the case. Therefore, the allele is not carried by the homologous segment of chromosomes X and Y.</p>	1.25
5	<p>II3 has a normal phenotype; since the normal recessive allele is only expressed under homozygous state then her genotype is: n/n II4 is diseased , and has a healthy child that should have inherited one normal allele from each of the two parents, thus she carries the normal allele which is masked by the allele causing the disease. Therefore she is heterozygous D/n.</p>	1
6	<p>III2 and III3 have necessarily inherited the normal allele from their healthy father and are thus heterozygous D//n, each of them gives two types of gametes 1/2n and 1/2 D.</p>	0.75

	<p>A heterozygous couple have $\frac{3}{4}$ of its children affected. Thus the risk of this couple to have an affected child is $1 \times 1 \times \frac{3}{4} = \frac{3}{4}$ Or the child will be sick if he inherit: D from the father and D from the mother ($\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$) or D from the father and n from the mother ($\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$) or n from the father and D from the mother ($\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$) $\frac{1}{4} + \frac{1}{4} + \frac{1}{4} = \frac{3}{4}$ children with retinitis. Or the child will be normal if he inherit n from the father and n from the mother ($\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$) thus the risk to have a sick child is $1 - \frac{1}{4} = \frac{3}{4}$ Or refer to a table of cross</p>	
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Part of the Ex	Exercise 2 (5 points)	Mark
1	Specific humoral immune response; since following the entry of the virus of variant 1, having the antigens A, B, C and D, the amount of antibodies specific to each of these antigens rises to 1a.u..	1
2	<p>a- During the first contact with the variant 1 of flu virus at the age of 2 years, the amount of antibodies specific for each of the antigens A, B, C and D was 1a.u. which corresponds to a primary immune response . However, during the second contact at the age of 5 years with the variant 2 of flu virus having the antigens C and A in common with variant 1, the amounts of antibodies specific to A and to C increase respectively to 5 a.u. and to 6 a.u($> 1\text{a.u.}$), while the amounts of antibodies specific to B and to D remains low (0.5 a.u.). This means that the immune response triggered upon the second contact with the same antigen or secondary immune response is more amplified than the primary immune response.</p> <p>b- During the 3rd contact at 20 years of age, with the variant 3 of flu virus having the antigens A and D in common with the variety 1, only the amount of antibody specific to these antigens increases respectively to 8 a.u ($> 1\text{a.u.}$) and 6 a.u ($> 1\text{a.u.}$) thus the secreted antibody is specific to the antigen and not to the variant of the virus.</p> <p>c- The organism keeps memory for an encountered antigen for more than ten years since the amount of antibodies has increased to 6 a.u($> 1\text{a.u.}$) 18 years after the first contact with the antigen D. Or Since the amount of anti B antibodies remains constant at 0.5 a.u from the age of 5 years to 20 years.</p>	2.25
3	<ul style="list-style-type: none"> - Macrophages: after phagocytosis of the antigen, they become APCs that activate specific T4L - T4L: once activated they secrete the interleukin 4 that activate the LB - LB: they identify the free antigens through their membrane antibodies to be activated - Plasma cells: secrete specific antibodies against the antigen. 	1
4	The specific antibodies neutralize their corresponding antigens of the flu virus by binding to them through their specific antigenic binding sites forming immune complexes. Thus the antibodies become able to bind through their constant part on macrophages that phagocytose the whole immune complexes thus destroying the virus (opsonization).	0.75
5	No. Since the infected cells cannot be identified by the antibodies which block only extracellular antigens.	0.5

Part of the Ex	Exercise 3 (5 points)	Mark
1	The amplitude of the electric activity of the extensor muscle is 0.18 mV during an achillian reflex in the absence of voluntary flexion of the foot . However, this amplitude decreases to 0.6mV upon the voluntary flexion of the foot. This means that the voluntary command inhibits the achillian reflex.	1
2	<p>case B corresponds to curve 2, because oscilloscope number 2 connected to the motor nerve fiber, issued from the encephalon and which is responsible for the voluntary command, shows 3 AP action potential only in case B revealing a voluntary intervention.</p> <p>Case A corresponds to curve 1 because oscilloscope number 2 connected to the motor nerve fiber, issued from the encephalon and which is responsible for the voluntary command, shows resting potential revealing no voluntary intervention.</p> <p>OR</p> <p>The student may refer to the activity of the motor neuron innervating the extensor muscle: the decrease in the frequency of action potential from 5 AP to 3AP indicates a decrease in the electric activity of the muscle.</p>	1
3	Oscilloscope number 3 shows a decrease in the frequency of action potential from 5 AP in case A to 3AP in case B and this could be explained by the fact that the motor neuron innervating the extensor muscle receives in case A only one excitatory nerve message from the T-shaped sensory neuron and records a series of 5 Ap. , while the motor neuron in case B receives in addition to the excitatory message from the T-shaped sensory neuron an inhibitory message from the encephalon through the interneuron. The motor neuron integrates these two messages, by spatial summation, this results in a decrease in the frequency of AP .	1
4	<p>Oscilloscope no. 1: </p> <p>Because the same stimulation at the level of achillian tendon records the same frequency of AP in the sensory fiber.</p> <p>Oscilloscope no.2: </p> <p>(Any drawing showing a frequency > 3 AP is accepted)</p> <p>Because the strong voluntary flexion reveals an increase in the frequency of AP in the nerve fiber coming from the superior centers responsible for voluntary command.</p> <p>Oscilloscope no. 3: </p> <p>(Any drawing showing a frequency < 3 AP is accepted)</p> <p>Because the inhibitory message is stronger than that in the case of slight flexion of the foot leading to a decrease in the excitatory message transmitted through the motor neuron innervating the extensor muscle.</p> <p>Oscilloscope no. 4: </p> <p>(Any drawing showing a frequency > 3 AP is accepted)</p> <p>Because the excitatory message is stronger than that in the case of slight flexion of the foot what leads to an increase in the the excitatory message transmitted through the motor neuron innervating the flexor muscle.</p>	2

Part of the Ex	Exercise 4 (5 points)	Mark																								
1	<p>The amount of glucose in the blood entering the muscle at rest :90 mg/100 ml of blood is higher than that in the blood leaving the muscle: 87 mg/100 ml of blood (3 mg/100 ml) , similarly, the amount of glucose in the blood entering the encephalon at rest :91 mg/100 mL of blood is higher than that in the blood leaving the encephalon:80 mg/100 ml of blood (11 mg/100 ml > 3 mg/100 ml). This shows that the muscle and the encephalon at rest consume glucose and that the encephalon consumes more glucose than the muscle.</p>	0.75																								
2	<p style="text-align: center;">\downarrow Injection of glucagon</p> <table border="1" data-bbox="198 673 1426 842"> <thead> <tr> <th data-bbox="198 673 436 729">Time (h)</th><th data-bbox="436 673 500 729">-0,5</th><th data-bbox="500 673 547 729">0</th><th data-bbox="547 673 611 729">0,3</th><th data-bbox="611 673 674 729">0,5</th><th data-bbox="674 673 722 729">1</th><th data-bbox="722 673 786 729">1,5</th><th data-bbox="786 673 833 729">2</th><th data-bbox="833 673 881 729">3</th><th data-bbox="881 673 928 729">4</th><th data-bbox="928 673 976 729">5</th><th data-bbox="976 673 1024 729">6</th></tr> </thead> <tbody> <tr> <td data-bbox="198 729 436 842">Hepatic glycogen ($\mu\text{mol.L}^{-1}$)</td><td data-bbox="436 729 500 842">200</td><td data-bbox="500 729 547 842">-</td><td data-bbox="547 729 611 842">190</td><td data-bbox="611 729 674 842">180</td><td data-bbox="674 729 722 842">120</td><td data-bbox="722 729 786 842">80</td><td data-bbox="786 729 833 842">40</td><td data-bbox="833 729 881 842">20</td><td data-bbox="881 729 928 842">20</td><td data-bbox="928 729 976 842">10</td><td data-bbox="976 729 1024 842">20</td></tr> </tbody> </table> <p>Variation of the hepatic glycogen in function of time before and after the injection of glucagon</p>	Time (h)	-0,5	0	0,3	0,5	1	1,5	2	3	4	5	6	Hepatic glycogen ($\mu\text{mol.L}^{-1}$)	200	-	190	180	120	80	40	20	20	10	20	1.5
Time (h)	-0,5	0	0,3	0,5	1	1,5	2	3	4	5	6															
Hepatic glycogen ($\mu\text{mol.L}^{-1}$)	200	-	190	180	120	80	40	20	20	10	20															
3	<p>Document 2 : the concentration of insulin increased from 200 to 700 a.u while that of glucagon decreased from 500 to 300 a.u. when glucose levels increased respectively from 0.6 to 0.8 g. L⁻¹. However the secretion of glucagon increased from 300 to 400 a.u. while that of insulin decreased from 300 to 100 a.u following a decrease of glucose from 0.8 to 0.7 g. L⁻¹ . This shows that the secretion of insulin varies in the same direction as glycemia while that of glucagon varies inversely with glycemia and that insulin and glucagon are antagonistic hormones.</p> <p>Document 3 : Before the injection of glucagon, glycemia was 90 mg/100 mL and hepatic glycogen was 200μ mol L⁻¹. Following the glucagon injection at time 0 h the glycemia increases to reach 210 mg/100 ml while the hepatic glycogen decreases to reach 80 μ mol L⁻¹ at 1.5 h. This shows that glucagon causes hyperglycemia by promoting the breakdown of hepatic glycogen.</p> <p>After that, glycemia decreases from 210 mg/100 ml to 100 mg/100 ml (remaining greater than 90 mg/100 ml), while the glycogen levels continues to decrease to a value of 10μ mol L⁻¹ from 1.5h until 5 h. Then glycemia remained constant from hour 5 to hour 6 meanwhile the hepatic glycogen reincreases from 10μ mol L⁻¹ to 20 μ mol L⁻¹.</p> <p>This shows that the action of glucagon is temporary.</p>	1.5																								
4	<p>In a medium with insulin, the amount of glucose taken from the blood by the muscle is greater (1.88 mg per gram of muscle every 10 minutes) than that taken in medium without insulin (1.43 mg per gram of muscle / 10 minutes). Similarly, the amount of glycogen content in the muscle after 10 minutes in medium with insulin is greater (2.85 mg / g muscle) than that in the medium without insulin (2.45 mg / g muscle).</p> <p>This indicates that insulin causes an increase in the absorption of blood glucose by the muscle and its storage as glycogen.</p>	0.75																								
5	<p>Experiment 2 shows that the consumption of glucose by encephalon cells is constant 6g/h in media with or without insulin. Thus insulin has no effect on nerve cells and thus these latter are not target cells for insulin</p>	0.5																								

الاسم : مسابقة في مادة "علوم الحياة"
الرقم : المدة ثلاثة ساعات

Answer the following exercises:

Exercise 1 (5 points)

The EBV virus infects 90% of the world population, but in a benign manner. This virus persists in the body. Its target cells are B lymphocytes.

Document 1 shows the activity of the EBV in “naive B Lymphocytes” (B lymphocytes that have never encountered the specific antigen) and in memory B lymphocytes specific for this antigen.

- Determine by referring to document 1, how the EBV virus persists and is produced in the body.

To better understand one of the immune responses triggered against the EBV virus, we follow up the evolution of anti-VCA and anti-EBNA antibodies directed respectively against two peptides VCA and EBNA that are found on the surface of this virus. The results are shown in document 2.

- Name the immune response revealed by these measurements. Justify the answer.
- Analyze the results of document 2. What can we draw out?

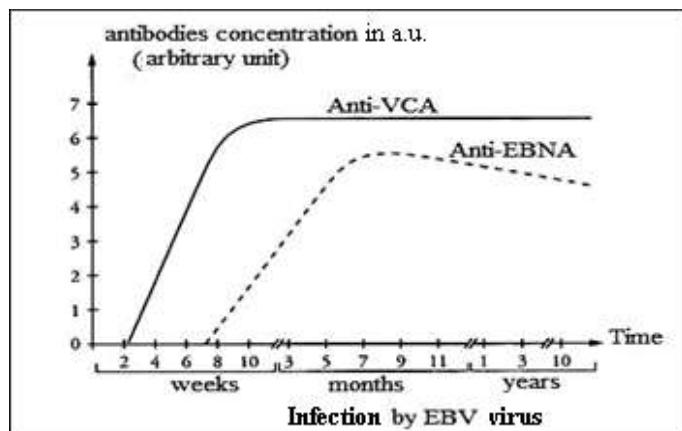
To Petri dishes containing appropriate culture medium, we add Lymphocytes (BL and TL) taken from different individuals infected or not by different viruses, EBV or other viruses. All the lymphocytes used in each experiment have the same HLA. Document 3 presents the conditions and the results of these experiments.

- Describe, in a short text, the experiments and the obtained results presented in document 3.
- Explain the obtained results of these experiments.

Immune responses against a virus

Activity of EBV	Naive B Lymphocyte	Memory B Lymphocyte
State of EBV in the lymphocyte	Active	Dormant
Presentation of viral peptides on the surface of the lymphocyte	Yes	No
Production of new viruses released into blood and able to infect other BL	Yes	No except if it is reactivated

Document 1



Document 2

Experiment	Experimental conditions	Results
1	T L of an individual infected by EBV ↓ BL infected by EBV	100% lysed BL
2	T L of an individual infected by EBV ↓ BL not infected by EBV	No lysed BL
3	T L of an individual infected by EBV ↓ memory BL infected by EBV	No lysed BL
4	T L of an individual infected by EBV ↓ BL infected by another virus	No lysed BL
5	T L of an individual not infected by EBV ↓ BL infected by EBV	No lysed BL

Document 3

Legend: → : Add

Exercise 2 (5 points)

The Xeroderma pigmentosum is a disease that results in skin lesions which can develop into cancerous tumors and eye lesions. We are interested in the causes of this disease and the relative influence of genes and environment on its appearance. The body cells have, in their nucleus, enzymes that can repair DNA whenever this latter shows alterations. One of these enzymes is the ERCC3 which is coded by the gene G-ERCC3.

We present in document 2 the nucleotides sequence of a fragment of the non-transcribed strand of the gene G-ERCC3 of a healthy individual (allele G1) and the sequence of the equivalent fragment of an individual affected by xeroderma pigmentosum (allele G2).

Document 2

Allele	nucleotides sequence of the fragment
G1	1 12 ...AAG AAG AGC AAC...
G2	1 12 ...AAG AAG AGA AAC...

DNA alteration

		NUCLEOTIDE POSITION 2							
		U	C	A	G				
NUCLEOTIDE POSITION 1	U	UUU UUC UUA UUG	phenylalanine serine leucine	UAU UAC UAA UAG	tyrosine stop	UGC UGC UGA UGG	cysteine stop tryptophane	U C A G	
	C	CUU CUC CUA CUG	leucine	CCU CCC CCA CCG	proline	CAU CAC CAA CAG	histidine glutamine	CGU CGC CGA CGG	
	A	AUU AUC AUA AUG	isoleucine methionine	ACU ACC ACA ACG	threonine	AAU AAC AAA AAG	asparagine lysine	AGU AGC AGA AGG	
	G	GUU GUC GUA GUG	valine	GCU GCC GCA GCG	alanine	GAU GAC GAA GAG	aspartic acid glutamic acid	GGU GGC GGA GGG	
								U C A G	
		A: Adenine		U: Uracile		G: Guanine		C: Cytosine	

Document 1

	Reference electrophoresis	Individual A	Individual B	Individual C
ERCC3 (coded by allele G1)	—		—	—
ERCC3 (coded by allele G2)	—	—	—	—

Document 3

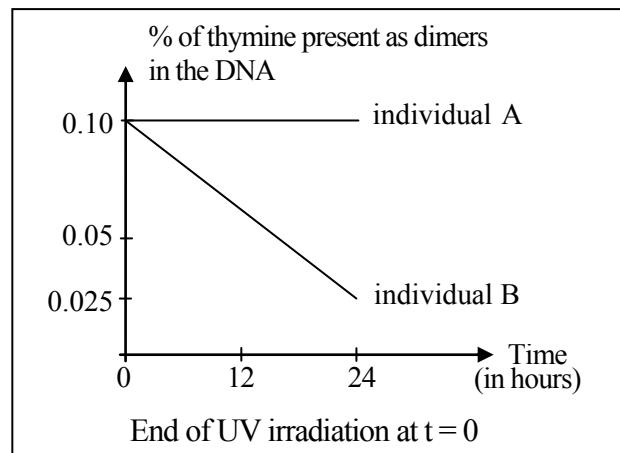
- 1- Determine using the genetic code table (doc.1) the amino acid sequence of the portion of each of the enzymes ERCC3 coded by the allele G1 and by the allele G2.

We can separate, by electrophoresis, the enzyme ERCC3 coded by the allele G1 and enzyme ERCC3 coded by allele G2. Electrophoresis is performed for three different individuals: A, B and C. Individual A is affected with Xeroderma pigmentosum, and individuals B and C are not. The results are presented in document 3.

- 2- Write the genotypes of individuals A, B and C. Justify the answer.
 3- Specify the dominant allele and the recessive one. Justify the answer.

Upon exposure to ultra violet sunlight rays, the DNA of skin cells undergo alterations, particularly the formation of dimers between two successive thymines T-T. We measure the evolution of the percentage of dimers in the two individuals A and B after being subjected to irradiation with ultraviolet rays. The measured results are presented in document 4.

- 4- Analyze the obtained results in document 4.
 5- Based only on the previous given:
 5-1- Explain the results of document 4.
 5-2- Specify the factors that determine the development of the studied disease. Justify the answer.



Document 4

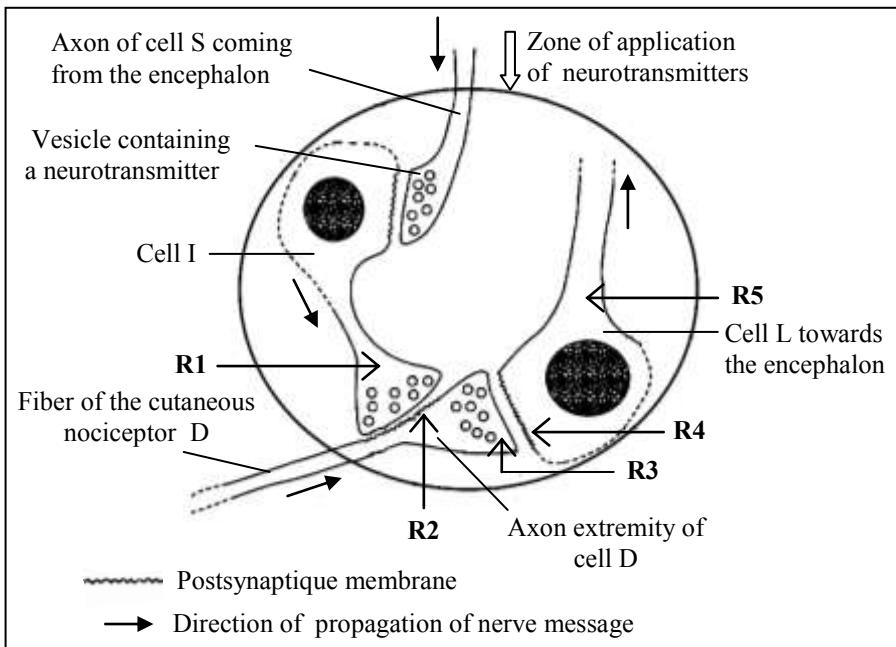
Exercise 3 (5 points)

Neurotransmitters and pain

In the posterior horn of the spinal cord, we observe cells I in addition to extremities of fibers of cells D and S as well as cell bodies of cells L (doc.1).

In the framework of studying the transmission of the pain message, we apply the same molar concentration of neurotransmitters, enkephalin or substance P, in the defined zone of document 1.

We record, using the microelectrodes R1, R2, R3 and R4, the membrane potentials of cells I, D, and L with respect to a reference potential. The results are presented in document 2.



Document 1

	Evolution of the membrane potentials at the level of the recording electrodes			
	R1	R2	R3	R4
Application of enkephaline	-70 ———	-70 ~~~~~	-70 ———	-70 ———
Application of substance P	-70 ———	-70 ———	-70 ———	-70 ~~~~~

Document 2

1- Specify the role and site of action of each of the used neurotransmitters. Justify the answer.

We stimulate a cutaneous nociceptor D whose fibres are responsible for the slow transmission of intense and prolonged pain. We stimulate again the same cutaneous nociceptor D with the application of serotonin neurotransmitter.

The obtained recordings of R1, R2, R3 and R5 of these experiments are shown in document 3.

	Evolution of the membrane potentials at the level of the recording electrodes			
	R1	R2	R3	R5
Case A: Stimulation of the cutaneous nociceptor D without application of any substance	-70 ———	AP 0 -70	0 -70	0 -70
Case B : Stimulation of the cutaneous nociceptor D with the application of serotonin	0 -70	-70 ~~~~~	-70 ———	-70 ———

Document 3

- 2- Interpret the obtained results in case A.
- 3- Compare the recordings obtained in case B to those obtained in case A, and draw out the role and the site of action of serotonin.
- 4- Explain, from what precedes, how the encephalon interferes in blocking the transmission of the pain message.

Exercise 4 (5 points)**Relations between the pituitary gland and the testis**

The testis produces testosterone in a constant manner due to a regulatory system that we aim to discover by performing the following experiments.

Experiment 1

We inject gonadotropins (anterior pituitary hormones) into a male animal that have not reached puberty and whose testicular cells are normally inactive. The consequences of these injections on three types of testicular cells are presented in document 1.

- 1- Specify the role of : Sertoli cells, spermatogonia and Leydig cells.
- 2- Analyze the results of experiment 1 and draw out the target cells of each of the pituitary hormones LH and FSH.

Experiment 2

Leydig cells are extracted from pig testes and cultured in vitro. We add different molecules, LH and/or TNF α , to the culture medium and we measure, at the same time, the production of testosterone. TNF α is a molecule that blocks the action of LH by binding the receptors of LH target cells. Document 2 shows the effects of LH on these cells.

- 3- Determine by referring to document 2, how are Leydig cells activated.

Experiment 3

In order to study the action of certain types of cells on the activity of pituitary cells, we prepare three appropriate culture media and we measure the level of gonadotropins released in these media after a period of incubation (document 3).

Experimental conditions	Medium 1	Medium 2	Medium 3
	Pituitary cells only	Pituitary cells + kidney cells or spleen cells	Pituitary cells + Leydig cells
Release of FSH	100%	100%	100%
Release of LH	100%	100%	60%

Document 3

- 4- Interpret the results of experiment 3.
- 5- Specify the type of feedback control revealed by experiment 3. Justify the answer.

الاسم :

اسس التصحيح

الرقم :

مادة "علوم الحياة"

Part of the Ex	Answer key	Mark
	Exercise1 (5 points)	
1	<p>The virus persists in the body because it remains in the dormant state in the memory BL(0.25pt)</p> <p>The virus is produced by naive B lymphocytes that are once infected and by the memory BL once reactivated.(0.25pt)</p>	0.5
2	Specific humoral immune response (0.25pt) because the actors in this response are antibodies anti-VCA and anti- EBVA . (0.25pt)	0.5
3	<p>Anti-VCA antibodies appear in blood two weeks after infection and reach their maximum concentration 6.5 a. u. within eight weeks after the infection then stabilizes for the following 10 years. However Anti-EBVA antibodies appear later at the 7th week (7 w > 2 w) and reach their maximum concentration 5.5a.u. (5.5 < 6.5 a. u.) after more than 7 months (7months>8 w) then their concentration decreased slightly to reach 4.5 a. u. (4.5 < 6.5 a.u.) after 10 years. (1pt)</p> <p>This shows that the body develops two different humoral immune responses against two different peptides (different antigens) of EBV virus, and that the response triggered against the VCA is faster, more amplified and more sustainable than the one triggered against the EBNA. (0. 5pt)</p>	1.5
4	<p>Experiment 1: Lymphocytes TL of an individual infected with the virus EBV are added into the medium containing BL infected with EBV, we obtain 100% of lysis LB.</p> <p>Experiment 2: Lymphocytes TL of an individual infected with the virus EBV are added into the medium containing BL non infected with EBV, no lysis of BL is obtained.</p> <p>Experiment 3: Lymphocytes TL of an individual infected with the virus EBV are added into the medium containing memory BL infected with EBV, no lysis of BL is obtained.</p> <p>Experiment 4: Lymphocytes TL of an individual infected with the virus EBV are added into the medium containing BL infected with another virus, no lysis of BL is obtained.</p> <p>Experiment 5: Lymphocytes TL of an individual non infected with the virus EBV are added in the medium containing BL infected with EBV, no lysis of BL is obtained .</p>	1.25 (5 x0.25)
5	<p>The Lymphocytes T cytotoxic having receptors that recognize infected cells presenting at with their surface self HLA having non self peptide which has activated the same T8 lymphocytes which is identified in experiment 1 (there is 100% of lysed BL).</p> <p>In experiment 2, non-infected BL do not present non-self peptides this is why we do not observe any lysis.</p> <p>In experiment 3, B memory cells infected by the same virus as TL do not present non-self peptides. They are not identified by Tc and they are not lysed.</p> <p>In experience 4, BL infected by another virus present another non-self peptides. They are not identified by Tc and they are not lysed.</p> <p>In experiment 5, TL from an individual non-infected with EBV are not activated and differentiated into Tc and do not cause the lysis of BL infected by the virus.</p>	1.25 (5 x0.25)

Part of the EX	Answer key	Mark
	Exercise 2 (5 points)	
1	<p>mRNA resulting from the transcription of the allele G1: AAG AAG AGC AAC Amino acid sequence of the polypeptide coded by the allele G1: Lysine – Lysine – Serine – Asparagine</p> <p>mRNA resulting from the transcription of the allele G2: AAG AAG AGA AAC Amino acid sequence of the polypeptide coded by the allele G2: Lysine – Lysine – Arginine – Asparagine Or We can obtain it directly from the non transcribed strand of DNA by replacing T by U. thus, we obtain the same sequence for both the mRNA and the DNA non-transcribed strand.</p> <p>Amino acid sequence of the polypeptide coded by the allele G1: Lysine – Lysine – Serine – Asparagine Amino acid sequence of the polypeptide coded by the allele G2: Lysine – Lysine – Arginine – Asparagine</p>	0.75
2	<p>The genotype of individual A G2//G2 (0.25 pt) because the result of his electrophoresis shows one type of enzyme ERCC3 that is coded by allele G2. (0.25 pt)</p> <p>The genotype of individual B is G1//G1 (0.25 pt) because the result of his electrophoresis shows one type of enzyme ERCC3 that is coded by allele G1. (0.25 pt)</p> <p>The genotype of individual C is G1//G2 (0.25 pt) because the result of his electrophoresis shows the two types of enzymes. (0.25 pt)</p>	1.5
3	<p>The allele G1 is dominant (0.25 pt) and the allele G2 is recessive (0.25 pt) because individual C who is heterozygous of genotype G1//G2 is not affected by Xeroderma Pigmentosum, Allele G2 is masked and not expressed phenotypically in the presence of allele G1 which dominates allele G2(0.25 pt).</p>	0.75
4	<p>The percentage of thymine dimers in the DNA remains constant (0.10%) through the 24 hours in individual A affected by xeroderma, while it decreases from 0.10% to 0.025% through the 24 hours in the healthy individual B after their exposition to ultraviolet irradiation.</p>	0.5
5-1	<p>Individual A (doc. 3) affected with xeroderma has no functional enzyme ERCC3 which is responsible of repairing the DNA alterations . The thymine dimers formed due to the exposition to ultra violet radiation cannot be repaired in this individual and thus the percentage of dimers T-T remains stable (0.25 pt). In the healthy individual B, which possesses functional ERCC3 enzyme, the altered DNA formed by ultraviolet irradiation is gradually repaired by this enzyme thus the percentage of thymine dimers decreases (0.25pt)</p>	0.5
5-2	<p>Two factors determine the development of Xeroderma pigmentosum:</p> <ul style="list-style-type: none"> - The genetic factor(0.25pt): the disease develops only in homozygous individuals with two mutant alleles of a gene coding for the enzyme ERCC3 involved in the repair of DNA damage(0.25pt); - The environmental factor(0.25 pt): exposure to sun ultraviolet rays provokes the alteration of DNA(0.25 pt). 	1

Part of the Ex	Answer key	Note
Exercise 3 (5 points)		
1	<p>Role of the enkephalin: inhibitory (0.25 pt) Site of action: synapse between cell I and cell D (0.25 pt) Because following the application of enkephalin we observe a hyperpolarization having an amplitude of 25mV only at the level of R2 while we observe a resting potential of -70mV at the levels of R1, R3 and R4. (0.5 pt)</p> <p>Role of substance P: excitatory(0.25 pt) Site of action : synapse between the cell D and cell L(0.25 pt) Because following the application of substance P we observe a hypopolarization having an amplitude of 20mV only at the level of R4 while we observe a resting potential of -70mV at the levels of R1, R2 and R3. (0.5 pt)</p>	2
2	<p>A nervous message of 3AP/6ms having the same amplitude (100mV) is recorded at the levels of R2 and R3. This shows that the stimulation is efficient and that the action potential propagates in the same cell keeping the same amplitude and the same frequency. (0.25 pt) Similarly, we observe a nervous message of the same amplitude as R2 and R3 at the level of R5 but with a lower frequency of 2AP/6ms following a depolarization of the membrane that reaches the threshold. This shows that the synapse between the cells D and L is excitatory and attenuates only the frequency of the nervous message and not its amplitude. (0.25 pt) However we observe always a resting potential of -70 mV at the level of R1. This shows that the nervous message triggered by the nociceptor doesn't propagate from cell D to cell I. (0.25 pt)</p>	0.75
3	<p>A single AP is recorded at the level of R1 in the presence of serotonin (case B) while no action potential is recorded in case A. (0.25 pt) A hyperpolarization is recorded at the level of R2 in the presence of serotonin (case B) while 3 AP/ 6ms is recorded in case A. (0.25 pt) No response is recorded at the level of in R3 and R5 in the presence of serotonin (case B) while 3AP/ 6ms is recorded at the level of R3(0.25 pt) and 2AP/ 6ms at the level of R5(0.25 pt) in case A. This shows that serotonin excites only the cell I thus inhibiting the propagation of the pain nervous message at the level of the cell D(0.25 pt) It acts between the axon of the cell S and the cell I. (0.25 pt)</p>	1.5
4	<p>The encephalon sends a nervous message through the cell S and provokes the release of serotonin at the level of the synapse between the cell S and the cell I. This triggers a nervous message at the level of cell I. This message propagates and induces the release of enkephalin at the level of the synapse I-D provoking a hyperpolarization at the level of the postsynaptic membrane of the cell D. Thus the propagation of the nerve message at the level of cell D is inhibited and the release of substance P is prevented thus stopping transmission of the pain nerve message.</p>	0.75

Part of the Ex	Answer key	Mark
	Exercise 4 (5 points)	
1	Spermatogonium: mother cell of male gametes.(0.25pt) Sertoli cell: nurturing role for germ cells.(0.25pt) Leydig cells : produce testosterone.(0.25pt)	0.75
2	Spermatogonia are only activated by FSH similarly Sertoli cells are only developed under the effect of FSH, however Leydig cells are not activated except by LH.(0. 5pt). We can draw out that the target cells of LH are Leydig cells. (0.25pt) Whereas Spermatogonia cells and Sertoli cells are the target cells of FSH.(0.25pt)	1
3	The presence of LH in the culture of Leydig cells, in absence of TNF α , has strongly increased the production of testosterone, which passes from 2 a.u. (without LH) to 50 a.u. (with LH). Thus LH activates Leydig cells. However, the production of testosterone decreases 50 a.u. to 5 a.u. when TNF α is added to LH, thus the activation of Leydig cells is done by the fixation of LH to their free receptors.	1
4	The secretion of gonadotropins is 100% for FSH and LH in media 1 and 2 where the pituitary cells are alone or with kidney cells or spleen cells. However, only the level of LH decreases to 60% in the medium where the pituitary cells are with leydig cells. This shows that only Leydig cells are able to inhibit the activity of pituitary cells that secrete LH and have no effect on those that secrete FSH.	1.25
5	It is a negative feedback control.(0. 5pt) Because the level of LH (produced by pituitary cells) and the level of testosterone (produced by Leydig cells) vary in an opposite manner.(0. 5pt) Or When the level of testosterone , that is produced by Leydig cells, increases the level of LH decreases.	1

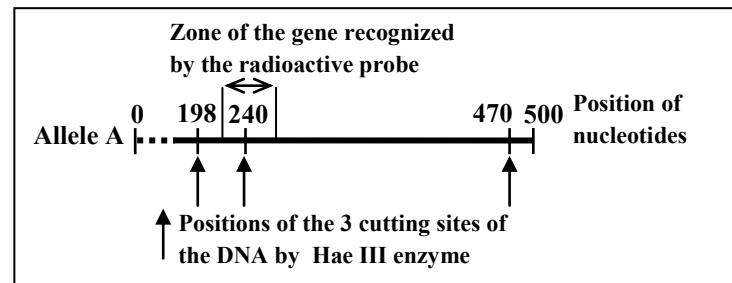
الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Answer the following exercises

Exercise 1 (5 points)

Albinism is a hereditary deficiency characterized by the absence of skin, eyes and hair pigmentation due to the absence of a black pigment: melanin. Tyrosinase is an enzyme involved in the biosynthesis of this pigment. The gene coding for tyrosinase exists in many forms of alleles and is carried by an autosome. Only two alleles are taken into consideration: allele A which codes for an active tyrosinase that is responsible for the synthesis of melanin and allele B that codes for an inactive tyrosinase that does not permit the synthesis of melanin.

Document 1 represents the map of the restriction sites recognized by Hae III enzyme in a portion of 500 base pairs (bp) of the allele A of tyrosinase gene.

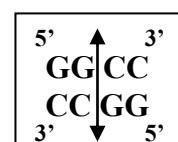


Document 1

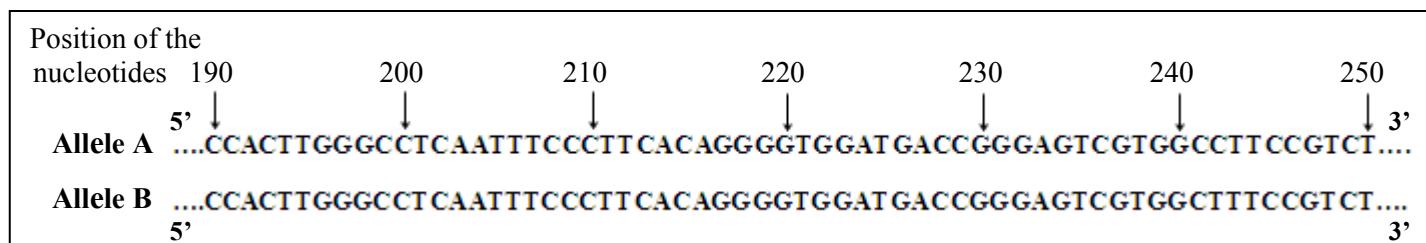
- Determine the number and the length of the restriction fragments obtained as a result of cutting allele A by Hae III enzyme.

Document 2 shows the restriction site of Hae III enzyme.

Document 3 reveals a partial single-stranded sequence of the two alleles A and B of tyrosinase gene.



Document 2

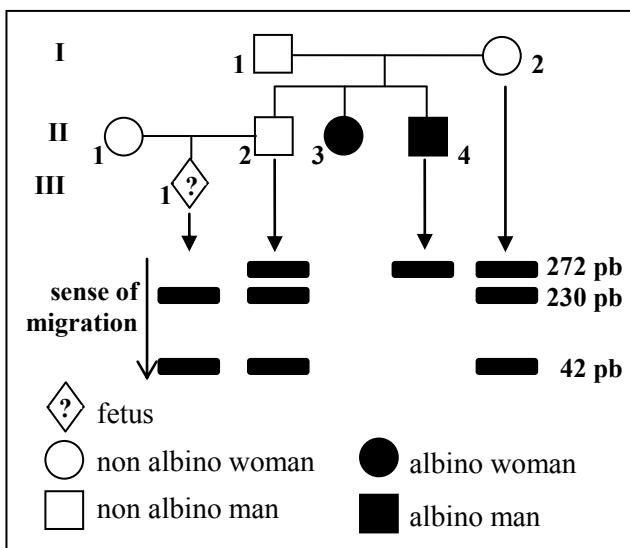


Document 3

- Compare these two sequences. Draw out the position and the type of mutation that took place.
- Determine the consequence of this mutation on the produced restriction fragments upon using Hae III enzyme on allele B.

Document 4 represents the pedigree of a family whose some members show albinism. It also shows the results of the electrophoresis of the restriction fragments obtained following the action of Hae III enzyme on a portion of the tyrosinase gene. These fragments are obtained by the Southern blot technique for four members of the family.

- Specify the respective alleles of individuals I₂ and II₄. Justify the answer by referring to the results of electrophoresis.
- Indicate, referring to document 4, whether the allele of albinism is dominant or recessive. Justify the answer.
- Establish a prenatal diagnosis of albinism for the fetus III₁.



Document 4

Exercise 2 (5 points)

Cellular Cooperation and Production of Antibodies

In the framework of determining the conditions of the production of antibodies during the immune response, we perform a series of experiments on mice of the same strain.

Experiment 1: Mice are subjected to the ablation of the thymus followed by irradiation that destroys all cells of the immune system. These mice are then divided into 4 lots and treated as shown in document 1.

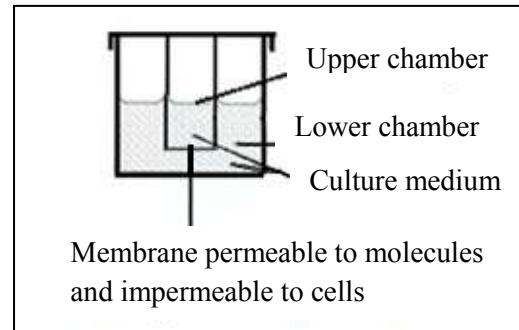
Ablation of the thymus then irradiation of the mice				
	Lot 1	Lot 2	Lot 3	Lot 4
Injection of lymphocytes removed from mice of the same strain	T	B and T	B and T	B
Injection of an antigen: SRBC (sheep red blood cells)	Yes	Yes	No	Yes
One week later, removal of serum from the mice and addition of SRBC to the serum				
Results : agglutination of SRBC	absence	presence	absence	absence

Document 1

- Interpret the experimental results of experiment 1.
- Specify the aim of destroying the cells of the immune system before starting the experiment.

Experiment 2: A mouse receives an injection of sheep red blood cells (SRBC). Three days later, we extract lymphocytes from its spleen. These lymphocytes are distributed into 4 identical lots then cultured in Marbrook chamber (document 2) according to the procedure described in document 3.

Few days later, the culture medium is filtered and the collected liquid is added to SRBC. The results are shown in document 3.



Document 2

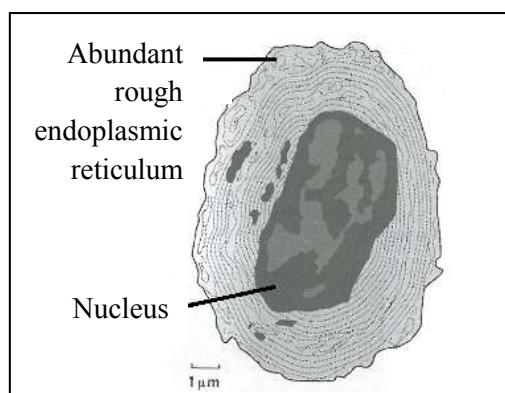
Culture medium	1	2	3	4
Lymphocytes placed in the upper chamber	none	T	none	none
Lymphocytes placed in the lower chamber	T and B	B	B	T
Results : agglutination of SRBC	Strong	Strong	Null	Null

Document 3

- Analyze the results of media 1 and 2. What can you draw out?

Document 4 illustrates an electronography of an antibody secreting cell that is found in large quantities, in media 1 and 2 of document 3 and absent in media 3 and 4.

- Name this cell. Justify the answer.
- Explain the variation in the quantity of this type of cells in the four media of experiment 2.



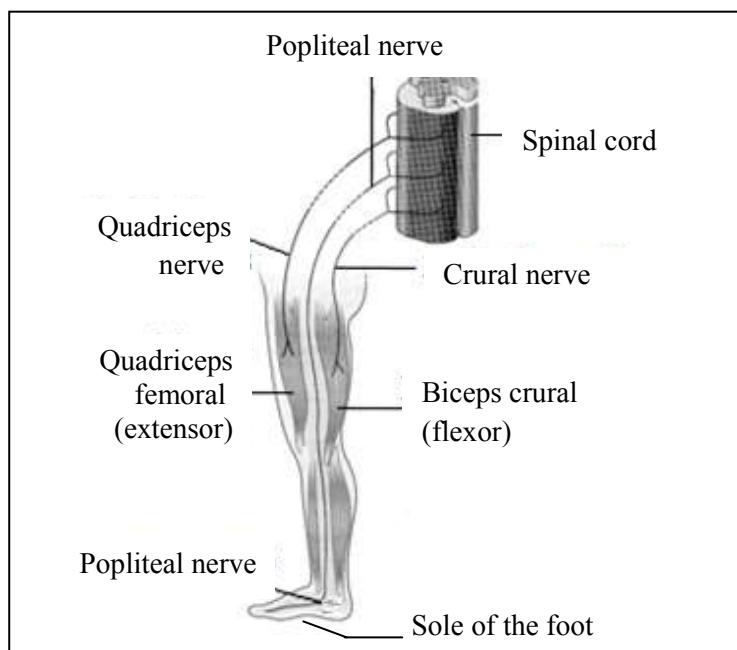
Document 4

Exercise 3 (5 points)

Protection Reflex

In a man who has been accidentally subjected to a section in the upper level of his spinal cord, the contact of a hot object with the skin of the sole of the foot causes systematically a protection reflex that is manifested by the flexion of the corresponding lower limb. We aim to study the mechanisms implicated in such a response.

Document 1 shows the muscles and the nerves involved in such a protection reflex. Document 2 represents the results of an experimental study performed on a spinal animal (cat) having only the spinal cord as a nervous center. The muscle structure and the innervation of this animal are similar to those of humans.



Document 1

Experiments	Popliteal nerve	Crural nerve	Nerve of the quadriceps
Sectioning of the nerve	disappearance of the flexion of the lower limb	disappearance of the contraction of the biceps crural	disappearance of the contraction of the quadriceps femoral
Excitation of the central end*	flexion of the lower limb	No reaction	No reaction
Excitation of the peripheral end *	No reaction	contraction of the biceps crural	contraction of the quadriceps femoral

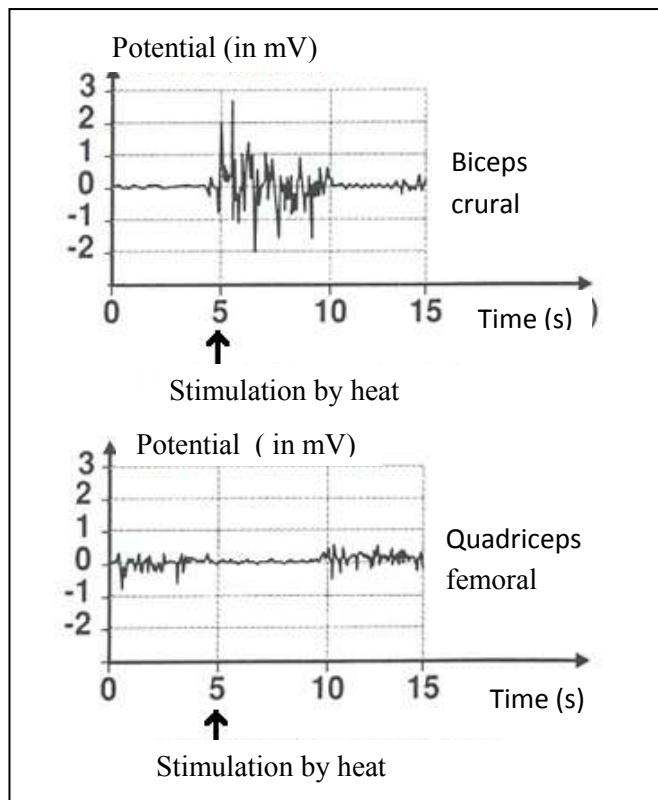
* At the level of the sectioning of a nerve, the end that is still attached to the nervous center is called the central end whereas the end that is still attached to the peripheral organs (muscle or skin) is called the peripheral end.

Document 2

- Specify, based on the experimental results, and for each nerve, whether it plays an afferent / sensory role or an efferent / motor role in this reflex. Justify the answer.

Document 3 represents the electromyograms recorded at the level of the biceps crural and the quadriceps femoral before and after stimulation by heat at time 5 seconds.

- Compare these electromyograms. What can you draw out?
- Draw a functional diagram relating the structures involved in this protection reflex.
- Give one difference between the protection reflex and the myotatic reflex.



Document 3

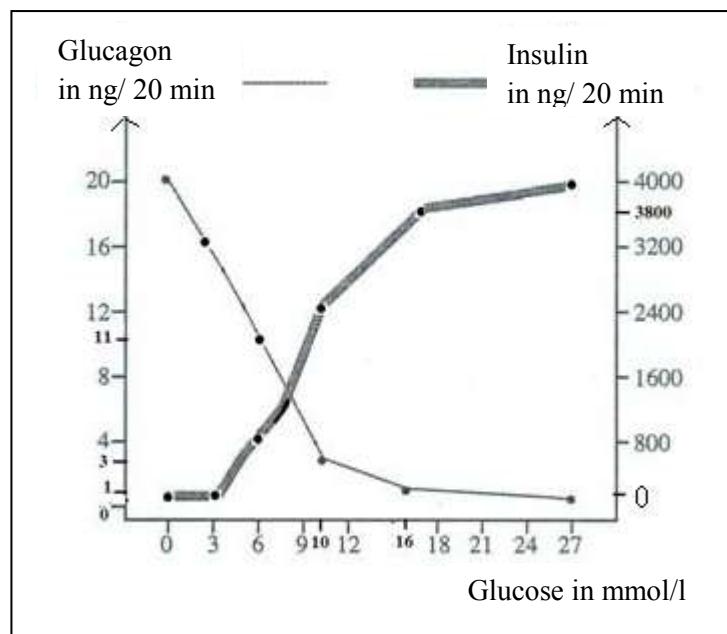
Exercise 4 (5 points)**Role of Liver and Pancreas in the Regulation of Glycemia**

In order to show the relation between glycemia, liver and the secretion of pancreatic cells, we perform the following experiments on a rat and a dog.

Experiment 1:

We isolate the pancreas of a rat and we perfuse it with glucose solution of increasing concentrations. Each test lasts for 20 minutes. For each glucose concentration, we measure the level of insulin and that of glucagon in the liquid leaving the pancreas. The results are represented in document 1.

- 1- Tabulate the variation of glucagon and that of insulin levels as a function of glucose concentration.
- 2- Analyze the results of document 1. What can you draw out concerning the role of pancreatic cells?

**Document 1****Experiment 2:**

An injection of insulin provokes, in a normal fasting dog, a rapid drop in the "hepatic balance". This balance corresponds to the difference between the concentration of glucose leaving the liver and that of glucose entering the liver. This balance drops, within less than an hour, from 42 mg / minute to approximately 0, following the injection of 1800 mg of insulin.

In a control dog, that is not subjected to insulin injection, the hepatic balance remains close to 42 mg/minute during the same period.

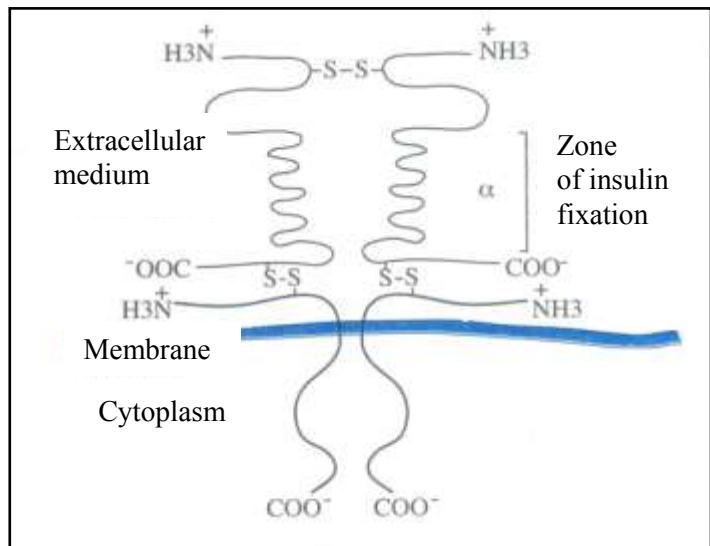
Experiment 3:

An injection of glucagon to a normal animal provokes hyperglycemia. This latter does not occur in an animal which liver was removed.

- 3- Interpret experiments 2 and 3. What can you deduce about maintaining normal glycemia in the body?

Document 2 shows a schematic representation of insulin membranous receptor of a hepatic cell.

- 4- Explain, referring to document 2, the mode of action of insulin on the hepatic cells.

**Document 2**

Part of ex	Answer key	Note
	Exercise 1 (5 points)	
1	<p>Allele A has 3 restriction sites of enzyme Hae III at the level of the nucleotide numbers 198, 240 and 470. Therefore, the enzyme cuts the allele into 4 fragments (1/4 pt). The length of each fragment is:</p> <p>a fragment of 198 base pairs (bp) (before the site 198), a fragment of 42 bp (between sites of 198 and 240), a fragment of 230 pb (between the sites of 240 and 470) and a fourth fragment which length is 30 bp (beyond the site 470) (1/2 pt).</p>	3/4
2	<p>The nucleotide sequences of the portions of the two alleles are identical except at the level of the nucleotide number 242 where nucleotide C in allele A is replaced by the nucleotide T in allele B(1/2 pt). It is a mutation by substitution (1/4 pt) at the level of nucleotide 242(1/4 pt).</p>	1
3	<p>Hae III enzyme cuts the DNA when encountering the sequence GGCC. The cutting is done between GG and CC (document 2).</p> <p>Document 3 shows that the restriction site at the level of the nucleotide 240 does no longer exist for allele B due to the mutation by substitution. Instead of the GGCC sequence for allele A there is a GGCT sequence for allele B.</p> <p>As a result, the enzymatic treatment of allele B will give:</p> <p>a fragment of 198 base pairs (bp) (before the site 198), a fragment of 272 bp instead of the two fragments (42 and 230 bp) a third fragment which length is 30 bp (beyond the site 470).</p>	3/4
4	<p>Document 4 gives the disposition of the fragments revealed by autoradiography for the four family member.</p> <p>Individual I₂ has two alleles A and B (1/4 pt) because the electrophoresis results show three fragments: 272 pb that corresponds to allele B and 42 and 230 pb that correspond to allele A(1/4 pt).</p> <p>Individual II₄ has two alleles B (1/4 pt) because the electrophoresis results show only the fragment of 272 pb that corresponds to allele B (1/4 pt).</p>	1
5	<p>Albinism allele is recessive with respect to the normal allele (1/4 pt) because individual I₂ having the two alleles A and B is of normal phenotype. Therefore allele A alone is expressed and allele B is masked (1/2 pt).</p> <p>Or</p> <p>Because II₃ and II₄ children with albinism arise from normal parents I₁ and I₂, then the allele of albinism is masked in the parents. Therefore allele B determining albinism is recessive with respect to the dominant allele A.</p>	3/4
6	<p>The fetus III₁ possesses only the fragments of 230 and 42 pb that correspond to the allele A. So, the fetus III₁ does not have except the allele A and he will not be albino but of normal phenotype.</p>	3/4

Part of ex	Answer key	Note
	Exercise 2 (5 points)	
1	<p>There is agglutination of SRBC in lot 2 where there was an injection of lymphocytes B and T and SRBC at the same time whereas there is no agglutination in lot 1 where there were only injection of lymphocytes T with SRBC and in lot 4 where there are only injection of B lymphocytes with SRBC; This shows that the agglutination requires the cooperation of TL and BL or the presence of the TL and BL at the same time.</p>	1 1/2
2	<p>There is agglutination of SRBC in lot 2 where there was an injection of lymphocytes B and T and SRBC at the same time however there is no agglutination in lot 3 where there were injection of lymphocytes B and T without SRBC. This shows that the contact with the antigen a week in advance is necessary to get agglutination.</p>	1/2
3	<p>There is strong agglutination of SRBC in the media 1 and 2 where B and T lymphocytes are found together whether they are in the same medium (medium 1) or separated by a membrane that is impermeable to cells but permeable to molecules (medium 2). (1/2 pt) Therefore, the agglutination of SRBC that is due to the production of anti-SRBC antibodies requires the cooperation of B and T lymphocytes via molecules and not by direct contact. (1/2 pt)</p>	1
4	<p>Plasmocyte (1/2 pt) because this cell has a voluminous cytoplasm that is rich in rough endoplasmic reticulum, cytoplasmic organelle that is indispensable for the synthesis of proteins such as antibodies. (1/2 pt)</p>	1
5	<p>Plasmocytes are derived from the differentiation of lymphocytes B which are absent in medium 4 where there is only TL, hence plasmocytes are absent in this medium. The differentiation of LB into plasmocytes is stimulated by IL 4 that is secreted by TL that are absent in medium 3, hence plasmocytes are absent in this medium. However B and T cells are present in media 1 and 2. IL 4 stimulates directly the B cells in the lower chamber (medium 1) or crosses the permeable membrane and stimulates B cells (medium 2). Hence the abundance of plasmocytes in these two media.</p>	1

Part of ex	Answer key	Note
	Exercise 3 (5 points)	
1	<p>The popliteal nerve is afferent (sensitive). (1/4pt) because the flexion of the lower limb disappears following the sectioning of popliteal nerve and the stimulation of its peripheral end however the flexion appears following the stimulation of its central end; This shows that the nervous message is transmitted by this nerve from the periphery to spinal cord (centripetal direction). (1/2pt)</p> <p>The crural nerve is efferent. (1/4 pt) because there is no more contraction of the biceps crural following the sectioning of the crural nerve and the stimulation of its central end while the biceps crural contracts following the stimulation of its peripheral end; This shows that crural nerve transmits the nervous message from the spinal cord toward the biceps crural. (1/2pt)</p> <p>The nerve of the quadriceps is efferent. (1/4pt) because there is no more contraction of the quadriceps femoral following the sectioning of the quadriceps nerve and the stimulation of its central end while the quadriceps femoral contracts following the stimulation of the peripheral end of this nerve; This shows that quadriceps nerve transmits the nervous message from the spinal cord toward the quadriceps femoral. (1/2pt)</p>	2 1/4
2	<p>From 0 to 5 and from 10 to 15 s, the electromyogram of the quadriceps femoral has a amplitude that fluctuates between -1 and 1mv greater than that of the electromyogram of the biceps crural that is almost null.</p> <p>From 5 to 10 mv, the electromyogram of the biceps crural has as amplitude that fluctuates between-2 and 2.5 mv greater than that of the electromyogram of the quadriceps femoral which is almost null. (1/2pt)</p> <p>Thus, the flexion of the lower limb is due to the activity of the biceps crural and the relaxation of the quadriceps femoral and that these two muscles are antagonistic. (1/2pt)</p>	1
3	<p>Functional diagram of the structures implicated in the protection reflex</p>	11/4
4	In the protection reflex, the receptor is the skin and the effector organ is the muscle, while in the myotatic reflex, the stretched muscle is, at the same time, the receptor organ and the effector organ.	1/2

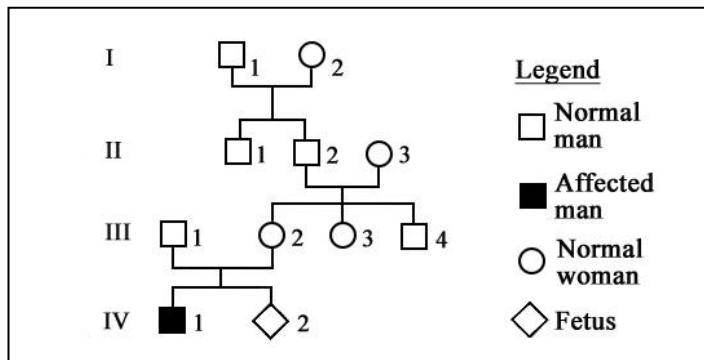
Part of ex	Answer key	Note																					
	Exercise 4 (5 points)																						
1	<p>Variation of glucagon and insulin levels as a function of the glucose concentration.</p> <table border="1" data-bbox="212 323 1387 570"> <tbody> <tr> <td data-bbox="212 323 700 406">Glucose (in mmol/l)</td><td data-bbox="700 323 763 406">0</td><td data-bbox="763 323 827 406">3</td><td data-bbox="827 323 890 406">6</td><td data-bbox="890 323 954 406">10</td><td data-bbox="954 323 1017 406">16</td><td data-bbox="1017 323 1387 406">27</td></tr> <tr> <td data-bbox="212 406 700 489">Glucagon (in ng/20 min)</td><td data-bbox="700 406 763 489">20</td><td data-bbox="763 406 827 489">16</td><td data-bbox="827 406 890 489">11</td><td data-bbox="890 406 954 489">3</td><td data-bbox="954 406 1017 489">1</td><td data-bbox="1017 406 1387 489">0</td></tr> <tr> <td data-bbox="212 489 700 570">Insulin (in ng/20 min)</td><td data-bbox="700 489 763 570">0</td><td data-bbox="763 489 827 570">0</td><td data-bbox="827 489 890 570">800</td><td data-bbox="890 489 954 570">2400</td><td data-bbox="954 489 1017 570">3800</td><td data-bbox="1017 489 1387 570">4000</td></tr> </tbody> </table>	Glucose (in mmol/l)	0	3	6	10	16	27	Glucagon (in ng/20 min)	20	16	11	3	1	0	Insulin (in ng/20 min)	0	0	800	2400	3800	4000	11/2
Glucose (in mmol/l)	0	3	6	10	16	27																	
Glucagon (in ng/20 min)	20	16	11	3	1	0																	
Insulin (in ng/20 min)	0	0	800	2400	3800	4000																	
2	<p>Glucagon levels decreases rapidly from 20 ng / 20 min till 16 ng / 20 min while the insulin level remains constant at 0 ng / 20 min when the concentration of glucose in the perfused liquid increases from 0 till 3 mmol/l. The glucagon levels continue to decrease to 1ng/20min however insulin level increases from 1 to 3800 ng / 20 min when the glucose concentration continues to increase to 16 mmol/l. The glucagon levels continue to decrease but less rapidly to 0ng/20min while insulin level continues to increase weakly to 4000 ng / 20 min when the glucose concentration continues to increase to 27mmol/l (1 pt)</p> <p>This shows that The pancreatic cells secrete insulin and glucagon, the insulin secretion varies in the same direction as the glucose concentration starting from 3 mmol/l while the glucagon secretion varies in opposite direction to the concentration of glucose. The pancreatic cells detect the variation of glucose and their sensibility varies according to the glucose concentration.(1/2pt)</p>	11/2																					
3	<p>The hepatic balance decreases from 42 mg/min to a value close to 0 mg/min, following the injection of 1800 mg of insulin opposite to what is noticed in the control dog without insulin injection where its hepatic balance remains constant of 42 mg/min. This shows that insulin decreases the liberation of glucose by the liver (facilitates the storage of glucose in the liver).(1/2pt)</p> <p>Hyperglycemia is observed following the injection of glucagon in a normal dog however this hyperglycemia does not occur after the same injection of glucagon in a dog that his liver was removed. This means that glucagon, a hyperglycemic hormone, acts on the liver to increase the liberation of glucose in the blood. (1/2pt)</p> <p>Therefore, insulin and glucagon act on the same target organ, the liver, in opposite manner (antagonist). The insulin promotes the storage of glucose while the glucagon favors his liberation which maintains the level of glucose normal. (1/2pt)</p>	11/2																					
4	<p>A hormone does not act except on its target cells that have receptors for this hormone. And since a part of the membranous receptor of the hepatic cells constitute the zone of fixation of insulin, thus insulin binds to its membrane protein receptor triggering the change in activity of hepatic target cell. This reduces the release of glucose into blood (or increases the storage of glucose in the liver).</p>	1/2																					

الاسم:
الرقم:مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات

الخميس 27 حزيران 2013

Answer the following exercises**Exercise 1 (5 points)****Fragile X Syndrome**

Fragile X syndrome is the most common cause of hereditary mental retardation. The gene FMR1 which is responsible for this disease is located on the non homologous segment of the X sex chromosome. The alleles at the origin of the abnormal phenotype are characterized by the repetition of CGG triplets for more than 200 times. Couple III₁- III₂ (document 1), who already had an affected child, expects another one and would like to know if it will be affected or not.



- Justify that the gene is localized on X chromosome.
- Propose an explanation for the appearance of the disease in individual IV1 (document 1).

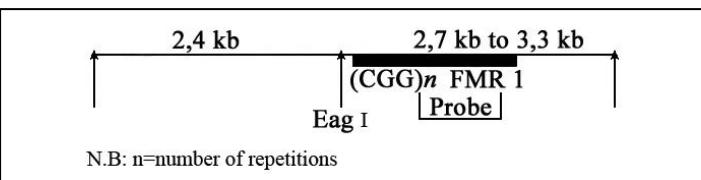
The fragment of DNA which carries the FMR1 gene is isolated. A very close site to this gene is recognized by the restriction enzyme EagI. For a complicated reason, this site is no more recognized by the enzyme when the number of repetitions of CGG triplets exceeds 200. Document 2 shows the position of this cleavage site in normal alleles.

The DNA of certain individuals of this family is cut and a specific radioactive probe of the FMR1 gene is used. The obtained bands are presented in document 3.

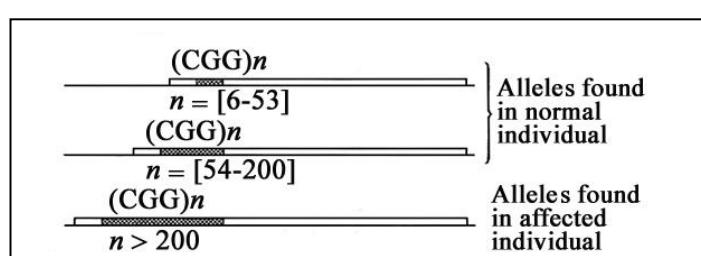
- Identify the band(s) corresponding to the alleles of the disease and those corresponding to the normal alleles.
- Determine whether the fetus IV2 will be affected or not by the fragile X syndrome.
- Pose the problem that arises from the study of document 3 concerning the origin of the disease in IV1.

Document 4 shows the position and the number of repetitions of CGG triplet for the allele of FMR1 gene. The alleles having a number of repetitions between 54 and 200 are expressed normally but might be subjected to instability during gametogenesis. This instability can be manifested by an increase in the number of triplets.

- Explain, based on what precedes, the real origin of the disease in IV1.

Document 1**Document 2**

Individuals	III3	III1	III2	IV1	IV2
5,8 kb				—	
3,2 kb			—		
2,8 kb	—	—	—		—

Document 3**Document 4**

Exercise 2 (5 points)

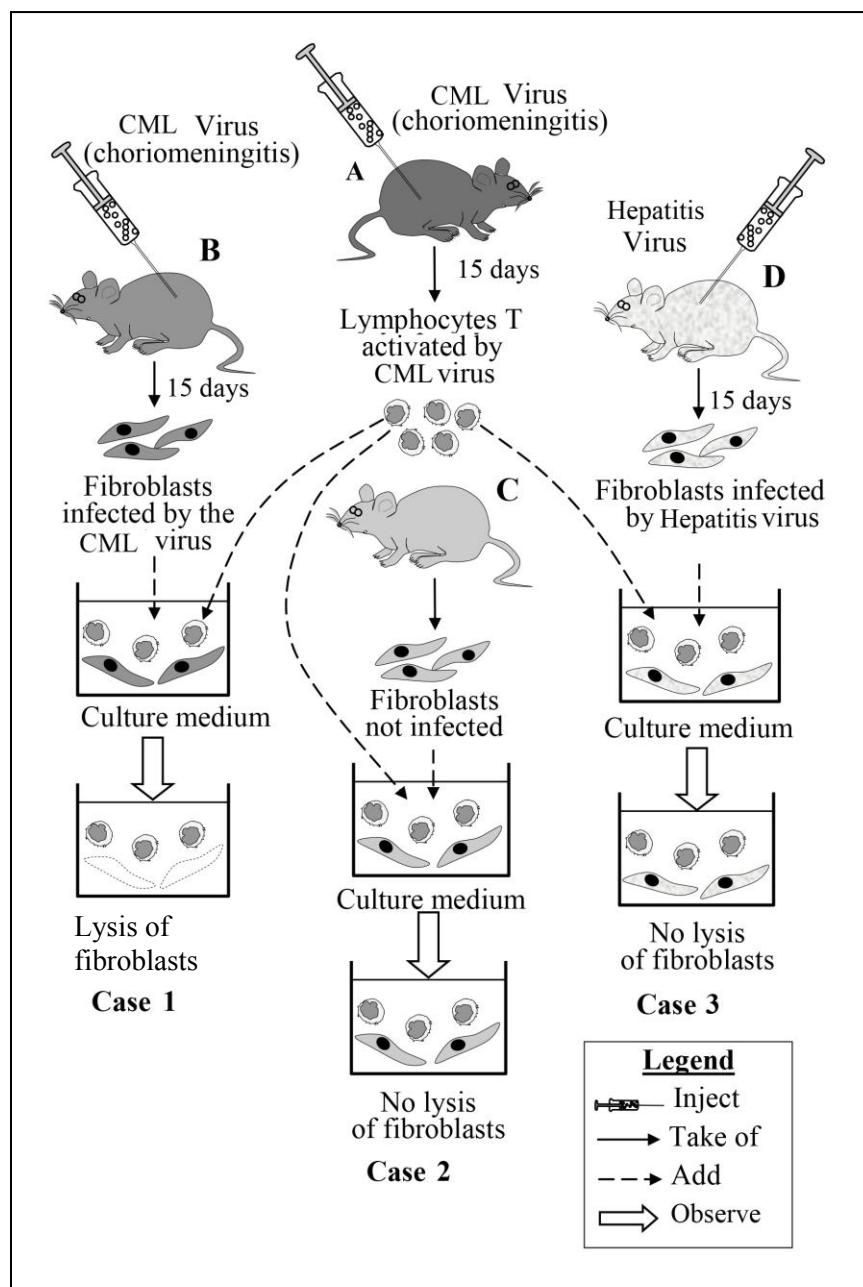
Cell Lysis

Choriomeningitis virus (CML) is a virus transmitted by rodents. The disease is manifested by symptoms similar to those of flu with fever. This disease is transmitted to humans by contaminated food or dust from infected mice.

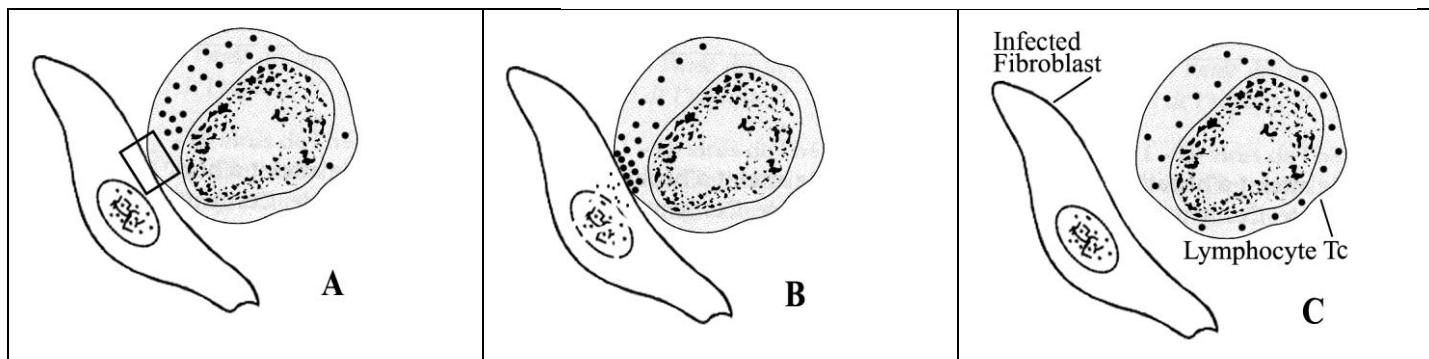
To better understand the immune mechanisms responsible for the lysis of infected cells, a set of experiments are performed on mice of the same line (document1).

- Pick out from the text the means of contamination of humans by CML virus.
- Describe the experiments schematized in document 1.
- Interpret the results of the experiments of document 1.

Document 2 shows the schematic representations of the cellular interactions observed in the culture medium in case1.



Document 1



Document 2

- Arrange, in chronological order, the schematic representations of document 2. Justify the answer.
- Explain the mechanism of cell lysis observed in document 2.

Exercise 3 (5 points)

Ecstasy: Euphoria or Depression?

Ecstasy is a synthetic drug derived from amphetamine. Its effects are described in the text below:

« ...if the quantity of the consumed ecstasy is limited, the consumer becomes euphoric, very talkative, and feels extreme happiness. This phase can last 2 to 4 hours depending on the dose and the individual's sensibility. It is followed by a "descent" period marked by exhaustion and even a strong depressive syndrome... »

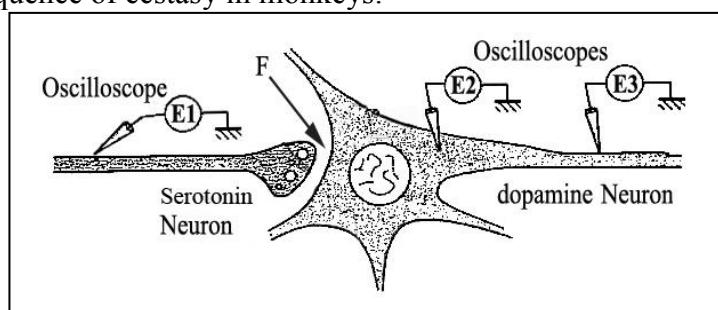
In monkeys, on the long term, ecstasy provokes irreversible destruction of neurons. In humans, we could assume that there is neuronal destruction that can be permanent ... »

1. Pick out from the text :

1.1- The effect of ecstasy 1 hour and 5 hours after consuming a limited dose of ecstasy.

1.2- The statement that shows the long term consequence of ecstasy in monkeys.

To better understand the effects of ecstasy on the nervous system, the activity of a dopamine-releasing neuron connected to a serotonin-releasing neuron (document 1) is studied. For this reason, two successive stimulations separated by different intervals of time are applied on the serotonin-releasing-neuron.



The obtained results are shown in document 2.

Document 1

Conditions	Recordings of E1	Recordings of E2	Recordings of E3
2 stimulations separated by a long time interval			
2 stimulations separated by a short time interval			

Document 2

- Determine if the synapse F is excitatory or inhibitory.
- Indicate, at the level of dopamine-releasing neuron, the type of summation revealed by this experiment. Justify the answer.
- Justify, by referring to document 2, the following expression: "Only the action potential propagates at the level of a neuron".

Pleasure sensation is related to the activity of certain dopamine-releasing neurons situated in the encephalon. Document 3 summarizes the effects of consuming ecstasy on the serotonin-releasing neurons and dopamine-releasing neurons.

Measured parameters at the level of neurons	Serotonin-releasing neurons				Dopamine-releasing neurons
	Frequency of action potentials at the level of serotonin-releasing neurons	Activity of serotonin synthesis	Amount of liberated serotonin	Activity of the pump that recaptures serotonin	Frequency of action potentials at the level of dopamine-releasing neurons
Without ecstasy	++	++	++	++	++
0 to 4 hours after the consumption of ecstasy	++	++	++++	+	++++
Beyond 4h from ecstasy consumption	++	0	0	Not measured	+

Document 3

N.B : the number of + indicates the importance of the phenomenon

- Explain the intervention of the serotonin-releasing neurons and the dopamine-releasing neurons after ecstasy intake in the cases:

5.1- sensation of euphoria.

5.2- state of depression.

Exercise 4 (5 points)

Role of the pancreas

Despite the different causes of the daily variations of glycemia, this latter fluctuates in a healthy individual around a value of 1 g/L. In order to understand the mechanism of the regulation of glycemia, several experiments were performed; some of these experiments are described below.

Experiment 1

A dog that has been subjected to pancreas ablation shows rapidly the symptoms of diabetes: severe hyperglycemia and an important decrease of hepatic glycogen.

Experiment 2

This dog is then subjected to a graft of a pancreas fragment rich in islets of Langerhans at the level of the neck. The previously manifested troubles disappear within few hours.

1. Indicate, by referring to these experiments, the role of pancreas, its mode of action, and its target organ. Justify the answer.

Analysis of pancreatic extracts showed the presence of two chemical messengers: insulin and glucagon. In order to determine their respective roles, experiments 3 and 4 are performed.

Experiment 3

The variations of glucose, insulin and glucagon concentrations in the blood are measured in 10 volunteers during four days of fast. The measurements are performed each morning between 8:00 and 9:00. The results are presented in document 1.

	Beginning of fast	24h	48h	72h
Glycemia (mg/dL)	86	78	72	70
Insulinemia (pg/mL)	10	5	4	3
Glucagonemia (mU/mL)	126	157	189	190

Document 1

2. Interpret the obtained results in document 1.

Experiment 4

These same volunteers receive:

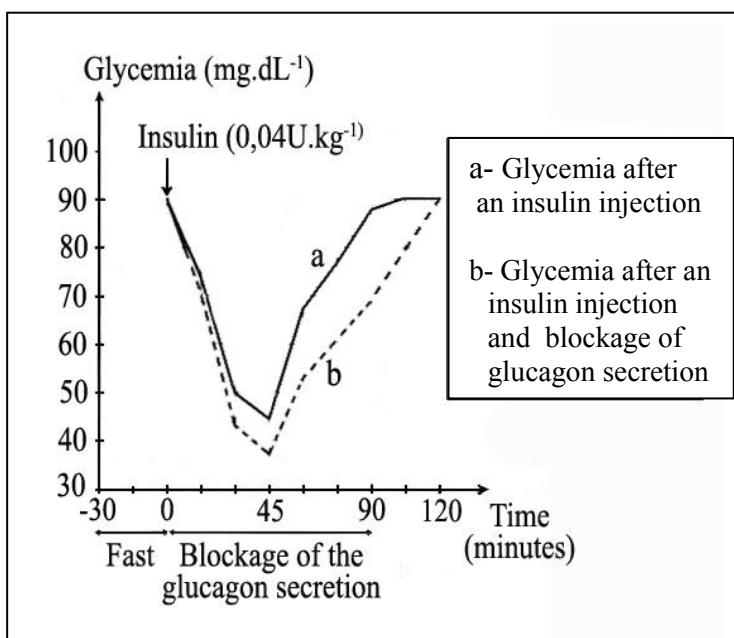
- first an intravenous injection of insulin; the obtained results are represented by curve **a**.
- later they receive an intravenous injection of insulin accompanied by a blockage of glucagon secretion from the moment of insulin injection till 90 minutes; the obtained results are represented by curve **b**.

3. Determine, by referring to document 2, the role of insulin and that of glucagon.

Experiment 5

Mice are given a meal enriched with radioactive sugar. After a certain period of time, radioactive glycogen is found in their hepatic cells and in their muscular cells. Similarly, radioactive triglycerides are found in their adipose tissues.

4. Explain, by referring to all preceding experiments, the regulatory mechanism of glycemia revealed in experiment 5.



Document 2

Part	Answer key Exercise 1	Grade
1	If the allele is located on the non homologous segment of Y, then every affected boy should have an affected father. However, child IV1 is affected but his father III1 is not. So, the gene is not located on the non homologous segment of Y but on the non-homologous segment of X.	0,5
2	Given that the gene is carried by the non homologous segment of chromosome X, the sick IV1 inherits obligatory the chromosome Y from his father and a chromosome X from his mother. Thus the mother with normal phenotype should carry an allele of the disease on one of its X gonosomes without expressing it. Therefore the possible origin of the disease of IV1 is a recessive allele masked form by the normal allele in the mother.	0,75
3	<p>Document two shows that the normal allele is cut by the restriction enzyme Eag1 into two fragments, and the probe fixes only on the fragment giving 2.7 to 3.3 kb. This means that the bands 2.8kb or 3.2kb correspond to a normal allele (0.75).</p> <p>The allele for the disease which has a number of repetitions of triplets that exceeds 200 can no more be cut by the restriction enzyme Eag1 and therefore, one fragment which length is more than 5.7kb is obtained. This mean the band of 5.8kb corresponds to the allele of the disease.</p> <p>Or</p> <p>The affected child IV1 possess only one allele of the gene since the gene is carried on non homologous segment of X and the male has one X chromosome. Document 3 presents only one band of length 5.8kb. Therefore, the latter band corresponds to the allele of the disease.</p> <p>The same reasoning for the normal male III1 indicating that the band which length is 2.8kb corresponds to the normal allele.</p> <p>Woman III2 who is normal possesses 2 alleles for the gene since she has two X chromosomes. Document 3 shows 2 bands of lengths 2,8 and 3,2 kb respectively that correspond according to document 2 to the normal alleles. Thus, the band 3,2kb corresponds to the normal allele.</p>	1,5
4	<p>Doc 3 shows that the fetus has only one band of 2,8kb length same as his normal father III1 thus he is normal.</p> <p>Or</p> <p>The fetus has only one band of 2,8kb length that corresponds to one of the fragment produced by the action of Eag1 on a normal allele thus he will be normal.</p>	0,75
5	<p>How come that the disease appeared in child IV1 although both of his parents carry only normal alleles?</p> <p>Or</p> <p>Both parents of IV1 have only the normal alleles, so where does the allele of the disease of child IV1 come from?</p>	0,75
6	The origin of the disease in child IV1 is due to an abnormality that occurred during meiosis in the mother. Actually the mother has two normal alleles, one of them has a large number of repetition that is subjected to an expansion of triplet CGG to more than 200 during oogenesis. This gamete carries the allele of the disease which upon fertilization has given birth to the affected child IV1.	0,75

Part	Answer key Exercise 2	Grade
1	The means of human contamination by the CML virus are: food and dust contaminated by infected mice.	0,5
2	<p>Mouse A is injected with CML virus (choriomeningitis). 15 days later T lymphocytes activated by CML of this mouse are taken of and added to three culture media.</p> <p>Mouse B is injected with CML virus (choriomeningitis). 15 days later, the fibroblasts infected by CML of this mouse are taken of and are added with the activated T lymphocytes of mouse A to a culture medium. Lysis of these fibroblasts is observed.</p> <p>Non infected fibroblasts of mouse C are taken of and added with the activated T lymphocytes of mouse A to a culture medium. No lysis of these fibroblasts is observed.</p> <p>Mouse D is injected with hepatitis virus. 15 days later, the fibroblasts infected by hepatitis virus of this mouse are added with activated T lymphocytes of mouse A to a culture medium. No lysis of these fibroblasts is observed.</p>	1,5
3	<p>There is lysis of the fibroblasts of mouse B that are infected by the CML virus in the medium containing T lymphocytes activated by the same virus, while there's no lysis of non-infected fibroblasts of the mouse C neither of the fibroblasts of the mouse D that are infected by another virus (hepatitis virus) which are placed in a culture medium containing the same T lymphocytes. This shows that activated T lymphocytes destroy only the cells that are infected by the same virus that led to their activation OR activated T lymphocytes destroy only the cells that are infected and that they are specific to the CML antigen.</p>	1
4	<p>1- The order is: C A B (0,25)</p> <p>The first scheme C shows near the infected fibroblast one T lymphocyte with vesicles that are spread in its cytoplasm.</p> <p>In the second scheme A, the T lymphocyte is in contact with the membrane of the infected fibroblast, what corresponds to the double recognition.</p> <p>In the third scheme B, we notice that the granules are in contact with the infected fibroblast and destroy its nucleus. (0,75 pt)</p>	1
5	<p>Tc recognizes the infected body cell and binds by its TCR to the self HLA-I non self peptide complex expressed on the membrane of the infected cell. Then it liberates perforin to form polyperforin channels through the membrane of the infected cell.</p> <p>After that the TcL releases granzymes that penetrates into the infected cell through the polyperforin channels leading to the degradation of its DNA, thus causing lysis of the infected cell.</p>	1

Part	Answer key Exercise 3	Grade
1	a- After 1h : euphoric, very talkative, and feels extreme happiness. After 5h: a “descent” period marked by exhaustion, and even a strong depressive syndrome... b- Irreversible destruction of neurons.	0,75
2	Following a nervous message propagated through the serotonin presynaptic neuron we observe at the level of E2 a hypopolarization of 10mV (EPSP) or an action potential of 100 mV at the level of the postsynaptic membrane. Thus the synapse F is excitatory.	0,5
3	Temporal summation since following the two successive stimulations separated by a long time interval, 2 AP separated by a long time interval are recorded at the level of the presynaptic neuron generating two distinct hypopolarizations (EPSP) of 10 mV each at the level of the postsynaptic neuron which didn't reach the threshold of depolarization. While following two stimulations separated by a short time interval, 2 AP separated by a short time interval are recorded at the level of the presynaptic neuron generating two hypopolarizations that add up reaching the threshold of depolarization and leading to an AP of 100 mV as amplitude. This shows that the postsynaptic neuron has summed the two EPSP.	0,75
4	In the case where the two stimulations are separated by a long time interval the EPSP recorded at the level of the cell body (E2), is not recorded at the level of the axon (E3) of the same neuron. Thus EPSP doesn't propagate. However, in the case where the two stimulations are separated by a short time interval the AP recorded at the level of E2 propagates and is recorded at (E3). Therefore, only the action potential propagates at the level of a neuron.	0,5
5-1	The euphoria sensation: Ecstasy consumption doesn't modify the frequency of AP at the level of the serotonin neuron (2+) nor the synthesis of serotonin (2+). Whereas, it increases the amount of serotonin released (from 2+ to 4+) and reduces the activity of the serotonin recapture pump (from 2+ to 1 +). This leads to a more important concentration and more persistent presence of serotonin in the excitatory synapse. Thus the activity of the dopaminergic neuron that is modulated by serotonin concentration increases (frequency of AP increases from 2+ to 4+), leading to a more important release of dopamine which explains the euphoria sensation 0 to 4 hours after ecstasy consumption.	1,25
5-2	State of depression : The serotonergic neuron stops the synthesis and release of serotonin, thus leading to a decrease in the activity of the dopaminergic neuron (frequency of AP decreases from 4+ to 1 +). Since in absence of serotonin, the dopaminergic neuron is no more stimulated, thus the release of dopamine which is responsible for pleasure sensation drops leading to exhaustion and to a state of depression.	1,25

Part	Answer key	Grade
Exercise 4		
1	<p>The pancreas has a hypoglycemic role since we observe hyperglycemia following pancreas ablation. (½ pt)</p> <p>The target organ is the liver since there is a decrease in hepatic glycogen amounts after pancreatectomy showing that the pancreas favors the storage of glycogen in the liver. (½ pt)</p> <p>The mode of action of the pancreas is through blood since connecting a pancreas fragment to a pancreatectomized dog at the level of the neck eliminates the symptoms of diabetes. (½ pt)</p>	1,5
2	<p>Starting from the fasting period, as glycemia decreases from 86 mg/dL to 70 pg/mL, insulinemia decreases from 10 to 2 pg/mL while glucagonemia increases from 126 mU/mL to 190 mU/mL. This shows that the variation of glycemia controls the (stimulus) secretion of insulin and glucagon: both glycemia and insulinemia vary in the same direction while glucagonemia varies in the opposite direction.</p>	1
3	<p>Since curve « a » shows a decrease of glycemia from 90 mg/dL to 45 mg/dL in 45 min after the injection of insulin at 0 min, this shows that insulin is a hypoglycemic hormone. (0.75 pt)</p> <p>Glucagon is hyperglycemic because curve « b » shows greater decrease of glycemia to 38 mg/dL which is lower than 45 mg/dL and a delayed return to the initial glycemia after 15 min (120 min > 105 min) after the injection of insulin with blockage of the secretion of glucagon. This shows that glucagon has lowered the hypoglycemic effect of insulin; thus, it has a hyperglycemic effect. (0.75 pt)</p>	1,5
4	<p>Following the ingestion of a meal rich in sugar, glycemia increases thus stimulating the pancreas. The latter increases its secretion of insulin (hypoglycemic hormone) and decreases its secretion of glucagon (hyperglycemic hormone). This variation of hormones acts on target organs to regulate glycemia, in the case of hyperglycemia the liver and muscles absorb glucose and store it in the form of glycogen. Similarly, the adipose tissues convert glucose to triglycerides and store it in this form. This explain the radioactivity detected at the level of organs mentioned in document 5.</p>	1

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Answer the following exercises

Exercise 1 (5 points)

Vaccine against AIDS

In the framework of researches concerning AIDS, scientists followed up 1600 non treated persons that are infected by HIV (Human Immunodeficiency Virus). They measured, at the beginning of the infection, the viral concentration in the blood and recorded the percentage of persons reaching the phase of AIDS. The results are presented in document 1.

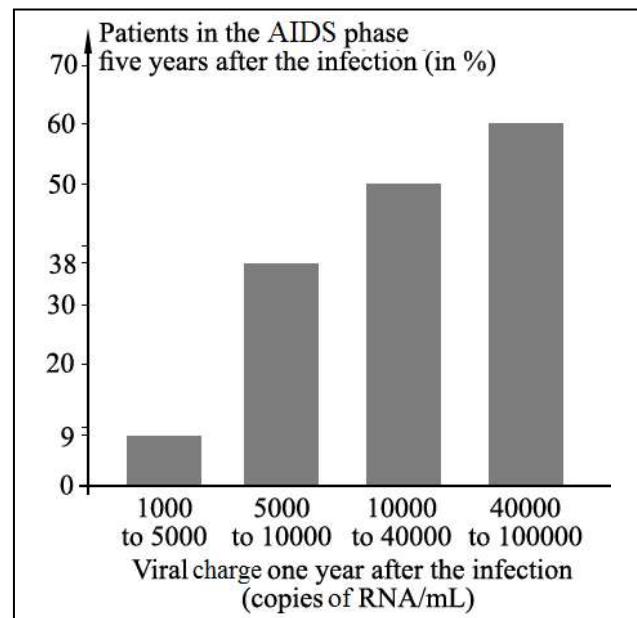
- Justify, by referring to document 1, the following statement: "in the absence of treatment, there is a relation between the onset of the phase of AIDS and the early evolution of the viral charge".

In the case of HIV, vaccines that activate only the production of anti-HIV antibodies don't protect against all the known strains of the virus. Currently, the scientific community agrees on the fact that: to be effective, a vaccine should also stimulate the production of cytotoxic T lymphocytes directed against HIV. This allowed the elaboration of vaccines against HIV.

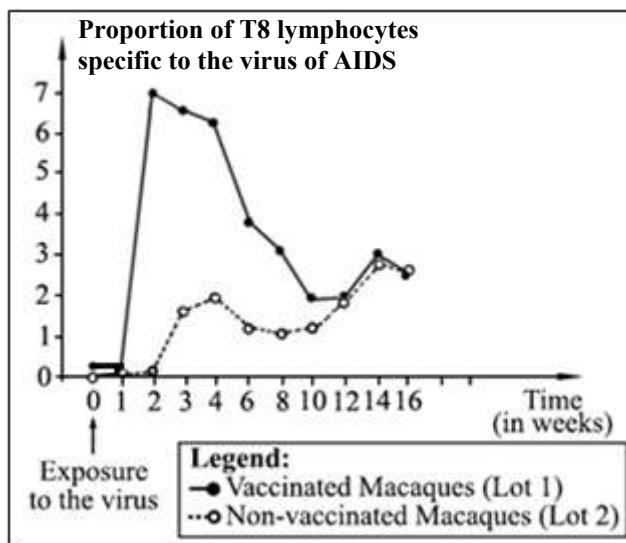
- Indicate how vaccination protects against a given antigen.

One of these vaccines was tested on two lots of macaques monkeys that are not infected by the virus of AIDS. The monkeys of the first lot (lot1) receives a series of five vaccine injections. The monkeys of the second lot (lot 2) are not vaccinated. Then, all the monkeys are exposed to the virus. The proportion of T8 lymphocytes specific to the AIDS virus is then evaluated in the blood of the monkeys (document 2). The viral charge is measured in the two lots of monkeys at the 8th and at the 24th week following the exposure to the virus (document 3).

- Show, by referring to document 2, that the immune response of the vaccinated monkeys is more rapid and more amplified than that of the non-vaccinated monkeys during the first 3 months of the infection.
- Determine if the immune response triggered in lot 1 is durable.
- Interpret the results of document 3.
- Show, by referring to what precedes, that the tested vaccine has a limited efficiency and doesn't allow the eradication of the disease.



Document 1



Document 2

Time after exposure to the virus.	Viral charge (number of viral RNA copies / ml of plasma)	
	Lot 1	Lot 2
8 th week	5.10^4	25.10^4
24 th week	5.10^4	50.10^4

Document 3

Exercise 2 (5 points)

Origins of Phenylketonuria

In hepatic cells, the enzyme phenylalanine hydroxylase, PAH, is responsible for the transformation of phenylalanine into tyrosine. Its absence or its inactivity results in the accumulation (increase in the amount) of phenylalanine in the blood which becomes toxic at a dose exceeding 20mg/dL which leads to the destruction of the nerve cells in individuals affected with phenylketonuria. This disease has different origins and is manifested by irreversible mental retardation.

- Pick out the consequence of the high amount of phenylalanine in the blood.

Document 2 represents a part of the gene coding for the enzyme PAH of a healthy individual and that of the equivalent fragment of an individual suffering from phenylketonuria.

- Determine, using the genetic code table (document 1), the sequence of amino acids of the part of the enzyme PAH coded by each of these two alleles.
- Explain how the modification in the nucleotide sequence of the allele leads to the appearance of phenylketonuria.

Two normal couples had two newborns with high plasma concentration of phenylalanine that exceeds 20mg/dL.

- Indicate if the allele of the disease is dominant or recessive. Justify the answer.

In order to determine the origin of the disease in these two newborns, N_1 and N_2 , these couples consulted a doctor who recommended DNA analysis for all the family members. The obtained results are presented in document 3.

Moreover, the doctor proposed another test, where he injected the newborns with phenylalanine followed by injection of BH_4 , an organic substance normally present in the organism and that is indispensable for the normal activity of PAH. The obtained results are presented in document 4.

- Indicate the possible origin of the disease in the case of the newborn (N_1). Justify the answer by referring to documents 3 and 4.
- Determine, by referring to documents 3 and 4, the possible origin of the disease in the case of the newborn (N_2).

Nucleotides position 2				U C A G	U C A G				
U	C	A	G						
U	UUU UUC UUA UUG	phenyl- alanine leucine	UCU UCC UCA UCG	serine	UAU UAC UAA UAG	tyrosine non-sens	UGU UGC UGA UGG	cysteine non-sens tryptophane	U C A G
	CUU CUC CUA CUG	leucine	CCU CCC CCA- CCG	proline	CAU CAC CAA CAG	histidine glutamine	CGU CGC CGA CGG	arginine	U C A G
	AUU AUC AUA AUG	isoleucine methionine	ACU ACC ACA ACG	threonine	AAU AAC AAA AAG	asparagine lysine	AGU AGC AGA AGG	serine arginine	U C A G
	GUU GUC GUA GUG	valine	GCU GCC GCA GCG	alanine	GAU GAC GAA GAG	aspartic acid glutamic acid	GGU GGC GGA GGG	glycine	U C A G
A : Adenine U : Uracil G : Guanine C : Cytosine.						Nucleotides position 3			

Document 1

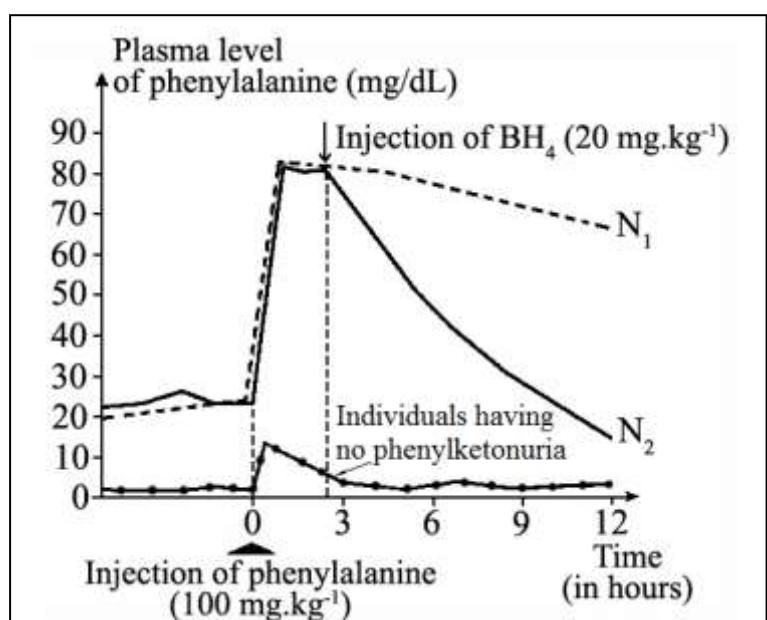
Alleles	Nucleotide sequence of the non-transcribed strand of DNA from codon 277 to codon 283
Normal	TAT ACC CCC GAA CCT GAC ATC
Diseased	TAT ACC CCC AAA CCT GAC ATC

Document 2

Alleles	F ₁	M ₁	N ₁	F ₂	M ₂	N ₂
Normal	—	—	—	—	—	—
Diseased	—	—	■	—	—	—

F: Father M: Mother N: Newborn

Document 3



Document 4

Exercise 3 (5 points)

LSD and Hallucinations

Albert Hofmann is best known for discovering a powerful synthetic drug, the LSD. In one of his books, he described his sensations after he voluntarily took this drug in the frame work of experimental automedication.

« Everything in my field of vision was oscillating and distorted as if seen in a curved mirror. I also had the sensation that the bike was not moving even though my assistant told me later that we have been moving fast. When I arrived home, dizziness and weakness sensation were more serious in a way that I couldn't stand up and was obliged to lie down on a sofa-bed.

Later, I noticed that the way all acoustic perceptions, such as the sound of a door handle or that of a car passing by the house, were transformed into visual perceptions. Every sound generated a corresponding animated image with a particular form and color. »

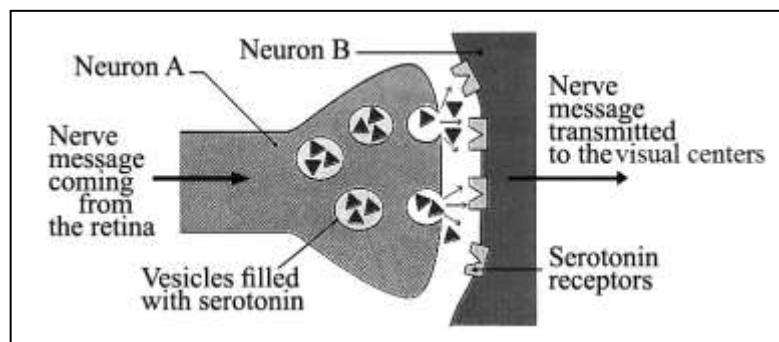
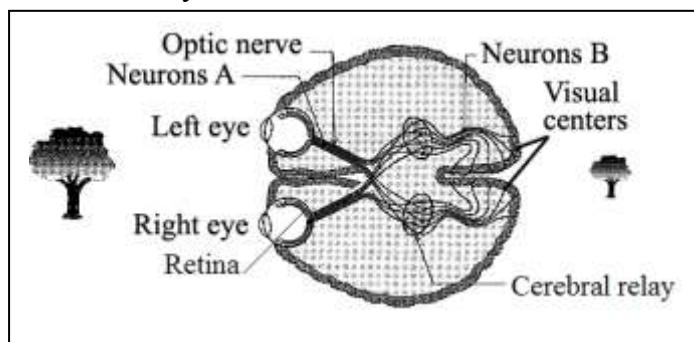
Document 1

- 1- Knowing that hallucination is defined as «perception without any object to perceive», show that the LSD is a powerful hallucinogen.
- 2- Justify that the LSD doesn't modify only the visual perceptions of the individual.

To better understand the action of LSD and its effects, the following studies are performed.

Stimulations applied on neurons A produce visual perceptions. Document 2 shows the encephalic visual pathways involved in these types of perceptions.

Document 3 represents the scheme of the synapse between the two types of neurons A and B at the level of the cerebral relay.



Document 2

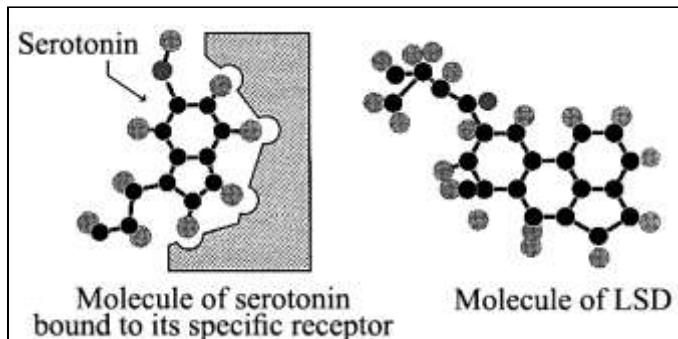
Effective stimulations of increasing intensities ($I_1 < I_2 < I_3$) are applied on neuron A. The amount of serotonin in the synaptic cleft is measured and the nervous message at the level of neurons A and B are recorded. The results are shown in document 4

Intensity	Frequency of AP at the level of the neuron A	Amount of serotonin (in AU)	Frequency of AP at the level of the neuron B
I_1	5	1.5	8
I_2	9	2.5	13
I_3	12	3	18

Document 4

- 3- Explain the steps of synaptic transmission of the nervous message coming from the retina via neurons of type A before reaching the visual centers.
- 4- Draw a histogram showing the variation of the amount of serotonin as a function of the intensity of stimulation.
- 5- Analyze the obtained results. Draw out the form in which the nervous message is coded at the level of the neuron as well as that at the level of the synapse.

Document 5 shows the molecular structure of serotonin and that of LSD.



Document 5

- 6- Suggest, referring to all what precedes, an explanation of the mode of action of LSD in the genesis of visual hallucinations.

Exercise 4 (5 points)

Ovaries and Sexual Cycles

Ovaries are active from puberty till menopause.

In order to understand the endocrine role of ovaries on the genital activity, the following experiments are performed.

Experiment 1:

Two lots of female rats which did not reach puberty, 2 and 3, are subjected to ovariectomy with or without injection of ovarian extracts: estradiol and progesterone. The conditions and the results of the experiment are presented in document 1.

- 1- Draw out the roles of ovaries and their mode of action as revealed in this experiment.

Experiment 2:

Four lots (A, B, C and D) of female rabbits that did not reach puberty receive daily injections of 5 µg of estradiol (E) and/or 200 µg of progesterone (P) during several days. Then, transverse sections of their uterus are prepared at the end of the experiment, at day 11. Document 2 presents the experimental conditions as well as the obtained results.

- 2- Interpret the results of document 2.

Experiment 3:

Protein receptors for progesterone were revealed at the level of endometrium cells. The injection of estradiol provokes an increase in the number of progesterone receptors in the day following the injection.

- 3- Explain the obtained results of lot A in experiment 2.

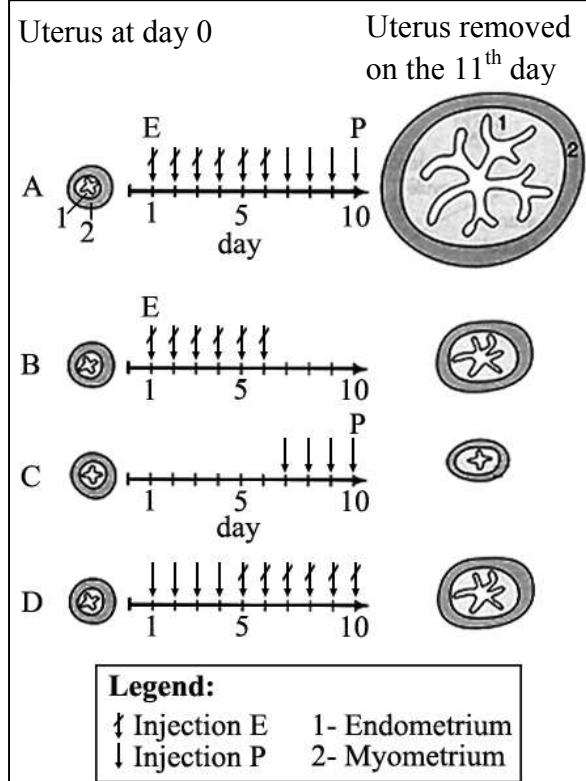
Experiment 4:

A lot of ovariectomized female mammals are subjected to injections of high amounts of estradiol with or without progesterone.

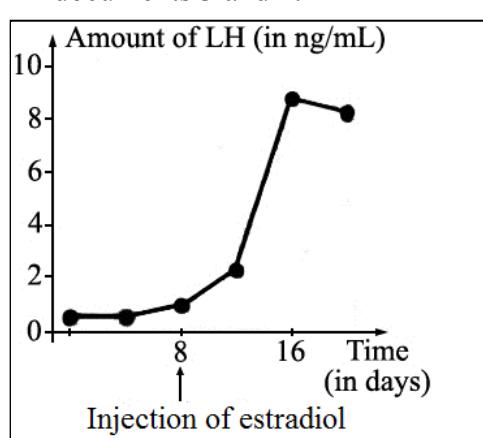
The evolution of the plasmatic concentration of the pituitary hormone LH is measured and the obtained results are shown in documents 3 and 4.

Groups	Conditions	Observed Results	
		Mass of the uterus	Uterine cycles
1	Control	710 mg	Cyclic variation
2	Ablation of two ovaries	120 mg	No variation
3	Bilateral ovariectomy + continuous and identical injections of ovarian extracts	705 mg	No cyclic variation

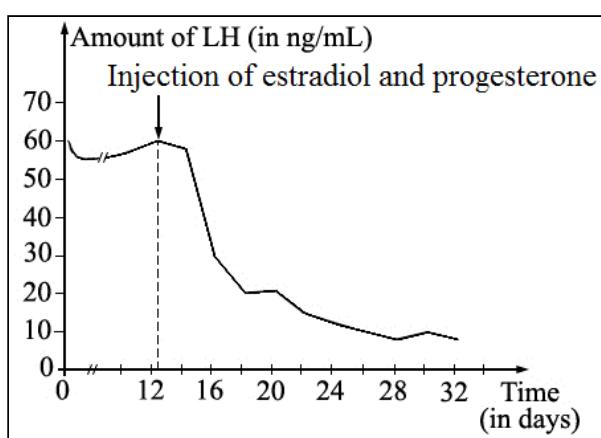
Document 1



Document 2



Document 3

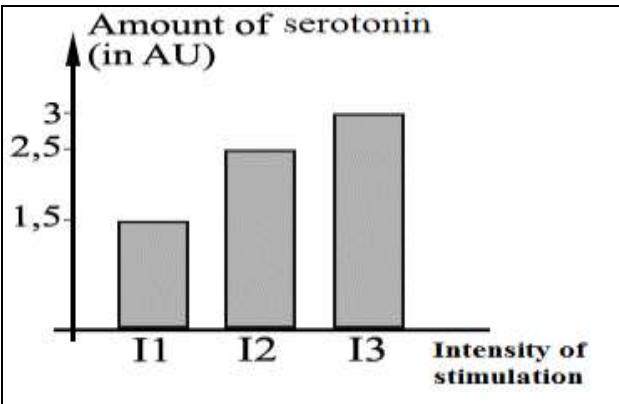


Document 4

- 4- Show, by referring to documents 3 and 4, that "the activity of the pituitary gland is under the control of ovarian hormones".
- 5- Establish, by referring to all what precedes, a functional diagram showing the relations existing between the ovaries and the two other organs: the pituitary gland and the uterus.

Part	Answer	Grade
	Exercise 1	
1	The percentage of untreated patients in the AIDS phase five years after the infection increases from 9% to 60% when the viral charge one year after the infection increases from 1000 up to 10000 copies of mRNA/mL. This shows that the onset of the AIDS phase and the early evolution of the viral charge vary in parallel to each other.	1
2	Vaccines immunize the organism against a specific antigen by inducing a durable immunological memory. Or Vaccines protect the organism by sensitizing the immune system against the pathogens in order to recognize and destroy them in a more rapid and more efficient manner upon a second contact with the same pathogen.	0,5
3	In vaccinated monkeys, the proportion of T8 lymphocytes increases from 0 to 6.5 greater than that of non-vaccinated monkeys which is 2. This shows that the response triggered in vaccinated monkeys is more amplified. (0,5 pt) After the exposure to the virus, the increase of the proportion of T8 lymphocytes in vaccinated monkey begins after a latency time of one week, less than that in the vaccinated monkeys which is 2 weeks. This shows that the response in vaccinated monkeys is more rapid than that of the non-vaccinated ones. (0,5 pt)	1
4	Between the fourth and the 12 th week, the proportion of T8 lymphocytes, in both lots 1 and 2 decreases while remaining higher in vaccinated monkeys and reach the same value of 2 at week 12. After the 12 th week, the variations of these proportions remain identical. This shows that the immune response triggered by the vaccine is not durable, it does not last except for 12 weeks.	0,5
5	Document 3 shows that the viral charge at the 8th week in vaccinated macaques is $5 \cdot 10^4$ viral RNA copies/ml inferior to that of the non-vaccinated ones $25 \cdot 10^4$ viral RNA copies/ml . At the 24 th week it increases (doubles) to $50 \cdot 10^4$ viral RNA copies/ml in the non-vaccinated macaques while it remains constant at $5 \cdot 10^4$ viral RNA copies/ml in vaccinated macaques value that is 10 times smaller than $50 \cdot 10^4$ viral RNA copies/ml. This shows that vaccine maintains the viral charge weak and constant at the beginning of the infection.	1
6	The chance of reaching the phase of AIDS diminishes in the case where the viral charge is weak at the beginning of the infection (doc.1). The vaccine maintains the viral charge low at the beginning of the infection (doc.3). This diminishes the evolutions of the disease toward the phase of AIDS thus extending the asymptomatic phase. Therefore there is a greater chance to prolong the life of seropositive individuals. From this point the vaccine is efficient. The vaccine amplifies the specific cell mediated immune response the first three months after infection (doc.2) however this amplification is not durable thus the efficiency is limited. In addition the vaccine doesn't ensure a total recovery and the disease is not eradicated.	1

Part of the ex.	Answer	Grade
	Exercise 2	
1	It is toxic, leads to the destruction of the nerve cells and is manifested by irreversible mental retardation.	0,5
2	<p>Portion of the amino acids sequence of the enzyme: We establish the mRNA sequence by replacing T by U Normal mRNA: UAU ACC CCC GAA CCU GAC AUC Amino acids sequence : Tyr-Thr-Pro-Glu-Pro-Asp-Ile Diseased m RNA: UAU ACC CCC AAA CCU GAC AUC Amino acids sequence : Tyr-Thr-Pro-Lys-Pro-Asp-Ile</p>	1
3	<p>The mutation by substitution at the level of the first nucleotide of the 280th codon of the DNA where G is replaced by A is transcribed at the level of mRNA by a new codon which is translated into a new amino acid, lysine instead of the glutamic acid. This new amino acid sequence affects the tridimensional structure of the enzyme PAH which becomes inactive (nonfunctional). Since this enzyme is responsible for the transformation of phenylalanine into tyrosine. This transformation doesn't occur any more leading thus to the accumulation of phenylalanine which in high amount becomes toxic and causes phenylketonuria.</p>	1
4	<p>The allele of the disease is recessive with respect to the normal allele. Since normal parents gave birth to an affected child, thus they carry the allele of the disease that is masked in the parents. Let N be the symbol of the normal allele. Let m be the symbol of the allele coding for the disease.</p>	0,5
5	<p>The origin of the disease in the case of N1 is a mutation that leads to the synthesis of an inactive PAH (non-functional). Document 3 shows that affected N1 is homozygous of genotype m//m. And document 4 shows that a slight decrease in the plasma level of phenylalanine in N1 from 80 to 70 mg/dL after the injection of 20 mg/Kg of BH4. This implies that even in the presence of functional BH4, the PAH remains nonfunctional.</p>	1
6	<p>Document 3 shows that the affected newborn N2 is homozygous of genotype N//N. His allele codes for a normal PAH. Document 4 shows that in N2, the constant plasma level of phenylalanine of 80 mg/dL decreases after the injection of 20 mg/Kg of BH4 to 15 mg/dL value that is inferior to the reference level of 20 mg/dL. Thus BH4 acts in N2 by decreasing the plasma level of phenylalanine toward its normal value. The PAH in the newborn N2 is functional but needs the presence of BH4 to be activated. Hence, his disease in N2 can be due to the absence of BH4 or to the presence of non-functional BH4.</p>	1

Part	Answer	Grade								
Exercise 3										
1	“Every sound generated a corresponding animated image”. The “acoustic perceptions such as the sound of a door handle or a passing automobile became transformed into optical perceptions”. Sounds are generating visual perceptions. Thus LSD is a powerful hallucinogen since it provokes perceptions without objects to perceive.	0,5								
2	LSD doesn’t modify only the visual sensations, but it also causes general disturbance. Hoffmann didn’t realize that the bike was moving, he felt dizzy and weak he couldn’t stand up and was forced to lie down on a sofa bed.	0,5								
3	The nerve message coming from the retina towards the extremity of the axon of presynaptic neuron A, leads to the influx of calcium in the terminal knob, this causes vesicles filled with neurotransmitters, serotonin, to migrate to the cell’s surface and to release their contents of serotonin into the synaptic cleft by exocytosis. Then, serotonin fixes on specific receptors of the membrane of the postsynaptic neuron B. the binding of the neurotransmitter to its specific receptor generates an EPSP in the postsynaptic neuron inducing the birth of a nerve message that is transmitted by neuron B towards the nerve centers.	1								
4	Histogram : variation of the amount of serotonin as a function of the intensity of stimulation  <table border="1"> <caption>Data from Histogram</caption> <thead> <tr> <th>Intensity of stimulation</th> <th>Amount of serotonin (in AU)</th> </tr> </thead> <tbody> <tr> <td>I1</td> <td>~1.5</td> </tr> <tr> <td>I2</td> <td>~2.5</td> </tr> <tr> <td>I3</td> <td>~3.0</td> </tr> </tbody> </table>	Intensity of stimulation	Amount of serotonin (in AU)	I1	~1.5	I2	~2.5	I3	~3.0	1,25
Intensity of stimulation	Amount of serotonin (in AU)									
I1	~1.5									
I2	~2.5									
I3	~3.0									
5	The frequency of AP at the level of the neuron A and at the level of neuron B increases respectively from 5 to 12 AP and from 8 to 18 AP as the intensitiy of stimulation increases from I1 to I3. Meanwhile, the amount of serotonin increases from 1.5 to 3 au. Thus the nerve message at the level of neuron is modulated by frequency of AP and at the level of the synapse by the amount of neurotransmitter.	0,75								
6	the molecule of serotonin and that of LSD have an identical part in their molecular structures, and this common part allows the serotonin to fix on its specific receptor. We can suggest that molecules of LSD fix on serotonin receptors due to their complementary form. Being agonist to serotonin, the LSD fixation on serotonin receptors generates an EPSP at the level of neuron B in absence of any message at the level of the presynaptic neuron A, and consequently the induced nerve message propagates towards the visual centers even though eyes don’t detect any object. This explains the visual hallucinations described by Hoffmann.	1								

Part	Answer Exercise 4	Grade
1	Ovaries are responsible for the development of the uterus and the cyclic variation of the uterine cycle. Ovaries act by secreting estradiol and progesterone in the blood in a variable or cyclic manner.	0.75
2	There is development in the endometrium of the uterus following the injection of estradiol for 6 days followed by an injection of progesterone for 4 days (lot A). However this development is less important following the injection of estradiol for 6 days alone (lot B). Thus estradiol stimulates the development of the endometrium and progesterone amplifies this action. While there is no development of the endometrium following the injection of progesterone alone from day 7 till day 10 (lot C). Hence, progesterone alone doesn't have any effect on the endometrium. On the other hand the endometrium shows a weak development less important than that in lot A following the injection of progesterone for 4 days followed by an injection of estradiol for 6days. Thus progesterone does not act on the endometrium unless it is preceded by estradiol.	2
3	In lot A, the injection of estradiol at the beginning of the cycle ensures slight development of the endometrium and increases the number of progesterone specific receptors. This increases the concentration of the progesterone bound to its receptors in the nucleus of target cells. this increases the synthesis of proteins and ensures the thickening of the endometrium, leading to the important development of the endometrium in lot A.	0.75
4	The amount of LH increases from 0.5ng/mL to 9ng/mL between the days 8 and 16 following the injection of estradiol alone on day8. This shows that estradiol exerts a positive feedback on the pituitary gland. On the contrary, the amount of LH decreases from 60ng/mL to 10ng/mL between day 12 and 28 after the injection of estradiol and progesterone at day 12. This shows that estradiol with progesterone exert a negative feedback on the pituitary gland. This shows that the activity of the pituitary gland is under the control of ovarian hormones.	0.75
5	Functional diagram showing the relations existing between the ovaries and the two other organs: the pituitary gland and the uterus	0.75
	<pre> graph TD PG[Pituitary gland] -- "+" --> LH[LH] LH --> Ov[Ovaries] Ov --> E[Estradiol] E --> WDE[Weak development of the endometrium] Ov --> EP[Estradiol + Progesterone] EP --> IDE[Important development of the endometrium] E -.-> FB[Feedback] FB -.-> PG </pre> <p>Legend: → Secrete → Lead to → Feedback </p>	

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Answer the following exercises:

Exercise 1 (5 points)

Mode of Action of Botox

Botulinum toxins are at the origin of a serious disease called Botulism. This disease affects all the muscles and may lead to paralysis of the respiratory muscles thus causing death. However, these toxins are frequently used by all men and women who want to eliminate the signs of aging (anti-wrinkles treatment). This is realized by injecting these toxins "Botox" every 6 months.

In order to determine the mode of action of Botox, the following experiments are performed.

Experiment 1: In a physiological culture medium, using an appropriate experimental set up, four effective stimulations of increasing intensities are applied on a motor neuron that innervates a skeletal muscle.

For each of the applied stimulations, a muscular contraction is observed. The frequency of action potentials at the level of the presynaptic motor neuron (doc.1), the concentration of calcium in the presynaptic terminal bud (doc.2), and the quantity of acetylcholine released in the synaptic cleft (doc.3) are measured for each of the applied stimulations.

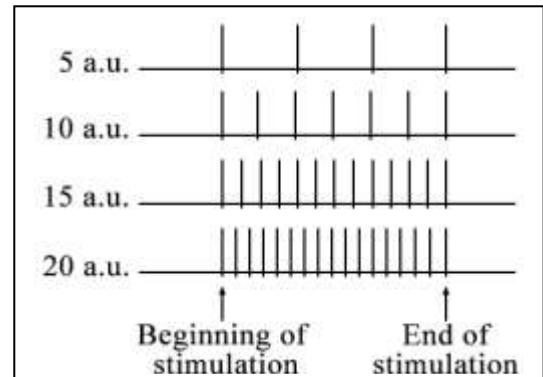
- Interpret the obtained results in document 1.
- Draw the curve that shows the variation of the quantity of acetylcholine as a function of the intensity of stimulation.
- Specify the type of coding of the nervous message that is revealed by each of the documents 2 and 3.

Experiment 2: Botox is added to the culture medium of the experimental set up of experiment 1. The same stimulations as well as the same measurements are repeated. Same results as those of experiment 1 are obtained except for the quantity of the released acetylcholine. In addition, no muscular contraction is observed for the 4 intensities of stimulation.

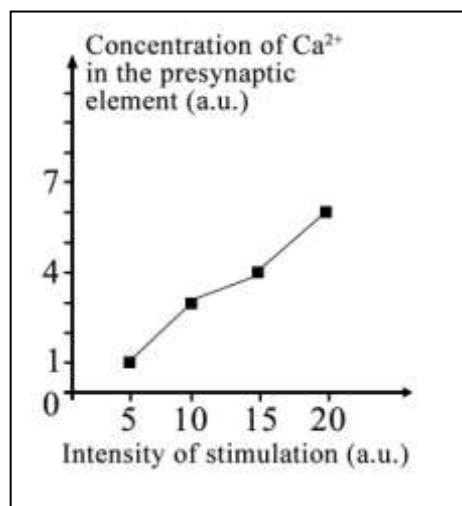
- Formulate a hypothesis explaining the mode of action of Botox on the transmission of the nervous message.

Experiment 3: The presynaptic vesicles of the motor neuron of a frog are labeled by a fluorescent dye. This neuron is placed in a medium with or without botulinum toxin. The intensity of fluorescence inside the presynaptic bud is measured before and after stimulating this neuron. The results are presented in document 4.

- Determine, referring to experiment 3, the quantity of acetylcholine that should be released in experiment 2.
- Explain how Botox eliminates the signs of aging without causing death by intoxication.



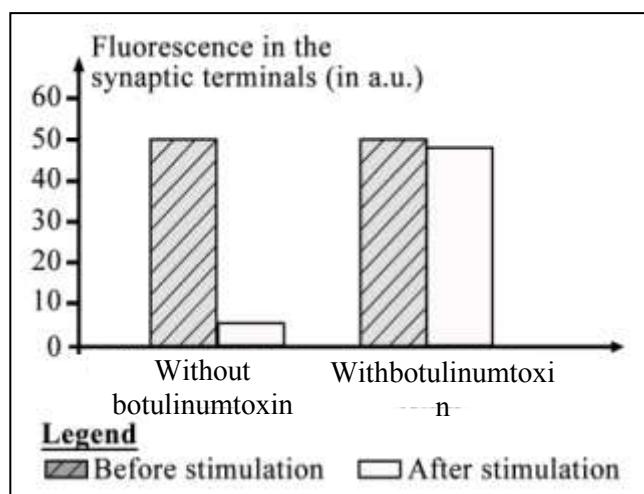
Document 1



Document 2

Intensity of stimulation (a.u.)	Quantity of released acetylcholine (a.u.)
5	30
10	40
15	50
20	60

Document 3



Document 4

Exercise 2(5 points)

Role of the Liver in Glycemia Regulation

Glycemia is a physiological constancy. To better understand how it is regulated, the following studies are carried out.

Clinical observations: Glycemia is measured as a function of time in two healthy individuals X and Y. Individual X who was fasting ingests 50 g of glucose at time 0 minute; his glycemia is then monitored during the two hours that follow the ingestion (document 1).

The glycemia of individual Y is monitored starting from the beginning of his fast at T0 and during the two days that follow (document 2).

- 1- 1-1- Analyze the results of each of the documents 1 and 2.
- 1-2- What can you conclude?

Histological observations: Document 3 shows histological sections (x1000) of the liver of an animal at two different times. They are colored by an dye that gives a brown color in the presence of glycogen. Section A was done on the liver of an animal fasting since 48h. Section B was done on the liver of the same animal after being fed recently with a meal rich in glucose.

- 2- Determine the role of the liver revealed by the histological sections.

Experiment 1: An experiment inspired by the historical experiment of the washed liver is performed. The experimental procedure and the obtained results are presented in document 4.

- 3- Describe the experiment illustrated in document 4.

Experiment 2: Experiment 1 is repeated with a final incubation in distilled water containing traces of insulin. The result of the glucose detection test remains negative.

Experiment 3: Experiment 1 is repeated with a final incubation in distilled water containing traces of glucagon. After only five minutes, the result of the glucose detection test becomes positive.

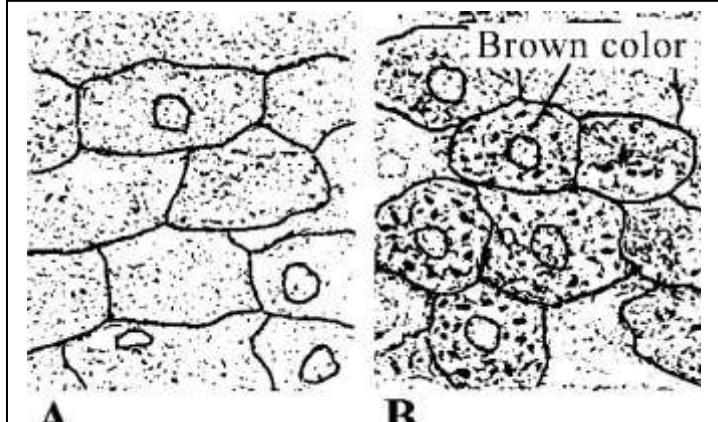
- 4- Show, by referring to the 3 experiments, that glycogenolysis is modulated by the action of hormones.
- 5- Explain, taking into consideration all what preceded, the results obtained in documents 1 and 2.

Time (in min)	↓ ingestion of glucose			
	0	60	90	120
Glycemia (in g/L) of individual X	0.8	1.7	1.3	0.9

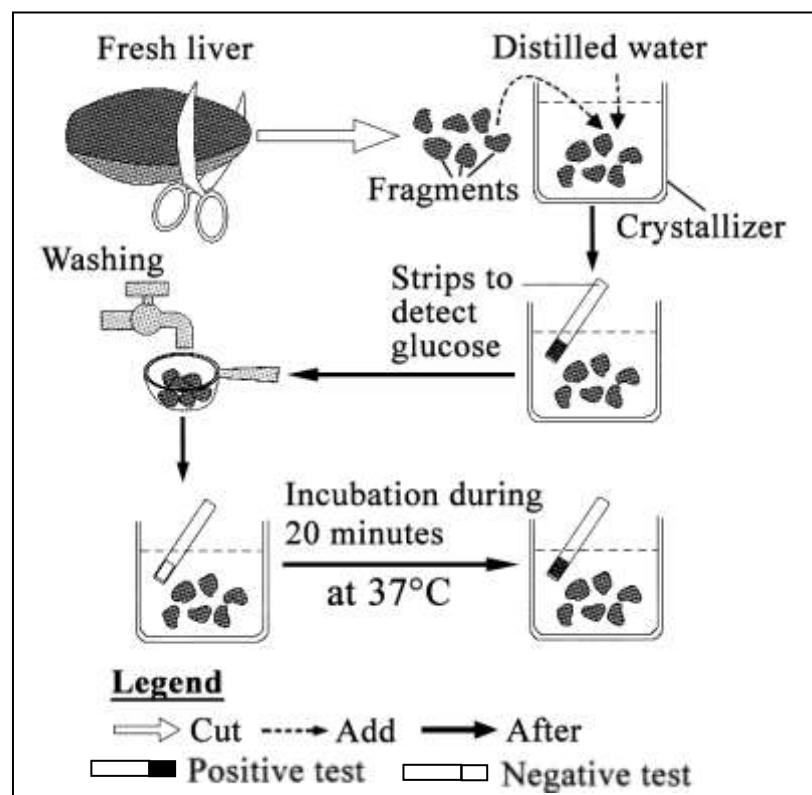
Document 1

Time	T0	T1	T2	T3
Glycemia (in g/L) of individual Y	1	0.9	0.7	0.8

Document 2



Document 3



Document 4

Exercise 3 (5 points)

Huntington Chorea

Huntington Chorea is a serious neurodegenerative hereditary disease. Its first symptoms appear in adults starting from the age of 25 years.

We seek to determine the mode of transmission of this disease as well as its origin.

Document 1 shows the pedigree of a family whose certain members are affected by this disease.

- 1- Indicate whether the allele determining this disease is dominant or recessive. Justify the answer.
- 2- Determine the localization of the gene responsible for this disease.

All the members of this family are over 25 years old except individuals III3 and III5. The latter are willing to get married but are afraid of being affected by this disease.

- 3- Determine the risk for each of individuals III3 and III5 to be affected by this disease.

Studies have shown that the gene coding for the functional protein, huntingtin, exists in many allelic forms that differ by the number of CAG triplets. The number of repetitions of CAG triplet in each allele is studied in healthy individuals as well as in affected ones. The obtained results are presented in document 2.

- 4- Deduce, based on the statistical results of document 2, the origin of this disease.

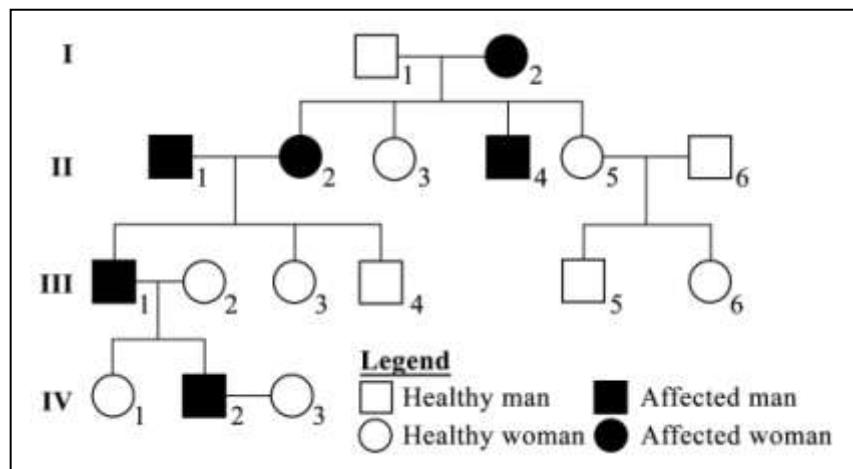
The analysis of the gene in woman III3 has revealed that she possesses two alleles. The number of repetitions of CAG in one of them is 10 and in the other it is 15.

- 5- Specify the real genotype of this woman.

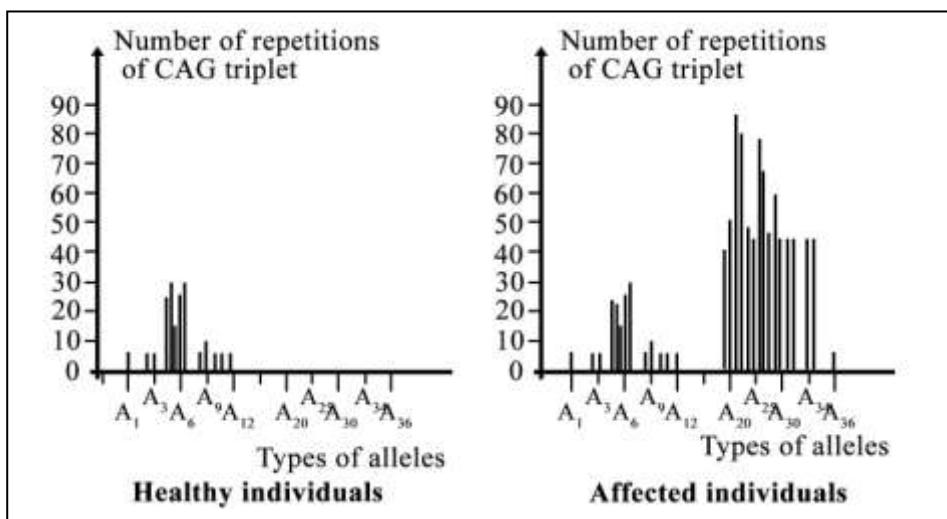
A statistical study has been performed concerning the age of appearance of this disease in function of the number of CAG triplets. The obtained results are shown in document 3.

- 6- 6-1-Analyze the obtained results.

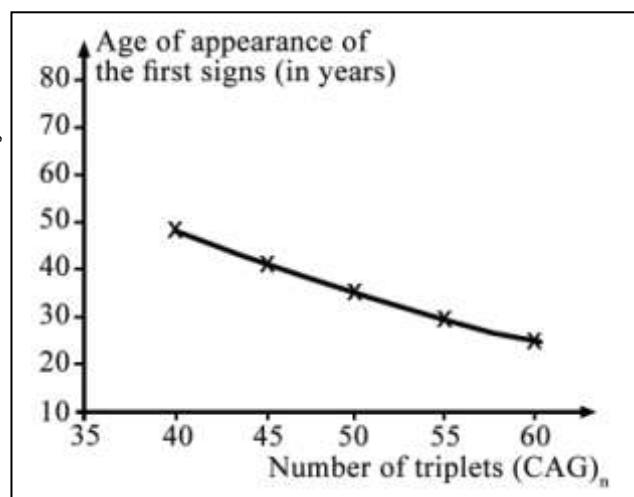
- 6-2- Conclude the factor that determines the age of appearance of this disease.



Document 1



Document 2



Document 3

Exercise 4 (5 points)**Fight Against Ebola**

Ebola is a very contagious and fatal virus that causes hemorrhagic fever. It is transmitted through blood, saliva, feces as well as through sexual contacts.

Infected individuals who survived, show first a high amount of specific anti-Ebola antibodies, followed by the disappearance of the virus with an important increase in specific cytotoxic T cells (TcL).

- 1-** Identify the immune response(s) triggered against Ebola.

In order to develop fighting or therapeutic modalities against this disease, researchers performed experiments that are described below.

- In December 2011, researchers developed a vaccine. They isolated a surface protein of the virus and injected it to a first lot of mice. To a second lot, they injected the same protein in the form of immune complexes called EIC (Ebola Immune Complexes). To a third lot they injected the EIC and a substance, the PIC. The injections are repeated four times for each lot. Two weeks after each injection, serum is collected from the mice and the antibodies amounts were measured. The obtained results are presented in document 1.

- 2-** Determine the most efficient vaccine against Ebola.

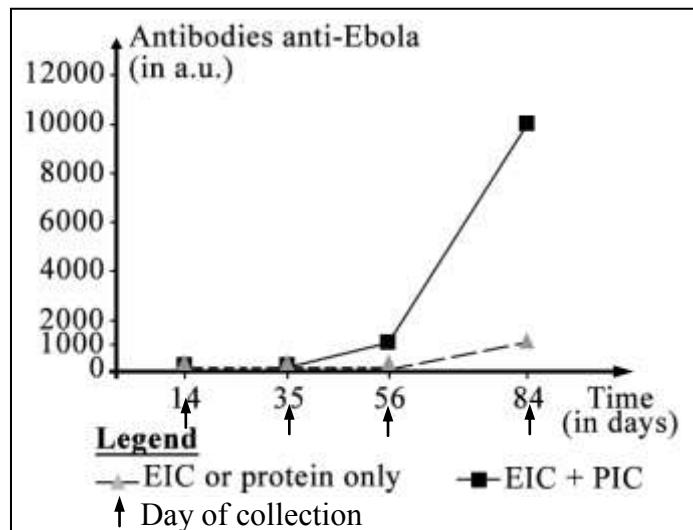
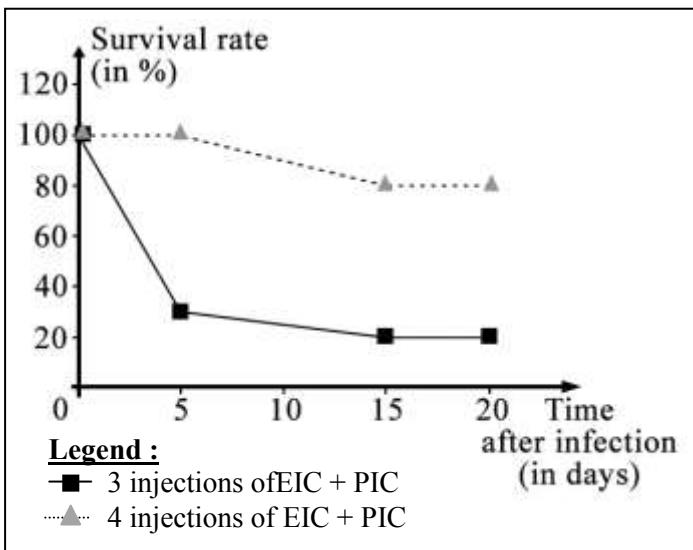
The molecule PIC is an agonist to proteins that are indispensable for phagocytosis.

- 3-** Indicate the roles and the moments where macrophages intervene in the specific immune response triggered against Ebola.

Two lots of mice have been vaccinated using the mixture EIC+PIC, the first lot received three boosters for the vaccine and the second received four boosters. After that both lots were contaminated by Ebola virus. The results concerning the survival of the mice are presented in document 2.

- 4-** Deduce one condition for the vaccination against Ebola to be successful.

- In June 2012, Canadian researchers performed the following experiment: two lots of monkeys, infected by the Ebola virus, received a mixture of three antibodies specific to particular epitopes of the virus. The obtained results are presented in document 3.

**Document 1****Document 2**

Lots of monkeys	Performed treatment	Number of monkeys	Number of surviving monkeys
A	Infection by the virus then injection of antibodies 24 hours after infection	4	4
B	Infection by the virus then injection of antibodies 48 hours after infection	4	2

Document 3

- 5-** Explain the obtained results.

- 6-** Distinguish serotherapy from vaccination concerning: the nature of the injected substance, the latency period and the duration of the protection established against Ebola.

Part of the ex	Exercise 1 Mode of Action of Botox	Grade 5 pts										
1	The recording obtained at the level of the axon of the presynaptic neuron of document 1 shows APs of same amplitude. However, the frequency of AP increases from 4 APs to 17 when the intensity of the stimulation increases from 5 a.u. to 20 a.u. This shows that the response of the axon is modulated by the frequency of APs as function of the intensity of stimulation.	1/2										
2	<p>Curve showing the variation of the quantity of acetylcholine as a function of the intensity of stimulation.</p> <table border="1"> <caption>Data points from the graph</caption> <thead> <tr> <th>Intensity of stimulation (in a.u.)</th> <th>Quantity of acetylcholine (in a.u.)</th> </tr> </thead> <tbody> <tr> <td>5</td> <td>30</td> </tr> <tr> <td>10</td> <td>40</td> </tr> <tr> <td>15</td> <td>50</td> </tr> <tr> <td>20</td> <td>60</td> </tr> </tbody> </table>	Intensity of stimulation (in a.u.)	Quantity of acetylcholine (in a.u.)	5	30	10	40	15	50	20	60	1
Intensity of stimulation (in a.u.)	Quantity of acetylcholine (in a.u.)											
5	30											
10	40											
15	50											
20	60											
3	<p>At the level of the presynaptic neuron, the nervous message is modulated by the concentration of calcium as function of the intensity of stimulation, since document 2 shows that the concentration of calcium in the presynaptic element increases from 1 a.u. to 6 a.u when the intensity of stimulation increases from 5 a.u. to 20 a.u.</p> <p>At the level of the synapse, the nervous message is modulated by the concentration of the released acetylcholine as function of the intensity of stimulation, since document 3 shows that the quantity of acetylcholine released increases from 30 a.u. to 60 a.u. when the stimulation intensity increases from 5 a.u. to 20 a.u.</p>	1										
4	<p>Hypothesis:</p> <ul style="list-style-type: none"> Botox inhibits the synthesis of Acetylcholine. Botox inhibits the exocytosis of Acetylcholine. Botox neutralizes Acetylcholine. Botox blocks the postsynaptic receptors. 	1/2										
5	<p>The fluorescence in the presynaptic bud decreases from 50 a.u. before stimulation to 5 a.u. after stimulation in a medium without Botulinum toxin. However, in a medium containing Botulinum toxin, it remains almost constant at 50 a.u. before and after stimulation. Thus, Botox blocks the release of neurotransmitters by exocytosis of the presynaptic vesicles. Hence, in a medium containing Botox, the quantity of released acetylcholine should be null.</p>	1										
6	<p>Botox blocks the transmission of the nervous message at the level of neuromuscular synapses by blocking the release of acetylcholine. Thus preventing the permanent muscular contractions that are responsible for the signs of aging.</p> <p>When Botox is injected in small doses, its action will be limited on the treated zone. However, when it is used in high doses, its action is generalized on other muscles especially respiratory ones which will be permanently relaxed leading to death by asphyxia.</p>	½										

Part of the exercise	Exercise 2 Role of the Liver in Glycemia Regulation	Grade 5 pts
1.1	<p>In individual X who was fasting, glycemia increases, following the ingestion of 50g of glucose, from 0.8 g/L at t= 0 min to 1.7 g/L at t= 60 min, while it decreases to 0.9 g/L between 60min and 120 min.</p> <p>In individual Y who is fasting, glycemia decreases from 1 g/L to 0.7 g/l between from T0 and T2 while it increases slightly to 0.8 g/l at T3.</p>	$\frac{1}{4}$ $\frac{1}{4}$
1.2	The body is provided with a system of regulation able to correct hypoglycemia or hyperglycemia.	1/2
2	We observe brown color indicating the presence of glycogen only in section B done on the liver after a meal rich in glucose. This shows that the liver stores glucose in the form of glycogen.	1/2
3	The fresh liver is cut into fragments, and then added into a crystallizer containing distilled water. After that a strip to detect glucose is introduced, the test is positive. After that the liver fragments are washed. After that the washed fragments are put in a crystallizer and a strip to detect glucose is introduced, the test is negative. After incubation during 20 minutes at 37°C, a strip to detect glucose is introduced, the test is positive.	1
4	<p>The glucose test becomes positive after the incubation of the washed liver fragments for 20 minutes. This shows that the liver releases glucose. On the contrary, the test remains negative when incubation is performed with insulin. This shows that insulin blocks the release of glucose by inhibiting glycogenolysis.</p> <p>The test becomes positive when incubation is performed with glucagon and this occurs only within 5 minutes, time that is less than the 20 minutes necessary to obtain a positive test in the medium containing only distilled water. This shows that glucagon accelerates the release of glucose; hence, it promotes glycogenolysis. So, glycogenolysis which is performed by the liver and which ensures the release of glucose is inhibited by insulin and promoted by glucagon. Knowing that insulin and glucagon are two hormones, so glycogenolysis is modulated by the action of these hormones, insulin and glucagon.</p>	$\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$
5	<p>Document 1 shows that the ingestion of glucose provokes hyperglycemia between t=0 min and t= 60 min. Which stimulates the secretion of insulin by β cells of the islets of Langerhans of the pancreas. This hormone stimulates hepatic glycogenesis (storage of glucose in the form of glycogen) and inhibits glycogenolysis leading to a decrease in glycemia at t=120min</p> <p>Fasting doesn't allow any exogenous supply of glucose while glucose is constantly used by the body cells. Thus, glycemia decreases between T0 and T1 (document 1). This decrease stimulates the α cells of the islet of Langerhans to secrete glucagon. The latter stimulates hepatic glycogenolysis (release of glucose from the stored glycogen). This maintains a normal almost constant level of glycemia close to its initial value starting from T3.</p>	$\frac{1}{2}$ $\frac{1}{2}$

Part of the exercise	Exercise 3 Huntington Chorea	Grade 5 pts
1	The allele of the disease is dominant with respect to the healthy allele, since normal children III3 and III4 have affected parents II1 and II2. Thus the normal allele is carried at least by one of the parents and masked by the allele of the disease. Let H be the symbol of the dominant allele of the disease and n the symbol of the recessive normal allele.	1/2
2	<p>If the allele is carried on the non-homologous segment of the chromosome Y, the disease would be transmitted from father to son, but the affected son II4 has a healthy father II1. Thus the gene is not carried on the non-homologous segment of the chromosome Y.</p> <p>If the gene is carried by the non-homologous segment of the chromosome X, the healthy girl IV1 must be homozygous of genotype $Xn//Xn$; she should have inherited the normal allele from her father III1 who should be healthy of genotype $Xn//Y$. But her father is affected. Thus the gene is not carried by non-homologous segment of X.</p> <p>If the gene is carried by the homologous segments of X and Y, healthy girl III3 should have inherited Xn from her father II1; the healthy boy III4 should have inherited Yn from his father II1. Father II1 should be healthy of genotype $XnYn$ which is not the case (II1 is affected). thus the gene is not carried by the homologous segments of X and Y.</p> <p>Therefore, the gene is carried by an autosome.</p>	$\frac{1}{4}$ $\frac{1}{4}$ $\frac{1}{4}$ $\frac{1}{4}$
3	<p>The mother II2 is affected by the disease and is heterozygous since she inherited the allele H from her mother and the allele n from her homozygous healthy father who produces only one type of gametes having the allele n.</p> <p>Thus she produces two types of gametes of equal probabilities: $\frac{1}{2} H$ and $\frac{1}{2} n$.</p> <p>The affected father II1 is heterozygous since he already has a healthy homozygous son III4 to whom he must have transmitted the recessive allele n.</p> <p>Thus he produces two types of gametes equal probabilities: $\frac{1}{2} H$ and $\frac{1}{2} n$.</p> <p>Since the affected allele of the disease is dominant; it is sufficient for III3 to have at least one allele of the disease in order to be affected. The genotype of III3 can be either $H//H \frac{1}{4}$ or $H//n \frac{1}{2}$. Thus the risk for III3 to be affected is 3/4 of the children.</p> <p>Couple II5- II6 is healthy and recessivity is a criterion of purity. These parents produce only one type of gametes carrying the normal allele n. Thus all their children will be healthy.</p> <p>Therefore the risk for III5 to be affected is null.</p>	$\frac{1}{2}$
4	In healthy individuals, the number of repetitions of CAG varies between 8 and 30 for the types of alleles A1 till A12. Thus these alleles are associated to the normal phenotype. However, affected individuals present two groups of alleles: the first is identical to that present in healthy individuals with a number of repetitions of CAG between 8 and 30. The second group corresponds to alleles having a number of repetitions of CAG between 39 and 70. Thus these alleles which have a number of repetitions of CAG higher than 39 are associated to the disease. The origin of the disease is the high number of repetitions of CAG greater than 39	1
5	The real genotype of III3 is $n//n$ or $A_6//A_9$. Since she has two alleles with a number of repetitions CAG that is respectively 10 and 15 which is less than 30 repetitions and thus correspond to the group of alleles of healthy individuals. These two alleles are among the ones that determine the normal phenotype.	$\frac{3}{4}$
6-1	The average age of appearance of the disease decreases from 49 years to 25 years, when the number of repetitions of CAG triplet increases from 40 to 60.	$\frac{1}{2}$
6-2	The factor determining the age of appearance of the disease is the high number of repetitions per allele(>40).	$\frac{1}{4}$

Part of the exercise	Exercise4 Fight against Ebola	Grade 5 pts
1	<p>A humoral specific immune response is triggered, since in case of Ebola the surviving individuals have a high amount of anti-Ebola antibodies that are released by plasma cells that are the effectors of humoral specific immune response.</p> <p>A cell-mediated specific immune response is triggered, since the surviving individuals show an important increase in the specific TcL which are the effectors of the cell mediated specific immune response.</p>	$\frac{1}{2}$ $\frac{1}{2}$
2	<p>The amount of anti-Ebola antibodies is nil and remains constant on the 14th and 35th day, after the first and the second injection of the three types of vaccine. After the 3rd injection of vaccine, this amount increases to 1000 a.u on the 56th day in individuals having received EIC + PIC, while it remains constant and nil in individuals having received the vaccine EIC or only the protein. This amount of antibodies increases in the three lots to reach 10000 a.u on the 84th day in individuals having received the vaccine EIC+PIC which is 10 times higher than the 1000a.u obtained when only the vaccine EIC or only the protein is administered. This shows that the vaccine EIC+PIC is the most effective.</p>	3/4
3	<p>At the beginning of the specific immune response, macrophages act as antigen presenting cells which induce the specific immune response.</p> <p>At the end of the specific humoral immune response, they perform phagocytosis of the immune complex in order to eliminate antigens.</p>	1
4	<p>Between day 0 and day 20, the percentage of survival decreases from 100% to 80% in the lot receiving 4 injections. This decrease is 4 times more significant than that obtained in the lot receiving only 3 injections which reaches 20 %. Thus, the condition for the vaccination against Ebola to be successful is to give 4 boosters.</p>	1/2
5	<p>The antibodies injected after 24 hours neutralize the antigen and slow down efficiently the propagation of virus which allows the body defenses to react and protect all the monkeys (4/4) which remain alive. However, when the injection is delayed to 48 hours, the viruses multiply more rapidly than the lymphocytes involved in the specific immune response and infect a great number of cells before being neutralized by the specific injected antibodies. This reduces the efficiency of the body defense and sometimes renders it insufficient. This explains the death of two out of the four infected monkeys.</p>	1
6	<p>In serotherapy, the injected substances are the specific antibodies while in vaccination, the injected substances are viral or antigenic proteins.</p> <p>In serotherapy, the latency time is null while in vaccination, the latency time is 2 weeks</p> <p>In serotherapy, the duration of protection time is short while in vaccination, the protection is more durable.</p>	$\frac{1}{4}$ $\frac{1}{4}$ $\frac{1}{4}$

الاسم: _____
الرقم: _____

مسابقة في مادة علوم الحياة
المدة: ثلاثة ساعات

Answer the following exercises

Exercise 1 (5 points)

Origin of Mental Retardation

Alain, son of Riad and Samar, is affected by a mental retardation. This couple who has no family history concerning mental retardation is expecting a second child and wishes to know whether he will be affected like his brother.

- 1- Formulate a hypothesis explaining the appearance of this retardation in Alain.

In order to understand the possible origin of this mental retardation, the following studies are performed.

Blood analysis of Alain concerning substances involved in mental retardation shows a high amount of purines of 118 mmol/L with respect to the normal level of 79 mmol/L.

The synthesis of purines is controlled by 5 enzymes. The pathway of this biosynthesis in the body is presented in document 1.

Three cell cultures are performed.

- Culture 1:** nerve cells are cultured in a medium rich in purines. These cells degenerate.
- Culture 2:** cells of CHO mice are cultured in a medium without purines. In these mice, the gene coding for enzyme E2 which is homologous to that of humans, is inactive. These cells degenerate.
- Culture 3:** human cells are fused with cells of CHO mice and Hybridoma are obtained. These hybridoma are cultured in a medium without purines. Spontaneously, some hybridoma lose with time their human chromosomes. Those that lose their chromosome n° 21 degenerate and those that conserve the chromosome n° 21 remain in the medium.

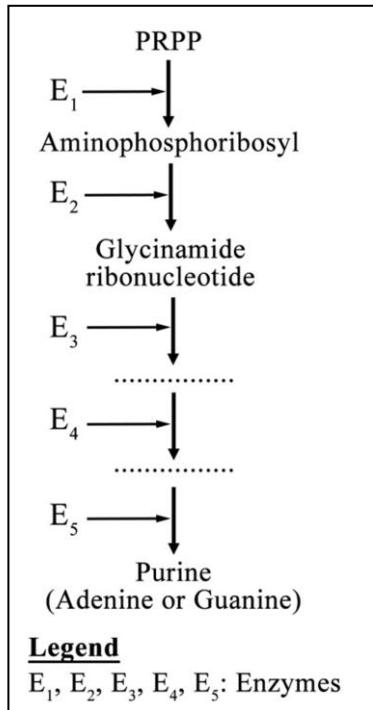
- 2- Interpret the results obtained in cultures 1 and 2.
3- Determine the location of the gene studied in this mental retardation.

The karyotype of Alain consists of 46 chromosomes. Document 2 shows the blood level of purines as well as the karyotype of Alain, those of his parents, and that of the fetus. In these karyotypes only the pairs of chromosomes 14 and 21 are schematized; the other pairs of chromosomes are normal.

Alain's Family	Mother : Samar	Father : Riad	Alain	Fetus
Karyotype	 14 14 21 21	 14 14 21	 14 14 21 21	 14 14 21
Blood level of purines (in mmol/L)	79	79	118.5	?

Document 2

- Determine, from all what precedes, the origin of the mental retardation revealed in Alain.
- Specify the diagnosis for the fetus.
- Make the factorial analysis to determine the phenotypic proportion of this couple's children who will suffer from a mental retardation identical to that of Alain.

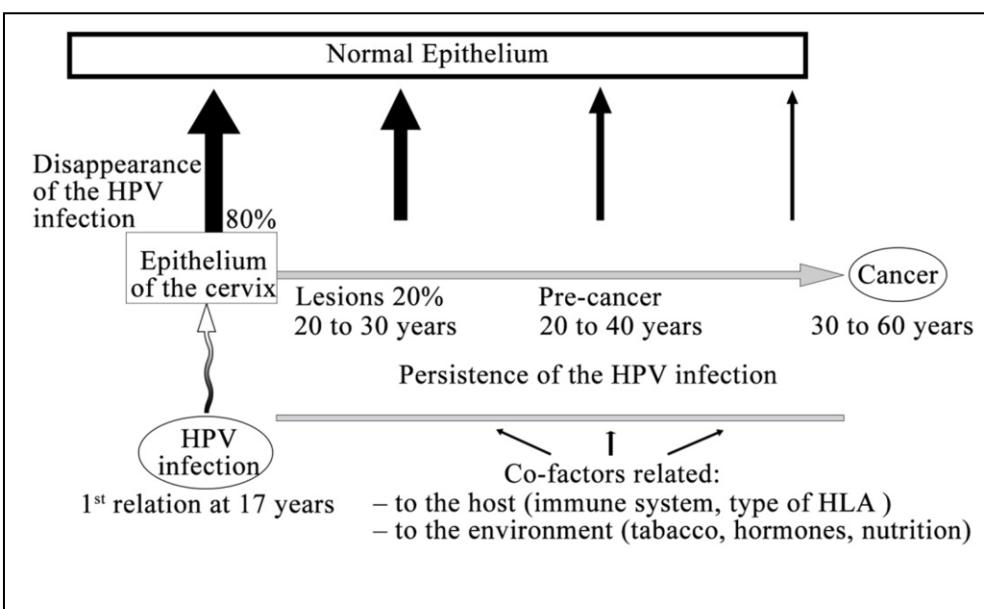


Exercise 2 (5 points)

Cervical cancer is considered to be the second cancer that affects women in the developing countries, and the eighth in the developed ones. To better understand the cause of this cancer and in order to prevent it efficiently, researchers performed different studies.

- A study involving thousands of women suffering from cervical cancer shows that 75% of these women have encountered the human papillomavirus (HPV) during their sexual life.

Document 1 shows the evolution of the state of the cervical epithelium after HPV infection.



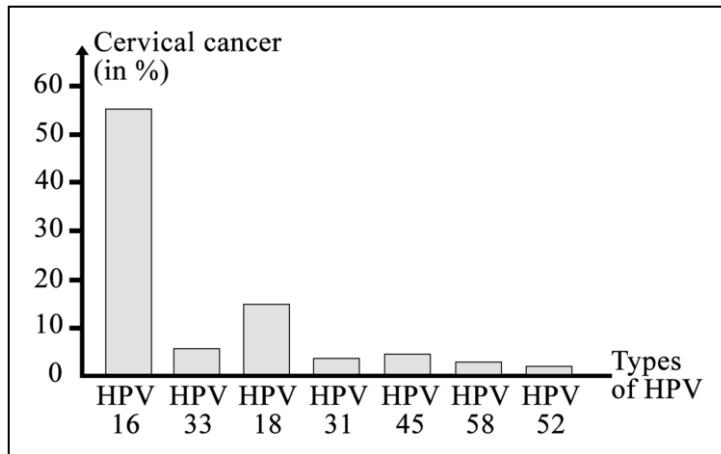
Document 1

- 1- Justify the following statement: « Cervical cancer is a slowly induced viral cancer ».
- 2- Pick out, from document 1, two other risk factors for the development of cervical cancer.

- 3- Indicate the type of specific immune response triggered to fight against a viral infection. Justify the answer.

- In a second study, 150 types of HPV have been identified, some of which are qualified as being of "high-risk", induce a genetic mutation which causes cervical cancer. Document 2 shows the percentages of women having cervical cancer as a function of the types of HPV that have infected them.

- 4- Deduce from document 2 the two types of HPV of high risk .



Document 2

- Researchers have elaborated prophylactic vaccines aiming to ensure a preventive protection of individuals against the infection. These vaccines stimulate the production of antibodies directed against certain types of HPV viruses. The characteristics of two of these vaccines are regrouped in document 3.

- 5- Determine the most efficient vaccine.
- 6- Explain how the antibodies produced during this vaccination allow protection against cervical cancer.
- 7- Suggest two preventive means against cervical cancer.

	Vaccine	
	Gardasil	Cervarix
Types of targeted HPV	Quadrivalent Vaccine HPV6, HPV11, HPV16 and HPV18	Bivalent Vaccine HPV16 and HPV18
Suggested amount	Almost 20 µg	20 µg
Vaccination Schedule	0, 2 and 6 months	0, 1 and 6 months
Amount of antibodies produced compared to that of the natural infection	8 times higher	100 times higher

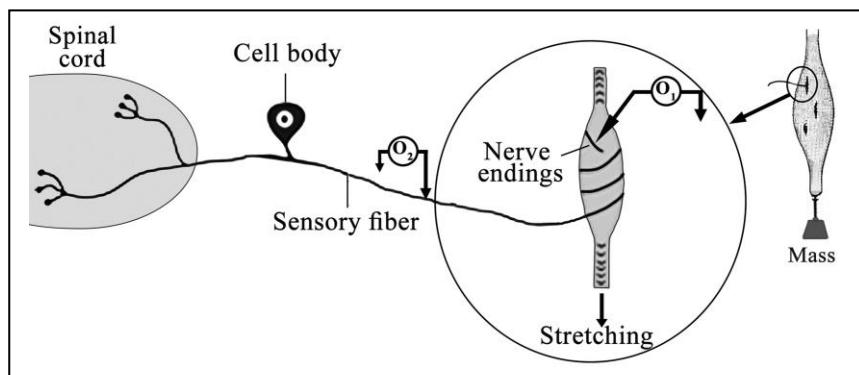
Document 3

Exercise 3 (5 points)

Nervous Coding of Sensory Information

When a dog is kept on a leash, the muscles of the arms react immediately to all the traction variations they undergo. This is a reflex.

In order to study the coding of the message involved in this reflex, the following experiments are performed using the experimental set up presented in document 1.



Document 1

Experiment 1: The arm muscle is stretched five times using increasing masses. The obtained responses are recorded by oscilloscope O1 at the level of the neuromuscular spindle (document 2) and by oscilloscope O2 at the level of the sensory nerve fiber (document 3). Meanwhile more and more important contractions are observed at the level of the stretched muscle.

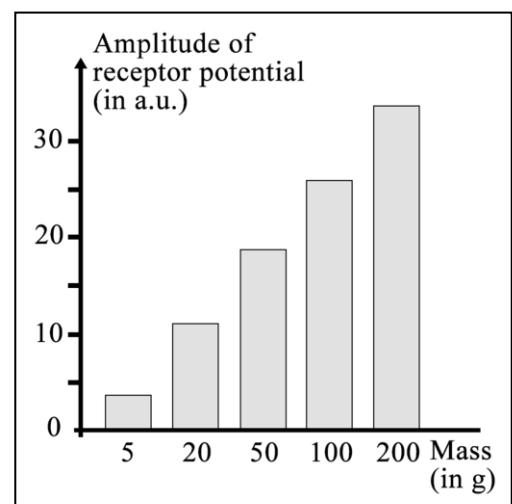
- 1- Show that this is a “myotatic reflex”.
- 2- 2-1- Analyze the results of each of the documents 2 and 3.
2-2 Conclude the type of coding of the nerve message at the level of the neuromuscular spindle and at the level of the sensory fiber.

Experiment 2: This muscle is subjected twice to the same effective stretching of $750 \mu\text{m}$ at different velocities. The response for each stretching is recorded at the level of the sensory fiber. The obtained results are presented in document 4.

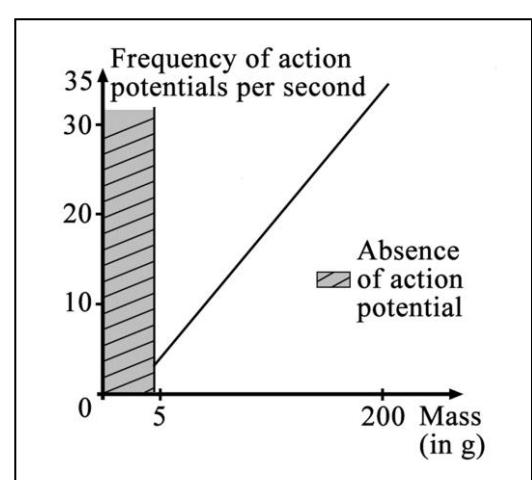
- 3- What can you draw out from document 4?

Experiment 3: The arm muscle, like in experiment 1, is stretched five times using increasing masses. The amount of the neurotransmitter (acetylcholine) that is released at the level of the synapse involved in the neural circuit of this reflex is measured. The obtained results are shown in document 5.

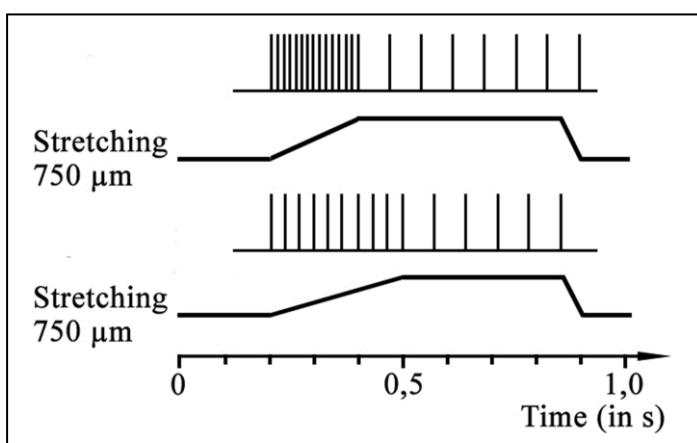
- 4- Draw the curve showing the variation of the amount of released acetylcholine as a function of the intensity of stretching.
- 5- Deduce the type of coding of the nervous message at the level of a synapse.
- 6- Show how the arm muscles react in an adapted way to each traction.



Document 2



Document 3



Document 4

Intensity of stretching (in a.u.)	5	10	15	20	25
Amount of released acetylcholine (in a.u.)	20	30	40	50	60

Document 5

- 7- Schematize the neuronal circuit and the structures involved in this reflex.

Exercise 4 (5 points)

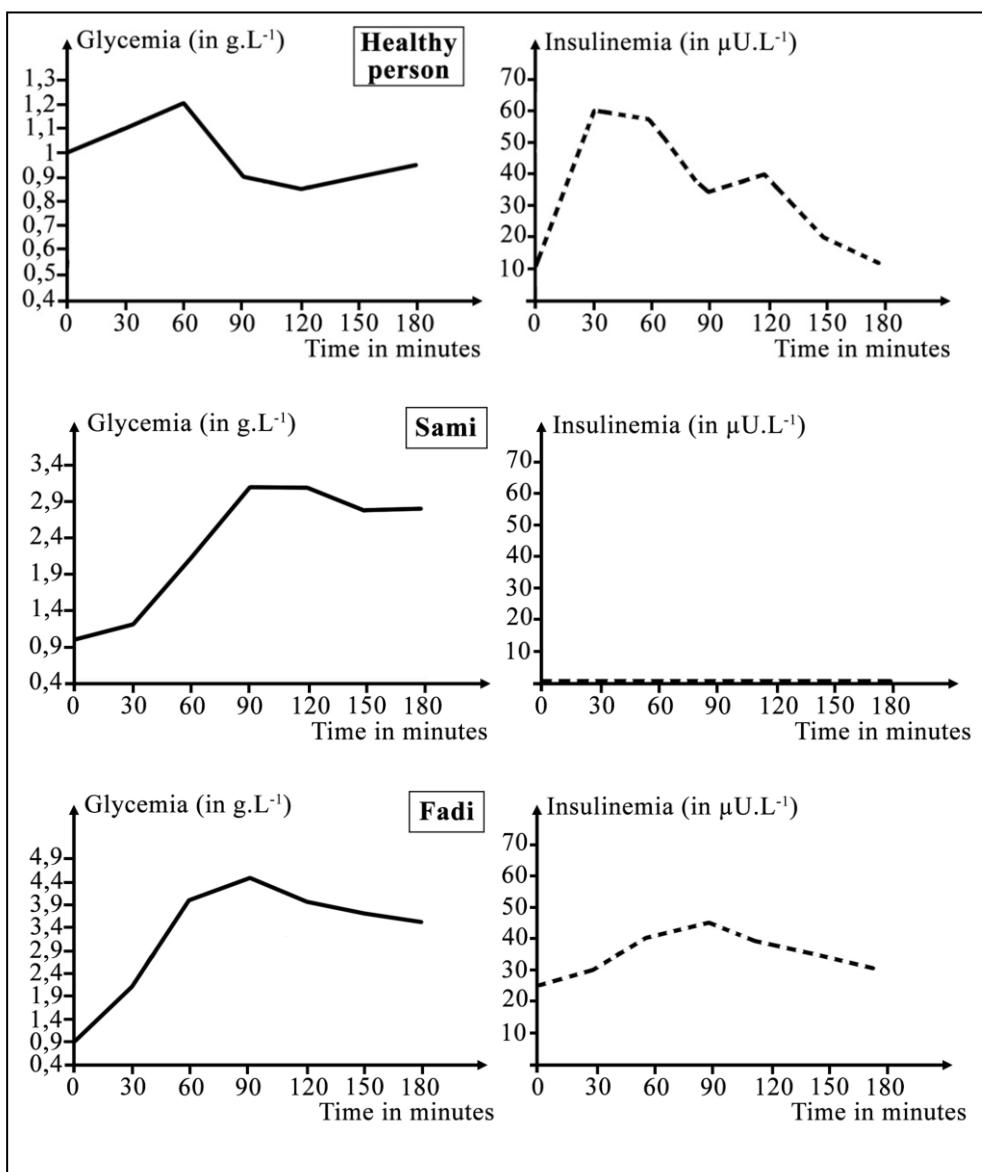
Two persons Sami and Fadi show the following symptoms: frequent urination, severe thirst, sensation of intense hunger, loss of weight and intense fatigue. They consult a doctor who monitors their glycemia and insulinemia after an ingestion of 50g of glucose at $t = 0$ min. The results obtained for Sami and Fadi as well as those of a healthy person are presented in document 1.

- 1- Specify, for the healthy person, the factor that determines the secretion of insulin on one hand, and the role of insulin on the other hand.
- 2- Determine the type of diabetes diagnosed for each of the two diabetic persons: Fadi and Sami.

Histological examination of the pancreas of each of the two diabetic persons, Sami and Fadi, shows the results presented in document 2.

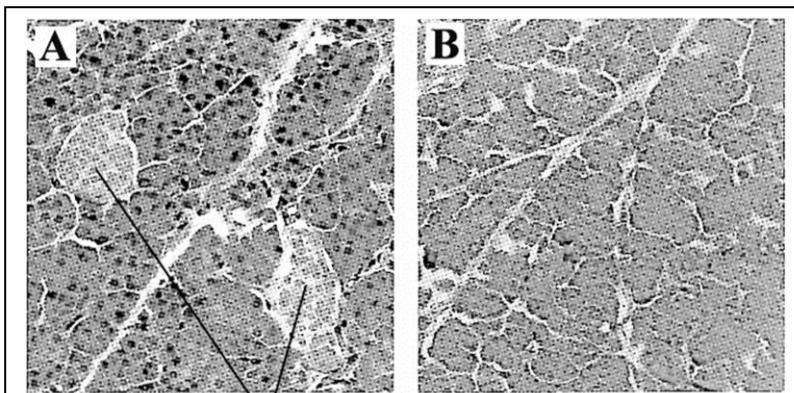
- 3- Match each of the sections A and B to each of these persons.
Justify the answer.

Diabetes: Type I or Type II?



Document 1

Nowadays a treatment by artificial monohormonal pancreas is prescribed to some diabetic patients. It consists of an apparatus inserted under the skin. This apparatus ensures a continuous automatic measurement of blood glucose and sends the data to a control programmed module. This module calculates the necessary amount of insulin and sends commands to an insulin pump to deliver the calculated amount.



Islets of Langerhans

Document 2

- 4- Justify the following statement: "the artificial monohormonal pancreas is a treatment that is more efficient than the treatment based on insulin injections".
- 5- Determine to which person the doctor will suggest the treatment with artificial pancreas.
- 6- Suggest an advice that the doctor will give to the second person. Justify the answer.

Part of the ex	Exercise 1 Origin of Mental Retardation	Grade 5 pts																							
1	<p>Hypothesis: The mental retardation of Alain is due to a recessive allele masked in parents. Or The mental retardation of Alain is due to the mutation of a gene implicated in the mental development and that occurred during his conception. Or The mental retardation of Alain is due to a chromosomal aberration (that occurred during meiosis in one or in both parents).</p>	3/4																							
2	<p>Nervous cells degenerate in the culture medium rich in purines (culture 1). Similarly cells which are unable to synthesize purines degenerate in the medium lacking purines (culture 2). This shows that the synthesis of purines in amounts far from its normal range is responsible for the degeneration of cells.</p>	1/2																							
3	<p>Culture 2 shows that the cells of CHO mice having inactive E2 are unable to synthesize purines and degenerate. Culture 3 shows that the hybridoma having lost their chromosome 21 degenerate. And since the degeneration of nervous cells may lead to a mental retardation, this allows us to say that the gene coding for E2 is localized on the chromosome 21 and that its inactivation is responsible for the mental retardation.</p>	1/2																							
4	<p>Culture 1 shows that cells degenerate in a medium rich in purines, and Alain possesses a high purines level of 118mmol/L. This excessive synthesis is due to an additional allele. However the karyotype of Alain shows two free chromosomes for each of the pairs 14 and 21 with one chromosome 14 that is longer than its homologous. And since the allele coding for E2 is carried by the chromosome 21. This can be explained by the presence of an additional chromosome 21 linked to the chromosome 14. Thus the origin of the mental retardation of Alain is a linked trisomy 21 leading to a high enzymatic activity of E2.</p>	1																							
5	<p>The fetus is normal, since as his normal father he possesses a free chromosome 21 and another chromosome 21 linked on the chromosome 14. He has conserved his genetic material, he has two alleles coding for the enzyme E2 and consequently will have a normal amount of purines of 79 mmol/l indicating a normal mental activity.</p>	3/4																							
6	<p>Factorial analysis</p> <table style="margin-left: auto; margin-right: auto;"> <tr> <td>Phenotype :</td> <td>normal mother</td> <td>X</td> <td>normal father</td> </tr> <tr> <td>Genotype :</td> <td>14//14 21//21</td> <td></td> <td>14//14²¹ 21//</td> </tr> </table> <p>Gametes and proportions : 14 21 14 21 , 14 , 14²¹ , 14²¹ 21 1 1/4 , 1/4 , 1/4 , 1/4</p> <p>Table of cross :</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td></td> <td>14 21 1/4</td> <td>14 1/4</td> <td>14²¹ 1/4</td> <td>14²¹ 21 1/4</td> </tr> <tr> <td>1421 1</td> <td>14//14 21//21 1/4</td> <td>14//14 21// - 1/4</td> <td>14//14²¹ 21// - 1/4</td> <td>14//14²¹ 21// 21 1/4</td> </tr> <tr> <td></td> <td></td> <td></td> <td></td> <td>Mental retardation like that of Alain</td> </tr> </table> <p>The Phenotypic proportion of children suffering from mental retardation like that of Alain is 1/4</p>	Phenotype :	normal mother	X	normal father	Genotype :	14//14 21//21		14//14 ²¹ 21//		14 21 1/4	14 1/4	14 ²¹ 1/4	14 ²¹ 21 1/4	1421 1	14//14 21//21 1/4	14//14 21// - 1/4	14//14 ²¹ 21// - 1/4	14//14 ²¹ 21// 21 1/4					Mental retardation like that of Alain	1 1/2
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				Mental retardation like that of Alain																					

Part of the exercise	Exercise 2 Cervical Cancer and the HPV Virus	Grade 5 pts
1	Studies involving thousands of women suffering from cervical cancer shows that 75% of them have encountered the human papillomavirus (HPV) at some point of their sexual life. Thus this cancer is induced by a virus. The development of this cancer is slow it requires the persistence of the HPV infection for more than 13 years before inducing a genetic mutation at the origin of the cancer (document 1). Thus, this cancer is induced by a virus and its development needs time.	3/4
2	The environment: tobacco, nutrition... Status of the host: immune system, type of HLA	1/2
3	The specific immune response is cell mediated. Since the virus integrates it's DNA into the genome of infected cells and modifies their immunological self. This modified self is only recognized by the LT8 which are the effectors of the cell mediated response.	3/4
4	Document 2 shows that the highest percentage of cancers is 54,5% due to HPV 16 and 16% due to HPV 18. These percentages are higher than those of cancers induced by all the other types of HPV (more than 100 type of HPV). Thus the two types HPV 16 and HPV 18 are of high risk.	3/4
5	Both vaccines, Cervarix and Gardasil , requires the same amount and the same number of repetitions (3times) and immunize the body against the two types of HPV of high risk(HPV16 and 18). On the contrary, the level of produced antibodies induced by the vaccine Cervarix (100 times higher) is more important than that induced by the vaccine Gardarix that is 8 times higher than that produced in the case of natural infection. Thus the more efficient vaccine is Gardarix.	1
6	The antibodies produced due to vaccination neutralize the viruses before they bind to the membrane receptors of the target cells of the cervix and inhibit the viruses from infecting them. The viruses are thus eliminated (the formed immune complexes will be phagocytized by macrophages) and the lesions leading to cancer do not appear . Thus antibodies inhibit the HPV infection and protect the epithelium from genetic mutations that are at the origin of cervical cancer.	3/4
7	Get vaccinated before the first sexual intercourse. Do not smoke, have balanced healthy nutrition.	1/2

Part of the exercise	Exercise 3 Nervous Coding of sensory information	Grade 5 pts												
1	A myotatic reflex is the contraction of a muscle in response to its own stretching, in this case (exp 1) the stretching of the muscle of the arm leads to its contraction. Thus it is a myotatic reflex.	1/2												
2.1	Document 2 shows that the amplitude of the receptor potential increases from 4 to 35 a.u. when the used mass increases from 5 to 200g. Document 3 shows that the frequency of APs per second increases from 4 to 35AP/s when the used mass increases from 5g to 200g.	1/2												
2.2	The coding of the nervous message at the level of the neuromuscular spindles is modulated in amplitude. The coding of the nervous message at the level of the fiber is modulated in frequency of APs.	1/2												
3	The response is modulated in frequency of APs as a function of the velocity in which the stretching is performed.	1/2												
4	<p>Curve showing the variation of the amount of released acetylcholine as a function of the intensity of stretching.</p> <table border="1"> <caption>Data points estimated from the graph</caption> <thead> <tr> <th>Intensity of stretching (a.u.)</th> <th>Amount of released acetylcholine (a.u.)</th> </tr> </thead> <tbody> <tr><td>5</td><td>20</td></tr> <tr><td>10</td><td>30</td></tr> <tr><td>15</td><td>40</td></tr> <tr><td>20</td><td>50</td></tr> <tr><td>25</td><td>60</td></tr> </tbody> </table>	Intensity of stretching (a.u.)	Amount of released acetylcholine (a.u.)	5	20	10	30	15	40	20	50	25	60	1
Intensity of stretching (a.u.)	Amount of released acetylcholine (a.u.)													
5	20													
10	30													
15	40													
20	50													
25	60													
5	The amount of the released neurotransmitter increases from 20 to 60 a.u. when the intensity of stretching increases from 5 to 25 a.u. Hence, the coding at the level of the synapse is modulated in concentration of neurotransmitters.	1/2												
6	The traction performed by the dog stretches the neuromuscular spindles of the arm muscle ensuring the contraction of the same stretched muscle (it's a myotatic reflex). Since the response of the sensory neuron: the receptor potential and the APs as well as the response at the level of the synapse, the amount of neurotransmitters, are modulated in function of the intensity and the velocity of the traction. This allows to adapt the response to each traction.	3/4												
7	<p>The neuronal circuit and the structures involved in this reflex.</p>	3/4												

Part of the exercise	Exercise 4 Diabetes: Types I or Type II?	Grade 5 pts
1	<p>The factor that determines the secretion of insulin is the hyperglycemia. Since after the ingestion of glucose, glycemia increases from 1g/L to 1,2 g/l within 60 minutes. And insulinemia also increases from $10 \mu\text{g.L}^{-1}$ to reach a maximum of $60 \mu\text{g.L}^{-1}$ within 30 min. 1/2pt</p> <p>Insulin is hypoglycemic. Since glycemia decreases from 1,2 g/L to 0,85 g/L between 60 and 120 min, when insulinemia reaches its optimum $60 \mu\text{g.L}^{-1}$ between 30 to 60 min. 1/2pt</p>	1
2	<p>In Sami, there is an important increase of glycemia following the ingestion of 50g of glucose from 1g/L to 3g/L (superior to the 1,2 g/L in the healthy individual), whereas insulinemia remains nil during the 90 min after the ingestion . This shows that Sami doesn't produce insulin that is indispensable for the decrease of hyperglycemia. Thus Sami has type I diabetes or insulin-dependent diabetes. 1/2pt</p> <p>In Fadi, there is an increase of glycemia following the ingestion of 50g of glucose from 0,9 g/L to 4,4 g/L 3 times higher than healthy individual. Similarly, Insulinemia increases from $25 \mu\text{g.L}^{-1}$ ($> 10 \mu\text{g.L}^{-1}$ in healthy individual) to $40 \mu\text{g.L}^{-1}$ ($< 60 \mu\text{g.L}^{-1}$ in healthy individual) during 90 min (> 30 min)</p> <p>This shows that Fadi secretes insulin more slowly than the healthy individual and in an amount that is insufficient to induce a decrease of the provoked hyperglycemia. Hence, Fadi has diabetes of type II, "non-insulin-dependent" that can be due to abnormal insulin or the absence or lack in insulin-receptors at the level of the target cells. 1/2pt</p>	1
3	<p>Section A corresponds to Fadi. It shows the presence of islets of Langerhans responsible for the secretion of insulin, which explains the secretion of insulin after the increase of glycemia.</p> <p>Section B corresponds to Sami. It shows the absence of islets of Langerhans, which are responsible for the secretion of insulin, which explains the nil insulinemia after the increase of glycemia.</p>	1
4	<p>The artificial monohormonal pancreas is more efficient than insulin injections. It permanently detects in an automatic manner the variation of glycemia allowing the administration of insulin in amounts adapted to this variation. This is not the case with injections done at specific times and constant doses that are sometimes inadequate to the needs.</p>	3/4
5	<p>The artificial pancreas administers insulin as a function of glycemia that is controlled constantly by a programmed module. It is only prescribed to persons who have a problem in insulin secretion: absence or insufficient amount of insulin or abnormal insulin. This corresponds to the case of Sami who has no insulin secretion and no islets of Langerhans.</p>	3/4
6	<p>The doctor prescribes to Fadi a diet that does not induce hyperglycemia. Since Fadi has normally (in absence of provoked hyperglycemia), an insulin secretion higher than that of the normal individual that allows him to maintain a normal constant glycemia. And the increase in this secretion in case of hyperglycemia is not efficient and doesn't lower glycemia.</p>	1/2

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Answer the following exercises:

Exercise 1 (5 points)

Dysuria

Dysuria is a disease that consists of a difficulty in urinating. It's related to excessive formation of urinary calculi ("stones" in urinary tracts). A family, which has twins suffering from dysuria, consults a doctor. He prescribed many tests whose results are represented in document 1.

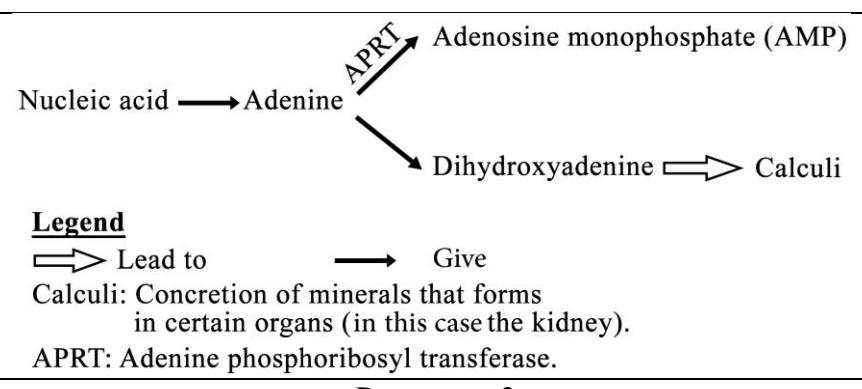
Document 2 shows the reactions of metabolism of adenine related to the formation of calculi.

Measurements	Control	Twins
Quantity of adenine in urine excreted within 24h	1.5 mg	40 mg
Dihydroxyadenine (constituent of calculi)	Not detected	High quantity
Amount of active enzyme APRT	100 %	0 %

Document 1

- 1- Justify, by referring to documents 1 and 2, the dysuria detected in the twins.

In order to clarify the problem observed in the twins, a more detailed analysis concerning members of their family was performed. The pedigree of their family is shown in document 3.



Document 2

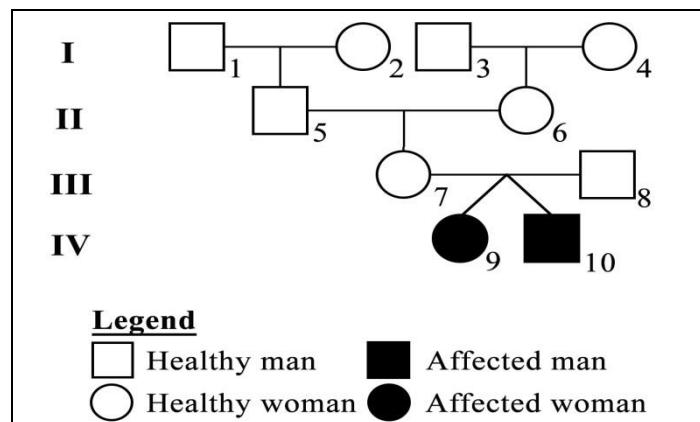
- 2- Formulate, by referring to document 3, two hypotheses explaining the appearance of the disease in the twins.

- 3- Knowing that the gene exists only in two allelic versions, specify if the allele responsible for the disease is dominant or recessive.

- 4- Show that this gene is not carried by a sex chromosome.

- 5- Indicate the possible genotype(s) of each of the individuals II5 and III8. Justify the answer.

Blood tests concerning the amount of active enzyme APRT were performed in members of this family. The results are represented in document 4.



Document 3

- 6- Show, by referring to document 4, that at the molecular level, the two alleles are codominant.

Member of the family	Amount of active APRT
III7	50 %
III8	50 %
II5	50 %
II6	100 %
IV9	0 %
IV10	0 %

Document 4

Exercise 2 (5 points)

AIDS and Treatments

The human immunodeficiency virus (HIV) is responsible for the weakness of immune defenses in the organism, which leads to the death of affected persons. Document 1 shows the evolution of the concentration of T4 cells, measured in patients contaminated by HIV.

- 1- Analyze the results of document 1.
- 2- Draw out, from document 1, the cause of the appearance of opportunistic diseases.

In order to find a treatment that limits the consequences of opportunistic diseases, a series of studies is performed, some of which are represented below:

Study 1: Lymphocytes are removed from a monkey and B, T4 and T8 cells are separated.

- B cells are placed in chambers of culture 1 (1a, 1b and 1c) where molecules of antigen X are present at their bottoms. Only 0.01% of B cells remains fixed to the bottom of each chamber and is not eliminated by rinsing.
- T8 cells are placed in chambers of culture 2 (2a and 2b) where monkey cancerous fibroblasts are present at their bottoms. Only 0.01% of T8 cells remains fixed to the bottom of each chamber and is not eliminated by rinsing.
- Then, lymphocytes activated by the same antigens (X or cancerous fibroblasts) are added to certain chambers.

Document 2 shows the experimental conditions as well as the results.

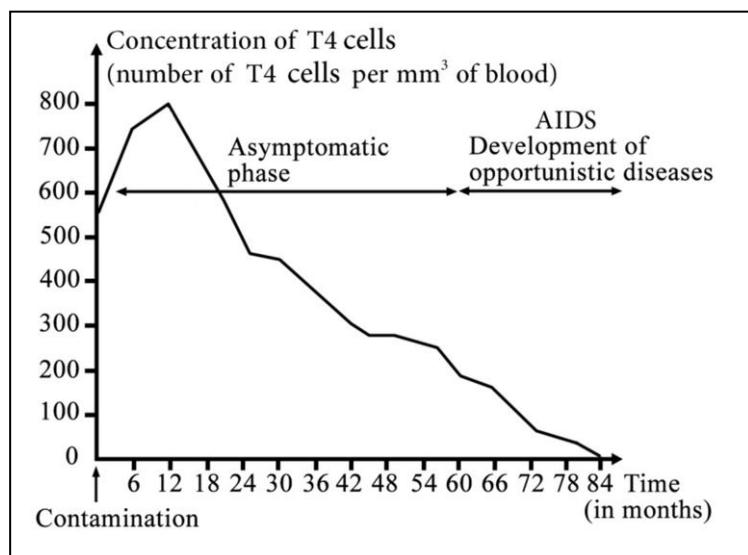
- 3- Interpret the results obtained in document 2.
- 4- Explain, by referring to all what precedes, the appearance of opportunistic diseases observed in document 1.

Study 2: Two groups of researchers have produced two treatments.

The first treatment is based on the principle of vaccination against some opportunistic diseases such as pneumonia. This treatment was tested on two categories of patients having a different number of T4 cells. The results are represented in document 3.

In the second treatment, three medicines are administered during 5 years to individuals whose number of T4 cells, at the beginning of treatment, is between 200 and 350 T4 cells/mm³ of blood. The results are shown in document 4.

- 5- Explain the importance of vaccination.
- 6- Determine if the first treatment is efficient against the development of opportunistic diseases.
- 7- Show that the second treatment may delay the AIDS phase.



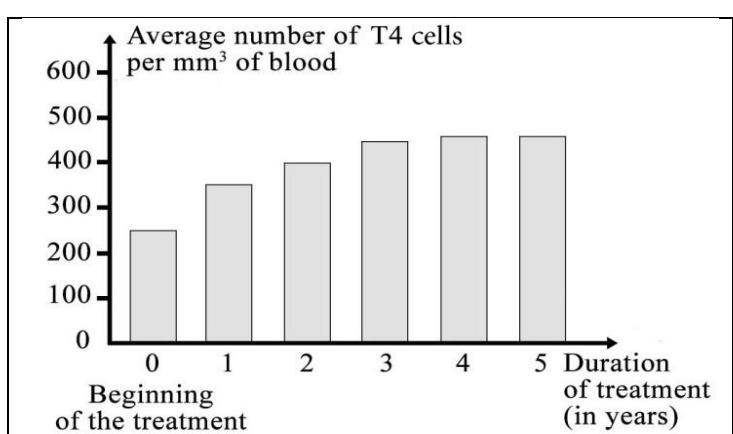
Document 1

Chamber	Existing lymphocytes	Added lymphocytes	Result
1a	B cells retained by antigen X	-	No antibodies
1b		Activated T4 cells	Presence of antibodies
1c		Activated T8 cells	No antibodies
2a	T8 cells retained by monkey cancerous fibroblasts	-	No lysis of fibroblasts
2b		Activated T4 cells	Lysis of fibroblasts

Document 2

Category	Average number of T4 cells/ mm ³ of blood	production of antibodies
1	> 500	Strong
2	< 200	Weak

Document 3



Document 4

Exercise 3 (5 points)**Analgesia without Morphine**

Morphine is an analgesic substance (pain-killer) that acts at the level of enkephalin and endorphin synapses. The latter substances are neurotransmitters that are naturally produced in the brain and in the spinal cord, while morphine is exogenous. Its excessive usage causes physical and psychological dependence as well as respiratory and digestive troubles.

- 1-** Explain how morphine acts at the level of enkephalin synapses.

In order to avoid the secondary effects of the use of morphine, researchers have tried to find other endogenous analgesic substances. Some of their studies are represented in the following experiments.

Experiment 1: Researchers have injected serum to rats without or with analgesic, morphine or endorphin.

Then, they put each rat in zone P of a box whose surface is divided into two zones: zone P (periphery) that is covered by sharp ends causing intense pain, and zone S (center) without sharp ends. Then, during three minutes, they measured the average duration during which the rats stayed in zone P. This duration indicates the analgesic effect of the studied substance. Document 1 shows the conditions as well as the results of the experiment.

Animals	Injections	Duration of staying in zone P (sec)
A	-	5
B	Morphine (6 mg/kg)	72
C	Endorphin (6 mg/kg)	5

Document 1

- 2-** What can you deduce from experiment 1?

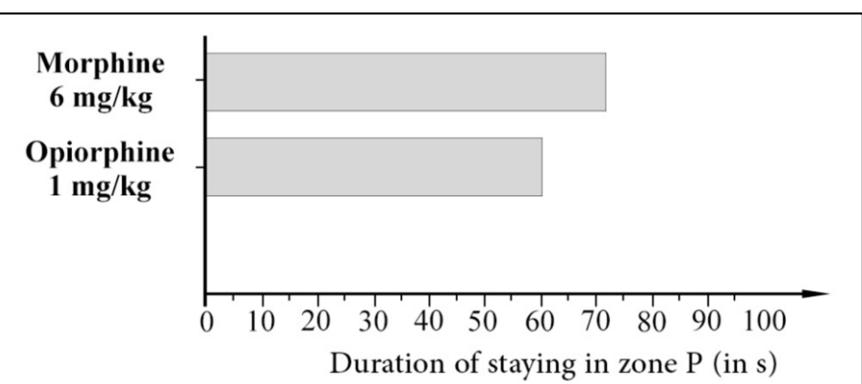
- 3-** Formulate a hypothesis explaining the result obtained upon the injection of endorphin.

Experiment 2: Researchers have injected endorphin marked by radioactive tritium in the blood of a group of animals. The performed tests reveal the absence of radioactivity in the brain and in the spinal cord. Moreover, even in blood, endorphin disappears rapidly, but other radioactive molecules appear.

- 4-** Show that experiment 2 explains the result obtained in rats C.

Experiment 3: Other researchers of Pasteur institute have identified a new analgesic substance, secreted naturally in the saliva of humans, the opiorphine. They have tested opiorphine on rats. They repeated experiment 1 but they injected opiorphine instead of endorphin. The experimental conditions as well as the results are represented in document 2.

Document 3 shows information concerning opiorphine.



Document 2

- 5-** Show, by referring to document 2, that opiorphine is an effective analgesic.
6- Explain how opiorphine acts as an analgesic.
7- Draw out two reasons why opiorphine seems to be a molecule whose therapeutic value is more important than that of morphine.

Opiorphine has an analgesic power for thermal and mechanical pain as well as chronic pain. Opiorphine seems to protect enkephalin from the effects of the enzyme NEP present in the cleft of enkephalin synapses. It is not necessary to increase the doses of opiorphine to obtain the same anti-nociceptive effect. It doesn't cause constipation and its addictive effect is much reduced.

Document 3

Exercise 4 (5 points)

Female Infertility

Fertilization is not an automatic phenomenon. Only 25% of the sexual intercourses occurring during the fertile period are followed by pregnancy.

Document 1 shows a part of a female genital duct.

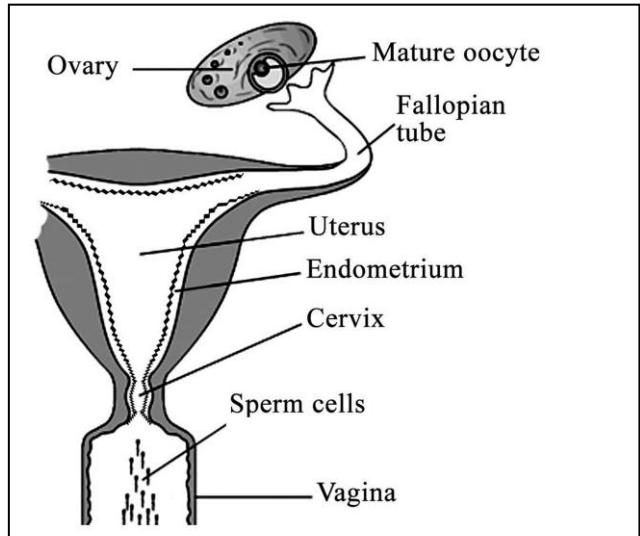
- 1- Indicate the site of fertilization and the role of the uterus.

- 2- Explain briefly the process of fertilization.

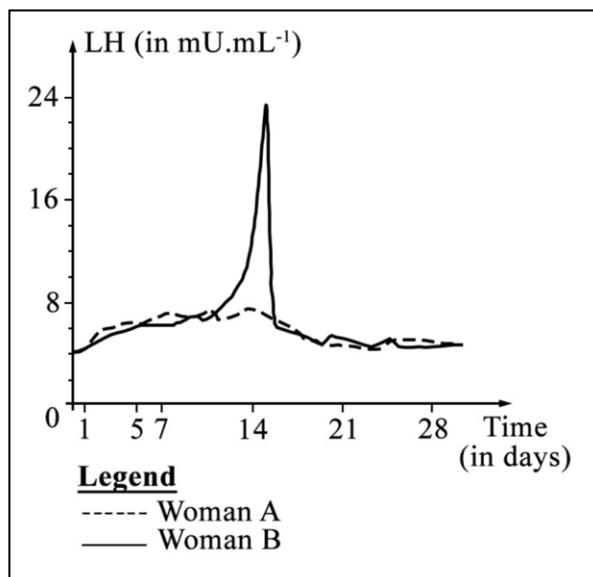
Two women, A and B, consult a gynecologist because of their infertility. In order to determine the origin of their infertility, the doctor prescribed the following tests:

- Measurement of the plasma concentration of LH hormone
- A radiologic exam of the genital duct after introducing an opaque liquid in the genital duct of each of the two women.

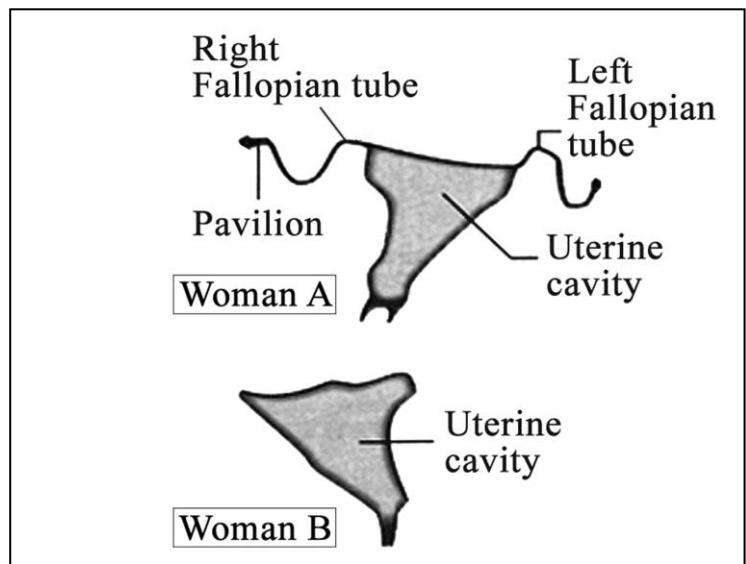
The results are shown respectively in documents 2 and 3.



Document 1



Document 2



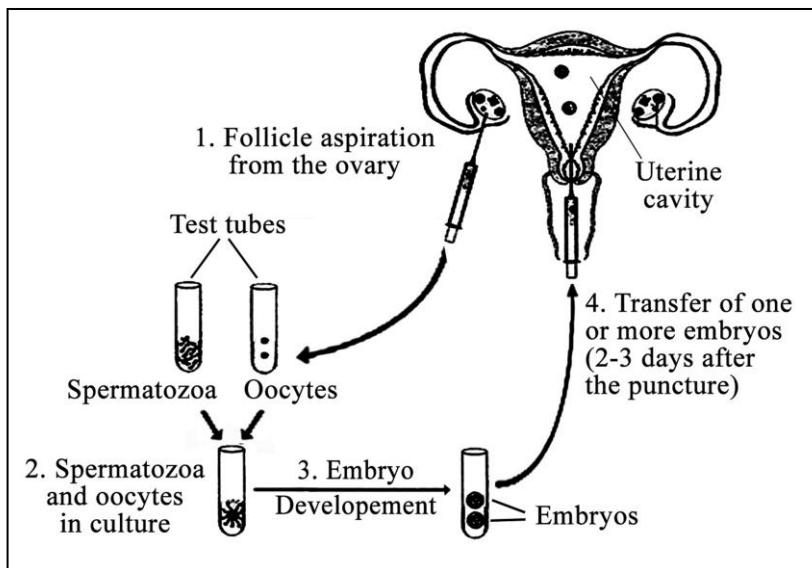
Document 3

- 3- Determine the cause of infertility of each of the women A and B.

After analyzing the results of the performed tests, the gynecologist decides to perform IVF and ET technique in order to solve the problem of one of the two infertile women.

Document 4 shows the different steps of this technique.

- 4- Describe, in a text, the IVF and ET technique.
- 5- Indicate which infertile woman A or B, the IVF and ET technique can solve her problem. Justify the answer.
- 6- Formulate a hypothesis explaining the probable origin of infertility in the second woman.



Document 4

Part of the ex.	Exercise 1 Dysuria	Grade 5 pts
1	<p>The difficulty of urinating in the twins is due to the presence of urinary calculi. The result shows that the amount of active APRT enzyme necessary for the transformation of adenine into adenosinemonophosphate is null (document 1). This blocks the transformation and leads to the accumulation of adenine and its elimination in high amounts in the urine, $40 \text{ mg} > 1.5 \text{ mg}$ (in control).</p> <p>The absence of APRT provokes the formation of dihydroxyadenine in high amounts (not detected) forming calculi leading to urinary difficulties in the twins.</p>	1
2	<p>Hypothesis :</p> <p>The disease is due to a recessive allele carried by the parents.</p> <p>The disease is due to congenital malformation.</p> <p>The disease is related to the mutation of the gene coding for APRT in the twins.</p> <p>The disease is due to a chromosomal aberration</p>	1
3	<p>Individuals IV9 and IV10 suffer from dysuria and descend from normal parents III7 and III8. So, the allele responsible for the disease is carried by the parents but it is masked. Therefore, the allele responsible for the disease is recessive, whose symbol is d, with respect to the normal allele whose symbol is N.</p>	3/4
4	<p>If the gene is located on the non-homologous part of Y, then the disease is transmitted from the father to son, but the male IV10 is diseased while his father is normal. Therefore, the gene is not located on the non-homologous part of Y.</p> <p>If the gene is located on the non-homologous part of X, then the diseased female IV9 having 2X chromosomes should carry 2 alleles for the disease. She should inherit one allele from each parent. So, her father III8 should be carrying the allele responsible for the disease and would be sick but this is not the case. Therefore, the gene is not located on the non-homologous part of X.</p> <p>If the gene is located on the homologous part of X and Y, then parent III8 must be sick and his genotype must be X^dY^d in order to give his daughter IV9 X^d and his son IV10 Y^d. But he is not sick .Therefore, the gene is not located on the homologous part of X and Y.</p> <p>Therefore, this gene is not carried by a sex chromosomes.</p>	3/4
5	<p>The possible genotypes of I1 is N/N or N/d since the normal allele is dominant and can be expressed in the homozygous or heterozygous state.</p> <p>The genotypes of III8 is N/d since the diseased twins IV9 and IV10 who exhibit the recessive phenotype have genotype dd. The recessive allele is only expressed in the homozygous state. They have surely inherited one allele for the disease d from their father III8 and since he is normal he has the allele N.</p>	1
6	<p>Since the gene is carried by an autosome and it has only two allelic versions then the presence of three different amounts of APRT, 100%, 50% and 0% shows the presence of three molecular phenotypes indicating codominance.</p>	1/2

Part of the ex.	Exercice 2 AIDS and Treatments	Grade 5 pts
1	During the asymptomatic phase, the concentration of T4 cells/mm ³ of blood increases from 550 up to 800 just within 12 months after the contamination. On the contrary, this amount decreases from 800 to 200 at the 60 th month, the beginning of the appearance of opportunistic diseases until it becomes nil at the 84 th month.	1/2
2	The cause of appearance of opportunistic diseases is the low amount of T4 cells, less than 200/mm ³ of blood.	1/2
3	<p>The presence of antibodies is observed in culture medium 1b containing B lymphocytes activated by antigen X and LT4 activated by the same antigen. On the contrary, neither antibody are produced in culture medium 1a containing only B cells activated by antigen X or culture medium 1c containing B cells activated by the antigen and activated T8 cells. This implies that the cooperation only between T4 cells and B cells is indispensable for the secretion of antibodies.</p> <p>Lysis of monkey cancerous fibroblasts is observed in medium 2b containing T8 cells and T4 cells activated by the same antigen. On the contrary, no lysis is observed in culture medium containing only activated T8 cells. This implies that cooperation between T4 and T8 cells is indispensable for cellular lysis.</p>	1
4	<p>Document 2 shows the importance of T4 cells in the activation of specific humoral immune responses whose effectors are B lymphocytes and in cellular immune responses whose effectors are T8 cells.</p> <p>Document 1 shows that the opportunistic diseases appear when the concentration of T4 cells decreases to an amount inferior to 200/mm³. Thus, this low amount of interleukin secreted is insufficient to activate proliferation of activated B and T8 cells. This blocks specific immune responses and reduces general immunity of the organism, which renders the environment favorable to the development of opportunistic diseases.</p>	3/4
5	Vaccine ensures the first contact with this antigen and triggers immunological memory. Consequently, the body, after a second contact, develops a secondary response which is more amplified, more rapid and more durable against this antigen.	3/4
6	<p>In the first treatment, the vaccine isn't effective unless the amount of T4 cells/mm³ of blood is superior to 500 T4 cells/mm³ (document 3).</p> <p>But opportunistic diseases develop only when the amount is less than 200 T4 cells/mm³ (2.5 times less than 500). The first treatment is only efficient against pneumonia, one of the multiple opportunistic diseases. Thus this treatment isn't efficient against the development of the opportunistic diseases.</p>	3/4
7	The second treatment ensures the increase in the concentration of T4 cells in blood from 250 to 480 T4 cells/mm ³ between the beginning of treatment and the 5 th year (document 4). This doesn't lead to a concentration less than 200 T4 cells/mm ³ characterizing the AIDS phase which prolongs the asymptomatic phase and delays the AIDS phase.	3/4

Part of the ex.	Exercise 3 Analgesia without morphine	Grade 5 pts
1	Morphine is agonist to enkephalin. Morphine has a shape complementary to that of enkephalin receptors. It binds to the enkephalin receptors and inhibits the release of substance P. Thus, it stops the transmission of nerve message associated with pain.	1/2
2	The duration of staying in zone P by animals that haven't received any injection or by animals that have received 6 mg/kg of endorphin is the same 5 sec. This duration is 14 times less than 72 sec which corresponds to the duration of staying in zone P of animals that have been injected by morphine. Therefore, endorphin seems not to have an analgesic effect in comparison with morphine which is a strong analgesic.	3/4
3	Hypothesis: Endorphin cannot cross the blood brain barrier to act at the level of endorphin synapses. OR Endorphin is decomposed rapidly before reaching the endorphin synapse. OR Endorphin has a short term effect.	1/2
4	Experiment 2: shows a rapid transformation of endorphin into other substances. This leads to its rapid disappearance in blood and prevents its arrival to the spinal cord and the brain. Similarly, it shows that the radioactivity remains at the level of the blood which explains the inability of endorphin to cross the blood brain barrier that is neither permeable to this substance nor to its products. This explains the ineffectiveness of endorphin as an administered exogenous analgesic.	3/4
5	The duration of staying in Zone P of animals injected by opiorphine is 62s which is less than 72s which is the duration of staying of animals injected by morphine, despite the injection of 1 mg/kg of opiorphine. This quantity is 6 times less than 6 mg/kg which corresponds to the amount of injected morphine. Thus opiorphine even in small doses is an efficient analgesic.	3/4
6	Since opiorphine seems to protect enkephalin from the effects of the enzyme NEP that is present in the cleft of enkephalin synapses, this analgesic decreases the degradation of this neurotransmitter after its fixation on its corresponding postsynaptic receptors. This leads to an increase in the concentration of enkephalin and its persistence in the synaptic cleft and on the receptors. Thus, the action of enkephalin that consists of inhibiting the transmission of pain messages is enhanced. That is why, the analgesic effect, observed in doc 2, revealed by the duration of staying, 62s, is close to that of morphine, 72s.	3/4
7	Opiorphine acts at small doses (6 times < than that of morphine) to have a certain analgesic effect. The secondary effects of opiorphine are reduced compared to that of morphine: no constipation, no addiction... Opiorphine is a natural substance secreted by the body unlike morphine which is exogenous. It acts by amplifying the natural analgesic capacities of the organism (amplifies the action of enkephalin that is also a natural endorphin), contrary to morphine which reduces them.	1

Part of the ex.	Exercise 4 Female Infertility	Grade 5 pts
1	Fertilization occurs at the level of the fallopian tubes. The uterus is the site of implantation of the embryo and the development of the fetus.	1/2
2	One of the spermatozoa that surround oocyte II blocked at metaphase II arrives to the zona pellucida. Pendunculated cells retract. The release acrosomal enzymes digest the zona pellucida. The head of the sperm binds to the oocyte membrane. Then, oocyte II gets activated and liberates the content of cortical granules thereby forming the fertilization membrane. Oocyte II continues the second division and releases the second polar body. The sperm is totally absorbed. Male and female pronuclei are formed and then they unite (karyogamy). The zygote is formed.	1
3	Document 2 shows that woman A has an amount of LH almost constant, fluctuating between 4 and 7 mU/mL without any peak on the 14 th day necessary for ovulation. However, document 3 shows that woman A has a uterus and 2 open tubes (oviducts) allowing opaque liquid to pass through. Thus this woman doesn't have any problem in her genital duct. Hence, Mrs A problem is the absence of ovulation due to the absence of LH peak. Document 2 shows that woman B has a normal variation of the amount of LH with a peak of 24 mU/mL in the middle of the cycle thereby provoking ovulation. On the contrary, document 3 shows a uterus without fallopian tubes. These tubes are invisible when radiology was performed, they didn't allow opaque liquid to pass through. Hence, the problem of woman B is blocked fallopian tubes and not hormonal.	1 1/2
4	Follicles are aspirated from ovaries; they are put in one test tube. Sperm cells are put in another one. Then, sperm cells and oocytes are cultured together. Embryos are obtained after embryo development. Two to three days after the puncture, one or more embryos are transferred to the uterine cavity.	1
5	Woman B can be treated by IVF and ET technique since this woman undergoes ovulation but her fallopian tubes are blocked, so sperm cells can't reach oocytes. This technique allows the sperm cells to fertilize the oocyte outside the woman's body.	1/2
6	Hypothesis : GnRH Receptors on pituitary cells are deficient. OR Amount of estradiol is not enough to exert a positive feedback on pituitary cells. OR Pituitary cells have a small number of estradiol receptor.	1/2

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Exercise 1 (5 points)

Hemochromatosis

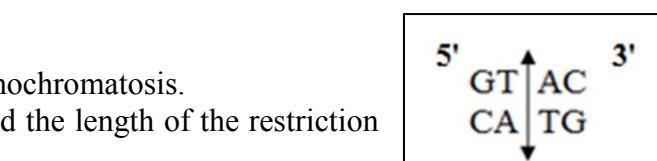
Hemochromatosis appears after the age of 40 years and is characterized by the accumulation of iron in the body. It is a recessive disease linked to the HFE gene which is located on chromosome 6. This gene has two alleles: the normal allele which codes for a membrane protein that regulates the entry of iron into the cells, and the mutated allele which codes for an abnormal protein that favors the accumulation of iron inside the cells.

Document 1 presents the partial sequence of nucleotides of the two alleles, the normal and the mutated ones.

Document 2 presents the restriction site of a restriction enzyme Rsa1.

Number of the nucleotide	1	240	250	270	278	387
Normal HFE Allele						
Mutated HFE Allele						

Document 1



Document 2

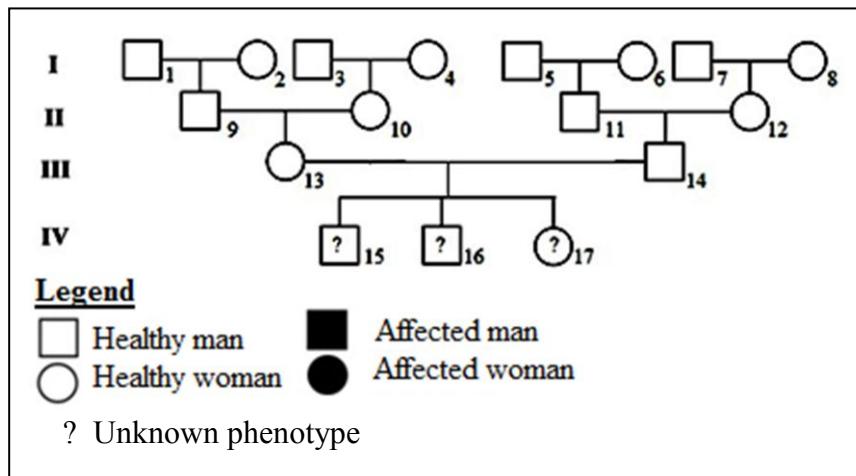
The frequency of heterozygotes in a certain population is 1/10.

A healthy couple, older than 40 years, belongs to this population. This couple would like to know if their three children, who appear healthy, have a risk to develop the disease. That's why they consult a doctor who, as a first step, establishes for this family a pedigree which is shown in document 3.

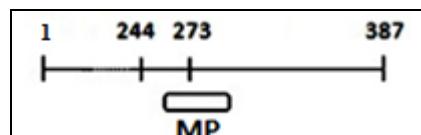
- 3- Calculate the risk for this couple, III13 and III14, to have an affected child.

As a second step, the doctor performs DNA analysis by applying the southern blot technique using the restriction enzyme Rsa1 and a radioactive molecular probe (MP) which is complementary to a specific sequence of HFE gene. This probe can fix to the whole or to a part of the recognized sequence as shown in document 4.

Document 5 shows the results obtained by this technique for certain members of this family.



Document 3



Document 4

Size of DNA fragments (bp)	III13	III14	IV15	IV16	IV17
29	=====	=====		=====	=====
114	=====	=====		=====	=====
143	=====	=====	=====	=====	=====

Document 5

- 4- Explain the absence of the 244 bp fragment in the electrophoregram presented in document 5.
5- Establish the diagnosis for each of the children in document 5.

Exercise 2 (5 points)

Conditions of LT8 Action

The Choriomeningitic leukemia virus (CML) is slightly pathogenic and infects nervous cells. In the framework of studying the immune response against the infection by this virus, two experiments were performed.

Experiment 1: different viruses are injected into mice of different strains, Y and Z. The experimental conditions as well as the obtained results are shown in document 1.

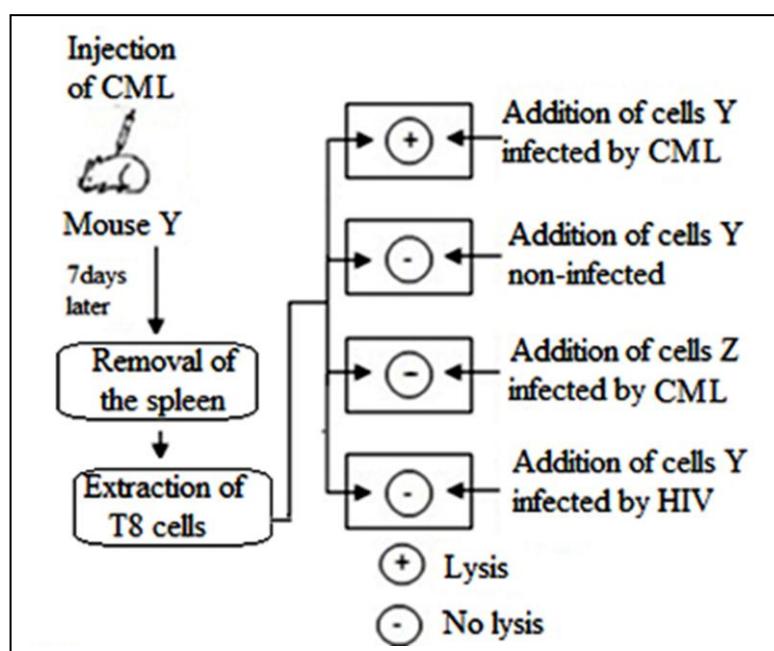
- 1- Name the specific immune response triggered against a virus and that triggered against a bacterium.
- 2- Interpret the results of experiment 1.

Experiment 2: T4 cells are cultured in the presence of macrophages and CML. The experimental conditions as well as the results are shown in document 2.

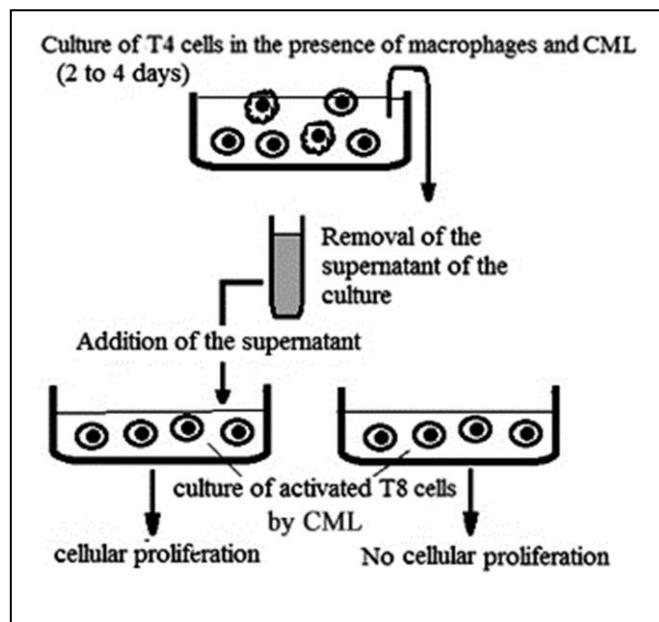
- 3- Determine the role and the mode of action of T4 cells as revealed in experiment 2.
- 4- Explain the role of macrophages in the culture of T4 cells in experiment 2.

Document 3 shows two electronographs, made at two successive times, of a target cell infected by CML in the presence of an activated T8 cell taken from experiment 2.

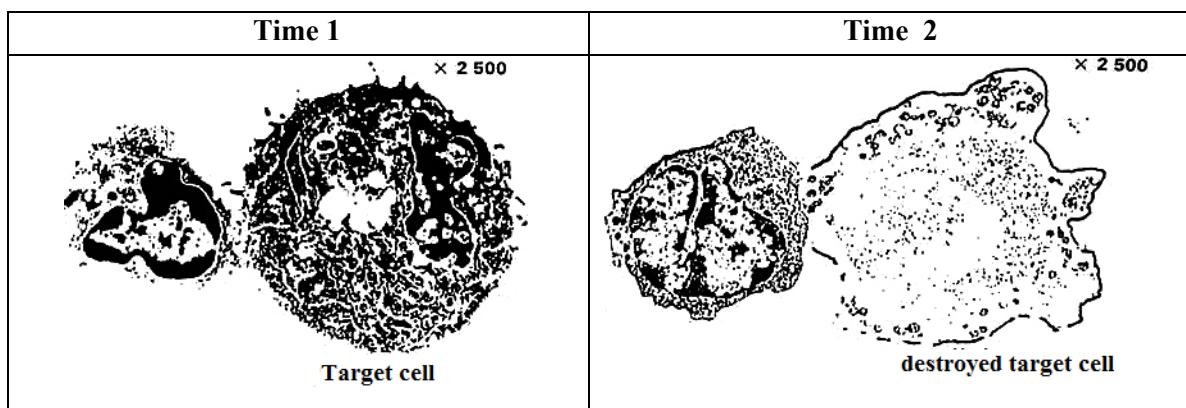
- 5- Draw a scheme showing the molecules involved in the recognition taking place between the activated T8 cell and the target cell.
- 6- Explain the mechanism shown in document 3.



Document 1



Document 2



Document 3

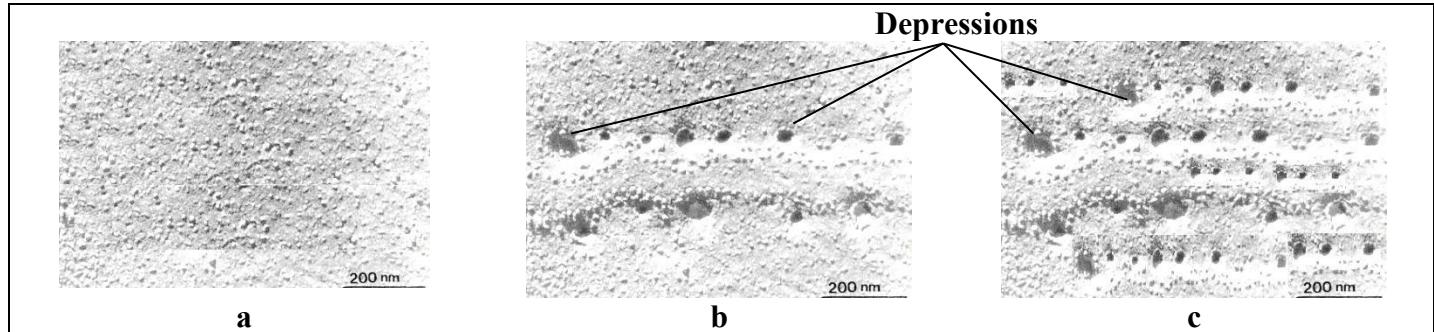
Exercise 3 (5 points)

Synaptic Transmission

Nervous messages are transmitted along the nerve fibers and traverse synapses.

In order to study the mechanisms of the synaptic transmission and the effect of certain exogenous substances, Norcuron and TEPP, the following studies were performed.

Study 1: electronographs of the external side of the presynaptic membrane were performed in different cases: case “a” where the presynaptic neuron is not stimulated, and cases “b” and “c” where this neuron is stimulated respectively with increasing intensities I_1 and I_2 which are above the threshold. The results are shown in document 1. The depressions represent the fusion of the vesicles with the presynaptic membrane.



Document 1

- 1- Justify, based on document 1, that the exocytosis of neurotransmitters at the level of a synapse is amplified with the increase of the intensity of stimulation.

Study 2: an experimental set up is used to measure the quantity of acetylcholine released in the synaptic cleft of a neuromuscular synapse as well as the amplitudes of the muscular contractions in the three cases “a”, “b” and “c” of study 1. The obtained recordings are shown in document 2.

- 2- Draw a histogram showing the variation of the quantity of acetylcholine and that of the amplitude of the muscle contraction in the three cases “a”, “b” and “c”.
3- Indicate the type of coding of the nervous message at the level of a synapse. Justify the answer by referring to document 2.

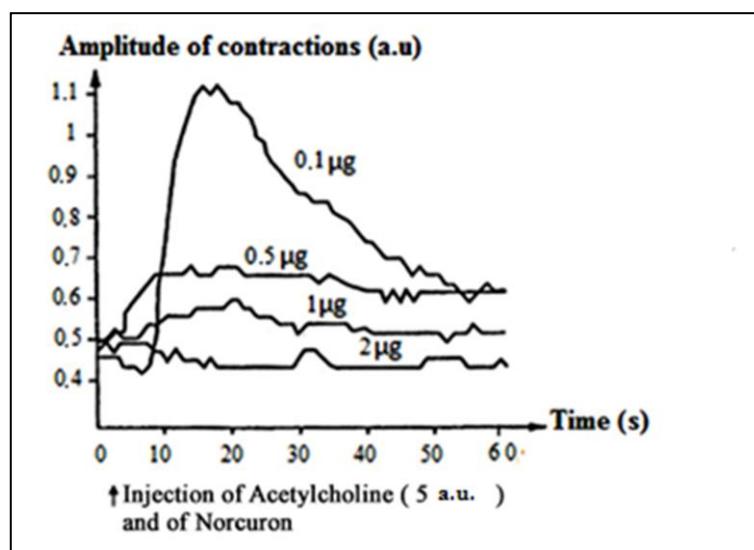
Case	a	b	c
Quantity of Acetylcholine (a.u.)	1	3	5
Amplitude of the contraction (a.u.)	0.1	0.5	1.5

Document 2

Study 3: in the synaptic cleft, 5 a.u. of acetylcholine are injected simultaneously with increasing amounts (from $0.1\mu\text{g}$ to $2\mu\text{g}$) of Norcuron, a substance that has similar molecular structure to that of acetylcholine. Document 3 shows the recordings of muscular contractions obtained for each amount of Norcuron.

- 4- What can be deduced from the results of document 3?

Study 4: TEPP is injected in insects. Symptoms characterized by a period of convulsions followed by permanent contraction of muscles are observed.



Document 3

- 5- Determine whether each of the substances TEPP and Norcuron is agonist or antagonist relative to acetylcholine.

Exercise 4 (5 points)

Infertility in a Woman

Many factors lead to sterility in women. Most of them are irreversible, but some can sometimes be solved. Mrs. A consults her doctor for a sterility problem. He asks her to measure her body temperature for a certain period of time. The obtained results of Mrs. A as well as those of a normal woman are shown in document 1.

- 1- Determine the cause of sterility of Mrs. A.

The gynecologist supposes that the sterility of Mrs. A is due, either to a lack of stimulation of the ovaries by the pituitary gland (hypothesis 1) or to the insensitivity of the ovaries to the pituitary gland secretions (hypothesis 2).

- 2- Justify the two hypotheses that are formulated by the doctor.

The doctor requests Mrs. A to perform an echography accompanied by ovarian biopsies as well as hormonal measurements.

The echography reveals two ovaries of normal size while the multiple performed biopsies show only primary follicles.

The results of the hormonal measurements of Mrs. A concerning the pituitary hormones (LH and FSH) and the ovarian hormones (estradiol and progesterone), show concentrations that are obviously lower than that of a non-sterile woman during a normal cycle.

- 3- Show that the above obtained results are insufficient to validate hypothesis 2.

The doctor then performs a treatment that consists of injecting a mixture of LH and FSH followed by LH. Estradiol measurements are performed during cycle 1 before treatment and during cycle 2 with treatment. The obtained results are shown in document 2.

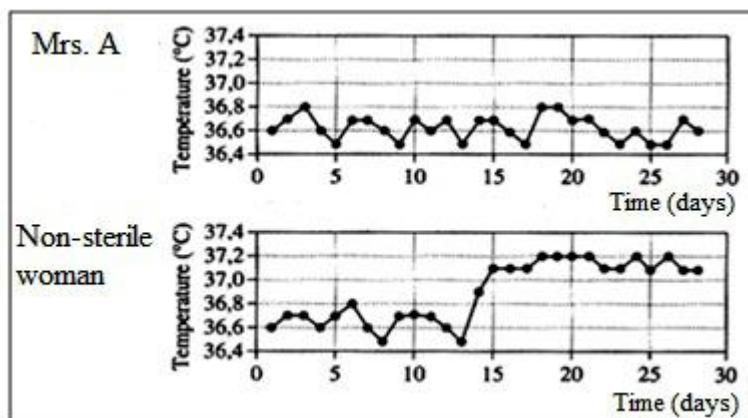
- 4- Specify which of the two hypotheses that are formulated by the doctor is validated by the above obtained results.

Following this treatment, the regular follow up of the development of the ovarian follicles gives the result presented in document 3.

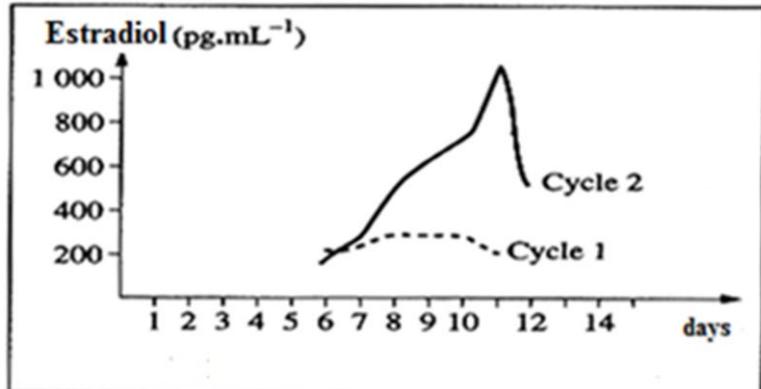
- 5- Did this treatment solve the sterility problem of Mrs. A? Justify the answer.

The doctor announces to Mrs. A that she may have fraternal twins.

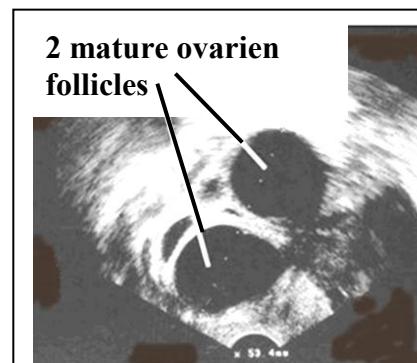
- 6- Justify this announcement concerning the possible birth of fraternal twins.



Document 1



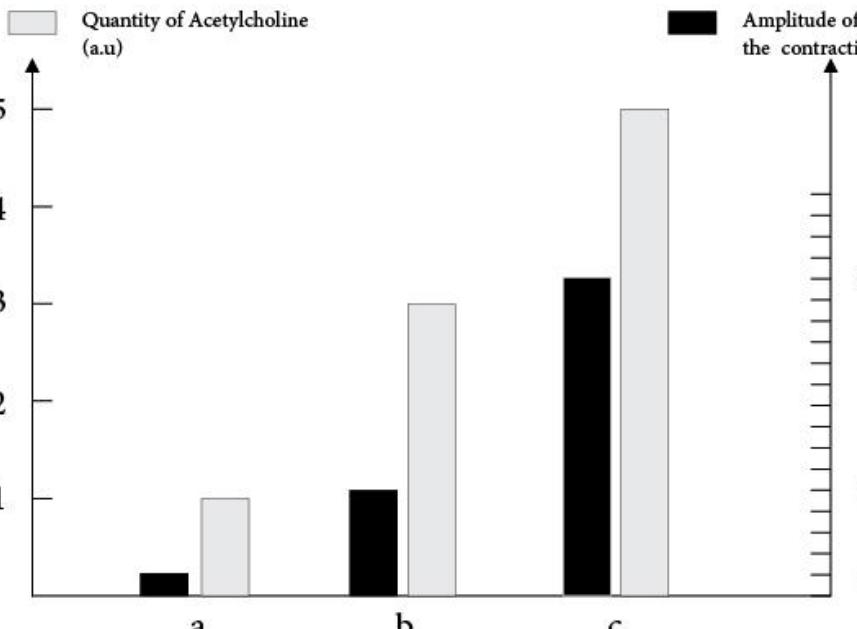
Document 2



Document 3

Q.	Exercise 1 (5 points)	Grade
1	<p>The origin of hemochromatosis is a mutation by substitution at the level of the HFE gene, Since the nucleotides of the normal allele HFE, presented in document 1, are identical to those of the mutated allele except for the nucleotide 274 where G in the normal allele is replaced by A in the mutated one. This mutation leads to the synthesis of an abnormal protein.</p>	3/4
2	<p>When treated by the restriction enzyme Rsa1, the normal allele which presents only one recognition site GTAC at the level of nucleotides 243 – 246 is cut once between T in position 244 and A in position 245, thus we obtain 2 fragments the first is of 244 bp length and the second of $387-244= 143$ pb length (3/4 pt)</p> <p>When treated by the restriction enzyme Rsa1, the normal allele which presents 2 recognition sites GTAC at the level of nucleotides 243-246 and at the level of nucleotides 272-275 is cut twice:</p> <ul style="list-style-type: none"> - between T in position 244 and A in position 245, giving the first fragment of 244 pb length, - between T in position 273 and A in position 274 which gives the second fragment $273 - 244 = 29$ bp length and the third fragment of $387 - 273 = 114$ bp length. <p>Therefore three fragments are obtained (3/4 pt)</p>	11/2
3	<p>Since each of the two parents has no family history for hemochromatosis, the frequency for each of them to be heterozygous is 1/10 (frequency in the considered population). Thus the risk for both of them to be heterozygotes is $1/10 \times 1/10 = 1/100$</p> <p>Since the allele responsible for the disease is recessive, the risk for a heterozygous couple to have an affected child is 1/4.</p> <p>Hence the risk for this couple to have an affected child is $1/100 \times 1/4 = 1/400$</p>	1/2
4	<p>The electrophoregram shows only the fragments to which the radioactive molecular probe is hybridized. Since the recognized sequence to which the MP gets fixed is localized only at the level of nucleotide 273, thus the 244 bp fragment is not hybridized and doesn't appear in the electrophoregram.</p>	3/4
5	<p>The electrophoregram shows 3 bands: band 143 bp characterizing the normal allele and bands 29bp and 114 bp characterizing the mutated one.</p> <p>The electrophoregram of child IV15 shows one thick band at the level of 143 bp corresponding to the normal allele. Hence he is healthy homozygote. (1/2 pt)</p> <p>The electrophoregram of child IV16 shows the 3 bands. Thus he is heterozygote and since the allele of the disease is recessive, he is healthy. (1/2pt)</p> <p>The electrophoregram of child IV17 shows two thick bands, 29 bp and 114 bp corresponding to the mutated allele. Thus she is recessive homozygote. She will be sick after the age of 40 years. Hence, among the three children, only the girl 17 will be sick after the age of 40 years. (1/2pt)</p>	11/2

Part of the Ex	Exercise 2 (5 points)	Grade
1	<p>The response triggered against a virus is a specific cell mediated immune response.</p> <p>The response triggered against a bacterium is a specific humoral mediated immune response.</p>	1/2
2	<p>Cells Y infected by CML undergo lysis by T8 cells which are taken from mice of the same strain Y injected by CML. Whereas, cells of the same strain Y that are not infected don't undergo lysis. This shows that T8 cells destroy only infected cells.</p> <p>On the other hand, T8 cells which are taken from mice of strain Y injected by CML lyse the cells of same stain that are infected by CML, but they don't lyse cells of a different strain Z infected by the same virus CML. This implies that T8 cells lyse only infected cells that belong to the same strain.</p> <p>Cells Y infected by CML undergo lysis by T8 cells which are taken from mice of the same strain Y injected by CML. On the contrary, cells of same strain Y which are infected by another virus, HIV, are not lysed. This implies that T8 cells destroy only the cells infected by the same virus that activated them</p>	11/2
3	<p>There's only proliferation of T8 cells when we add the supernatant taken from a culture of T4 cells which are activated by CML in the presence of macrophages. Thus activated T4 cells stimulate the multiplication of T8 cells that recognized the same antigen, by secreting a substance, chemical messengers.</p>	3/4
4	<p>The macrophage phagocytizes and digests the CML virus, the obtained peptides get associated to HLA class II molecules and expressed at the cell surface. The macrophage becomes an antigen presenting cell APC. The APC fixes to T4 cells having specific receptors to the HLA- CML peptide complex thus activating the T4 cells leading to the formation of TH cells that secrete IL-2.</p>	3/4
5	<p>Scheme of the recognition site between T8 cells and the target cell</p> <p>The diagram illustrates the interaction between a T8 cell and a target cell. The T8 cell, at the top, has a T-cell receptor (TCR) labeled in blue. This TCR is shown binding to a specific protein complex on the surface of the target cell, labeled as the "HLA- CML peptide complex". The target cell is represented by an orange shape with a black oval inside. The interaction is depicted as a close proximity between the T8 cell and the target cell.</p>	3/4
6	<p>The T8 cell performs the double recognition by fixing to the HLA- CML peptide complex of the target cell (time 1). It secretes perforin molecules that form a channel through the plasma membrane of the target cell; then it releases granzymes that penetrate the target cell through the perforin channel leading to the degradation of its DNA and consequently to its lysis (time 2).</p>	3/4

Part of the Ex	Exercise 3 (5 points)	grade												
1	<p>The depressions observed at the level of the presynaptic membrane correspond to the fusion of vesicles with the membrane, exocytosis. Since the number of depressions increases between cases b and c as the intensity of stimulation increases from I1 to I2, thus the number of vesicles undergoing exocytosis increases with the intensity of stimulation. This justifies that the exocytosis of neurotransmitters at the level of a synapse is amplified with the increase of the intensity of stimulation.</p>	3/4												
2	<p>Variation of the amplitude of the muscle contraction as a function of Acetylcholine dose.</p>  <table border="1"> <caption>Data from Figure 2</caption> <thead> <tr> <th>Category</th> <th>Quantity of Acetylcholine (a.u.)</th> <th>Amplitude of the contraction (a.u.)</th> </tr> </thead> <tbody> <tr> <td>a</td> <td>~1.0</td> <td>~0.2</td> </tr> <tr> <td>b</td> <td>~3.0</td> <td>~1.0</td> </tr> <tr> <td>c</td> <td>~5.0</td> <td>~1.1</td> </tr> </tbody> </table>	Category	Quantity of Acetylcholine (a.u.)	Amplitude of the contraction (a.u.)	a	~1.0	~0.2	b	~3.0	~1.0	c	~5.0	~1.1	1 3/4
Category	Quantity of Acetylcholine (a.u.)	Amplitude of the contraction (a.u.)												
a	~1.0	~0.2												
b	~3.0	~1.0												
c	~5.0	~1.1												
3	<p>The nervous message at the level of the synapse is coded in concentration of neurotransmitters. Since the amplitude of the muscle contraction increases from 0.5 to 1.5 a.u. when the amount of acetylcholine increases from 3 a.u up to 5 a.u. which corresponds to an increase in the intensity of stimulation from I1 to I2.</p>	3/4												
4	<p>The amplitude of the contraction increases to a maximum of 1.1 a.u within 15 s in the presence of 0.1μg of Norcuron and 5 au of acetylcholine. On the contrary, this amplitude decreases and become almost constant at 0.45 a.u when we increase the amount of injected Norcuron up to 2μg with the same injection of 5 au of acetylcholine. Thus, Norcuron inhibits the action of acetylcholine and decreases the amplitude of the muscle contractions and its action varies in parallel to its concentration.</p>	3/4												
5	<p>Since Norcuron reduces the muscle contraction while acetylcholine provokes the muscle contraction. Thus Norcuron has an opposite (reverse) effect to acetylcholine. Hence they are antagonistic substances. TEPP provokes the permanent contraction of muscles like acetylcholine. Thus it has the same effect as acetylcholine on the muscle. They are agonistic substances.</p>	1												

Part of the ex.	Exercise 4 (5 points)	Grade
1	<p>The temperature fluctuates in the 2 women around a value of 36.6°C, from day zero till day 14 of the cycle. This temperature increases abruptly on the 14th day up to 37.1°C in the non-sterile woman indicating ovulation and remains high around 37.2°C for the rest of the cycle. On the contrary in Mrs. A, and throughout the whole cycle, the temperature undergoes variations which stay always slight around a value of 36.6 °C indicating the absence of ovulation in Mrs. A what causes her sterility.</p>	1
2	<p>The pituitary gland secretes two hormones FSH and LH: FSH triggers the follicle development and LH triggers ovulation. In case where one of these two hormones is deficient, there will be no ovulation, nor formation of corpus luteum and thus no secretion of progesterone which is responsible for the increase of temperature to above 37°C. This justifies the first hypothesis.</p> <p>Similarly, if the pituitary gland secretes hormones that cannot fix on the follicular cells due to the absence of receptors, we obtain the same results in the first case. This justifies the 2nd hypothesis.</p>	1
3	<p>The echography shows ovaries whose size is normal and containing primary follicles. Thus maybe these follicles can develop in the presence of pituitary hormones if they exist or maybe these follicles are not sensitive to these hormones.</p> <p>The results of hormone measurement show low concentrations of pituitary and ovarian hormones. Thus, maybe there's no control of the pituitary gland on the ovaries or maybe there is no positive feedback of ovarian hormones on the pituitary gland what maintains the low level of pituitary hormones.</p>	3/4
4	<p>Hypothesis 2 is validated by the results of document 2 since following the injections of FSH and LH followed by LH, the level of estradiol increases from 200 pg/ml to around 1000 pg/ml indicating follicular development. Thus the ovaries are sensitive to the pituitary secretions and hence it is the levels of FSH and LH in Mrs. A that are insufficient to stimulate the ovaries. What allows the rejection of hypothesis 2 and the validation of hypothesis 1.</p>	1
5	<p>Yes, the treatment has solved the problem of Mrs.A. Since the ovaries have, starting from the primary follicles, developed into two mature ovarian follicles that may undergo ovulation releasing two oocytes II blocked at metaphase II that have the possibility to be fertilized.</p>	1/2
6	<p>The birth of fraternal results from two different zygotes formed by the fertilization of two oocytes II issued from the two mature follicles presented in document 3 by two different sperm cells.</p>	3/4

Exercise 1 (5 points)**Cystic Fibrosis**

Certain mutations which are at the origin of genetic diseases may protect against other diseases. In order to clarify this observation, the following studies are performed.

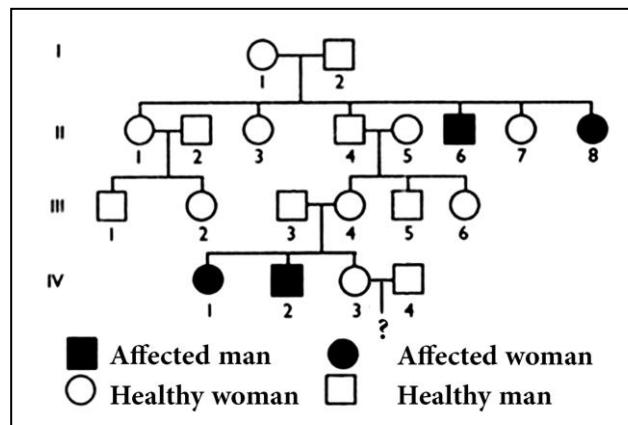
Study 1:

Cystic fibrosis is a severe disease manifested by respiratory and digestive troubles.

The origin of the disease is a mutation of the gene coding for the protein CFTR leading to the modification of amino acid 508.

The protein CFTR is present in the plasma membrane of the cells. It allows the exchange of Cl^- ions and therefore, the exchange of water. The alteration of this protein blocks the passage of the Cl^- ions and water leading to an increase in the viscosity of the mucus, particularly at the level of the lungs and the digestive tract. In a well-defined population, 1 out of 20 persons are heterozygous.

Document 1 shows the pedigree of a family whose some members are affected by cystic fibrosis.

**Document 1****1- Pick out:**

- 1-1** The origin of cystic fibrosis.
- 1-2** The consequences of the mutation at the cellular level.
- 2-** Indicate if the allele responsible for the disease is dominant or recessive. Justify the answer.
- 3-** Determine the chromosomal localization of the gene responsible for cystic fibrosis.
- 4-** Specify the genotype of each of the individuals II8, III3, IV2 and IV3.
- 5-** Determine the risk for couple IV3 and IV4 to have a child affected by cystic fibrosis.

Study 2:

Three lots of mice are genetically modified by integrating the human gene coding for CFTR protein in their genome. The mice of lot 1 are homozygous for the normal allele, the mice of lot 2 are homozygous for the mutated allele, and the mice of lot 3 are heterozygous.

Salmonella typhi bacteria have been ingested by the mice of the three lots. The number of intestinal cells infected by *Salmonella typhi* is estimated. The results are shown in document 2.

The infection by this bacterium leads to Typhoid fever which is manifested by a very serious inflammation of the digestive tract leading to death in the absence of any antibiotic treatment.

- 6-** Justify, referring to what precedes, that some mutations which are at the origin of genetic diseases may protect against other diseases.

	Lot 1	Lot 2	Lot 3
Mice	Homozygous for the normal allele	Homozygous for the mutated allele	Heterozygous for this gene
Results	Numerous infected intestinal cells	No infected intestinal cells	Few infected intestinal cells

Document 2

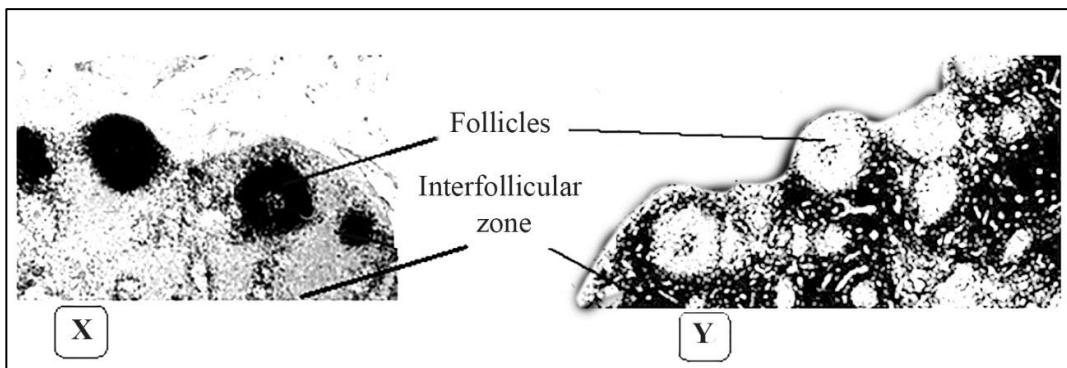
Exercise 2 (4 points)

Hypertrophy of Lymph Nodes

A temporary hypertrophy (swelling) of the lymph nodes is observed in an individual infected by an antigen like the tetanus toxin. In order to better understand the mechanisms involved in this hypertrophy, the following experiments are performed.

Experiment 1: The constituents of the lymph nodes of this individual are studied by using radioactive markers. Microradiographs are then performed.

The radioactive labeled zones appear in black on the microradiographs.



Document 1

Document 1 shows the results of labeled radioactive B lymphocytes (X) and of labeled radioactive T lymphocytes (Y).

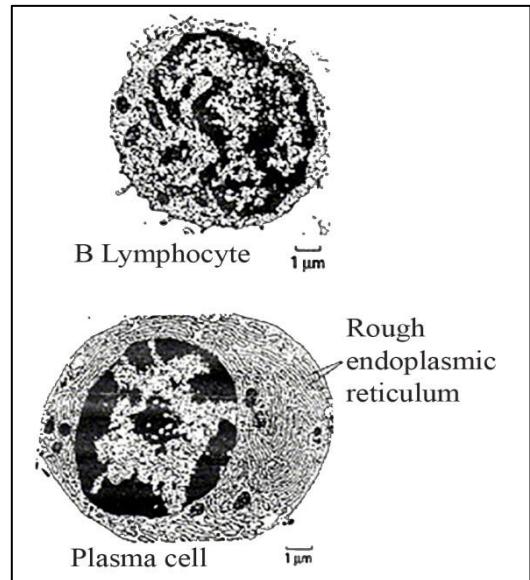
- 1- Deduce the localization of each lymphocyte population at the level of lymph nodes.

Document 2 shows microphotographs of the cells identified in the lymph nodes of the individual who is infected with tetanus toxin.

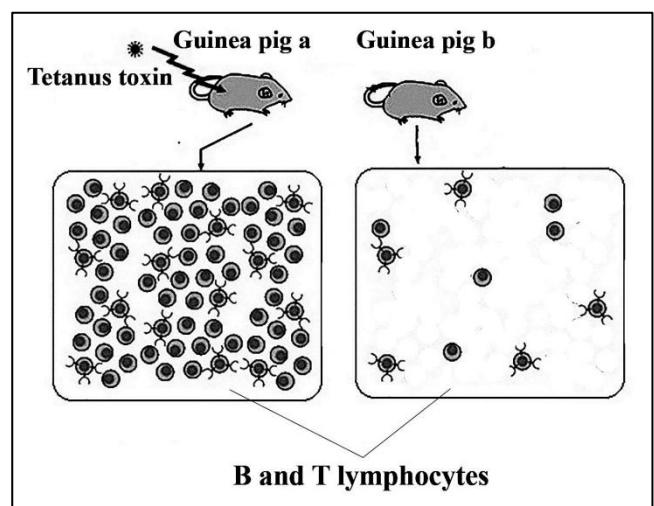
- 2- Specify the type of the immune response triggered against this antigen and revealed in document 2.
- 3- 3-1- Name the molecules secreted by this plasma cell.
3-2- Explain how the plasma cell is a cell adapted to the secretion of these molecules.

Experiment 2: cells are extracted from the lymph nodes of a guinea-pig (a) which is injected with tetanus toxin and from the lymph nodes of a healthy guinea pig (b). They are then purified to obtain only B and T lymphocytes. The results are schematized in document 3.

- 4- Interpret the results presented in doc 3.
- 5- Justify, referring to what precedes, the temporary hypertrophy of the lymph nodes observed in this individual.
- 6- Explain the role of TL involved in the immune response revealed in document 2.



Document 2



Document 3

Exercise 3 (5 points)

Effect of An Insecticide

Farmers use organophosphorous insecticides to kill insects. Some of these insecticides such as pyrethrum alter the function of the nervous system thus blocking respiration leading to death by asphyxia. In fact, the respiratory movements are ensured by contractions followed by relaxations of the respiratory muscles. In order to better understand the mode of action of pyrethrum, the following experiments are performed.

Experiment 1: the gastrocnemius muscle of a frog and the nerve connected to it are immersed in a physiological medium. An effective stimulation of intensity I is applied on this nerve in the presence and absence of pyrethrum. For each stimulation, the amplitude and the duration of the muscle contraction are recorded. The results are presented in document 1.

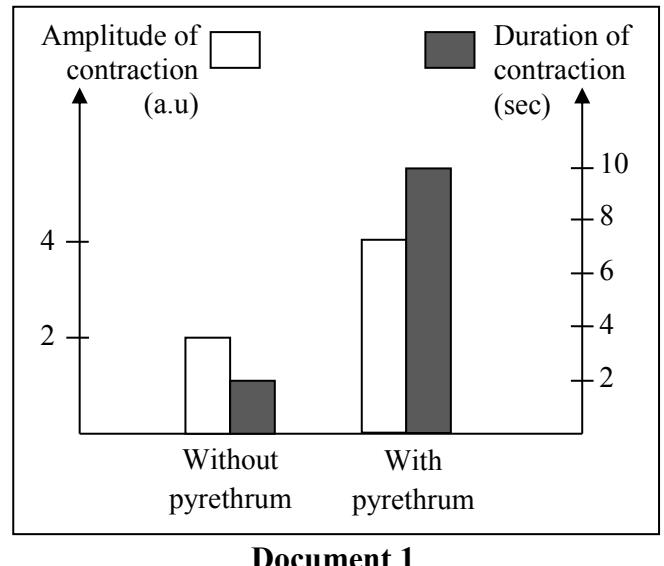
- 1- Represent in a table the results of document 1.
- 2- 2-1- compare the obtained results.
2-2- what can you conclude?
- 3- Formulate two hypotheses explaining the mode of action of pyrethrum.

Document 2 shows the ultrastructure of the neuromuscular synapse.

- 4- Identify, which of the structures 1, 2, or 3 corresponds to the presynaptic neuron.

Experiment 2: a micro-drop of pyrethrum marked by radioactive phosphorus is injected at the level of the neuromuscular synapse. Concentrated radioactivity is observed at the level of the synaptic cleft. Profound analyses show that the pyrethrum molecules are associated with acetylcholinesterase, an enzyme that degrade acetylcholine molecules that are fixed on the receptors of the postsynaptic membrane.

- 5- Explain, referring to what precedes, how can pyrethrum lead to death by asphyxia.



Document 1



Document 2

Exercise 4 (6 points)

What Determines the LH Peak?

The secretion of the hormone LH by the pituitary gland varies in a cyclic manner. In a woman having a 28-days cycle, the LH peak on the 13th day of the cycle triggers the ovulation of the oocyte II blocked at metaphase II. Searching for the factors that determine the LH peak, different experiments are performed on female mammals.

Series of experiments 1: different treatments are performed on 4 lots of adult female rats, then the level of the secreted LH is measured.

Lot 1: the female rats are not subjected to any treatment. There is secretion of LH.

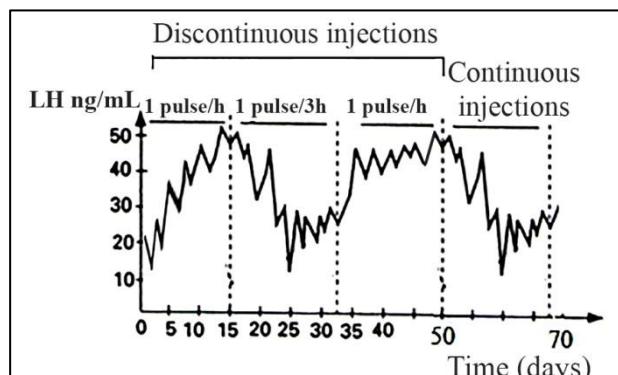
Lot 2: the female rats are subjected to the lesion of the hypothalamus. There is no secretion of LH.

Lot 3: the female rats are subjected to ablation of the pituitary gland followed by the graft of the pituitary gland in the anterior chamber of the eye. There is no secretion of LH.

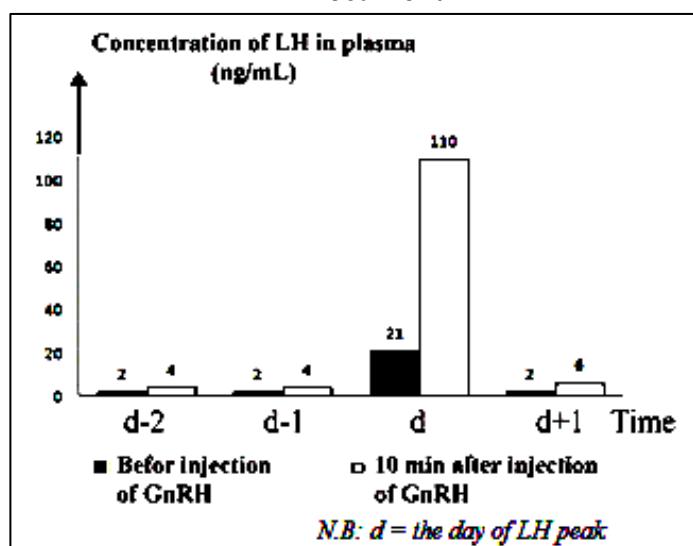
Lot 4: the female rats are subjected to ablation of the pituitary gland followed by the graft of this gland in an area connected to the pituitary duct. There is secretion of LH.

- 1- Interpret the results of the series of experiments 1.

Experiment 2: In a female macaque, the arched nucleus of the hypothalamus has been destroyed and the secretions of FSH and especially of LH have dropped. This female is injected by GnRH (substance extracted from the hypothalamus) in a continuous manner and in a pulsatile manner at two different frequencies using an automatic micropump. The obtained results are represented in document 1.



Document 1



Document 2

Experiment 3: female rats are injected on daily basis of the cycle at 16:00 o'clock with the same quantity of GnRH. The plasma level of LH is measured immediately before the injection and ten minutes after the injection of GnRH. The results are presented in document 2.

- 3- What can you deduce concerning the sensitivity of the pituitary gland to GnRH?

Experiment 4: the same number of pituitary LH secreting cells extracted from female rats in the morning of day (d-1) is incubated in vitro. At the end of the incubation, the quantity of LH in the medium is measured. The experimental conditions as well as the results are presented in document 3.

Quantity of LH (μ g)	Pituitary cells with estradiol	Pituitary cells without estradiol
	With GnRH	Without GnRH
3.3	3.3	0.7
< 0.2	< 0.2	< 0.2

Document 3

- 4- Name the structures that secrete estradiol during the sexual cycle.
 5- 5-1- Analyze the results of document 3.
 5-2- What can you conclude?
 6- Explain how the peak of LH is triggered.

الاسم:	مسابقة في مادة علوم الحياة
الرقم:	المدة: ثلاثة ساعات

Exercise 1 (5 points)

Genetics and Cancer

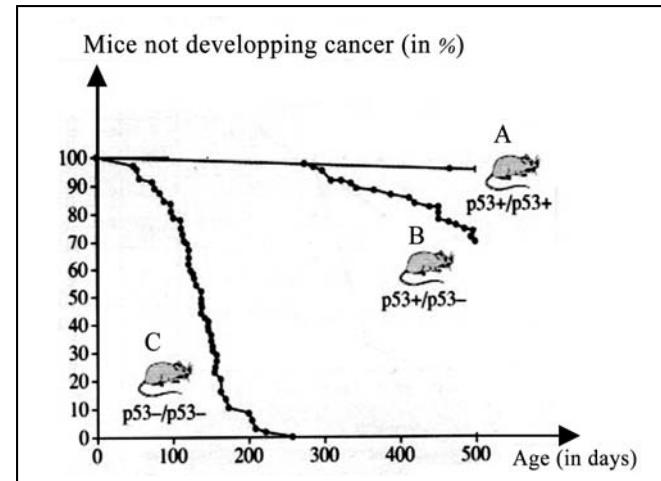
Billions of cells of the organism, having a limited lifespan, are continuously renewed due to cellular divisions controlled by a system of regulation. The dysfunction of this system of regulation can produce a clone of cells, thus forming a tumor. This latter, is benign as long as it is controlled but it can evolve into malignant tumor: cancer.

The cancerous cells lose their contact with their neighboring cells; they tend to migrate and colonize in other tissues: this is metastasis.

1- Pick out from the text :

- 1-1- The cause of the appearance of tumor.
- 1-2- The definition of metastasis.

In order to better understand the origin of this type of cancer, several studies have been carried out on the gene p53 coding for the protein p53. This protein intervenes in the regulation of the cell divisions.



Study 1:

The development of this type of cancer is studied in three lots of mice as a function of their genotypes concerning the gene p53. Two alleles of this gene, p53+ (normal) and p53- (mutated) are only considered. The results of this study are shown in document 1.

2- Interpret the obtained results shown in document 1.

Document 2 shows the nucleotides sequence of the non-transcribed strand of each of the two alleles involved in this study.

3- Specify the type of mutation at the origin of this cancer.

4- Explain how the modification in the nucleotide sequence leads to the appearance of this type of cancer.

Study 2:

Researchers have studied the mutations detected in three groups of individuals: individuals of group 1 are non-smokers and non-alcohol consumers, those of group 2 are smokers but non-alcohol consumers and those in group 3 are smokers and alcohol consumers. The results are shown in document 3.

5- Show that the consumption of tobacco is a risk factor for cancer.

6- Justify the high number of individuals affected by cancer in the case of the simultaneous consumption of alcohol and tobacco.

Gene p53	Sequence of the nucleotides of the non-transcribed DNA strand	
N° of codon	↓244	↓250
Allele p53+	GGC GGC ATG AAC CGG AGG CCC	
Allele p53-	GGC GGC ATG AAC CGG AGT CCC	

Document 2

Mutations detected at the level of gene p53	Number	Group 1	Group 2	Group 3
	Type	Substitution	Substitution	Substitution, deletion and insertion
Result : Number of individuals affected by cancer		Low	Moderate	High

Document 3

Exercise 2 (5 points)

Roles of Macrophage

The monocytes circulate in the blood and can migrate to the tissues where they become macrophages.

- 1- Indicate the origin of monocytes.

In order to study the mode of action of macrophages and their cooperation with certain cells of the immune system, the following experiments are performed.

Experiment 1 :

Cells are extracted from the ganglia of a guinea pig which is immunized against antigen X. T4 lymphocytes and macrophages are isolated and placed in different culture media. The experimental conditions and the results are shown in document 1.

- 2- Determine the conditions indispensable for the proliferation of T4 lymphocytes.

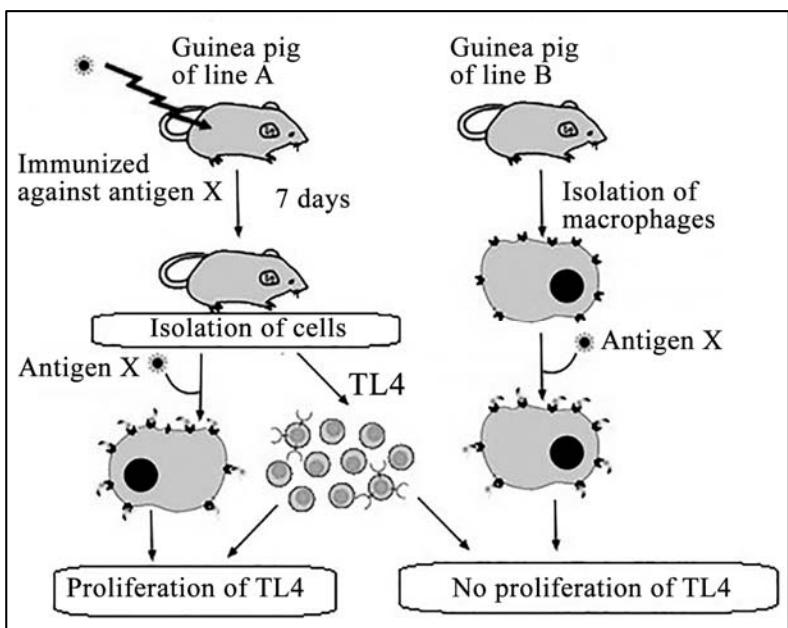
Culture medium	Conditions of the culture	Results
1	T4 lymphocytes and antigen X	No proliferation of T4 lymphocytes
2	T4 lymphocytes and macrophages	No proliferation of T4 lymphocytes
3	T4 lymphocytes, macrophages and antigen X	Proliferation of T4 lymphocytes
4	T4 lymphocytes	No proliferation of T4 lymphocytes

Document 1

Experiment 2 :

An experiment is performed on two different strains of guinea pigs, A and B. The experimental conditions as well as the results are shown in document 2.

- 3- Indicate the condition indispensable for the proliferation of T4 lymphocytes shown in this experiment. Justify the answer.



Document 2

Experiment 3:

Macrophages are incubated with the same antigen X labeled with radioactive ^{131}I odine isotope. At phase I, radioactivity is detected inside the macrophage, and in phase II a rapid degradation of antigen X is noticed. After some time 80% of radioactivity is detected in the culture medium in the form of ^{131}I odine isotope linked to peptides, while the remaining 20% are found attached to the cell surface.

- 4- Draw out the role of macrophages shown at phase I of experiment 3.
- 5- Explain the results obtained at phase II in experiment 3.
- 6- Explain the mode of action of the macrophages that permits the proliferation of T4 lymphocytes.
- 7- Specify the consequence of the absence of the macrophages on the specific immune responses.

Exercise 3 (5 points)

Reflex Control

In order to understand how the myotatic reflex can be controlled, many studies are carried out on different fibers, the sensory fibers and motor fibers involved in this reflex.

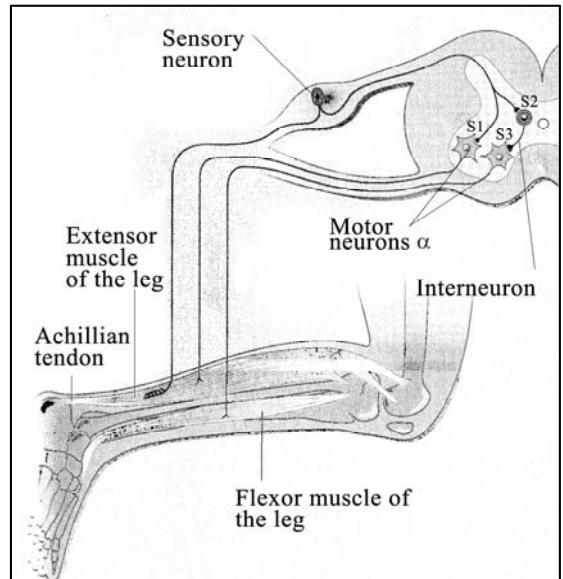
- 1- Define the myotatic reflex.

Study 1:

The extensor muscle is stretched and the sensory and the motor messages are recorded in two different situations: in the first situation the flexor muscle is at rest, and in the second situation the flexor muscle is strongly stretched.

Document 1 shows the concerned muscles with their nervous connections.

Document 2 shows the experimental conditions as well as the obtained recordings during the same duration in the two situations.



Document 1

- 2- Compare the neuronic circuits innervating these antagonistic muscles involved in this reflex.

		Situation 1 Flexor muscle at rest	Situation 2 Flexor muscle strongly stretched
Electrical recordings	Fiber issued from the neuromuscular spindle of the extensor muscle		
	Fiber issued from the motor neuron α innervating the extensor muscle		_____
	Fiber issued from the motor neuron α innervating the flexor muscle	_____	

Document 2

- 3- Determine, based on the results of the first situation (doc 2), the contracted muscle and the relaxed one.
- 4- Indicate the role of the interneuron.
- 5- Explain the role of the motor neuron α of the extensor muscle in the second situation.

Study 2

The extensor muscle is stretched and the activities of the sensory fiber and the motor fiber of this muscle are recorded with or without the voluntary contraction of the flexor muscle. The results are presented in document 3.

		Stretching of the extensor muscle	
		Flexor muscle at rest	Voluntarily contracted flexor muscle
Electrical recordings	Fiber issued from the neuromuscular spindle of the extensor muscle		
	Fiber issued from the motor neuron α innervating the extensor muscle		

Document 3

- 6- Deduce the action of the superior nerve centers on the studied reflex.

Exercise 4 (5 points)**Determination of Ovulation**

The first phase of the menstrual cycle is marked by important development of the follicles. Out of these follicles, only one becomes mature and ready for ovulation. In order to better understand the factors and the mechanisms that cause ovulation to occur, the following studies are performed.

Study 1: the variation of the level of estradiol, an ovarian hormone, is monitored during a sexual cycle. The results are shown in document 1.

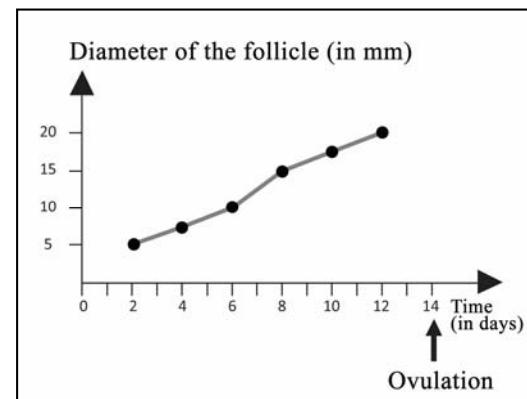
Time(days)	0	4	10	12	14	18	21	28
Level of estradiol (pg/mL)	60	75	150	240	75	100	150	60

Document 1

- 1- Draw the curve which represents the variation of the level of estradiol as a function of time.

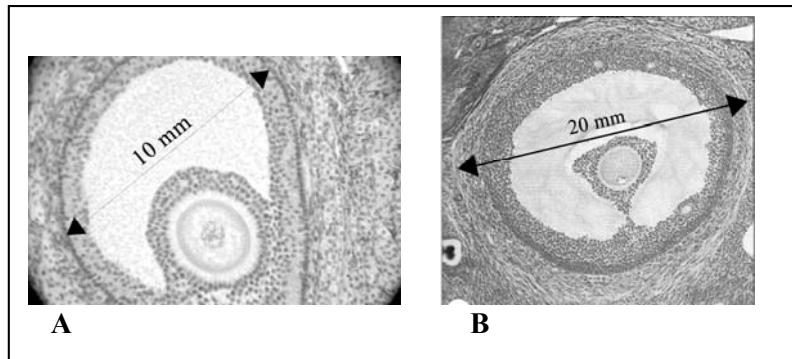
Document 2 shows the variation of the diameter of a cavitary follicle during maturation until ovulation. Note that the diameter of the follicle is proportional to the number of follicular cells.

- 2- Define ovulation
- 3- Explain how the transformation of the follicle (doc 2) leads to the variation of the level of estradiol during the follicular phase (doc 1).
- 4- Name the follicle represented in each of the photos A and B. Justify, by referring to document 2, the answer.

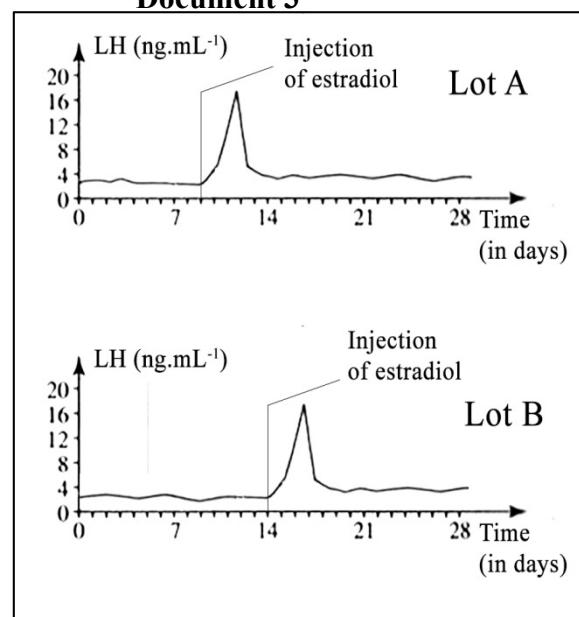
**Document 2****Study 2**

Two lots A and B of female monkeys which are subjected to the ablation of their ovaries, receive a continuous injection of moderate level of estradiol, which keeps LH at a low level.

The monkeys of each lot receive later a unique injection of a high dose of estradiol, on a specific day. The variation of the LH level is monitored in these monkeys. The results are represented in document 4.

**Document 3**

- 5- Interpret the results in document 4.
- 6- Explain that the stimulus leading to ovulation is given by the ovarian follicle.

**Document 4**

Q.	Exercise 1 Genetics and Cancer	mark
1-1	The dysfunction of this system of regulation can produce a clone of cells, thus forming a tumor.	1/4
1-2	The cancerous cells lose their contact with their neighboring cells; they tend to migrate and colonize in other tissues: this is metastasis.	1/4
2	In lot A of genotype p53+/p53+, the percentage of mice that don't develop cancer stays constant at 100% during 500 days. However, the percentage decreases from 100% to 70% in mice of lot B of genotype p53+/p53- between day 280 and day 500. This shows that the allele p53- favors the development of cancer when it's present in one copy. However, in lot C having the genotype p53-/p53-, the percentage of mice not developing cancer begins to diminish from 100% on day 50 to null on day 250 days which is less than day 280 corresponding to the appearance of cancer in lot B. Therefore, the allele p53- accelerates the appearance of cancer and its action is amplified when it exists in 2 copies.	1
3	The origin of cancer is a mutation by substitution of gene p53. Since the nucleotides of alleles are identical except at the level of the 3rd nucleotide of codon 249 where the G nucleotide in the allele p53+ is substituted by the nucleotide T in the allele p53-.	1
4	The mutation by substitution at the level of codon 249 leads to an amino acid different than that translated by the normal allele. This modification of amino acid has as a consequence the synthesis of an abnormal and non-functional protein. As a consequence, the regulatory system of cellular divisions becomes nonfunctional and the cells divide in an uncontrollable manner, producing thus a clone of cells forming tumors.	1
5	Document 3 shows that in the smokers, the number of mutation by substitution is 5 and the number of individuals affected by cancer is moderate; whereas, the non-smokers present 3 ($3 < 5$) mutations by substitution that limits the development of cancer in them. This shows that tobacco is a risk factor for cancer.	3/4
6	In the smokers and consumers of alcohol, the number of mutation is 7, a value greater than 5, which is the number of mutation in the smokers (and also greater than 3, in non-smokers and non-alcohol consumers). In addition to the increase in number, the mutations exist in different types: deletions and insertions in addition to substitutions, the only type mutations revealed in the two groups 1 and 2. Since the mutations at the level of gene p53 is at the origin of tumors, the increase in the number of mutations as well as the occurrence of new types of mutations, favor the appearance of cancer and therefore justifies the high number in individuals affected by cancer which are smokers and alcohol consumers.	3/4

Q	Exercise 2(5 Points)	Role of Macrophages	Mark
1-	Bone marrow		1/4
2-	Proliferation of T4 lymphocytes takes place only in culture of medium 3 in the presence of T4, macrophages and antigen X. Hence the proliferation of T4 lymphocytes necessitate the association or cooperation between T4 and macrophages in the presence of antigen X.		1
3-	The macrophages and the T4 cells must descend from the same strain. Since there is no proliferation of T4 lymphocytes when T4 cells of strain A are incubated with macrophages from another strain B. However proliferation takes place when T4 cells and macrophages previously in contact with an antigen X that descend from the same strain A.		1
4-	Phagocytosis		1/2
5-	At phase II of the experiment, 20% of radioactivity is detected on the surface of the cell, because a part (80%) of the degraded radioactive protein is eliminated out of the cell; The remaining 20% is degraded into peptides that are associated with MHC II on the surface of macrophages.		1/2
6-	The macrophages that are transformed into APC present the non-self-peptide associated with MHC II on its surface. So TCR of T4 lymphocytes bind to this complex and the T4 becomes activated.		3/4
7-	The induction of specific immune response ceases because the activation of T4 lymphocytes necessitate its binding to APC. So in the absence of activated T4 lymphocytes, no more secretion of interleukin 2 takes place which is responsible for launching the specific cell mediated immune response. Moreover, no interleukin 4 secretion takes place which is responsible for launching of the specific humoral immune response.		1

Q	Exercise 3 (5 points)	Control of Reflex	Mark
1	The myotatic reflex is the contraction of the muscle due to its own stretching		1/2
2	The two circuits posses the same sensory neuron . Each of the two circuit have a unique motor neuron α . The circuit of the flexor muscle possesses an interneuron between its sensory neuron and its motor neuron α . However the circuit of the extensor muscle does not have interneuron . The number of synapses in the extensor muscle(1) circuit is less than that in the circuit of the flexor muscle (2).		1

	<p>3 The muscle which receives the excitatory nerve message, contracts. Since the fiber issued from the motor neuron α which innervates the extensor muscle shows the propagation of the nerve message of frequency equals to 15 A.Ps. Hence the muscle which contracted is the extensor muscle.</p> <p>The muscle which does not receive any nerve message does not contract. Since the fiber issued from the α motor neuron and which innervates the flexor muscle shows resting potential (or absence of action potential). Hence this muscle stays at rest.</p>	1
4	The interneuron plays an inhibitory role on the α motor neuron of flexor muscle.	1/2
5	The recordings at the level of the fibers issued from the motor neuron of the extensor muscle shows disappearance of the frequency of action potential (previously shows recording at the level of the same fiber in the absence of contraction of the flexor muscle). Although there exists sensory nerve message of frequency of 8 APs at the level of the sensory neuron, and this is explained by the spatio-temporal summation of an excitatory nerve message coming from sensory neuron and an inhibitory message coming from the flexor muscle resulting in the disappearance of the recording (algebraic sum).	1
6	<p>Only the frequency of APs of the fiber issued from the motor neuron innervating the extensor muscle decreases from 15 to 3 APs after the voluntary contraction of flexor muscle. (On the contrary, sensory nerve message stays at the same frequency of 8 APs in the two cases with and without voluntary contraction of flexor muscle) .</p> <p>Thus the superior nerve center inhibits only the motor nerve message at the level of the motor neuron innervating the extensor muscle. This results in decreasing the stimulation of the muscle consequently its contraction and attenuates the myotatic reflex. .</p>	1

Q.	Exercise 4 (5 Points)	Determining Ovulation	Mark
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1	<p>level of estradiol (in pg/mL)</p> <table border="1"> <thead> <tr> <th>Time (days)</th> <th>Level of Estradiol (pg/mL)</th> </tr> </thead> <tbody> <tr><td>0</td><td>60</td></tr> <tr><td>5</td><td>70</td></tr> <tr><td>10</td><td>150</td></tr> <tr><td>12</td><td>240</td></tr> <tr><td>15</td><td>70</td></tr> <tr><td>20</td><td>150</td></tr> <tr><td>28</td><td>50</td></tr> </tbody> </table> <p>Variation of the level of estradiol as a function of time</p>	Time (days)	Level of Estradiol (pg/mL)	0	60	5	70	10	150	12	240	15	70	20	150	28	50	11/2
Time (days)	Level of Estradiol (pg/mL)																	
0	60																	
5	70																	
10	150																	
12	240																	
15	70																	
20	150																	
28	50																	
2	Ovulation is the liberation or release of oocyte II from the mature ruptured graafian follicle into the pavilion duct.	1/4																
3	The increase in the diameter of the follicle from 5mm at day 2 of the follicular phase into 20 mm at day 12 of the same phase is followed by an increase in the number of follicular cells. Knowing that these cells are responsible for estradiol secretion, as the number of these cells increases, the estradiol level increases from 60 pg/ml into 240 pg/ml between day 0 and 12 of the follicular phase (as shown in document 1)	3/4																
4	<p>A= cavitary (tertiary) follicle. Since the diameter is 10 mm which corresponds to a follicle at day 6 of the cycle during its development.</p> <p>B= Graafian follicle. Since the diameter is 20 mm which corresponds to a follicle at day 12 of a follicular phase that's a mature follicle tends to ovulate.</p>	1																
5	In both ovariectomized female monkeys of lot A and B a peak of LH of 16 ng/ml at day 12 and day 17 for the females that are subjected respectively at day 9 (lot A) and day 14 (lot B) to a unique injection of high dose of estradiol. However this level of LH is constantly maintained about 3 ng/ml following the injection of a continuous moderate level of estradiol. This shows that a high quantity of estradiol favors the peak of LH.	3/4																
6	The ovary secretes a high concentration of estradiol (at the level of threshold) that stimulates by positive feedback the pituitary gland. Hence the peak of LH is responsible for ovulation. Moreover the follicle undergoing mature emits a stimulus, high dose of estradiol that favors its rapturing corresponding to ovulation.	3\4																

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ثلاثة ساعات

Exercise 1 (5.5 points)

Diagnosis of Galactosemia

Galactosemia is a genetic disease which results from a deficiency in the enzyme transforming galactose to glucose. Several days following the consumption of milk or milk products, the following clinical signs appear: vomiting, diarrhea, On the long term, infants would show retarded growth and later they may have mental retardation.

Mr. and Mrs. G are expecting a child. Mrs. G is worried because several members in her family are affected by this disease as shown in the pedigree presented in document 1.

- Indicate if the allele responsible for the disease is dominant or recessive. Justify the answer.
- Determine the chromosomal location of the gene responsible for this disease.
- Specify the possible genotype(s) of Mrs. G and individual IV-4.

Worldwide, the probability of individuals to be heterozygous for the gene responsible for this disease is 1/100.

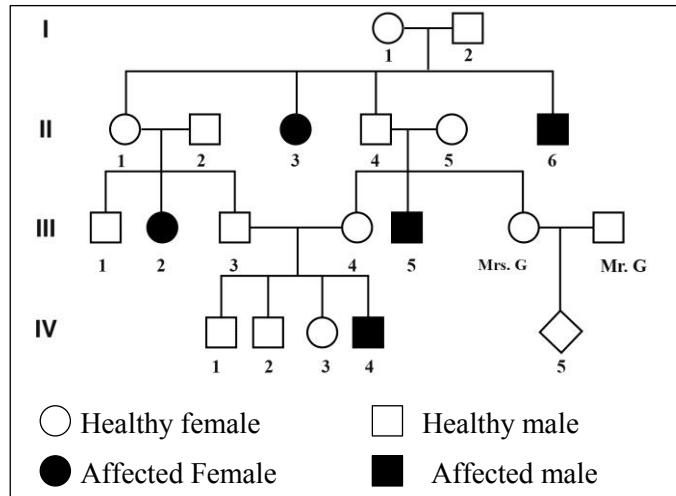
- Determine the risk for the expected child, IV5, to be diseased.

The GALT gene is responsible for galactosemia.

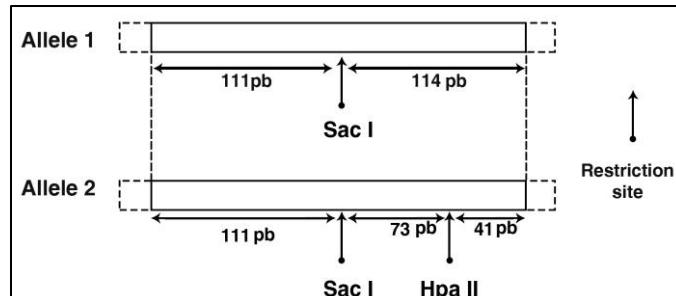
Document 2 shows the cleavage sites of two restriction enzymes, Sac I and Hpa II, at the level of a part (from nucleotide 1367 to nucleotide 1605) of two alleles of this gene: Allele 1 and allele 2.

Document 3 represents the results of electrophoresis obtained after the combined action of enzymes, Sac 1 and Hpa II on allele 1 and allele 2 of GALT gene of certain family members.

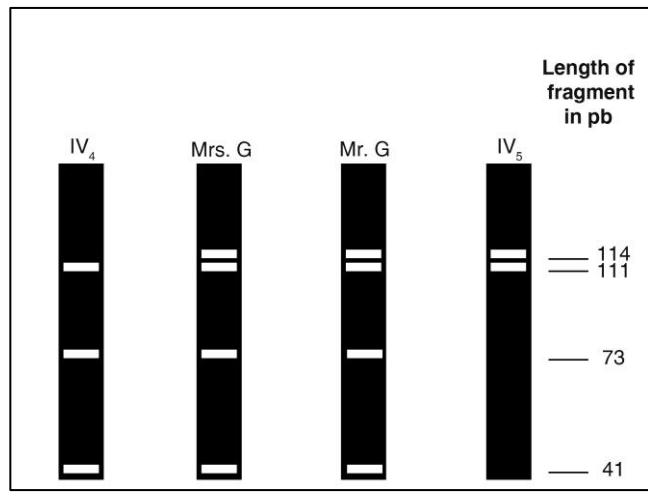
- Indicate, by referring to document 2, the number and size of restriction fragments obtained by the enzymatic digestion of allele 1 and allele 2.
- Determine the allele which corresponds to the mutant one.
- Verify if the fetus IV5 will be affected by galactosemia.



Document 1



Document 2

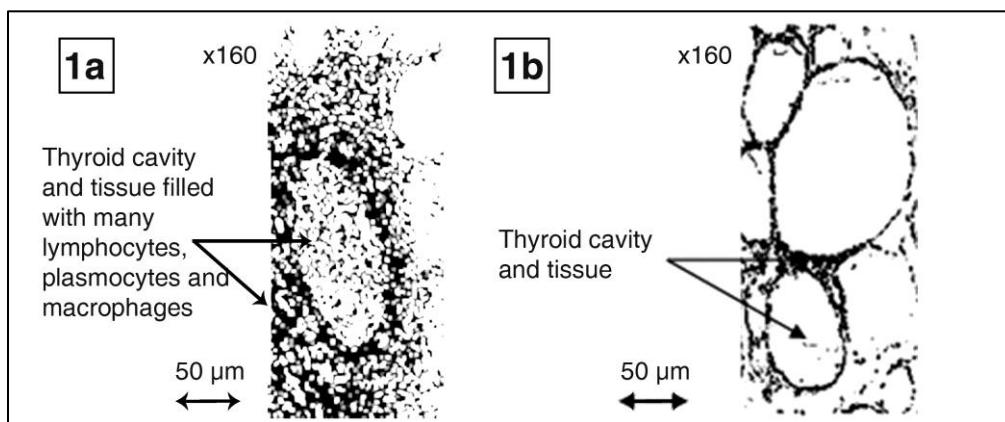


Document 3

Exercise 2 (5 points)**A Case of Thyroiditis**

Sarah has a swelling of the neck at the level of thyroid gland and suffers from many troubles of metabolic origin. Blood analysis of Sarah shows that the concentration level of the thyroid hormones is noticeably lower than the normal values. The synthesis of these thyroid hormones necessitates the presence of a protein named thyroglobulin.

A biopsy is performed on the thyroid gland of Sarah. Document 1 represents the results of the microscopic observations of the sections of thyroid gland of Sarah (1a) and those of the normal thyroid gland (1b).

**Document 1**

- Formulate a hypothesis that can explain the results of biopsy of the thyroid gland of Sarah.

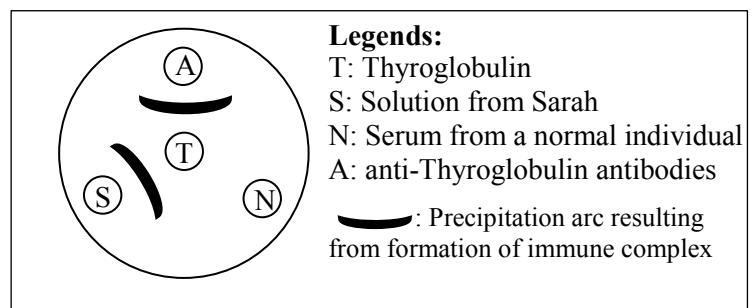
The immune and thyroid cells extracted from the thyroid gland of Sarah, are cultured in 3 different media. The conditions as well as the results are shown in document 2.

- Interpret the results shown in document 2.
- Identify the nature of the specific immune response revealed in document 2.
- Explain the following statement: "Macrophages induce specific immune response".

Culture	Cultivated Cells	Results
1	Thyroid cells + B Lymphocytes	Absence of antibodies
2	Thyroid cells + B Lymphocytes + Macrophages	Absence of antibodies
3	Thyroid cells + B Lymphocytes + Macrophages + T ₄ Lymphocytes	Presence of a large amount of antibodies

Document 2

Afterwards, immunodiffusion gel test is applied. A solution containing the protein thyroglobulin (T) is deposited in the central well, and three other different solutions are separately deposited in three peripheral wells: A solution of antibodies from Sarah (S), anti-thyroglobulin antibodies (A), and serum from a normal individual (N). The results are shown in document 3.

**Document 3**

- Show that Sarah suffers from an auto-immune disease directed against the self.

Exercise 3 (4.5 points)**Cause of Muscle Paralysis**

In the framework of studying certain cases of muscle paralysis, researchers carried on experiments on animals which exhibit complete paralysis of their muscles. In order to determine the origin of this paralysis, the following experiments are performed on a normal animal another paralyzed one. These experiments are performed on the motor neuron N connected to muscle M by synapse F.

Experiment 1:

Effective stimulations are directly applied on muscle M in each of the two animals. Muscular contraction is observed in both cases.

Experiment 2:

Effective stimulations are applied on motor neuron N innervating muscle M in each animal. The results and the experimental conditions are shown in document 1.

1. Show that the paralysis of this animal is due to dysfunctioning of the synapse.

A group of researchers formulate the following hypotheses concerning the cause of the synaptic dysfunctioning in the animal affected by muscle paralysis.

Results of effective stimulation of motor neuron N		
Normal animal	Nerve Message at the level of motor neuron N	Contraction of muscle M
Paralyzed Animal	Nerve Message at the level of motor neuron N	No contraction of muscle M
Document 1		

H1: Muscle paralysis is due to the blockage of exocytosis of acetylcholine in the synaptic cleft.

H2: Muscle paralysis is due to nonfunctional postsynaptic receptors of acetylcholine.

H3: Muscle paralysis is due to a deficiency in the production of acetylcholine by the presynaptic neuron.

These researchers performed experiments 3, 4, and 5 to verify these hypotheses.

Experiment 3:

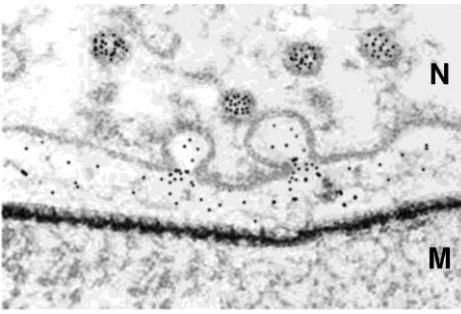
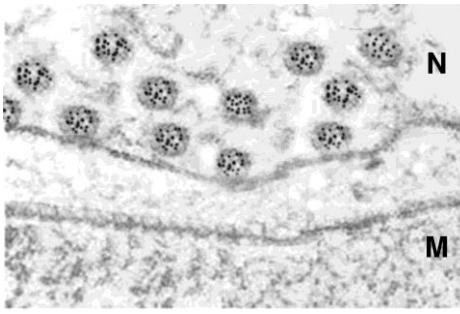
The analysis of the content of the synaptic vesicles of the neuromuscular synapse in the paralyzed animal reveals the presence of acetylcholine, similar to that in the normal animal.

Experiment 4:

Acetylcholine in the neuromuscular synapse of the paralyzed animal is extracted and injected into the synaptic cleft between N and M, in both the paralyzed animal and the normal animal. Contraction of muscle M is observed in both animals.

2. Determine, after studying the results of each of the experiments 3 and 4, the two rejected hypotheses.

Experiment 5: Radioactive choline, a substance transformed by the neuron into acetylcholine, is injected into neuron N of the normal and paralyzed animals. Then, neuron N in both animals is stimulated. Document 2 shows the electromyographies of the synapse after nervous stimulation. The radioactivity appears in the form of black spots.

Structure of neuro-muscular synapse	Normal animal	Paralyzed animal
		

Document 2

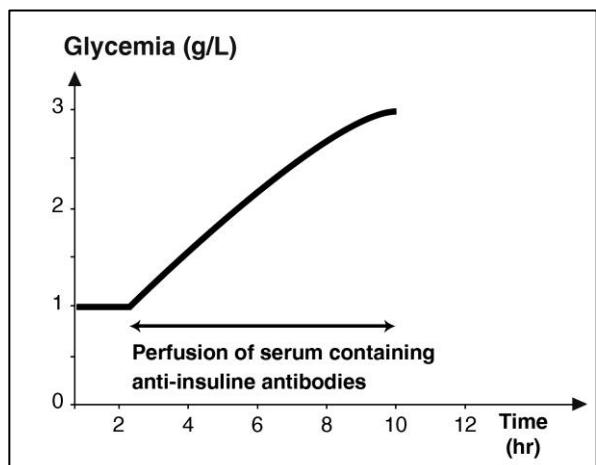
3. Specify the cause of muscle paralysis in the animal.

Exercise 4 (5 points)**Role and Mode of Action of Insulin**

In order to understand the role and mode of action of insulin in an organism, the following experiments are performed.

Experiment 1 :

A rat is perfused (continuously injected) by a serum containing anti-insulin antibodies. These antibodies neutralise insulin, thus preventing it from binding to its receptors on target cells. Afterwards, the variation of glycemia is studied. The results are presented in document 1

**Document 1**

- 1.1. Analyse document 1.

- 1.2. Conclude the role of insulin evident in the document.

Experiment 2:

The rate of absorption of glucose by hepatic cells and the hepatic enzyme activity involved in glycogenesis are measured as a function of the concentration of insulin. The results of the experiment are presented in document 2.

Concentration of insulin ($\mu\text{g/L}$)	5	10	15	20	40
Rate of absorption of glucose by the hepatic cells (a.u.)	10	20	40	60	90
Activity of hepatic enzyme E (%)	15	45	60	75	85

Document 2

2. Interpret the results represented in document 2.

Experiment 3:

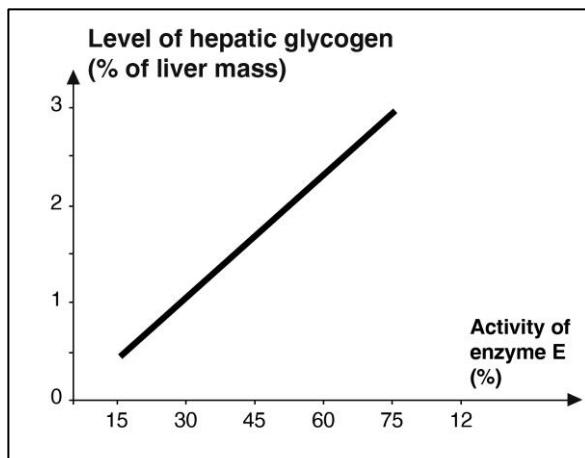
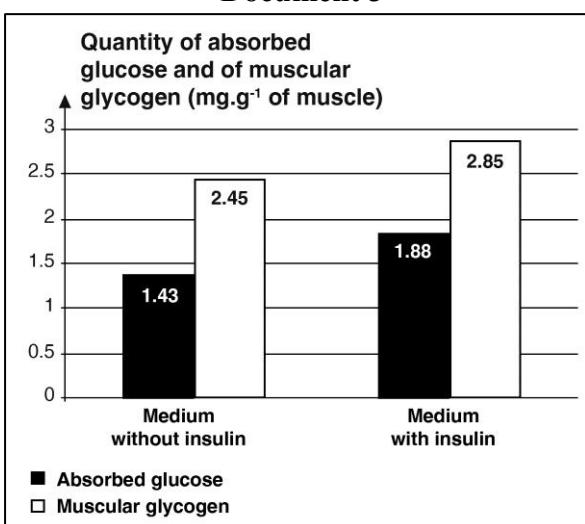
The evolution of hepatic glycogen reserve is studied. The results are shown in document 3.

3. Draw out, from document 3, the role of enzyme E.

Experiment 4:

A muscle is placed in a medium containing glucose with or without insulin for 10 minutes. Then, the quantity of glucose absorbed by the muscle and the quantity of stored glycogen are measured in both media. The results are shown in document 4.

4. Draw a table showing the variation of the quantity of absorbed glucose and that of muscular glycogen with and without insulin.
5. What can be deduced concerning the effect of insulin on the muscle?

**Document 3****Document 4**

Q1	Exercise 1 : Diagnosis of Galactosemia Correction	Marks
1	The allele of the disease is recessive. Couple 1 and 2 are normal but have affected children 3 and 6. This shows that the allele of the disease is carried at least by one of the parents who do not show phenotypically, so the allele is masked, that's why it is recessive (g) with respect to the normal allele N.	0.5
2	If the gene is carried by the proper part of Y , First argument: there are no girls affected because	
3		

Exercice 2 (5 points)**Case of Thyroditis**

Q 2	Correction	Marks
1	Hypothesis : Sarah may have an infection in the thyroid gland. Sarah may have an auto-immune disease. Sarah may have a cancer at the level of the thyroid gland	

2	A large amount of antibodies is secreted in culture 3 in the presence of four types of cells : thyroid gland, B lymphocytes , macrophages and T4 lymphocytes. On the contrary, no antibodies are secreted in the absence of T4 lymphocytes (culture 2) and absence of macrophages (culture 1). This means that secretion of antibodies by B lymphocytes nécessitâtes the presence of T4 lymphocytes and macrophages in the presence of an antigen, in this case the infected thyroid cells of sara.	0.5
3	Document 2 reveals the secretion of antibodies, therefore, the nature of specific immune response is humoral.	
4	When a macrophage phagocytoses and digests a cell or protein, resulting peptides are attached to HLA class II molecules and presented on the cell surface. The macrophage migrates to the closest lymph node, where it becomes an antigen presenting cell or APC. The T helper cells that are specific for the peptides presented by this APC remain attached to it. Then they are activated and they proliferate	
5	The anti-thyroglobulin antibodies in well A moves along the gel where it recognizes the thyroglobulin protein, fix to it and form an immune complex which appears as a precipitation arc. A similar precipitation arc (immune complex) is formed between well S and T which means that serum of sara contains antibodies specific to the protein thyroglobulin where they move along the gel and forms an immune complex . No such arc is revealed between well A and well N which lacks the anti-thyroglobulin antibodies since well N contains serum of a normal individual. Thus. Sara cells secrete antithyroglobulin antibodies which attack the thyroglobulin protein in her thyroid gland leading to problems in metabolism and swollen neck. This shows that Sara has auto immune disease.	

Q4.	Correction	Marks
1.1	At T=0 , the level of glycemia of the rat is 1g/l> This glycemia rests constant at this level till t=2 hr, time of injecting the rat with a continuous amount of anti-insulin antibodies. This perfusion induces an augmentation of glycemia from 1g/l to 3 g/l at t=10 hr.	0.5
1.2	Insulin plays a hypoglycemic role.	0.25

2	<p>With an insulin concentration of 5 Mg/l...., the absorption rate of glucose by the hepatic cells is 10 a.u. and the activity of hepatic enzyme E is 15% . As insulin concentration increases, the absorption rate of glucose increases to reach 90 a.u (about 90 % increase) and also the activity of enzyme E increases to reach 85% (about 5 times increase) when insulin concentration reaches 40 Mg/l . This shows that insulin acts on the hepatic cells and favors the absorption of glucose by the hepatic cells and activate enzyme E.</p>	1									
3	The hepatic enzyme E favors the augmentation of hepatic reserves.	0.5									
4	<p>Table showing the variation of the quantity of glucose absorbed by hepatic cells and quantity of muscular glycogen in the absence and presence of insulin.</p> <table border="1" data-bbox="231 552 1306 777"> <thead> <tr> <th></th><th>Glucose stored (mg/g)</th><th>Muscular glycogen (mg/g)</th></tr> </thead> <tbody> <tr> <td>Medium without insulin</td><td>1.43</td><td>2.45</td></tr> <tr> <td>Medium with insulin</td><td>1.88</td><td>2.85</td></tr> </tbody> </table>		Glucose stored (mg/g)	Muscular glycogen (mg/g)	Medium without insulin	1.43	2.45	Medium with insulin	1.88	2.85	
	Glucose stored (mg/g)	Muscular glycogen (mg/g)									
Medium without insulin	1.43	2.45									
Medium with insulin	1.88	2.85									
5	<p>In a medium without insulin, the quantity of stored glucose is 1.43 mg/g and that of muscular glycogen is 2.45 mg/g. However, in the presence of insulin, the quantity of stored glucose increases to 1.88 mg/g and that of muscular glycogen also increases to reach 2.45 mg/g. We deduce that insulin allows the muscle to absorb glucose and store it in the form of glycogen</p>	1									

Q	Exercise 3	Marks
1	<p>Experiment 1 shows that both muscle contracts when they receive direct effective stimulations. So Both muscles are functional.</p> <p>Experiment 2 shows a nervous message at the level of motor neuron N when it is stimulated .So motor neuron N is functional.</p> <p>On the contrary, muscle M of the paralyzed animal doesn't show any contraction as a consequence of a nervous message that it receives contrary to the muscle of the normal animal. This shows that the paralysis in the animal is neither due to a disfunctioning of motor neurone N nor due to disfunctioning of muscle M. So it is due to dysfunctioning at the level of neuromuscular synapse.</p>	1.5
2	<p>As vesicles are filled with acetylcholine as in the normal animal, this permit us to reject H3 which proposes a deficiency in acetylcholine.</p> <p>The muscular contraction of the paralysed animal upon injection of acetylcholine in the synaptic shows that these neurotransmitters are fixed on their postsynaptic receptors at the level of the muscle .This evident rejects H2.</p>	2
3	<p>The electromyographies show black traces at the level of synaptic membrane.</p> <p>A- Synaptic vesicles are marked in black. This indicates the presence of acetylcholine.</p> <p>B- The vesicles during exocytosis and the black spots of acetylcholine are observed uniquely in the normal individual.</p> <p>A radioactive marker is observed uniquely on the post Synaptic membrane of the muscle of the control animal.</p> <p>So the cause of the paralysis of the animal is due to the absence of exocytosis, and consequently absence of liberation of acetylcholine. The message is not transmitted and the muscle remains relaxed.</p>	1

Exercise 1 (4.5 points)

Patau Syndrome

Patau syndrome is caused by an excess of genetic material of chromosome 13 in the cells of the body. It affects one newborn in 10000 births. The affected children show certain abnormalities: small head, malformation of the hands and eyes, as well as various perturbations in the functioning of the organs.

- Formulate a hypothesis explaining the presence of the excess genetic material in individuals affected by Patau syndrome.

Mr. and Mrs. H, a healthy couple who already have a child affected by Patau syndrome, are expecting another child. They are worried that the fetus might be affected by this syndrome.

The doctor requests certain tests to be performed.

Test 1: The fluorescent in situ hybridization technique (FISH) is applied on fetal cells.

In this prenatal diagnosis technique, two fluorescent single-stranded molecular probes are used:

- Probe A complementary to a specific DNA sequence of chromosome 10.
- Probe B complementary to a specific DNA sequence of chromosome 13 that is involved in Patau syndrome.

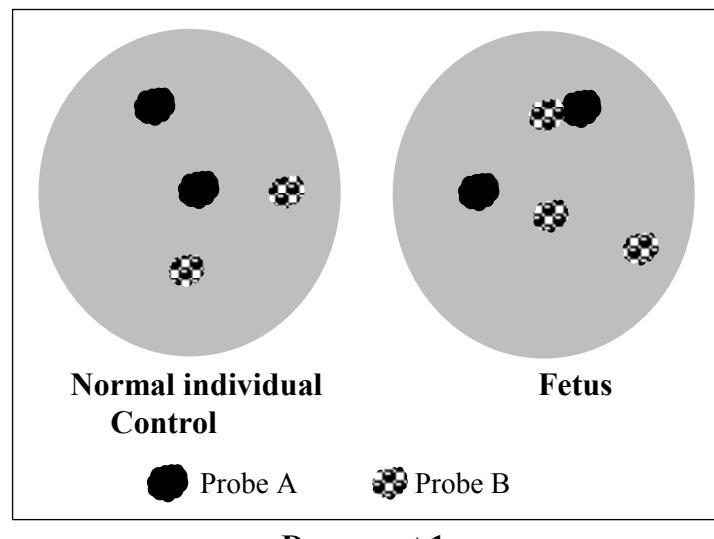
The obtained results are shown in document 1.

Based on the analysis of the results, the doctor assures for the parents that their expected child is affected by Patau syndrome.

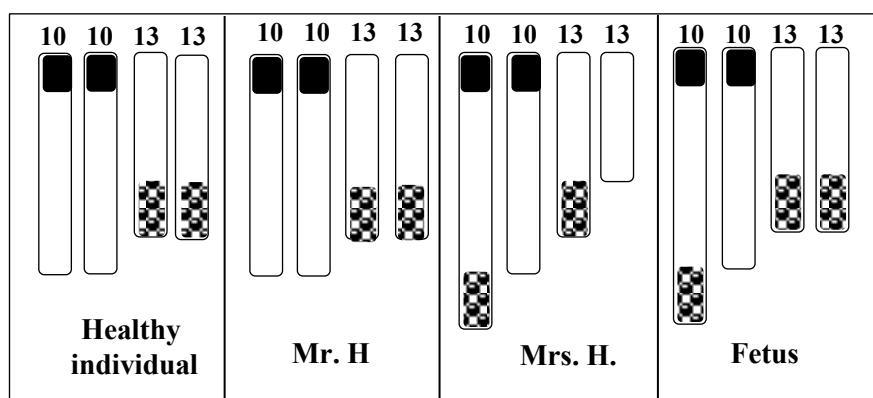
- Justify, by referring to document 1, the doctor's diagnosis.

Test 2: The doctor orders additional tests for each of the two parents and their fetus. Document 2 shows only the pairs of chromosomes 10 and 13 of the mother, the father, the fetus and those of a healthy individual. The other pairs are all normal.

- Justify why the mother presents no phenotypic abnormalities.
- Show that the chromosomal abnormality of the fetus is an abnormality in structure and not in number.
- Schematize chromosomes 10 and 13 in the gametes produced by each of the two parents.
- Indicate the two parental gametes that are at the origin of the karyotype of the fetus.



Document 1



Document 2

Exercise 2 (5 points)

Therapy against an autoimmune disease

Type 1 diabetes (T1D) is due to an autoimmune disease. The current treatment that is based on insulin injection attenuates the symptoms of type I diabetes disease without curing it. For this reason, a research is carried out to verify the effectiveness of a new therapeutic approach to stop the progression of the autoimmune disease which is at the origin of this type of diabetes.

Measurements of the mass of certain components of the pancreas are performed during autopsies in healthy individuals and in individuals suffering from type 1 diabetes. Document 1 shows the obtained results.

1.1. Compare the obtained results.

1.2. Draw out the cause of type 1 diabetes.

The NOD mice (Non Obese Diabetic) develop a disease similar to T1D starting from the age of 10 weeks.

Document 2 represents islets of Langerhans of NOD mice at two different stages of diabetes: an early stage of diabetes (2 a) and a more advanced stage (2b). In this document, T8 lymphocytes appear in the form of black spots.

Note that these mice are not subjected to any viral infection.

2. Identify the type of the immune response involved in this autoimmune disease.

3. Explain the mode of action of T8 Lymphocytes on their target cells.

A new treatment for T1D is tested on two lots of NOD mice at the age of 4 weeks, before the onset of the disease:

- Lot A receives an injection of a saline solution that has no effect (control lot).
- Lot B is subjected to this new treatment.

Document 3 shows the occurrence of diabetes in these two lots of NOD mice.

4. Draw a table representing the results of document 3.

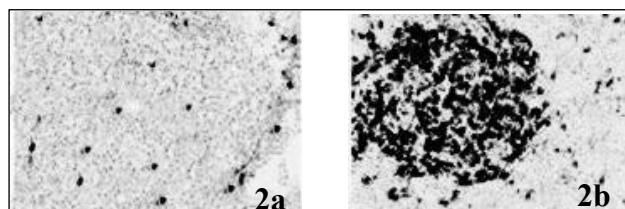
5. Verify if this new treatment is effective against type 1 diabetes.

Document 4 presents the results of labeling cytotoxic T8 lymphocyte in the pancreas of beginning of treatment. T8 lymphocytes appear in form of black spots inside the islet of Langerhans.

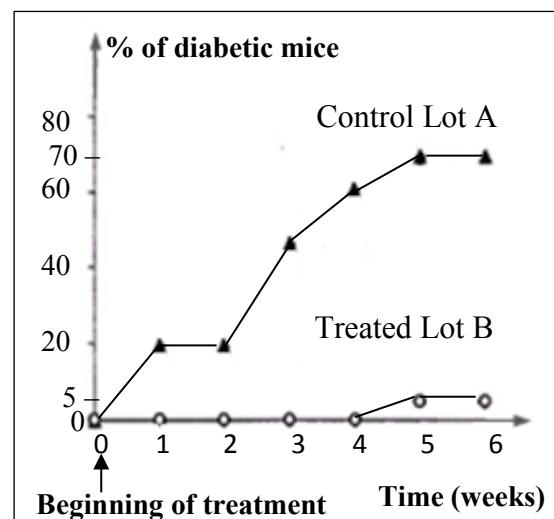
6. Draw out how this new treatment slows down the progression of T1D.

	Healthy individual	Individual suffering from type 1 diabetes
Mass of the islets of Langerhans (mg)	1400	415
Mass of alpha cells (mg)	220	200
Mass of beta cells (mg)	850	0

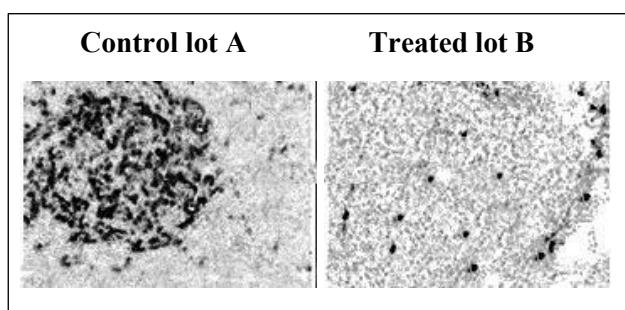
Document 1



Document 2



Document 3



Document 4

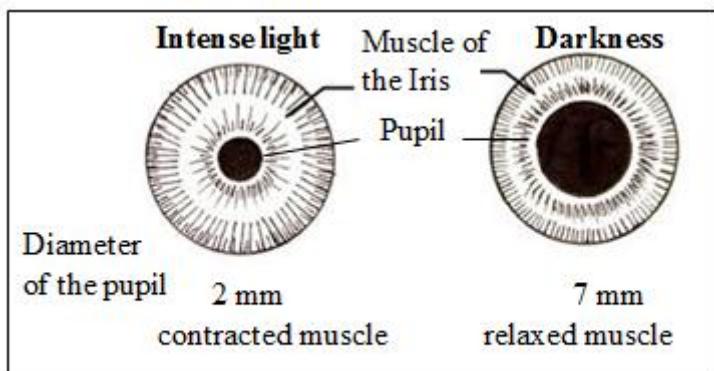
Exercise 3 (5 points)

The diameter of the pupil, an orifice in the eye through which the light penetrates, is controlled by a muscle (the iris). This diameter varies with light intensity, document 1.

Ophthalmologists use medicine such as "atropine" which allows the examination of the eye.

A study is performed to determine the mode of action of atropine.

Action of Atropine



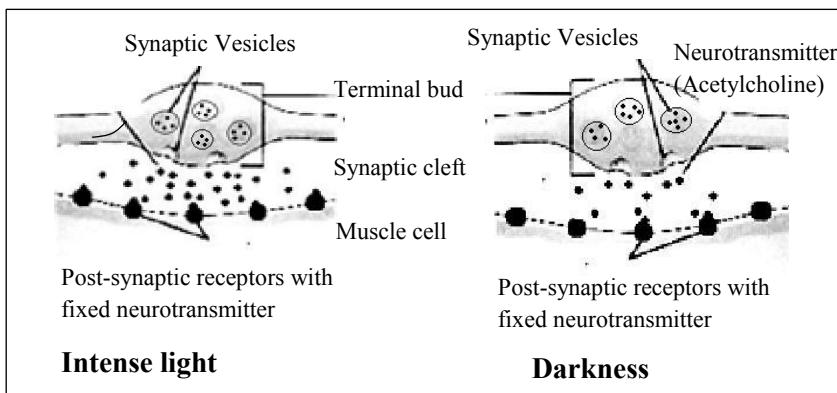
Document 1

- 1.1. Compare the aspect of the pupil and the muscle of the iris in light and in darkness.

- 1.2. Draw out the effect of light on the muscle of the iris.

At the level of the iris, the muscle fibers form excitatory cholinergic synapses with the ends of motor neurons.

Document 2 shows the functioning of these neuromuscular synapses in intense light and in darkness.



Document 2

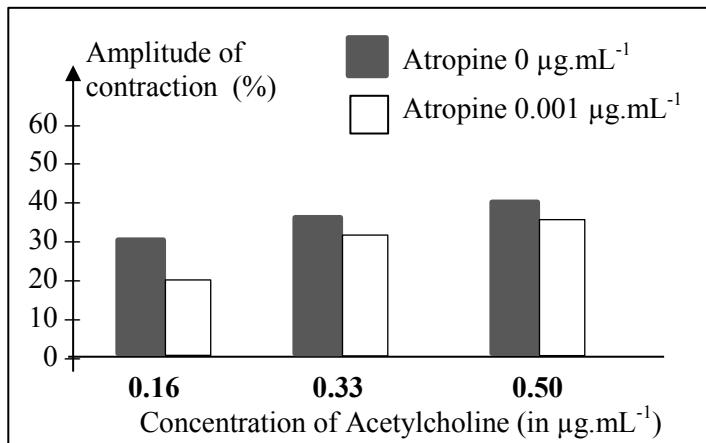
2. List the steps of the synaptic transmission.
3. Justify, referring to document 2, the amplified muscle contraction in the presence of light.

Document 3 shows the amplitude of contraction of the muscle of the iris, in the presence and absence of atropine, as a function of the concentration of acetylcholine in the synaptic cleft.

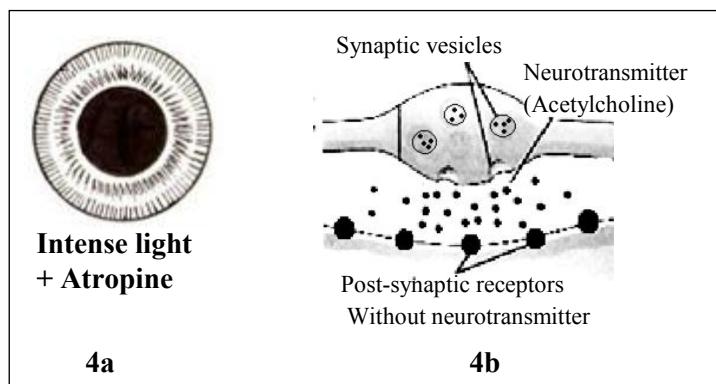
- 4.1. Analyze the obtained results.
- 4.2. Conclude the effect of atropine on muscle contraction.

Document 4 shows the aspect of the pupil (4a) and the functioning of the neuromuscular synapse (4b) in intense light, after the application of a droplet of atropine in the eye of an individual.

5. Compare the aspect of the pupil in document 4a to each of the two aspects shown in document 1.
6. Draw out the step of the synaptic transmission at the level of which atropine acts.
7. Explain, based on what precedes, the use of atropine by ophthalmologist to provoke dilation of the pupil even in the presence of intense light.



Document 3



Document 4

Exercise 4 (5.5 points)

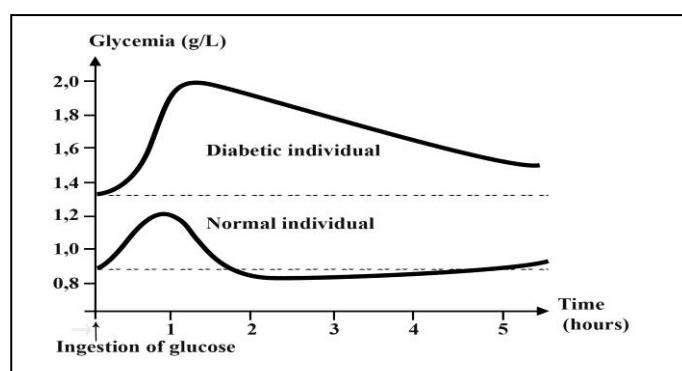
Hypoglycemic Treatment

According to a predicting study performed recently by the World Health Organization (WHO), the number of the individuals affected by diabetes will become 300 million in 2025. The results of this study lead to the research for new medicines for diabetes.

Document 1 shows two major characteristics of diabetes by comparing the development of glycemia in two individuals, one is diabetic and the other is non-diabetic, after the ingestion of glucose solution.

1. Interpret the results of document 1.

Researchers have discovered a hormone, GLP1, secreted into the blood by the intestinal cells after a meal. In the framework of studying the action of GLP1, the following experiments are carried out:



Document 1

Experiment 1: Diabetic individuals are divided into two groups. One group receives a perfusion (continuous injection) of GLP1 during 240 minutes. The other group receives a placebo perfusion, a neutral substance that has no action. The results are represented in document 2.

- 2.1. Analyze the results represented in document 2.
- 2.2. What can you conclude?

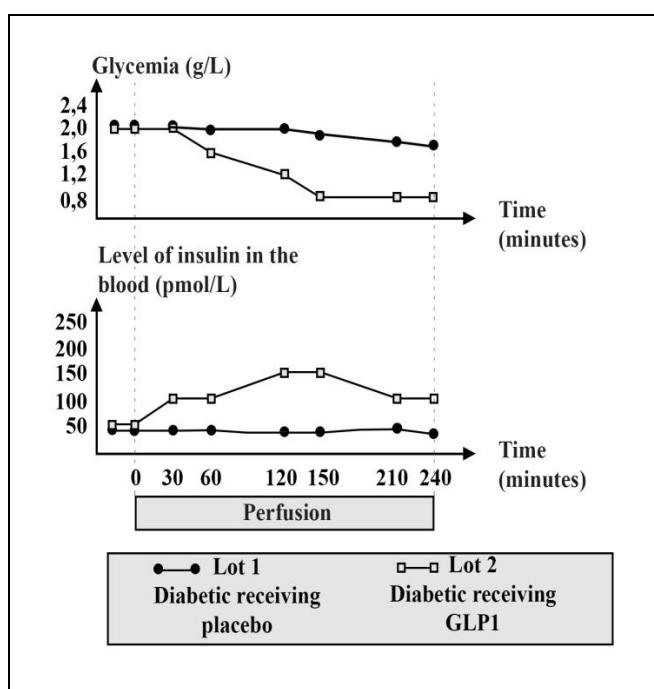
Experiment 2: Zucker rats are obese rats that develop diabetes. Document 3 shows the effect of GLP1 on pancreatic beta cells of two groups of Zucker rats, one treated with GLP1, while the other is a control which is not treated with this hormone.

3. Deduce the effect of GLP1 on Beta cells of the pancreas.

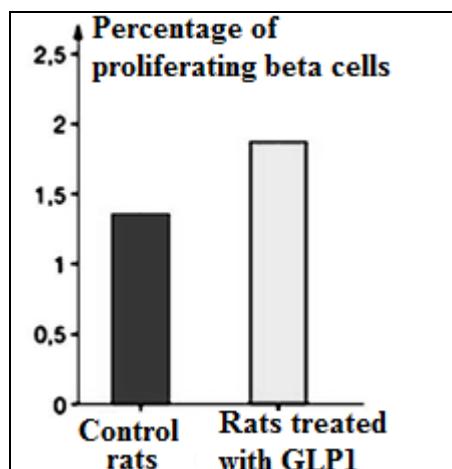
The hormone GLP1 is degraded in the body within two minutes by an enzyme, DPP4. It is thus transformed into inactive substances. Sitagliptin, a new medicine, is an inhibitor of DPP4: When Sitagliptin is administered, it blocks the action of DPP4.

4. Draw out the effect of the administration of Sitagliptin on the blood level of GLP1.

5. Explain how Sitagliptin molecule can improve the health state of certain diabetic individuals.



Document 2



Document 3

مسابقة في مادة علوم الحياة

Q.	Exercise 1 Patau syndrome	Mark															
1	<p>Hypothesis:</p> <ul style="list-style-type: none"> - The excess of genetic material might be due to trisomy 13 (linked or free). - The excess of genetic material might be due to a translocation of part of chromosome 13. - The excess of genetic material might be due to a certain mutation at the level of chromosome 13 (duplication of a fragment of a chromosome). 	0.5															
2	<p>Document 1 shows that each of the healthy individual and the fetus possesses two fluorescent probes A which correspond to the two chromosomes 10. However, the fetus presents three fluorescent probes B corresponding to three chromosomes 13 , unlike the healthy individual who presents two fluorescent probes B (two chromosomes 13). Moreover, one of the three probes B is attached to probe A. The fetus thus has an excess of genetic material of chromosome 13. Since this excess of genetic material of chromosome 13 in the cells causes Patau syndrome, then the doctor's affirmation that the fetus will be affected by Patau syndrome is justified.</p>	0.75															
3	<p>Since one of the pair of chromosomes 13 of the mother shows a missing part , and one of chromosomes 10 has an excess of the same part, and since all the other chromosome pairs are normal; then the mother presents neither gain nor loss in the genetic material and her DNA mass is conserved. As a result, the mother has a normal phenotype.</p>	0.75															
4	<p>The fetus only presents abnormalities in chromosomes 10 and 13. Document 2 shows that the fetus possesses a pair of chromosomes 10 and a pair of chromosomes 13; so the exact total number of chromosomes is normal. Therefore, the fetus' abnormality is not in number.</p> <p>However, one of the chromosomes 10 of the fetus is longer than the pair of chromosomes 10 of the healthy individual, but the other copy of chromosome 10 and both copies of chromosome 13 of the fetus have equal lengths as those of the healthy individual. It is therefore the structure of the chromosomes that is abnormal.</p>	1															
5.1	<p>Scheme showing the types of the parental gametes</p> <table style="width: 100%; text-align: center;"> <tr> <td style="width: 33%;">Types of gametes of the mother</td> <td style="width: 33%;">Gamete of the father</td> <td style="width: 33%;"></td> </tr> <tr> <td>10⁺13 10⁺13- 10 13 10 13-</td> <td>10 13</td> <td></td> </tr> <tr> <td>Gametes:</td> <td></td> <td></td> </tr> <tr> <td> </td> <td> </td> <td></td> </tr> <tr> <td>25% 25% 25% 25%</td> <td>100%</td> <td></td> </tr> </table>	Types of gametes of the mother	Gamete of the father		10 ⁺ 13 10 ⁺ 13- 10 13 10 13-	10 13		Gametes:						25% 25% 25% 25%	100%		1
Types of gametes of the mother	Gamete of the father																
10 ⁺ 13 10 ⁺ 13- 10 13 10 13-	10 13																
Gametes:																	
25% 25% 25% 25%	100%																
5.2	<p>The gametes at the origin of the karyotype of the fetus are:</p> <table style="width: 100%; text-align: center;"> <tr> <td style="width: 50%;">mother 10⁺ 13</td> <td style="width: 50%;">father 10 13</td> </tr> <tr> <td> </td> <td> </td> </tr> </table>	mother 10 ⁺ 13	father 10 13			0.5											
mother 10 ⁺ 13	father 10 13																

Q	Exercise 2 Therapy against an Autoimmune Disease	Mark																										
1.1	The mass of the islets of Langerhans in a healthy individual is 1400mg which is greater (3.38 times more) than 415mg in individual suffering from T1D. While the mass of alpha cells in a healthy individual is 220mg which is slightly greater than that of alpha cells in the affected individual (200 mg). However, the mass of beta cells in a healthy individual is 850mg which is greater than 0mg in the affected individual.	0,5																										
1.2	Type 1 diabetes is due to a lack of beta cells.	0,25																										
2	Document (2a) shows few T8 lymphocytes which appear in the form of black spots in islets of Langerhans of NOD mice at an early stage of diabetes. At a more advanced stage of diabetes (document 2b), the concentration of T8 lymphocytes represented by black spots in the islets of Langerhans of NOD mice increases. As T8 cells have a cytotoxic action against cells, these results show that beta cells are being attacked by T8 cells causing their disappearance in the individual affected by T1D (document 1). Since T8 cells are the effector cells involved in cell mediated immune response, then the immune response involved is specific cell mediated.	1																										
3	<p>During a cell mediated specific immune response:</p> <ul style="list-style-type: none"> - TL8 recognizes the antigenic peptides presented by MHC found on the membrane of target cells, through its TCR. - They are then activated by double recognition. - Once activated, and under the action of IL-2, T8 cells proliferate and form a clone. - Activated T8 cells differentiate into killer cells or cytotoxic TL which : <ul style="list-style-type: none"> • Secrete perforin which forms hollow channels through the plasma membrane of target cells. • Secrete granzymes that penetrate the polyperforin channels, leading to the degradation of its DNA. <p>This leads to apoptosis of target cells.</p>	1																										
4	<table border="1"> <thead> <tr> <th colspan="2">Time (weeks)</th> <th>0</th> <th>1</th> <th>2</th> <th>3</th> <th>4</th> <th>5</th> <th>6</th> </tr> </thead> <tbody> <tr> <td rowspan="2">% of diabetic mice</td> <td>Control Lot A</td> <td>0</td> <td>20</td> <td>20</td> <td>50</td> <td>60</td> <td>70</td> <td>70</td> </tr> <tr> <td>Treated Lot B</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>5</td> <td>5</td> </tr> </tbody> </table> <p style="text-align: center;">↑ Beginning of treatment</p> <p>Title: Table showing the variation of percentage of T1D in NOD mice with or without treatment, as a function of time.</p>	Time (weeks)		0	1	2	3	4	5	6	% of diabetic mice	Control Lot A	0	20	20	50	60	70	70	Treated Lot B	0	0	0	0	0	5	5	1,25
Time (weeks)		0	1	2	3	4	5	6																				
% of diabetic mice	Control Lot A	0	20	20	50	60	70	70																				
	Treated Lot B	0	0	0	0	0	5	5																				
5	<p>The results of document 3 show that the percentage of diabetic mice in control lot A (injected with a saline solution that has no effect) increases between 0 and 6 weeks from 0% to reach 70% which is a value much greater than that of treated lot B (subjected to the new treatment), where the percentage of diabetic mice remains null till 4 weeks, and then increases slightly to 5 % between 4 and 6 weeks.</p> <p>The new treatment has thus reduced the risk of developing type 1 diabetes, which confirms its effectiveness against this disease.</p>	0,75																										
6	This treatment seems to protect the beta cells of islets of Langerhans from the cytotoxic action of T8 lymphocytes; consequently slowing down the occurrence of type 1 diabetes in individuals at risk.	0,25																										

Q	Exercise 3 Action of Atropine	Mark
1.1	In darkness, the diameter of the pupil is more dilated, 7mm , a value greater than 2 mm obtained in intense light. Furthermore, the muscle of the iris is contracted in the presence of intense light, but it is relaxed in darkness.	0, 5
1.2	Light stimulates the contraction of the muscle of the pupil.	0,25
2	The steps of the synaptic transmission are : 1. Arrival of the nervous message to the terminal bud of the presynaptic neuron. 2. Opening of the voltage dependent calcium channels and entrance of calcium ions into the terminal bud. 3. Exocytosis of the neurotransmitter from the synaptic vesicles. 4. Fixation of the neurotransmitter on its specific receptors found on the post-synaptic membrane. 5. Generation of PSP. 6. Recapture or degradation of neurotransmitter.	1,25
3	In the presence of intense light, the quantity of neurotransmitter found in the synaptic cleft and that fixed on the post-synaptic receptors is much higher than the quantity observed in darkness. Since these synapses are excitatory, then the muscle will be more contracted in light.	0, 5
4.1	In the presence of atropine, the amplitude of muscle contraction increases from 20% to 36% as the concentration of acetylcholine increases from $0.16 \text{ } \mu\text{g.mL}^{-1}$ to $0.50 \text{ } \mu\text{g.mL}^{-1}$, which is less than the amplitude recorded in absence of atropine which increases from 31% to 41%.	0,5
4.2	Thus, atropine reduces (attenuates) the muscle contraction.	0,25
5	In the presence of atropine and intense light, the pupil shows a diameter similar to that obtained in darkness, and which is larger than that obtained in intense light without atropine.	0,5
6	The step of the synaptic transmission at the level of which atropine acts is the binding (fixation) of acetylcholine on its post-synaptic receptors.	0,25
7	Atropine is antagonistic with acetylcholine (document 3), it prevents the fixation of acetylcholine on its specific receptors (document 4). Consequently, the muscle of the iris does not contract in the presence of light. When it is relaxed, the pupil dilates, allowing the passage of more light which permits the ophthalmologist to examine the eye even in the presence of intense light.	1

Q	Exercise 4 Hypoglycemic treatment	Mark
1	<p>At time 0 hours, upon ingestion of glucose, glycemia in normal individual is 0.9 g/l which is less than that in diabetic individual which is 1.3 g/L.</p> <p>One hour after the ingestion of glucose, glycemia increases in both individuals; however it increases in the normal individual to 1.2 g/L which is less than 2 g/L obtained in diabetic individual. This shows that the ingested glucose is absorbed in the blood which provokes a hyperglycemia.</p> <p>However, glycemia in the normal individual decreases rapidly after 40 minutes to regain its initial value (0.9 g/L), unlike the glycemia in the diabetic individual which decreases slowly from 2g/L to 1.6 g/L during a longer duration of time (around 4 hours) and remains higher than its initial value (1.3g/L). This shows that these individuals have a hypoglycemic system that permits the regulation of glycemia, but this system is slower in diabetic individuals than in normal ones.</p>	1,25
2.1	<p>Before the perfusion of GLP1, the level of glycemia and the level of insulin are constant at 2g/L and 50 pmol/L respectively in both lots 1 and 2.</p> <p>While during the perfusion, the level of insulin remains almost constant at 50 pmol/L in diabetics of lot 1 receiving placebo which is less than the level of insulin in diabetics of lot 2 receiving GLP1 which increases to 150 pmol/L after 120 minutes, and then it decreases to reach 100 pmol/L which is higher than its initial value between 210 and 240 minutes.</p> <p>However, glycemia decreases slightly in diabetics of lot 1 after perfusion of placebo to reach 1.8g/L after 240 minutes, which is higher than the glycemia in diabetics of lot 2 receiving GLP1, which decreases to reach a constant value of 0.8g/L after 210 minutes.</p>	1
2.2	GLP1 stimulates the secretion of insulin, and it has a hypoglycemic effect.	0,5
3	The percentage of proliferating beta cells in rats treated with GLP1 is 1.85 % which is greater than that in control rats which is 1.4%. Therefore, GLP1 favors the proliferation of beta cells.	0,75
4	Sitagliptin increases the level of GLP1 in blood.	0,5
5	<p>Sitagliptin inhibits the degradation of GLP1 by DPP4, resulting in the increase of the level of GLP1 in the blood. This hormone stimulates rapidly the secretion of insulin, a hypoglycemic hormone, thus presenting a hypoglycemic action. On the long term, GLP1 leads to an increase in the number of beta cells. Since these cells secrete insulin, the production of insulin by the pancreas of diabetics will increase. This hypoglycemic property allows the use of Sitagliptin as a treatment of diabetes.</p>	1.5

الاسم: الرقم:	مسابقة في مادة علوم الحياة المدة: ثلث ساعات
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Exercise 1 (5 points)

Transmission of Two Genetic Anomalies

Majida and Tarek wish to marry. Majida belongs to a family whose some members are affected by albinism, while Tarek has in his family affected cousins by another anomaly, daltonism.

This couple consults a doctor to determine the risk of having children affected by the two concerned anomalies.

Document 1 represents the pedigree of Majida's family.

- Indicate whether the allele of albinism is dominant or recessive. Justify the answer.
- Determine the chromosomal localization of the gene responsible for this anomaly.

The gene of daltonism is localized on the non- homologous segment of chromosome X and exists in two allelic forms, a normal allele and a mutant allele responsible for daltonism.

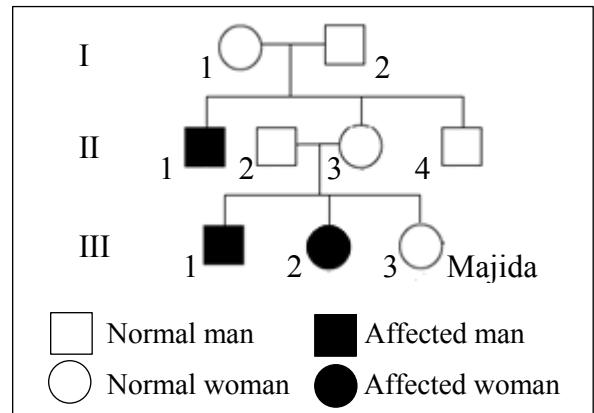
Document 2 shows the obtained results of the electrophoresis performed on the two alleles of the daltonism gene of Tarek and his parents who are all non daltonian.

- Show that the allele responsible for daltonism is recessive.
- Specify which of the two alleles, 1 or 2, is responsible for this anomaly.

The doctor requests several tests to detect the presence of the mutant allele of albinism and the mutant allele of daltonism for Majida and Tarek.

The results are represented in document 3.

- Write, referring to document 3, the genotype of Majida and that of Tarek for the two studied genes.
- Indicate the gametes produced by Majida and those produced by Tarek.
- Verify if this couple could have a child affected by the two studied anomalies at the same time.



Document 1

	Tarek	Father	Mother
Allele 1	—	—	—
Allele 2		—	

Document 2

	Majida	Tarek
Gene of albinism	Normal allele	—
	Mutant allele	—
Gene of daltonism	Normal allele	—
	Mutant allele	—

Document 3

Exercise 2: (5points)

Mode of Action of a Relaxant: Valium

To determine the mode of action of valium, a relaxant prescribed against anxiety, the following experiments are performed.

An effective stimulation **S** is applied on neuron **N**. Document 1 shows the utilized experimental set-up and the results recorded by the oscilloscope connected to the recording electrode **R_N** at the level of axon of neuron **N**, and that recorded by the oscilloscope connected to the recording electrode **R_M** at the level of the cell body of the motor neuron **M**.

- 1- Specify the nature of the synapse between neuron **N** and the motor neuron **M**.
- 2- List the steps of the synaptic transmission.

In the absence of any stimulation, we inject in the synaptic cleft **F**:

Situation A: a dose **D** of a neurotransmitter, GABA.

Situation B: the same dose **D** of GABA with an equivalent dose of valium.

The recordings obtained at level of **R_M** are presented in document 2.

- 3- Interpret the obtained results in document 2.

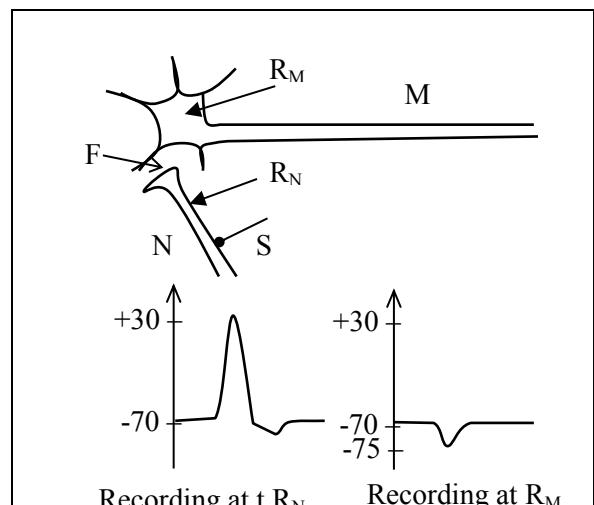
The postsynaptic membrane of the motor neuron **M** has chemical-dependent channels of Cl^- ions. Document 3 shows the ionic concentrations of Cl^- ions in the intracellular and extracellular media of this motor neuron in the absence of any stimulation.

- 4- Define chemical-dependent channels.
- 5- Specify the direction of movement of Cl^- ions across the postsynaptic membrane of the motor neuron **M** following the opening of the Cl^- channels.

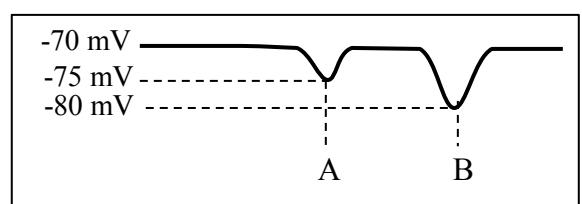
The state of chemical-dependent Cl^- channels of the postsynaptic membrane of the motor neuron **M** is monitored in the two previously listed experimental situations, A and B. Document 4 reveals the obtained results.

Valium fixes on specific sites of the Cl^- channel receptors. This binding activates the fixation of GABA on other sites of the same Cl^- channel receptors.

- 6- Determine the mode of action of valium.



Document 1



Document 2

	Extracellular medium	Intracellular medium
Ionic concentration of Cl^-	560 mol.L ⁻¹	40 mol.L ⁻¹

Document 3

	Duration of the opening of Cl^- channels (ms)	Number of opening of Cl^- channels per second
Situation A	23	48
Situation B	29	92

Document 4

Exercise 3 (5 points)

Immune Responses Against Flu Virus

In the framework of studying the immune responses against the flu virus, several observations and experiments are performed.

First observation: Individuals who are infected by the flu virus show signs of an inflammatory reaction.

- 1- List the four signs of the inflammatory reaction.

Second observation: Document 1 presents the variation of the concentration of anti-flu antibodies as a function of time, following the infection by the flu virus.

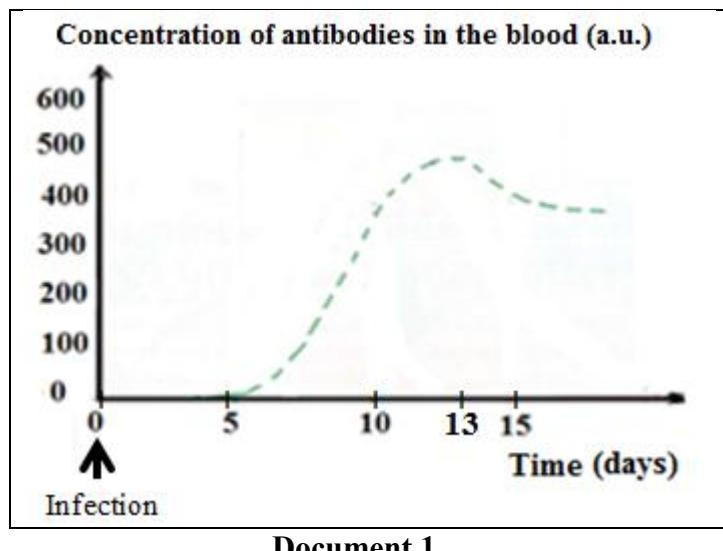
- 2- Identify the type of the specific immune response revealed by the results of document 1.

Experiment 1: The flu virus and anti-flu antibodies of increasing concentrations, C_1 , C_2 and C_3 , are added to different culture media containing human cells. The concentration of the infected cells is measured and the obtained results are presented in document 2.

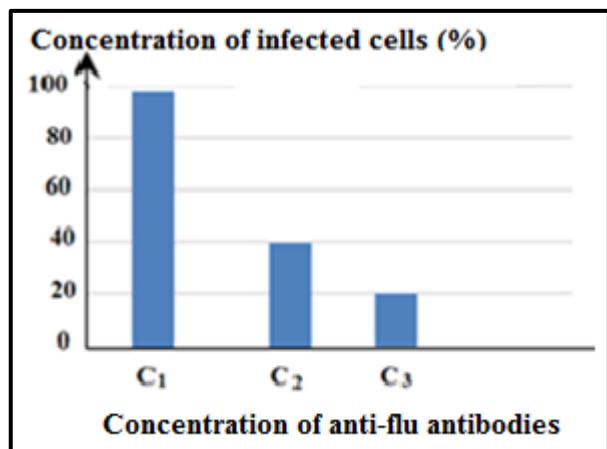
- 3- Interpret the obtained results.

Experiment 2: The action of antibodies does not permit the elimination of the cells infected by the flu virus. The monitoring of the number of cytotoxic T lymphocytes (Tc) and the infected cells in an individual infected by the flu virus shows the results presented in document 3.

- 4- Draw the graph showing the variation of the number of infected cells and that of Tc cells as a function of time.
- 5- Specify the type of the specific immune response revealed by the results presented in document 3.



Document 1



Document 2

Time (days)	0	3	7	9	13	15
Number of Tc cells	0	0	300	500	100	50
Number of infected cells	50	100	200	150	10	0

Document 3

Third observation:

Clinical observations show that the flu virus may be fatal in some individuals showing deficiency in T_H lymphocytes (case of AIDS).

- 6- Explain this observation.

Exercise 4 (5 points)

Hormonal Origin of a Disease

Sara, a 16 year-old girl consults a doctor to check the cause of the following symptoms: absence of breast development and absence of menstruation. The doctor requests hormonal tests and biopsies for Sara's ovaries to know the origin of these symptoms. The results of the blood concentration of estradiol during 28 days are presented in document 1.

	Sara	16 year-old normal girl (control)
Concentration of estrogen (Estradiol) (Pg / mL)	Around 15	Follicular phase : 30 to 90 Ovulatory peak : 90 to 400 Luteal phase : 50 to 20.

- 1- Draw out, referring to document 1, a possible cause of the observed symptoms.

The results of biopsies performed on Sara's ovaries at different time intervals reveal the presence of only primary follicles.

- 2- How do the results of the biopsies explain the concentration of estradiol noticed in Sara's blood?

The FSH and LH concentrations in the blood are measured during 28 days. The results are shown in document 2.

- 3- Indicate the roles of FSH and LH hormones.
4- Analyze the obtained results.

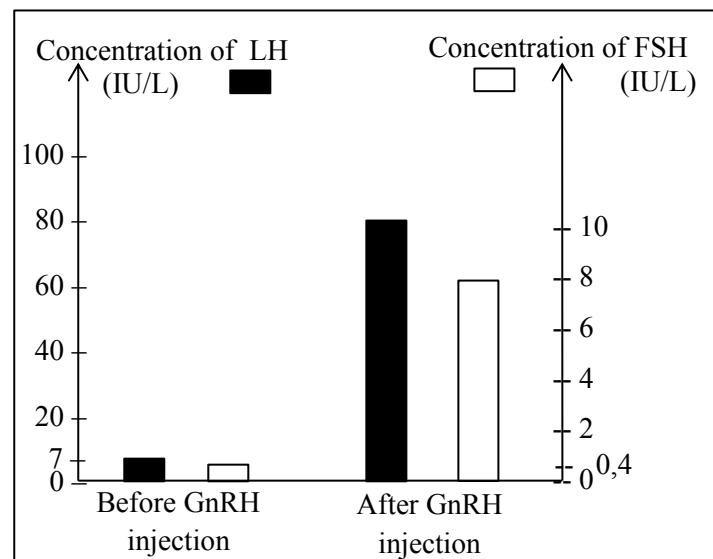
The origin of these hormonal results could be due to either a defect in the secretion of GnRH by the hypothalamus or to a defect in the receptors specific to GnRH located at the level of the anterior pituitary gland.

To determine the origin of these hormonal troubles, the concentrations of FSH and LH in Sara's blood are measured before the injection of 100 microgram of GnRH and 30 minutes following this injection. The results are shown in document 3.

Document 1

Blood concentration of hormones	Sara	16 year-old normal girl (control)
LH (IU/L)	5 to 7	Follicular phase: 1.5 to 10 Ovulatory Peak: 18 to 90 Luteal phase: 1 to 16
FSH (IU/L)	< 0.5	Follicular phase : 2 to 17 Ovulatory peak : 9 to 26 Luteal phase : 2 to 8

Document 2



Document 3

- 5- Pick out the formulated hypotheses.
6- Which hypothesis is validated by the results of document 3? Justify the answer.

Part de l'ex	Exercice 1 Transmission de deux anomalies génétiques	Note
1	<p>L'allèle de l'albinisme est récessif.</p> <p>Car, les parents II₂ et II₃ de phénotype sain ont eu des enfants III₁ et III₂ malades. Ces enfants ont hérités l'allèle de la maladie au moins de l'un des parents. Ce parent possède l'allèle morbide à l'état masqué.</p> <p>Soit A le symbole de l'allèle normal, dominant.</p> <p>Soit a le symbole de l'allèle muté responsable de l'albinisme.</p>	0,5
2	<p>Si le gène est localisé sur la partie propre du chromosome Y:</p> <p>La maladie serait présente uniquement chez les hommes puisque les filles ne possèdent pas le gonosome Y. Or. L'arbre montre une fille, III₂, malade.</p> <p>OU: Père et fils seraient de même phénotype car le garçon hérite son Y de son père.</p> <p>Or. Le garçon II₁ (ou III₁) est malade de génotype X/Y^a, son père devrait être malade de génotype obligatoire X//Y^a, ce qui n'est pas le cas.</p> <p>Le gène n'est pas porté par Y.</p> <p>Si le gène est localisé sur la partie commune à X et Y.</p> <p>Le garçon III₁ serait de génotype X^a//Y^a avec Y^a d'origine paternelle, et sa sœur III₂ de génotype X^a//X^a, dont l'un d'eux hérité du père. Leur père II₂ serait de génotype X^a//Y^a et de phénotype malade. Or, il ne l'est pas.</p> <p>Le gène n'est pas porté sur X et Y.</p> <p>Si le gène est porté par X:</p> <p>Le génotype de la fille malade III₂ serait X^a//X^a avec un X^a d'origine paternelle. Le père II₂ serait malade de génotype X^a//Y. Or, il est de phénotype normal.</p> <p>Le gène n'est pas porté par X.</p> <p>D'où, le gène de l'albinisme est autosomal.</p>	1
3	<p>L'électrophorégramme de la mère de Tarek montre qu'elle a les deux allèles, 1 et 2, du gène du daltonisme avec l'un d'eux est « normal » et l'autre est responsable du daltonisme. Comme la mère est de phénotype normal, donc seul l'allèle « normal » s'exprime alors que l'allèle « morbide » est masqué. Par conséquent, cet allèle est récessif.</p> <p>Soit « d » le symbole de l'allèle responsable du daltonisme et « N » le symbole de l'allèle « normal ».</p>	0,5
4	<p>D'après l'électrophorégramme, Tarek (ou son père) possède l'allèle 1. Comme il est de phénotype normal, cet allèle doit correspondre à l'allèle « normal ».</p> <p>L'allèle 2 est donc celui responsable du daltonisme.</p>	0,5
5	<p>Concernant le gène de l'albinisme, Majida et Tarek possèdent les deux allèles, normal et morbide. Ils sont donc hétérozygotes.</p> <p>Quant au gène du daltonisme, Majida possède les deux allèles, normal et morbide alors que Tarek possède uniquement l'allèle normal.</p> <p>Sachant que le gène de l'albinisme est autosomal et que le gène du daltonisme est gonosomal, porté par la partie propre du gonosome X :</p> <p>Le génotype de la mère de Tarek serait A//a X^N//X^d.</p> <p>Le génotype du père de Tarek est A//a X^N//Y.</p>	1
6	<p>Les gamètes maternels sont : (A,X^N) ; (A,X^d) ; (a,X^N) ; (a,X^d).</p> <p>Les gamètes paternels sont : (A,X^N) ; (A,Y) ; (a,X^N) ; (a,Y).</p>	1

7	<p>Un enfant serait atteint des deux anomalies s'il a l'allèle récessif de l'albinisme en double exemplaire et s'il a l'allèle du daltonisme seul ou en double exemplaire.</p> <p>Cet enfant doit donc hériter un allèle a et un allèle d de chaque parent ou un allèle a de chaque parent et un allèle d d'un parent sans recevoir l'allèle N de l'autre parent.</p> <p>Or, parmi les gamètes parentaux, la seule combinaison possible serait entre le gamète maternel de génotype (a,X^d) et le gamète paternel de génotype (a,Y). Ceci détermine le génotype a//a X^d//Y et donc le phénotype albinos et daltonien.</p>	0,5
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Part de l'ex	Exercice 2	Mode d'action d'un relaxant : le valium	Note
1	La synapse étudiée est inhibitrice car suite à la stimulation de la fibre nerveuse présynaptique S, un PPSI d'amplitude -5 mV apparaît en R _M		0,5
2	<ul style="list-style-type: none"> - Arrivée d'un potentiel d'action à la terminaison nerveuse présynaptique. - Ouverture des canaux calciques et entrée des ions Ca²⁺ dans la membrane présynaptique. - Libération des neurotransmetteurs par exocytose dans la fente synaptique. - Fixation des neurotransmetteurs sur les récepteurs postsynaptiques. - Ouverture des canaux chimiodépendants. - Apparition d'un PPS dans l'élément post-synaptique. - Recapture des neurotransmetteurs par le neurone présynaptique ou dégradation. 		1
3	<p>Suite à l'injection d'une dose D de GABA dans la fente synaptique et en dehors de toute stimulation, une hyperpolarisation (PPSI) d'amplitude 5 mV apparaît en R_M, ceci montre que le GABA est le neurotransmetteur de cette synapse et qu'il a un effet inhibiteur.</p> <p>De plus, suite à l'injection de la même dose D de GABA avec une dose équivalente de valium, l'amplitude de l'hyperpolarisation augmente à 10 mV ceci signifie que le valium amplifie l'action inhibitrice du GABA (OU, le valium est agoniste du GABA).</p>		1
4	Les canaux chimiodépendants sont des canaux ioniques dont l'ouverture dépend de la fixation de substances chimiques (les neurotransmetteurs).		0,5
5	<p>Les ions Cl⁻ diffusent du milieu extracellulaire au milieu intracellulaire.</p> <p>En effet, ces ions passent selon le gradient de concentration, c-à-d du milieu où ils sont concentrés au milieu où leur concentration est plus faible. Or, d'après le document, la concentration extracellulaire de Cl⁻ est de 560 mL.L⁻¹, supérieure à 40 mL.L⁻¹ qui est la concentration intracellulaire.</p>		0,5
6	<p>La durée d'une ouverture du canal Cl⁻ dans la situation A où une dose D de GABA est injectée dans la fente synaptique, est de 23 ms, inférieure à 29 ms dans la situation B où cette dose D de GABA est associée à une dose équivalente de valium. De même, le nombre d'ouverture du canal Cl⁻ est 48 par seconde dans la situation A, plus petit que 92 ouvertures par seconde dans la situation B.</p> <p>Alors, lorsque le valium se fixe sur ses sites spécifiques au niveau des canaux Cl⁻, il facilite la fixation du GABA au niveau de ces canaux et amplifie l'action du GABA en prolongeant la durée et le nombre des ouvertures des canaux Cl⁻ et par suite, augmente la diffusion des ions Cl⁻.</p>		1,5

Part de l'ex	Exercice 3 (5 points)	Réponses immunitaires contre les virus	Note																				
1	Les signes de la réaction inflammatoire sont : une rougeur, un œdème, une douleur et une chaleur.		0,5																				
2	La concentration sanguine en anticorps antigrippaux augmente de 0 à 450 u.a pendant 13 jours. Comme les anticorps sont les effecteurs de la réponse immunitaire humorale, donc la réponse immunitaire spécifique mise en évidence contre le virus de la grippe est à médiation humorale.		0,5																				
3	La concentration des cellules infectées par le virus de la grippe diminue de 100% à 20% lorsque la concentration des anticorps antigrippaux ajoutée aux cultures de cellules humaines en présence du virus de la grippe, augmente de C ₁ à C ₃ . Ceci montre que les anticorps empêchent l'infection des cellules par le virus.		1,25																				
4	<p>Titre : Graphique montrant les variations du nombre de LTc et de cellules infectées en fonction du temps.</p> <p>Echelle:</p> <p>Sur X: 1cm pr 3 jours</p> <p>Sur Y:</p> <p>1 cm pour 100 LTc</p> <p>1 cm pr 50 cellules infectées</p> <table border="1"> <caption>Data extracted from the graph</caption> <thead> <tr> <th>Temps (en jours)</th> <th>Nombre de LTc</th> <th>Nombre de cellules infectées</th> </tr> </thead> <tbody> <tr><td>0</td><td>50</td><td>0</td></tr> <tr><td>3</td><td>150</td><td>0</td></tr> <tr><td>7</td><td>300</td><td>200</td></tr> <tr><td>9</td><td>300</td><td>200</td></tr> <tr><td>13</td><td>50</td><td>50</td></tr> <tr><td>15</td><td>0</td><td>0</td></tr> </tbody> </table>	Temps (en jours)	Nombre de LTc	Nombre de cellules infectées	0	50	0	3	150	0	7	300	200	9	300	200	13	50	50	15	0	0	1,5
Temps (en jours)	Nombre de LTc	Nombre de cellules infectées																					
0	50	0																					
3	150	0																					
7	300	200																					
9	300	200																					
13	50	50																					
15	0	0																					
5	La réponse immunitaire spécifique mise en jeu dans le document 3 est à médiation cellulaire, car le nombre de LTc, acteurs de la réponse à médiation cellulaire, augmente de 0 à 500 pendant les 9 jours qui suivent l'infection par le virus de la grippe.		0,5																				
6	<p>Suite à une infection, les LT4 se différencient en lymphocytes Th qui sécrètent les interleukines IL2 et IL4 indispensables à la différenciation des lymphocytes T8 en LTc et des lymphocytes B en plasmocytes sécréteurs d'anticorps.</p> <p>Les personnes ayant une déficience en LT4 n'auront plus d'interleukines et par conséquent, elles n'auront pas de LTc ni de plasmocytes. Ceci entraîne une déficience immunitaire qui se traduit par la disparition de la réponse spécifique indispensable dans la lutte antivirale.</p>		0,75																				

Part de l'ex	Exercice 4 (5 points)	Origine hormonale d'une maladie	Note
1	La cause possible des symptômes observés est un déficit de sécrétion d'œstrogènes par les ovaires....		0,5
2	La biopsie des ovaires de Sara montre seulement des follicules primaires. Or, les follicules en développement, surtout aux stades avancés, sont responsables de la sécrétion ovarienne des œstrogènes. Ainsi, en leur absence, cette sécrétion est très limitée, ce qui explique le taux faible d'oestradiol chez Sara.		0,5
3	La FSH stimule le développement folliculaire et la maturation du follicule cavitaire ; La LH déclenche l'ovulation et stimule la transformation du follicule en corps jaune.		0,5
4	La concentration de FSH chez Sara est inférieure à ceux observées chez la fille saine tout au long du cycle : <0.5 UI/L est inférieure à 2-26 UI/L. De même, la concentration de LH, 5-7 UI/L, est inférieure aux valeurs obtenues durant le pic ovulatoire, 18-90 UI/L. Par contre, ces valeurs sont incluses dans les valeurs observées pendant la phase folliculaire, 1,5-10 UI/mL, et 1-16 UI/mL durant la phase lutéale.		1
5	Hypothèses : <ol style="list-style-type: none"> 1. L'origine du faible taux de gonadostimulines chez Sara est un déficit de sécrétion de GnRH par l'hypothalamus. 2. L'origine du faible taux de gonadostimulines chez Sara est un défaut de récepteurs spécifiques de la GnRH au niveau de l'hypophyse. 		1
6	L'injection de GnRH provoque une forte augmentation de la sécrétion de LH (de 7 UI/L à 80 UI/L). De même, elle induit une augmentation de la concentration de FSH de 0,4 à 8 UI/L entre 0 et 60 minutes. Ainsi, les cellules hypophysaires sécrétrices de FSH et de LH sont stimulées par la GnRH. Donc l'hypothèse 1 est validée.		1,5

الاسم:	مسابقة علوم الحياة
الرقم:	المدة: ثلاثة ساعات

Exercise 1 (5 points)

Pulmonary Emphysema

Pulmonary emphysema is a fatal disease characterized by an increasingly severe respiratory failure. This disease is due to a progressive destruction of the lung tissue by the proteases of the white blood cells. In fact, in the normal case, there are substances in the blood plasma called alpha antitrypsin (aT) which protect the pulmonary cells from being destroyed by inhibiting the action of proteases.

- 1- Pick out from the text the cause of pulmonary emphysema.

Alpha antitrypsin (aT) is a protein composed of 418 amino acids produced by liver cells. Document 1 shows the nucleotide sequence of a fragment of the non-transcribed strand of the normal allele (M1) and that of the allele of the disease (M2) of the gene responsible for the synthesis of "aT".

Allele	Nucleotide sequence of the fragment of the non-transcribed strand			
M ₁	181	184		
 ATC AAC GAT TAC ...			
M ₂	181	184		
 ATC AAC GAT TAG ...			

Document 1

- 2- Determine, using the genetic code table (document 2), the amino acid sequence of the portion of the alpha antitrypsin coded by the fragment of the allele M1 and that coded by the fragment of the allele M2.

- 3- Explain how the modifications in the nucleotide sequence of the allele (document 1) lead to the appearance of pulmonary emphysema.

Document 3 represents the pedigree of a family of which some members are affected by pulmonary emphysema.

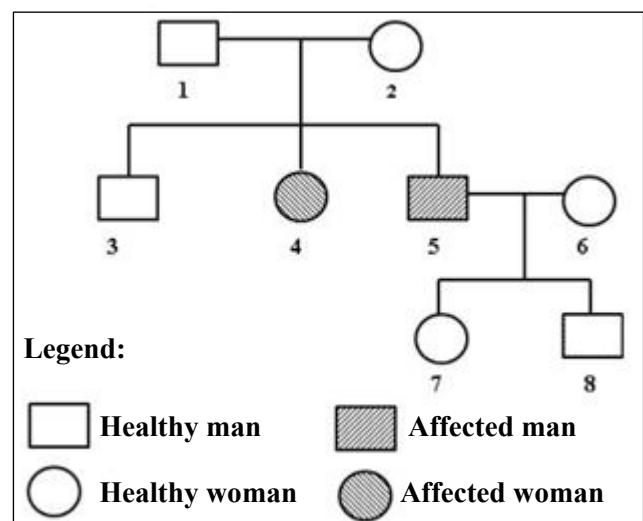
- 4- Specify whether the allele M2 which is responsible for this disease is dominant or recessive.
 5- Determine the chromosomal localization of the gene responsible for pulmonary emphysema.
 6- Write the genotype of individual 8. Justify the answer.

Individual 8 is a heavy smoker and has manifested the same symptoms of pulmonary emphysema.

- 7- Show that there is a factor other than the genetic factor that could provoke this disease.

		Second letter													
		U	C	A	G										
First letter	U	UUU	Phe	UCU	Ser	UAU	Tyr	UGU	Cys	U					
		UUC		UCC		UAC		UGC	stop	C	A				
	C	UUA	Leu	UCA		UAA	stop	UGA	Trp						
		UUG		UCG		UAG	stop	UGG							
	C	CUU		CCU		CAU	His	CGU		U					
		CUC	Leu	CCC		CAC		CGC		C	A				
	A	CUA		CCA	Pro	CAA	Gln	CGA							
		CUG		CCG		CAG		CGG		A	G				
	A	AUU		ACU		AAU		AGU		U					
		AUC	Ile	ACC		AAC	Asn	AGC		C	A				
	A	AUA		ACA		AAA	Lys	AGA							
		AUG	Met	ACG		AAG		AGG		A	G				
	G	GUU		GCU		GAU		GGU		U					
		GUC	Val	GCC		GAC	Asp	GGC		C	A				
	G	GUA		GCA		GAA	Glu	GGG							
		GUG		GCG		GAG				A	G				

Document 2



Document 3

Exercise 2 (5 points)

AIDS

The acquired immunodeficiency syndrome (AIDS) is due to a retrovirus, human immunodeficiency virus (HIV). HIV recognizes and binds to CD4 and CCR5 proteins present on the surface of T4 cells, resulting in the entrance of viral RNA into the host cell. Inside T4 cells, the viral RNA undergoes reverse transcription into viral DNA by the reverse transcriptase enzyme. The viral DNA is then integrated into the DNA of the host cell. In the nucleus of the host cell, the viral DNA is transcribed into mRNA by the cellular mechanism of transcription. This mRNA contributes to the synthesis of the viral proteins (viral constituents) by translation. The RNA and the obtained proteins are necessary for the multiplication of the virus.

Document 1

- 1- Draw out from document 1:

- 1.1- The molecules recognized by HIV.
- 1.2- The target cell of HIV.

Mrs. Y, who is seropositive for HIV, has two children whose HIV seropositivity has been monitored from birth till the age of 18 months. Document 2 represents the electrophoregrams of anti-HIV antibodies of Mrs. Y and of her two children at three different ages. These antibodies, anti-GP160, anti-GP120, anti-GP41 and anti-GP24, are directed against the proteins Gp160, Gp120, Gp41, Gp24 of HIV.

- 2.1- Analyze the obtained results.

- 2.2- Draw out who of the two children is seropositive for HIV at the age of 18 months.

- 3- Propose a hypothesis concerning the origin of the antibodies present at birth in the two children.

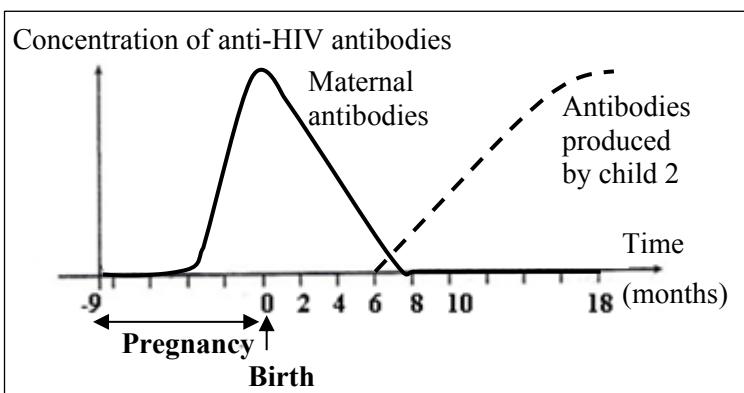
Document 3 represents the evolution of the concentration of anti HIV antibodies in child 2 before and after birth.

- 4- Do the results of document 3 validate the hypothesis formulated in question 3? Justify the answer.

- 5- Explain the reappearance of anti-HIV antibodies after the age of 6 months in child 2.

Mrs. Y	Child 1			Child 2		
	Birth	6 th month	18 th month	Birth	6 th month	18 th month
Anti-GP160	—	—	—	—	—	—
Anti- GP120	—	—	—	—	—	—
Anti-GP 41	—	—	—	—	—	—
Anti-GP 24	—	—	—	—	—	—

Document 2



Document 3

Exercise 3 (5 points)

GABA and Baclofen

Baclofen is a chemical substance, known for its relaxant action.

In order to study the action of baclofen at the level of certain neurons, several experiments are performed using the same setup shown in document 1a.

Experiment 1

An effective stimulation is applied on nerve fiber 1 and then on nerve fiber 2. The obtained results recorded at the level of the cell body of the motor neuron are represented in document 1b.

- 1- Specify the nature of each synapse S1 and S2.

Experiment 2

Acetylcholine is deposited at the level of synapse S1. Another time, GABA is deposited at the level of the synapse S2. The results recorded at the level of the cell body of the motor neuron are represented in document 1b.

- 2- Show that the motor neuron has different types of membrane receptors for neurotransmitters.

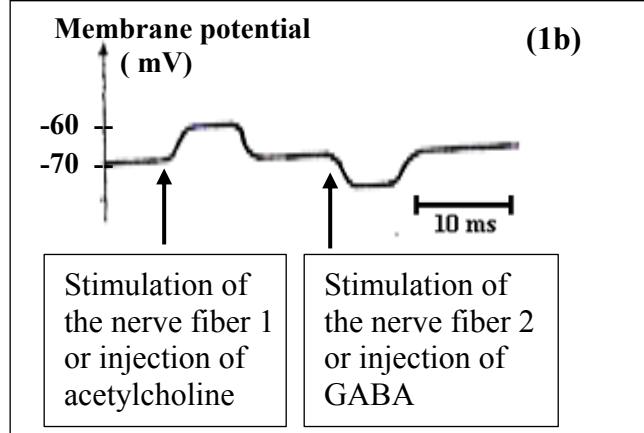
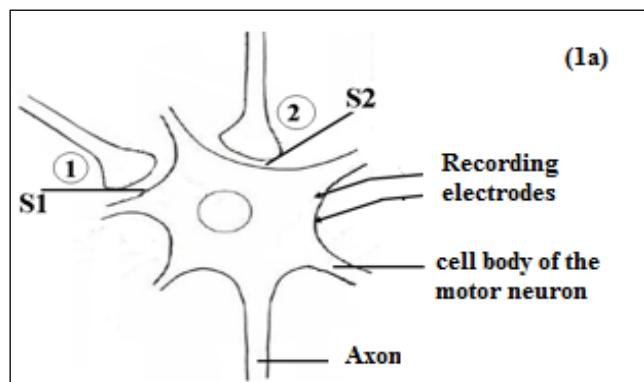
Experiment 3

GABA or baclofen of same concentration are deposited at the level of S2. The variations of the membrane potential at the level of the cell body are represented in document 2.

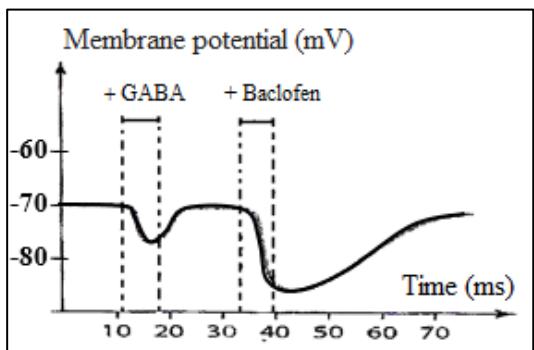
- 3- Interpret the obtained results.
- 4- Explain, referring to the acquired knowledge, the mode of action of GABA.

In order to verify if baclofen acts on GABA receptors, experiment 3 is repeated, but the motor neuron is placed in a medium without Cl^- . The results are presented in document 3.

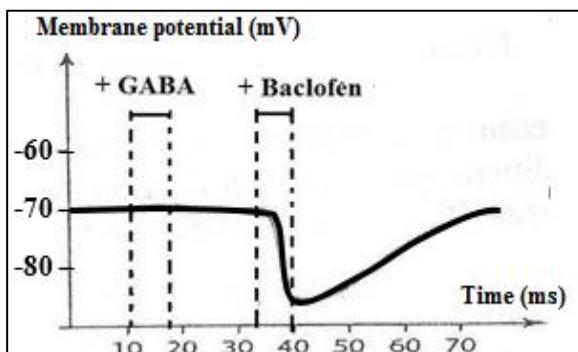
- 5- Verify if baclofen and GABA act on the same receptors.



Document 1



Document 2



Document 3

Exercise 4 (5 points)**Regulation of the Sexual Cycle**

In the framework of studying the functional relations between hypothalamus, pituitary gland, ovaries and uterus, a series of experiments are performed on the same female chimpanzee (A). The conditions and the obtained results are represented in document 1.

Experiment	Conditions	Results
1	Ablation of the pituitary gland in female chimpanzee (A)	Disappearance of ovarian and uterine cycles.
2	Ablation of the pituitary gland then periodic injections of anterior pituitary extracts in female chimpanzee (A)	Reestablishment of the ovarian and uterine activities.
3	Ablation of the pituitary gland then ablation of the ovaries followed by periodic injections of extracts from the anterior pituitary gland in female chimpanzee (A).	No reestablishment of the uterine activity.

Document 1

- 1- Interpret the obtained results.

In order to study the effect of the hypothalamus on the pituitary secretion, the following experiment is performed:

In a female chimpanzee (B), some specific cells of the hypothalamus are destroyed. The secretions of FSH and LH by the anterior pituitary gland decreased.

This female is injected with GnRH (hormone of the hypothalamus) in two manners: continuous and discontinuous.

The results are shown in document 2.

Hormones (ng.mL⁻¹)	Experimental conditions	Destruction of specific cells of the hypothalamus	Discontinuous injections of GnRH	Continuous injection of GnRH
	FSH	10	100	10
	LH	2.5	15	2.5

Document 2

- 2- Construct a histogram that represents the results obtained in document 2.
 3- Justify the following statement: “The secretion of FSH and LH is stimulated by the discontinuous secretion of GnRH by the hypothalamus”.

The ovaries secrete the hormones estrogen and progesterone.

- 4- Indicate one role for each of these ovarian hormones.

A moderate level of estrogen provokes a decrease in the level of FSH and LH (case 1). On the contrary, the high level of estrogen provokes an increase in the secretion of FSH and LH (case 2).

- 5- Name the type of feedback control revealed by each of the two cases, case 1 and case 2.
 6- Establish, by referring to all what precedes, a functional diagram showing the relations existing between the different organs involved in the regulation of the sexual cycles.

مسابقة في مادة علوم الحياة
اسس التصحيح

الاسم:
الرقم:

Part of the ex	Exercise 1 Pulmonary Emphysema	Grade
1	A progressive destruction of the lung tissue by the proteases of the white blood cells.	0.5
2	mRNA resulting from the transcription of the allele M1: ... AUC AAC GAU UAC ... Sequence of the amino acids of the polypeptide coded by the allele M1: ... -Ile - Asn - Asp - Tyr - ... mRNA resulting from the transcription of the allele M2: ... AUC AAC GAU UAG... Sequence of the amino acids of the polypeptide coded by the allele M2: ... -Ile - Asn - Asp	1
3	The mutation by substitution at the level of the 3rd nucleotide of triplet number 184 (C is replaced by G) is transcribed at the level of mRNA gives a truncated polypeptide having 183 amino acids instead of 418, leading to an non-functional protein alpha-antitrypsin (aT). This explains why alpha-antitrypsin is not found in the blood of an individual affected by pulmonary emphysema and consequently the pulmonary tissue is not protected against protease degradation and the patient shows manifestation of pulmonary emphysema.	1
4	The allele of the disease is recessive. The parents 1 and 2 are normal but gave two affected children 4 and 5. These children have taken the mutant allele from at least one of the parents. This parent does not phenotypically express the disease, so the mutant allele is being masked by the normal one. N: normal dominant allele. m: mutant recessive allele.	0.5
5	If the studied gene is carried on the non-homologous part of Y, in this case, any affected boy would necessarily have a sick father. For example, the affected boy 5 must have taken Y^m from his father who would have as genotype XY^m . Possessing such genotype, father 1 should be affected, which is not the case. If the studied gene is carried on the non-homologous part of chromosome X: in this case, the affected daughter 4 would have $X^m // X^m$ as genotype (purity is the criterion of recessivity). She should have taken one of her mutant alleles X^m from her father 1 who would have as genotype $X^m // Y$ who phenotypically should be affected, which is not the case. If the studied gene is carried on the homologous parts of X and Y: in this case, the affected boy 5 would have as genotype $X^m // Y^m$, and his sister 4 would have as genotype $X^m // X^m$. They have taken respectively Y^m and X^m from their father 1. This latter should have as genotype $X^m // Y^m$ and would be phenotypically affected. It's not the case. Therefore, the studied gene is not gonosomal but it is autosomal.	0.5
6	The genotype of individual 8 is $N//m$. He is phenotypically normal, possessing the normal dominant allele and the affected allele m is obligatory inherited from the homozygous diseased father 5.	0.5
7	Despite the presence of a normal allele in his genotype (heterozygous), individual 8 develops the same symptoms of pulmonary emphysema. Being a heavy smoker promotes the development of the disease. This shows that smoking is an environmental factor other than the genetic factor that could provoke this disease.	0.5

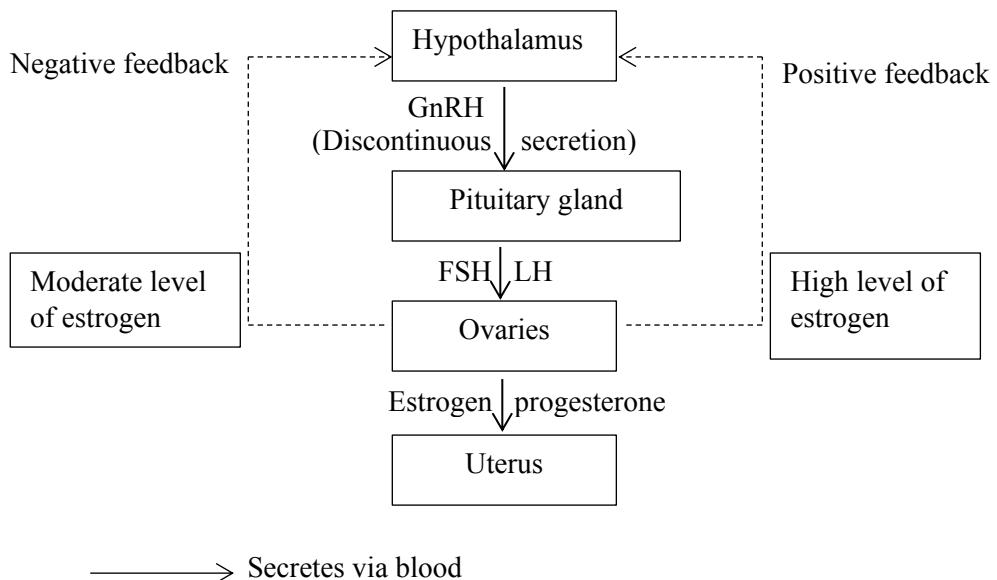
Part of the ex	Exercise 2 AIDS	Grade
1-1	The molecules recognized by HIV are: CD4 and CCR5	0.5
1-2	The target cell is: T4 cell	0.5
2-1	At birth, the electrophoreograms of both children 1 and 2 show the same 4 bands of the electrophoreogram of Mrs. Y. These bands are observed at the levels of anti-GP160, anti-GP120, anti- GP41 and anti-GP24 antibodies. However, at 6 th month, the number of these bands decreases in both electrophoreograms of children 1 and 2 to show bands at the levels of anti-GP160 and anti-GP120 antibodies only. On the other hand, at the 18 th month, the electrophoreogram of only child 2 shows the reappearance of the two bands which correspond to anti- Gp160 and anti-Gp120 compared to the electrophoreogram of child 1 that does not show any band.	1
2-2	Child 2, because he is seropositive at the age of 18 th month.	0.5
3	Hypothesis: The origin of antibodies present at birth in both children is maternal.	1
4	Yes, because the concentration of anti-HIV antibodies of maternal origin appears in the child at the fifth month during pregnancy and increases to reach maximum at birth. Then, this concentration of the produced antibodies decreases to null after 8 months of birth when the concentration of the antibodies produced by child 2 is approximately null. Therefore, the origin of anti-HIV antibodies present at birth is exclusively maternal.	1
5	The appearance of anti-HIV antibodies is a consequence of his contamination by HIV. In fact, after the infection by HIV, the immune system develops a reaction against the virus which is translated in the production of specific antibodies against the diverse viral protein Gp160, Gp120, Gp41, and Gp24.	0.5

Part of the ex	Exercise 3 GABA and Baclofen	Grade
1	Synapse S1 is excitatory since a hypopolarisation of amplitude 10 mv is obtained following stimulation of this nerve fiber 1. Synapse S2 is inhibitory since hyperpolarization of amplitude 5 mv is obtained following the stimulation of nerve fiber 2.	1
2	An EPSP is recorded at the level of the membrane of the motor neuron following the stimulation of the nerve fiber 1 or the injection of acetylcholine at the level of the synapse S1. This indicates that the acetylcholine fixes on its specific receptors on the membrane, while an IPSP is recorded at the level of the membrane of the motor neuron following the stimulation of the nerve fiber 2 or the injection of GABA at the level of the synapse S2. This indicates that the fixation of GABA on its specific receptor of the same motor neuron. Therefore, the motor neuron possesses different types of membrane receptors of neurotransmitters.	1
3	A hyperpolarization of amplitude 5mv is obtained at the level of the membrane of motor neuron following the injection of GABA, similarly a hyperpolarization but of higher amplitude (15 mV) is obtained after the injection of baclofen. This shows that baclofen has an inhibitory effect more amplified than that GABA.	1
4	GABA fixes on its specific post-synaptic membrane receptors of the chemical dependent Cl ⁻ channels. This provokes the opening of these channels followed by the entrance of Cl ⁻ ions leading to hyperpolarization. That's why GABA has an inhibitory effect.	1
5	In a medium deprived from Cl ⁻ , no variation in the membrane potential is observed at the level motor neuron in the presence of GABA. However, a hyperpolarization of 15 mv is recorded in the presence of baclofen. Hence, these two act on different receptors.	1

Part of the ex	Exercise 4 Regulation of the Sexual Cycle	Grade												
1	<p>A disappearance of the ovarian and uterine cycles is obtained following the removal of the pituitary gland of the female chimpanzee (A). This shows that the pituitary gland is indispensable for the ovarian and uterine cycles.</p> <p>However, the periodic injections of anterior pituitary extracts in female chimpanzee (A) restores the ovarian and uterine cycles. This shows that the anterior pituitary acts on the ovaries and uterus by releasing chemical substances in blood.</p> <p>On the other hand, the periodic injections of the extracts of the anterior pituitary at chimpanzee (A) that is submitted to the ablation of the ovaries do not restore the uterine cycle. This shows that the control of the pituitary on the uterus is indirect; it necessitates the intervention of the ovaries.</p>	1												
2	<table border="1"> <caption>Quantity of FSH and LH (ng/mL)</caption> <thead> <tr> <th>Injection Type</th> <th>FSH (ng/mL)</th> <th>LH (ng/mL)</th> </tr> </thead> <tbody> <tr> <td>Destruction of specific cells of the hypothalamus</td> <td>~10</td> <td>~2</td> </tr> <tr> <td>Discontinuous injections of GnRH</td> <td>~100</td> <td>~15</td> </tr> <tr> <td>Continuous injection of GnRH</td> <td>~10</td> <td>~2</td> </tr> </tbody> </table> <p style="text-align: right;"> 10 ng.mL⁻¹</p> <p>Variation of the amount of FSH, LH according to GnRH injection types</p>	Injection Type	FSH (ng/mL)	LH (ng/mL)	Destruction of specific cells of the hypothalamus	~10	~2	Discontinuous injections of GnRH	~100	~15	Continuous injection of GnRH	~10	~2	1.5
Injection Type	FSH (ng/mL)	LH (ng/mL)												
Destruction of specific cells of the hypothalamus	~10	~2												
Discontinuous injections of GnRH	~100	~15												
Continuous injection of GnRH	~10	~2												
3	<p>After the continuous injection of GnRH into a female chimpanzee (B), which is submitted to the destruction of specific cells of the hypothalamus, the levels of FSH and LH remain low (10 mg.mL^{-1} and 2.5 mg.mL^{-1}).</p> <p>On the contrary, when this chimpanzee is injected by GnRH discontinuously, the level of LH increases to 15 ng.mL^{-1} and that of FSH increases to 100 ng.mL^{-1}.</p> <p>This indicates that a moderate level of estrogen reduces the secretion of FSH and LH but the high level of estrogen stimulates the secretion of FSH and LH.</p>	0.75												
4	<p>Estrogen stimulates the proliferation of the uterine and vaginal mucosa. OR: Estrogen stimulates the development of the tube-like glands of the endometrium. Progesterone stimulates the gland secretions of the uterine mucosa and the cervix. OR: Progesterone stimulates the development of the spiral arterioles of the endometrium.</p>	0.5												
5	<p>Case 1: Negative feedback Case 2: Positive feedback.</p>	1												

6

0.75



Functional diagram showing the relation between the different organs participating in the regulation of the sexual cycles

الاسم:	مسابقة في مادة علوم الحياة
الرقم:	المدة: ثلاثة ساعات

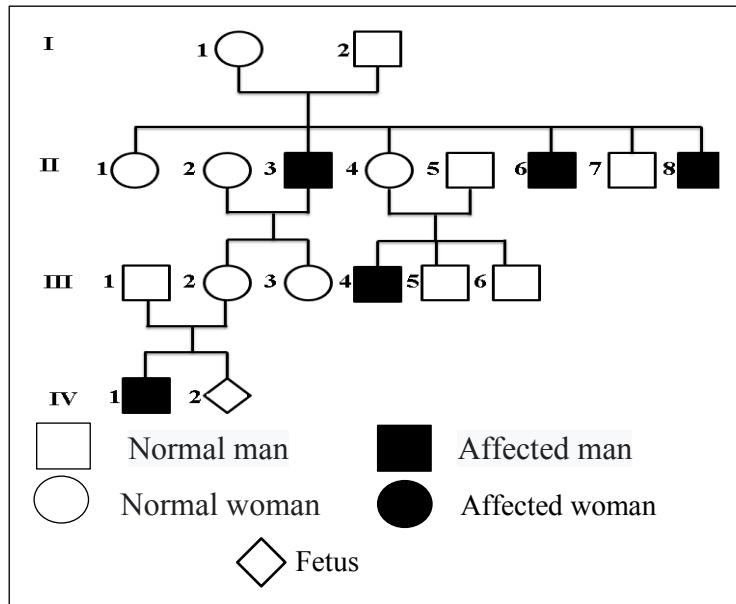
Exercise 1 (5 points)

Bruton Disease

Bruton disease is a genetic disease that affects one newborn in 200,000 birth. It is manifested by recurrent bacterial infections of the respiratory tract, starting from the age of six years. The disease predisposes patients to the risk of having chronic infections with viruses that attack particularly the digestive tract and the nervous system.

Document 1 represents the pedigree of a family, whose certain members are affected with Bruton disease.

- Indicate whether the allele responsible for Bruton disease is dominant or recessive. Justify the answer.
- Show that the gene responsible for the disease is localized on the non-homologous segment of chromosome X.
- Indicate the genotypes of individuals III₁, III₂ and IV₁.
- Determine the risk for the fetus IV₂ to be affected with this disease.



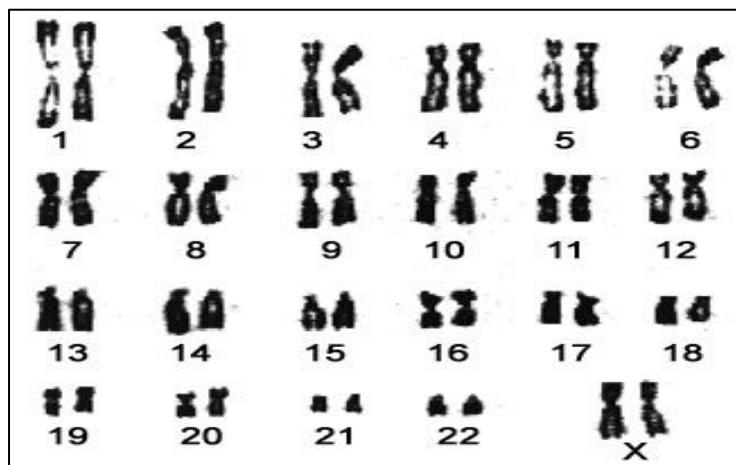
Document 1

To find out if the fetus IV₂ will be affected with Bruton disease, the doctor requests establishing the karyotype of the fetus. The obtained result is shown in document 2.

- Determine, referring to documents 1 and 2, if the fetus IV₂ will be affected by Bruton disease.

The doctor completes the diagnosis by performing DNA analysis using the Southern Blot technique. The used probe makes it possible to distinguish between the mutant allele and the normal allele of the gene involved in Bruton disease. The obtained results are shown in document 3.

- Specify the band that corresponds to the mutant allele.
- Draw out the genotype as well as the phenotype of the fetus IV₂.



Document 2

	III ₁	III ₂	IV ₁	Fetus IV ₂
A	—	—		—
B		—	—	—

Document 3

Exercise 2 (5 points)

Action of Antibodies and the Complement

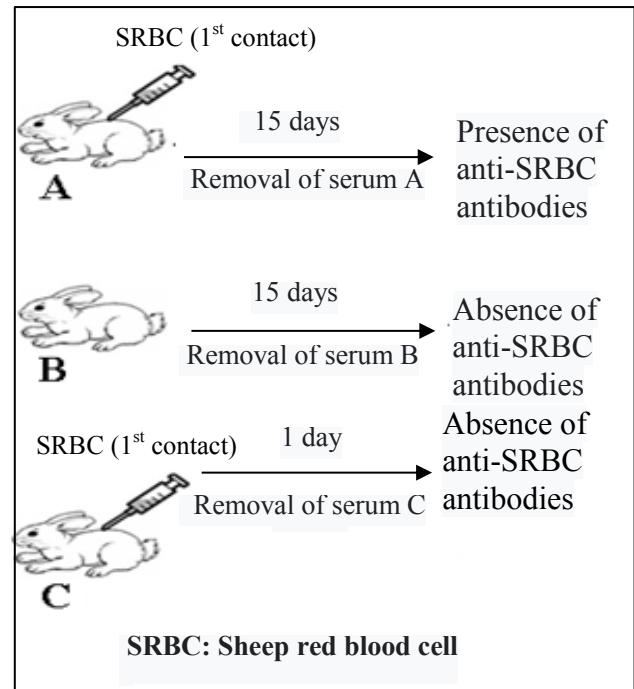
In order to study certain aspects of the action of antibodies, researchers performed experiments on three rabbits A, B and C. The experimental conditions and the obtained results are represented in document 1.

- 1- Show, by referring to document 1, that:
 - 1.1- The production of antibodies necessitates a prior contact with the concerned antigen.
 - 1.2- The production of antibodies necessitates a definite duration.
- 2- Explain the necessity of the 15 days, time delay to obtain the agglutination of SRBC by the serum of rabbits immunized against SRBC.

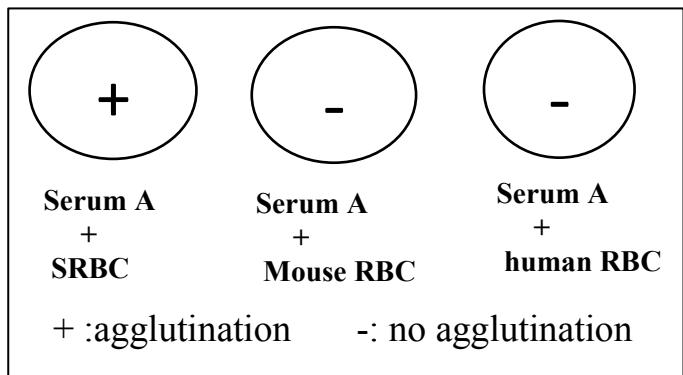
The serum taken from rabbit A, 15 days after being injected with SRBC, is placed in the presence of different substances. The conditions and the obtained results are shown in document 2.

- 3- Specify, referring to document 2, a characteristic of the humoral-mediated immune response.
- 4- Schematize the mechanism of agglutination of SRBC.

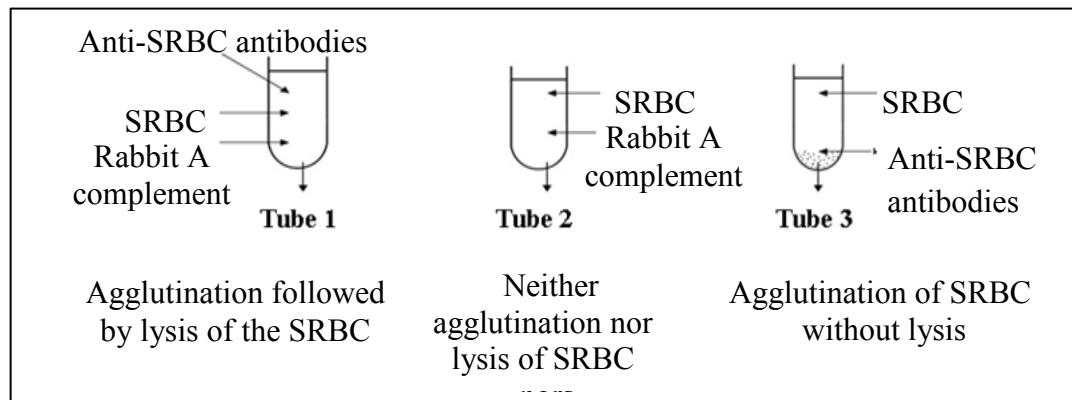
The complement is a set of plasma proteins. Researchers aim to show the role of the complement in the humoral immune response.



Document 1



Document 2



Document 3

- 5.1- Analyze the obtained results.
- 5.2- What can you conclude?

The experiment in tube 1 is repeated; the complement of rabbit A is replaced by the complement taken from a mouse. Agglutination of SRBC followed by their lysis is obtained.

- 6- Draw out a characteristic concerning the action of the complement.

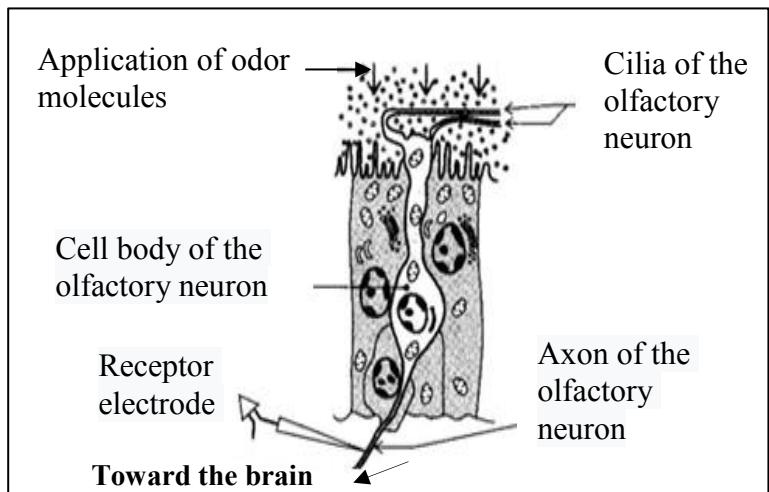
Exercise 3 (5 points)

Coding of the Olfactory Stimulus

Through olfaction (sense of smell), an organism is able to detect the presence of odor molecules in the air, estimate their concentration, and distinguish among different odors.

Odor molecules in the air bind to chemoreceptors found on the cilia of olfactory neurons. This results in a nerve message that propagates toward the brain (document 1).

- Pick out from the text, the localization of the olfactory chemoreceptors.



Document 1

Researchers expose cilia of olfactory neuron to increasing concentrations of an odor molecule. The frequency of the obtained AP is measured by a receptor micro-electrode placed on the axon of the olfactory neuron. Document 2 represents the obtained results.

- Represent, in a table, the results shown in document 2.
- Analyze the obtained results.
- What can you draw out concerning the coding of an olfactory stimulus?

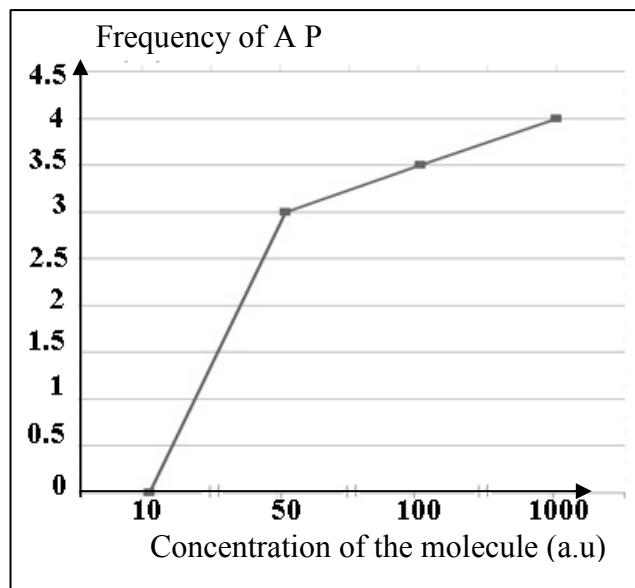
Researchers aim to study the action of three different odor molecules A, B and C on an olfactory neuron (document 3).

In the absence of any stimulus, the olfactory neurons show spontaneous activity that corresponds to a frequency of action potential of 100 a.u.

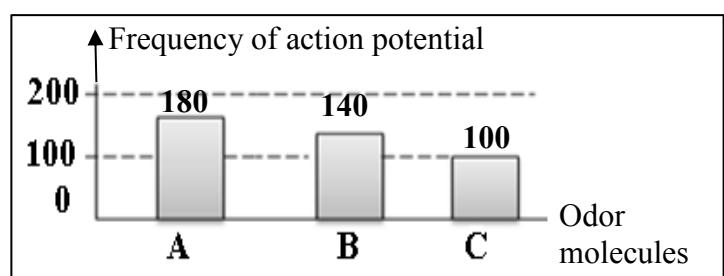
- Specify the molecule(s) which stimulate the olfactory neuron.

Document 4 represents the response of 4 different olfactory neurons in response to two odor molecules A and B.

- Show that the activity of the olfactory neuron depends on the nature of odor molecules.



Document 2



Document 3

Olfactory neurons		1	2	3	4
Frequency of A.P.	Odor molecule A	200	100	150	100
	Odor molecule B	100	150	100	180

Document 4

Exercise 4 (5 points)**Hormonal Regulation**

In order to study the relations existing between the hypothalamus, pituitary gland and ovaries, researchers performed the following experiments on adult female mammals.

Experiments	Conditions	Results
1	Electric stimulation on a specific zone of the hypothalamus	Hyperactivity of the anterior pituitary with hypersecretion of pituitary hormones, FSH and LH
2	Hypophysectomy (ablation of the anterior pituitary)	Atrophy of the ovaries and the uterus with disappearance of the sexual cycles

Document 1

- 1- Draw out the role of:

1.1- the hypothalamus.

1.2- the anterior pituitary.

Experiment 3: researchers inject the ovarian hormones to ovariectomized adult female mammals and then they measure the variations of the levels of LH and FSH. The conditions and the results are shown in document 2.

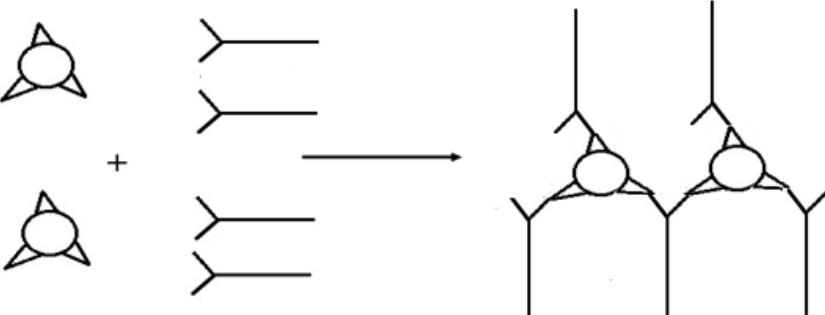
Experiment	Conditions	Results																			
3	Case A: Injection of a moderate dose of estrogen. Case B: Injection of a high dose of estrogen. Case C: Injection of high dose of estrogen and progesterone.	<table border="1"> <thead> <tr> <th></th> <th>Before the injection</th> <th>Case A</th> <th>Case B</th> <th>Case C</th> </tr> </thead> <tbody> <tr> <td>FSH (ng/mL)</td> <td>12</td> <td>3</td> <td>18</td> <td>3</td> </tr> <tr> <td>LH (ng/mL)</td> <td>40</td> <td>10</td> <td>65</td> <td>10</td> </tr> </tbody> </table>		Before the injection	Case A	Case B	Case C	FSH (ng/mL)	12	3	18	3	LH (ng/mL)	40	10	65	10				
	Before the injection	Case A	Case B	Case C																	
FSH (ng/mL)	12	3	18	3																	
LH (ng/mL)	40	10	65	10																	

Document 2

- 2- Draw a histogram that represents the data of experiment 3.
- 3- Indicate the types of the feedback revealed in experiment 3. Justify the answer.
- 4- Name the structures that secrete estrogen during a sexual cycle.
- 5- Establish, by referring to the preceded experiments, a functional diagram showing the relations between the ovaries, the anterior pituitary and the hypothalamus.

الاسم:	علوم الحياة
الرقم:	أسس التصحيح

Part of ex	Exercise 1 (5 points)	Bruton Disease	Grade
1	The allele responsible for the disease is recessive, since individuals 3, 6 and 8 are affected from healthy parents. This means that the mutant allele is present in the parents but masked by the normal allele (N).		0.5
2	There is discrimination of sex, only males are affected, so the allele of the disease is sex-linked. If the studied gene were localized on the non-homologous segment of chromosome Y, any affected boy should have an affected father, which is not the case (normal father I2 has affected boys). So the studied gene is localized on the non-homologous segment of chromosome X.		1
3	N: normal allele dominant m : affected allele recessive III ₁ : X ^N Y III ₂ : X ^N X ^m IV ₁ : X ^m Y		0.75
4	The fetus IV ₂ has a heterozygous mother (III ₂) of genotype X ^N X ^m . If the fetus were a boy, the risk of this fetus, in this case, to receive the chromosome Xm from his mother will be ½. He obligatory will receive chromosome Y from his father. If the fetus were a girl, the risk will be null since she will receive obligatory X ^N from her healthy father of genotype X ^N Y. Therefore, the fetus will be necessarily healthy regardless of the gamete received from her father, N is dominant.		0.75
5	The karyotype (document 2) shows that the fetus is a girl with two chromosomes X. Referring to the pedigree (document 1), the fetus has a healthy father of genotype X ^N Y. Thus, he must have obligatory received X ^N from his father and then the fetus will be a healthy girl since allele N is dominant, then the female is healthy		1
6	Band B corresponds to the mutated allele, because document 3 shows that male IV ₁ of genotype X ^m Y possesses a single band B.		0.5
7	Fetus: genotype X ^N X ^m Phenotype : Healthy [N]		0.5

Part of ex	Exercise 2 (5points) Mode of Action of Antibodies and the Complement	Grade
1.1	<p>Anti-SRBC antibodies are only found in the serum taken from rabbit A which is injecting with SRBC 15 days after the injection. However, no anti-SRBC antibodies are found in the serum taken from rabbit B, being not injected with SRBC.</p> <p>This shows that, the production of antibodies necessitates a prior contact with the concerned antigen.</p>	0.5
1.2	<p>Anti-SRBC antibodies are only found in serum of rabbit A 15 days being injected with SRBC but not after 1 day from the injection (rabbit B). Therefore, the production of antibodies necessitates a definite duration.</p>	0.5
2	<p>15 days is the time delay necessary for the induction of a humoral immune response during which :</p> <ul style="list-style-type: none"> activation of B lymphocytes by IL4 secreted by activated T4 lymphocytes and following their multiplication and differentiation to plasmocytes that secrete anti-SRBC antibodies. 	1
3	<p>The humoral immune response is specific, because agglutination occurs only when adding the serum of an immunized rabbit against the SRBC with the same antigen, SRBC.</p> <p>(no agglutination occurs neither with RBC of a mouse nor with RBC of a human).</p>	0.5
4	 <p style="text-align: center;">SRBC Anti-SRBC antibodies Immune complex (Agglutination)</p>	0.5
5.1	<p>There is agglutination with no lysis, when the SRBCs are mixed with only anti-SRBC antibodies (having no complement). However; there is neither agglutination nor lysis, when the SRBCs are mixed with complement in the absence of antibodies.</p> <p>On the other hand, there is agglutination followed by lysis when anti- SRBC antibodies are mixed with SRBC and the complement rabbit A.</p>	1
5.2	<p>Antibodies are responsible for the agglutination of the antigen but the complement is responsible for the lysis of only antigens previously agglutinated by antibodies.</p>	0.5
6	<p>The action of complement protein is not specific.</p>	0.5

Part of ex	Exercise 3 (5 points) Coding of the Olfactory Stimulus	Grade										
1	The cilia of the olfactory neuron.	0.5										
2	<table border="1"> <tr> <td>Concentration of the molecules (a.u.)</td> <td>10</td> <td>50</td> <td>100</td> <td>1000</td> </tr> <tr> <td>Frequency of AP (a.u.)</td> <td>0</td> <td>3</td> <td>3.5</td> <td>4</td> </tr> </table> <p>Variation of the frequency of AP (a.u.) as a function of the concentration of the molecules (a.u.)</p>	Concentration of the molecules (a.u.)	10	50	100	1000	Frequency of AP (a.u.)	0	3	3.5	4	1
Concentration of the molecules (a.u.)	10	50	100	1000								
Frequency of AP (a.u.)	0	3	3.5	4								
3.1	The frequency of AP increases sharply from 0 to 3 a.u. with the increase in concentration of the odor molecules from 0 to 50 a.u. Then, such variation shows slight increase from 3 to 4 a.u. when the concentration of the molecules increases from 50 to 1000 a.u.	1										
3.2	The intensity of the stimulation is modulated by frequency of AP.	0.5										
4	The frequency of AP for the odor molecule A (approximately 180 a.u.) is greater than that obtained by the odor molecule B (approximately 140 a.u.). However, the frequency of action potentials for odor molecule C is low (100 a.u) which corresponds to the spontaneous activity of the olfactory neuron. Thus, odor molecules A and B stimulate this olfactory neuron.	1										
5	Neurons 1 and 3 are excited by the odor molecule A with 200 AP and 150 AP respectively, but not excited by the odor molecule B, they show an activity (100 AP) similar to the spontaneous activity in the absence of any stimulation. However, neurons 2 and 4 are excited by the odor molecule B with 150 and 180 AP respectively; they are not excited by the odor molecule A with 100 AP. Thus, the activity of the olfactory neurons depends on the nature of the odor molecule.	1										

Part of ex	Exercise 4 (5points)	Hormonal Regulation	Grade															
1.1	The hypothalamus stimulates the secretion of the pituitary hormones FSH and LH.		0.5															
1.2	The anterior pituitary is responsible for the development of the ovaries and uterus, and for their cyclic activities.		0.5															
2	<p>The variation of FSH and LH as a function of the injected hormones.</p> <table border="1"> <caption>Data from Figure 2: Variation of FSH and LH levels (ng/mL)</caption> <thead> <tr> <th>Condition</th> <th>FSH (ng/mL)</th> <th>LH (ng/mL)</th> </tr> </thead> <tbody> <tr> <td>Before injection</td> <td>12</td> <td>40</td> </tr> <tr> <td>Case A</td> <td>2</td> <td>10</td> </tr> <tr> <td>Case B</td> <td>18</td> <td>65</td> </tr> <tr> <td>Case C</td> <td>2</td> <td>10</td> </tr> </tbody> </table>	Condition	FSH (ng/mL)	LH (ng/mL)	Before injection	12	40	Case A	2	10	Case B	18	65	Case C	2	10		1
Condition	FSH (ng/mL)	LH (ng/mL)																
Before injection	12	40																
Case A	2	10																
Case B	18	65																
Case C	2	10																
3	<p>Negative feedback: moderate dose of estrogen alone (case A) or the presence of high dose of estrogen coupled with progesterone (case C) decreases respectively the secretion of FSH and LH from 12 to 3 and from 40 to 10 mg/mL</p> <p>Positive feedback: High dose of estrogen (case B) increases respectively the secretion of FSH and LH from 12 to 18 and from 40 to 65 mg/mL.,</p>		1															
4	<p>Estrogen is secreted by:</p> <p>Follicular cells and those of the internal theca during the follicular phase.</p> <p>Luteal cells during the luteal phase.</p>		0.5															
5	<p>Functional diagram showing the relations between the ovaries, the anterior pituitary and the hypothalamus.</p> <p>(-): Negative feedback (+): Positive feedback</p>		1.5															

المادة: علوم الحياة الشهادة: الثانوية الفرع: علوم الحياة نموذج رقم: ١-١ المدة: ٣ ساعات	الهيئة الأكademية المشتركة قسم : العلوم	 المركز التعليمي لبحوث والإنماء
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نموذج مسابقة (يراعي تعليق الدروس والتوصيف المعدل للعام الدراسي ٢٠١٩-٢٠١٨ وحتى صدور المناهج المطورة)

Exercise 1 (5 points) Young Girls Becoming Males!

Some girls in Salinas, a village in the Dominican Republic Islands, become boys around the age of 12 years by developing their external genital organs.

The parents of Jeanne, a 7-year-old girl from Salinas, consulted a doctor to know if their daughter will suffer from this abnormality. The doctor initially demanded a karyotype for Jeanne and her parents. The results are presented in document 1 that shows only the sex chromosomes X & Y.

- What problem is posed upon studying the karyotype of Jeanne?

Chromosome Y carries a gene named SRY which is responsible for determining the masculine phenotype. The doctor performed a DNA analysis for the family members. The obtained electropherogram is presented in document 2.

- Show that Jeanne's anomaly is not due to the absence of the SRY gene.

SRY gene codes for "TDF protein" which activates testosterone during embryonic life leading to the development of testicles in an embryo of karyotype XY.

Document 3 shows the partial sequences of amino acids of a functional TDF protein (A), a non-functional TDF protein (B) and a TDF protein (C) of Jeanne.

- Does the result of document 3 reveal the origin of Jeanne's anomaly? Justify the answer.

In males, testosterone hormone favors the development of primary and secondary sexual characteristics.

During embryonic life, testosterone becomes active in the presence of 5 α reductase enzyme.

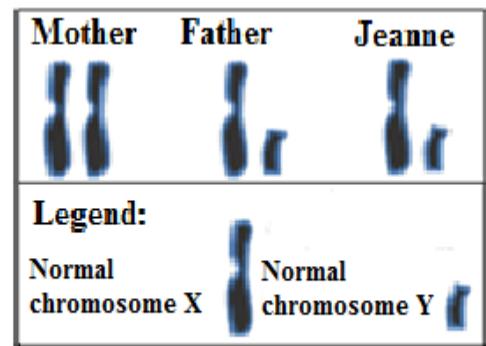
At puberty, around the age of 12, testosterone is active without the presence of this enzyme.

The pedigree in document 4 shows the family members of Jeanne with active or inactive form of 5 α reductase enzyme. Individuals 5, 12 and 15 show feminine phenotype before the age of 12. Jeanne's mother 8 and the woman 11 have similar karyotypes.

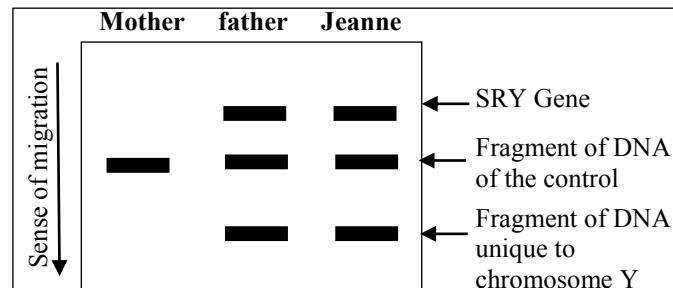
- Specify if the allele that determines the inactive form of 5 α reductase is dominant or recessive.

- Determine the chromosomal location of the gene responsible for the synthesis of 5 α reductase enzyme.

- Explain why Jeanne who is born with a feminine phenotype becomes a boy at the age of 12.



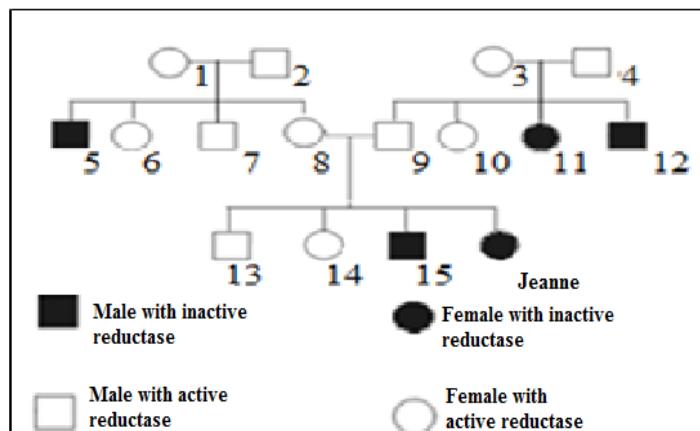
Document 1



Document 2

1	5	10
A :Met-Gln-Asp-Arg-Val-Lys-Arg-Pro-Met-Asn...		
B :Met-Gln-Asp-Arg-Val-Lys-Arg-Pro- Ile- Asn...		
C :Met-Gln-Asp-Arg-Val-Lys-Arg-Pro-Met-Asn...		

Document 3



Document 4

Exercise 2 (5 points)

Graft and Immunological memory

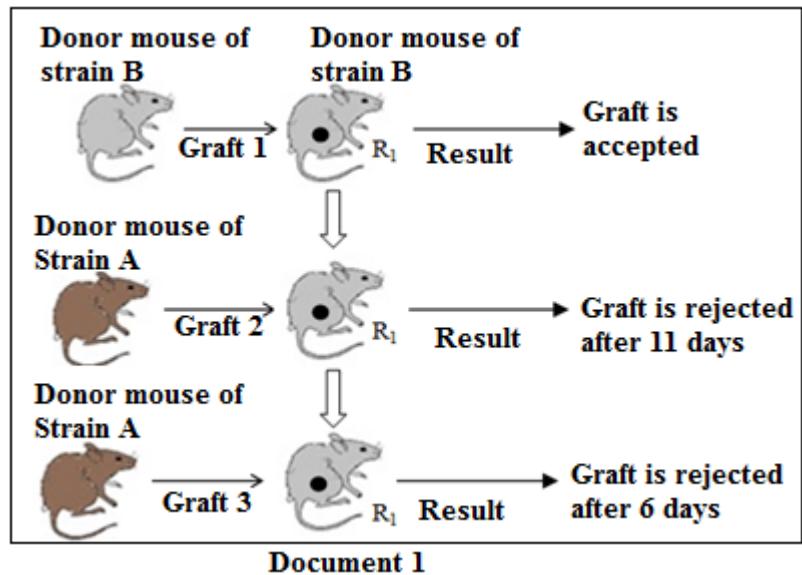
A study is performed to specify the mechanism of immunity involved in the rejection of skin graft in mice. Skin grafts are performed between different strains of mice, strain A and strain B. Document 1 shows the experimental conditions as well as the obtained results. The receiver mouse R1 is the same in the three cases of grafting.

1. Interpret the obtained results.

In order to explain the results of the third graft, two hypotheses are proposed:

Hypothesis 1: Mice B possess memory T lymphocytes against the antigens carried by the cells of mice A.

Hypothesis 2: Mice B possess antibodies against the antigens carried by the cells of mice A.



Mice of strain B are hyper-immunized by grafting them for three times by, three weeks apart, by skin from mice of strain A. Then, the researchers extracted from these hyper-immunized mice of strain B serum (blood plasma) or lymphoid cells from lymphatic ganglia close to the graft on the other hand.

An experiment is performed on mice of strain B called “Nude” (named NB), which are not subjected to any prior treatment. The conditions and the results are shown in document 2.

Day 1 : Injection of mice NB	Day 3 : Grafts done on mice NB	Result
Serum from the hyper-immunized mice of strain B	Skin from mice of strain A	On Day 6: Acceptance of the graft On day 11: Rejection of the graft
Alive lymphoid cells from the hyper-immunized mice of strain B	Skin from mice of strain A	On day 6: Rejection of the graft
Dead lymphoid cells from the hyper-immunized mice of strain B	Skin from mice of strain A	On day 6: Acceptance of the graft On day 11: Rejection of the graft

Document 2

2. Verify, by referring to doc.1 and doc.2, which of the preceding formulated hypotheses is valid.

The analysis of the lymphoid cells, responsible for graft rejection, present in the hyper-immunized mice gives the results presented in document 3.

3. Identify the cells X and Y in document 3.

4. Explain, by referring to all what precedes, the result of graft 3 in document 1.

	Hyper-immunized mice	
	Lymphoid cells X	Lymphoid cells Y
Percentage	95 %	5 %
Life Span	few days to few dozens of days	few months to few dozen of years
Proliferation	No	Yes

Exercise 3 (5 points)

Anesthesia and curare

Muscle relaxants, such as D-tubocurarine, a synthetic curare molecule administered as part of general anesthesia. They allow muscle relaxation. In cosmetic surgery, they utilize muscular relaxant by injecting them into muscles to reduce facial wrinkles.

In order to explain the role and the mode of action of D-tubocurarine in cosmetic surgery, the following experiments are performed.

Experiment 1:

The axon of a motor neuron is effectively stimulated in the absence and then in the presence of curare injected in the neuromuscular junction. The electrical activity of the muscle fiber is measured. The experimental setup is represented in document 1 and the obtained recordings in document 2.

1. Indicate the role the neuromuscular junction.
2. Justify, by referring to document 2, the role of curare as a muscular relaxant.

Experiment 2:

A skeletal muscle is isolated from a frog. It is placed in a physiological bath, in the presence of increasing concentrations of acetylcholine, a neurotransmitter of the motor neuron. The amplitude of muscular contraction of the muscle, under different concentrations of acetylcholine, is recorded. The measurements are performed in the absence or in the presence of same amount of D-tubocurarine. The results are shown in document 3.

3. Construct, on the same graph, the curves that show the variation of the amplitude of muscular contraction as a function of acetylcholine concentration, with and without D-tubocurarine.

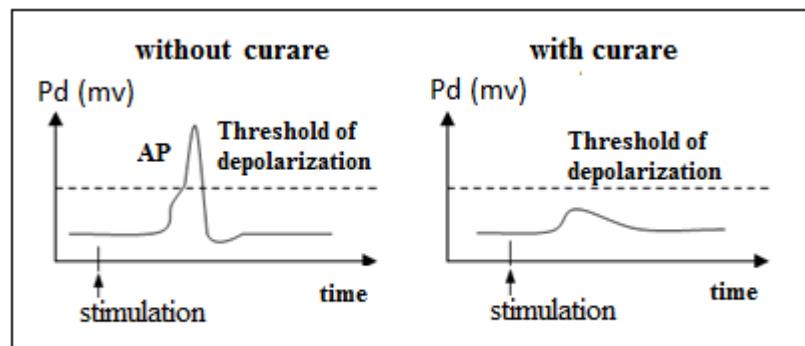
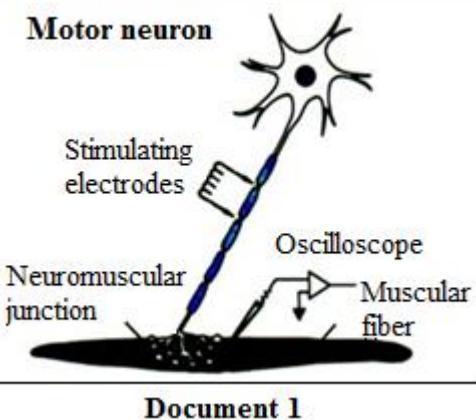
4.1. Analyze the obtained results.

4.2. Conclude the effect of D-tubocurarine on acetylcholine.

Acetylcholine interacts at the level of the postsynaptic membrane with a specific receptor consisting of 5 protein subunits named A, B, C, D and E.

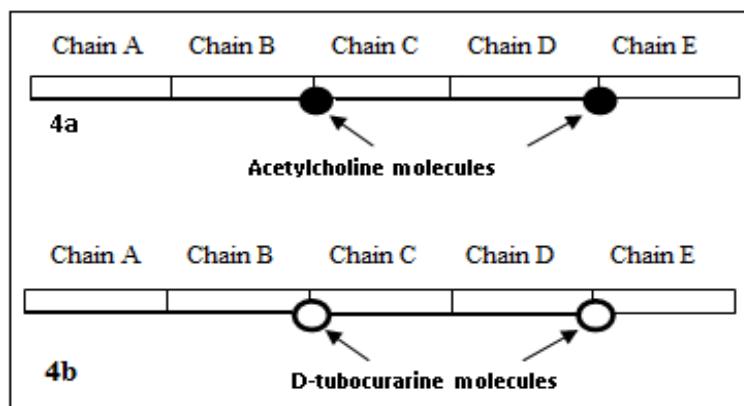
Document 4 represents the 5 subunits of the receptor in the presence of acetylcholine (**4a**) or D-tubocurarine (**4b**).

5. Determine, based on document 4, the mode of action of D-tubocurarine.
6. Explain, from what precedes, the usage of D-tubocurarine in cosmetic surgery to reduce facial wrinkles.



Concentration of acetylcholine (in M.L ⁻¹)	Amplitude of Contraction (a.u)	
	without D-tubocurarine	with D-tubocurarine
10 ⁻⁴	5	0
10 ⁻³	10	3
10 ⁻²	20	12
10 ⁻¹	25	20

Document 3



Document 4

Exercise 4 (5 points)

Type 2 Diabetes

وقف العمل بهذا المhour (التعيم رقم ٢٨٠١٨/٥/٢١ تاريخ ٢٠١٨/٥/٢٨)

Type 2 diabetes (T2D) often affects obese people and individuals who consume high amounts of lipids. It develops gradually throughout the years.

In the framework of studying the physiological causes of T2D, researchers performed the following experiments.

Experiment 1

Non-diabetic individuals and individuals affected by T2D are subjected to provoked hyperglycemia test during which each of them ingests 75 g of glucose. Then, the glycemia rate is measured in each of them during 120 minutes. The results are represented in document 1.

- Interpret the obtained results.
- Formulated two hypotheses concerning the origin of type 2 diabetes.
- Show, by referring to document 2, that treating individuals with T2D with insulin is not effective.

Experiment 2:

Fragments of identical masses of muscle tissues, target cells of insulin, are isolated from normal mice and from obese mice affected by diabetes which is similar to T2D in humans.

Each fragment of tissue is then placed in a medium containing the same concentration of insulin. 10 minutes later, the amount of glucose absorbed by the muscular cells of these tissues is measured.

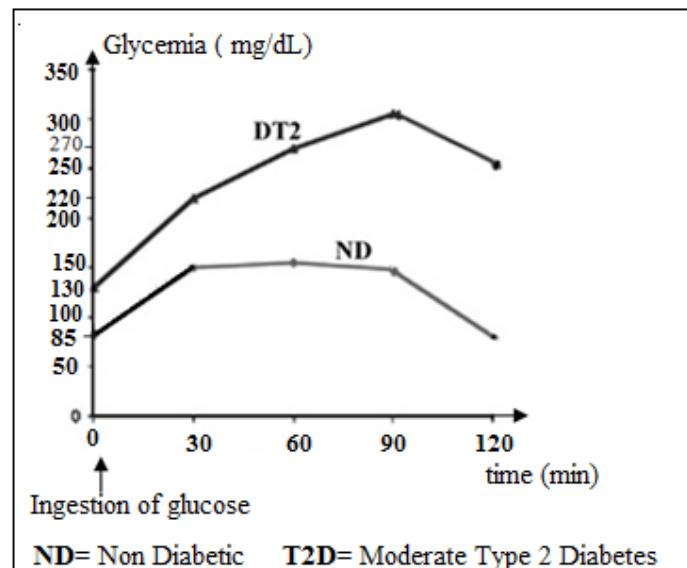
The results are presented in document 3.

- What can be deduced from the results in document 3?

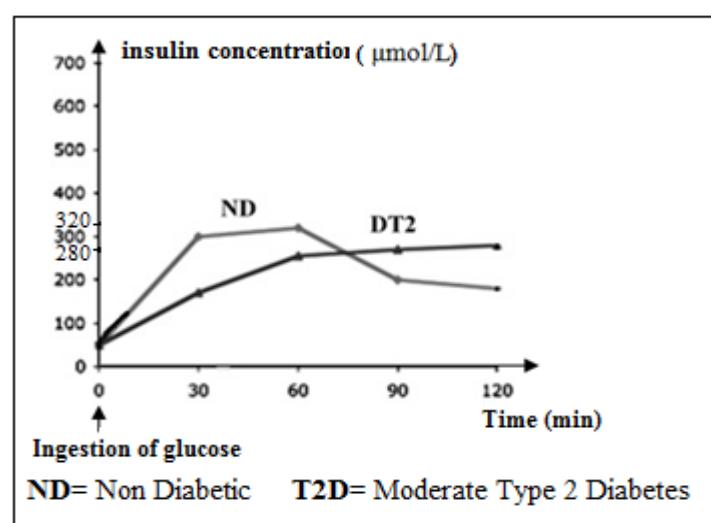
Experiment 3:

The plasma membranes of muscle cells isolated from normal mice and from obese mice affected with diabetes are placed in two culture media in the presence of the same concentration of radioactive insulin. The quantity of insulin fixed on the receptors of these membranes is measured and presented in document 4.

- Determine, by referring to document 4, the origin of type 2 diabetes.



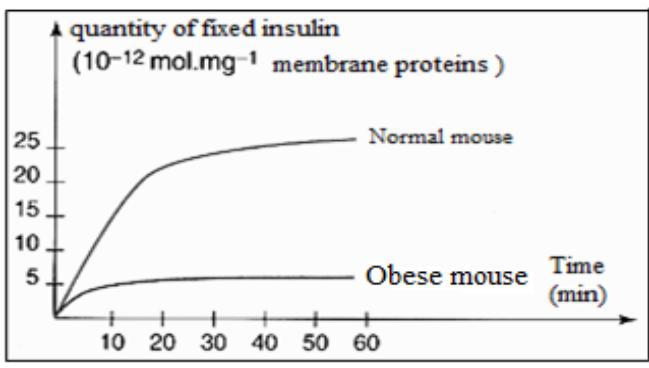
Document 1



Document 2

Quantity of glucose absorbed by the muscle cells ($\text{nmol} \cdot \text{mg}^{-1}$ of tissue)	Normal Mice	Obese Mice
	5	3

Document 3



Document 4

المادة: علوم الحياة الشهادة: الثانوية الفرع: علوم الحياة نموذج رقم ١-١ المدة: ٣ ساعات	الهيئة الأكاديمية المشتركة قسم : العلوم	 المجلس الأكاديمي للبحوث والإنماء
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أسس التصحيح (تراري تعليق الدروس والتوصيف المعدل للعام الدراسي ٢٠١٨-٢٠١٩ و حتى صدور المناهج المطورة)

Parts of ex	Exercise 1 (5 points)	Mark
1	Why Jeanne shows a feminine phenotype although she possesses X and Y sex chromosomes?	0.5
2	The DNA analysis of Jeanne and her father shows a band corresponding to SRY gene and another corresponding to a DNA fragment unique to Y chromosome. On the other hand, The DNA analysis of the mother shows the absence of both bands. Therefore, Jeanne possesses the SRY gene and her anomaly is not due to the absence of this gene.	0.75
3	The result in document 3 doesn't reveal the origin of Jeanne's anomaly. In fact, the partial sequence of amino acids of TDF protein in Jeanne (C) is identical to that of the functional TDF protein (A). This shows that Jeanne possesses the functional TDF coded by a normal allele of SRY gene.	0.25 0.5
4.1	The allele that determines the inactive form of 5α reductase is recessive with respect to the allele that determines the active form, since the affected individual 5 (or 11, 12 and 15) have normal parents 1 and 2. So, these parents have the allele responsible for deficiency but it is not expressed phenotypically and masked. Let d be the symbol of the allele responsible of the inactive form of 5 α reductase enzyme. Let N be the symbol of the allele responsible of the active form of 5 α reductase enzyme.	0.5
4.2	If the gene that determines the abnormality is carried by the non-homologous segment of the Y chromosome, then boys inherit the chromosome Y ^d from their fathers and should have the same phenotype. However, all the affected boys (5, 12 and 15) have "normal" fathers (2, 4 and 9) respectively. So, the gene is not located on the non-homologous segment of Y chromosome. If the gene that determines the abnormality is carried by the non-homologous segment of the X chromosome, the affected female 11 of recessive phenotype should be homozygous having received one chromosome X ^d from each of the parent 3 and 4. So, Her father 4 would be of genotype X ^d Y and then he will have abnormal phenotype. However her father 4 is normal. Hence, the gene is not located on the non- homologous segment of X chromosome. If the allele that determines the anomaly is carried by the homologous segment of the X and Y chromosomes, the affected male 12 should be homozygous of genotype X ^d Y ^d and then he should have inherited the Y ^d chromosome from his father 4. His affected sister 11, of recessive phenotype, should also be homozygous of genotype X ^d X ^d and inherited X ^d chromosome from each of her parents. For this reason, their father 4 should be homozygous and affected. However, he is normal. So, the gene is not located on the homologous segment of chromosomes X and Y. Therefore, the gene responsible of the synthesis of 5 α reductase enzyme is localized on an autosome.	1.25

5	<p>Jeanne possesses the gonosomes (sex chromosomes) X Y and the normal allele of the SRY gene on the Y chromosome, but based on the pedigree, she possesses the inactive 5 α reductase enzyme. However, this enzyme is indispensable for activating testosterone during the embryonic life. So, testosterone remains inactive during embryonic life thus leading to the inhibition of the masculine phenotype appearance before the age of 12.</p> <p>On the other hand, the secretion of active testosterone increases just before puberty without the need of the 5 α reductase enzyme. Since testosterone favors the development of primary and secondary sexual characteristics, the masculine phenotype will appear and Jeanne will become a boy at the age of 12.</p>	1.25
Parts of ex	Exercise 2 (5 points)	Mark
1	<p>The skin graft is accepted when it is performed from a mouse of strain B to a mouse of the same strain B (graft 1). However, it is rejected after 11 days if the skin tissue is done between two mice of different strains: a donor mouse of strain A and a receiver mouse of strain B (graft 2).</p> <p>This shows that the graft is only accepted between individuals of the same strain.</p> <p>The graft rejection (graft 3) between two different strains A and B happens after 6 days, less than 11 days for graft 2 when the mouse of strain B has previously rejected the first skin graft issued from mouse of strain A.</p> <p>This shows that the immune response responsible for graft rejection is much faster during the second contact with the same antigen.</p>	1.5
2	<p>When serum from hyper-immunized mice of strain B is injected into "nude" mice (BN) of strain B followed by transplanting in them skin graft from mouse of strain A; 11 days later, the graft is rejected at the same duration as a control mouse in graft 2 in document 1, which has never been in contact with the antigen of mouse A.</p> <p>This means that, the serum of hyper-immunized strain B has no effect in the rejection of the graft.</p> <p>Therefore, the hypothesis, which states that mice B have antibodies which are at the origin of graft rejection, is invalid.</p> <p>When alive lymphoid cells taken from hyper-immunized mice B are injected into "nude" mouse of strain B (BN) then, followed by transplanting in them skin tissue from mouse A ; after shorter duration of time, 6 days later, the graft is rejected, similar to duration of time required by the mouse which receives the same graft for the second time, graft 3 of document 1. Moreover, the graft is always rejected at day 11 in the control mice of strain B that are injected by killed lymphoid cells taken from hyper-immunized mice of strain B.</p> <p>This means that the lymphoid cells are responsible for triggering response against the antigen.</p> <p>Hence, the hypothesis which states that mice B possess immune memory cells which are at the origin of graft rejection is valid.</p>	1.5
3	<p>Cells X are short-lived cells which life span range from days to tens of days and are involved in the cell-mediated immune response. Since differentiated immune cells have a short life span, hence these cells are the effector cells, Tc.</p> <p>Cells Y have a long life span, few months to tens of years, and they can proliferate. Since the cells having these characteristics are memory cells which appear after the first contact with the antigen, so cells Y are memory cells. And since this is a cell-mediated specific immune response, then cells Y are Tc memory cells.</p>	1

4	<p>Mice R1 of strain B develops a primary specific cell mediated immune response against graft A (graft 1). The activated Tc cells proliferate and give a clone of lymphocytes. Some of the daughter cells differentiate into "effector" cytotoxic Tc and others become memory cells specific against antigen A.</p> <p>After the second contact with the same graft A, memory Tc cells proliferate rapidly and differentiate to cytotoxic Tc, ensuring the rapid rejection of the graft.</p> <p>Since, the triggered secondary immune response is faster, the rejection of the skin tissues in graft 3 of document 1 is obtained after 6 days instead of 11 days.</p>	1																		
Parts of ex	Exercise 3 (5 points)	Mark																		
1	The role of the neuromuscular junction is that it permits the transmission of the motor message to the muscle.	0.5																		
2	<p>In the absence of curare, an action potential (A.P) is observed, upon effectively stimulating the axon of motor neuron. However, in the presence of curare, the post synaptic membrane shows slight hypo-polarization (EPSP) less than the threshold of depolarization, with no recording of action potential at the level of muscle fiber</p> <p>Hence, curare prevents the genesis of Action potential at the level of the muscle fiber and consequently the contraction of the muscle thus playing the role of a muscular relaxant.</p>	0.75																		
3	<p>Title: Graph representing the variation of the amplitude of contraction of the muscle as a function of the acetylcholine concentration, with and without D-tubocurarine.</p> <table border="1"> <caption>Data points estimated from the graph</caption> <thead> <tr> <th>Concentration of Ach ($M.L^{-1}$)</th> <th>Amplitude (a.u.) without D-tubocurarine</th> <th>Amplitude (a.u.) with D-tubocurarine</th> </tr> </thead> <tbody> <tr> <td>10^{-5}</td> <td>5</td> <td>0</td> </tr> <tr> <td>10^{-4}</td> <td>10</td> <td>2</td> </tr> <tr> <td>10^{-3}</td> <td>20</td> <td>5</td> </tr> <tr> <td>10^{-2}</td> <td>25</td> <td>10</td> </tr> <tr> <td>10^{-1}</td> <td>-</td> <td>20</td> </tr> </tbody> </table>	Concentration of Ach ($M.L^{-1}$)	Amplitude (a.u.) without D-tubocurarine	Amplitude (a.u.) with D-tubocurarine	10^{-5}	5	0	10^{-4}	10	2	10^{-3}	20	5	10^{-2}	25	10	10^{-1}	-	20	1.75
Concentration of Ach ($M.L^{-1}$)	Amplitude (a.u.) without D-tubocurarine	Amplitude (a.u.) with D-tubocurarine																		
10^{-5}	5	0																		
10^{-4}	10	2																		
10^{-3}	20	5																		
10^{-2}	25	10																		
10^{-1}	-	20																		
4.1	<p>The amplitude of muscle contraction increases from 5 a.u to 25 a.u.in the absence of D-tubocurarine and similarly the amplitude of muscle contraction increases from 0 to 20 a.u in the presence of D-tubocurarine when the concentration of acetylcholine increases from $10^{-4} M.L^{-1}$ to $10^{-1} M.L^{-1}$. However, the latter amplitudes of contraction remain all the time less than that obtained in the absence of D-tubocurarine for each concentration of acetylcholine.</p>	0.5																		
4.2	We can conclude that D-Tubocurarine attenuates the action of acetylcholine on the muscle fibers.	0.25																		

5	<p>Document 4a shows that two molecules of acetylcholine bind to the receptor, one molecule of acetylcholine between chains B and C and another between chains D and E. Document 4b shows that D-Tubocurarine molecules bind to the same acetylcholine receptor between the same chains.</p> <p>Therefore, D-Tubocurarine replaces acetylcholine on postsynaptic receptors at the level of the muscle fiber and prevents the effect of acetylcholine.</p>	0.5
6	<p>The binding of D-Tubocurarine molecules to acetylcholine receptors prevents this neurotransmitter from binding to its receptors and stimulating muscle fibers. Thus, the molecules of D-Tubocurarine attenuate the action of acetylcholine on the facial muscles. The latter do not contract anymore and relax, which leads to the disappearance of the facial wrinkles.</p>	0.75
Parts of ex	وقف العمل بهذا المحور (التعليم رقم ٢٨/م تاريخ ٢٠١٨/٥/٢١). Exercise 4 (5 points)	Mark
1	<p>At $t = 0$ min, the glycemia level is 85 mg / dL in the non-diabetic individual, lower than that in the diabetic individual which is 130 mg / dL. So, the glycemia level is more important in a diabetic individual than in a healthy individual.</p> <p>Following ingestion of glucose, the glycemia level increases in both individuals, non-diabetic and diabetic, reaching 150 mg / dL in the non diabetic individual, and 220 mg / dL, a value which is 1.5 times higher in DT2 individual, at $t = 30$ min. This shows that the ingestion of glucose causes higher hyperglycemia in the individual with DT2 than in the unaffected individual.</p> <p>On the other hand, the glycemia level remains constant around 150 mg / dL in the non-diabetic individual from 30 to 90 min while it continues to increase in the individual with DT2 up to a maximum of 300 mg / dL during the Same duration.</p> <p>This shows that only the non-diabetic subject has a functional hypoglycemic regulation system.</p> <p>The glycemia level decreases and returns to its initial value of 85 mg / dL between $t = 90$ min and $t = 120$ min, in the non-diabetic individual. However, in the diabetic individual, the glycemia levels begin to decrease only after 90 min with a 60-min delay from the non-diabetic individual and reaches 250 mg / dL at 120 min, a value which is still much higher than the initial value. This shows that the hypoglycemic system in DT2 individual is slower than that in non diabetic individual.</p>	1.25
2	<p>Hypothesis 1: Type 2 diabetes is due to a lack of insulin.</p> <p>Hypothesis 2: Type 2 diabetes is due to a lack of insulin receptors at the level of target cells.</p>	1
3	<p>The level of insulin in blood increases to 280 $\mu\text{mol} / \text{L}$ in the diabetic individual during a period of 120 minutes, slightly lower than the maximum insulin level of 320 $\mu\text{mol} / \text{L}$ reached in the non-diabetic individual, during a shorter duration of time, 60 minutes.</p> <p>This shows that the individual DT2 secretes an almost sufficient quantity of insulin but with a delay of time of 60 min.</p> <p>Thus, the high hyperglycemia observed in the individual with DT2 after ingestion of glucose cannot be attributed to a lack of insulin. Consequently, treatment with insulin, a hypoglycemic hormone, would remain ineffective.</p>	1
4	<p>In obese mice, the amount of glucose absorbed by the muscle cells is $3 \text{ nmol} \cdot \text{mg}^{-1}$ of tissue, smaller than that absorbed by the muscle cells of the normal mouse which is $5 \text{ nmol} \cdot \text{mg}^{-1}$. It can be deduced that muscle cells in obese mice are less sensitive to insulin than those in normal mice.</p>	0.75

5	At t = 0, the amount of fixed insulin is null in both groups of mice. In normal mice, this amount increases up to 25×10^{-12} mol.mg ⁻¹ at t = 60 min; whereas in obese mice, it increases to 5×10^{-12} at t = 60 min, a value which is 5 times less than in normal mice. This shows that there are fewer receptors on target cells of insulin in obese mice with diabetes. Therefore, type 2 diabetes is due to a deficiency in insulin receptors at the level of these muscle cells.	1
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<p>المادة: علوم الحياة - لغة إنكليزية الشهادة: الثانوية العامة الفرع: علوم الحياة نموذج رقم ٢٠١٩/١ المدة: ثلاثة ساعات</p>	<p>الهيئة الأكاديمية المشتركة قسم : العلوم</p>	 <p>المركز التربوي للبحوث والإنماء</p>
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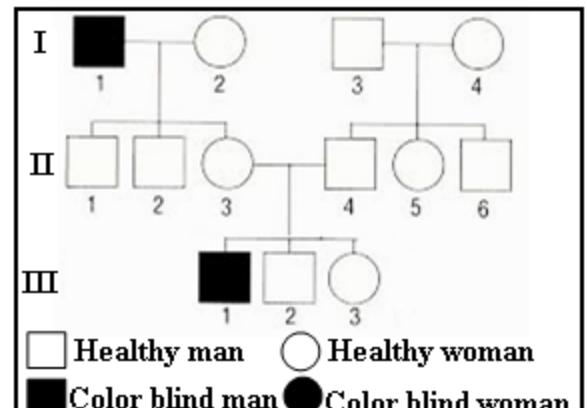
Answer the following four exercises.

Exercise 1 (5 points)

Case of Two Phenotypic Abnormalities

Color blindness or Daltonism, a hereditary abnormality, is characterized by difficulty in distinguishing certain colors. This abnormality is due to a gene located on X chromosome. Document 1 shows the genealogical tree of a family where a couple with normal vision (II₃-II₄) has a color blind boy (III₁), a normal vision boy (III₂) and a normal vision girl (III₃).

1. Specify if the allele coding for this abnormality is dominant or recessive.
2. Indicate the genotypes of the individuals II₃, II₄, and those of their children.
3. Determine the risk for this couple to have:
 - 3.1. A color blind girl.
 - 3.2. Another color blind boy.



Document 1

This couple gave birth to another girl who is color-blind.

4. Explain why the birth of this color blind girl from this normal couple is unexpected.

Document 2 shows the karyotype of this color blind girl.

5. Show that this karyotype reveals in this girl an abnormality other than color blindness.
6. Determine, in this girl:
 - 6.1. The parental origin of daltonism.
 - 6.2. The parental origin of her other abnormality.
7. Schematize, taking into consideration only one pair of autosomes and the sex chromosomes:
 - 7.1 The karyotype of the color blind girl.
 - 7.2 The karyotypes of the parental gametes which are responsible for the birth of this color blind girl.



Document 2

Exercise 2 (5 points)

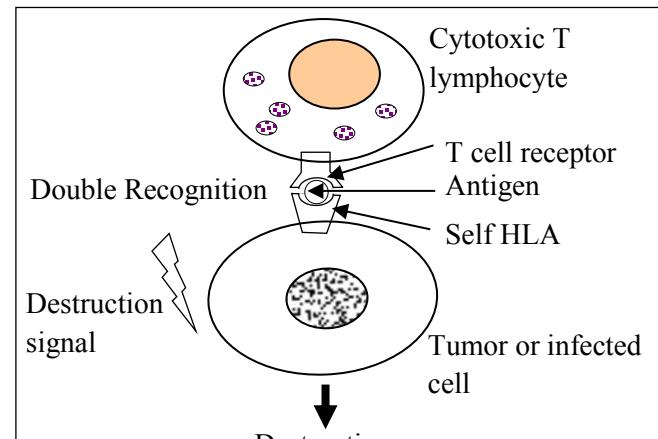
Pregnancy and Immune Defense

The fetus is a kind of temporary allograft that survives for nine months. However, fetal cells should be non-self for the mother's immune system because they express protein markers different from those of the mother. A research is performed to discover some mechanisms that allow the fetus to escape the mother's immune system during pregnancy.

The fetus is surrounded by a tissue called trophoblast, which isolates it from the maternal immune system.

The trophoblast cells do not express HLA class I proteins that are mainly involved in the cytotoxicity of certain lymphocytes against the non-self (Document 1).

1. Explain the mechanism of cellular cytotoxicity of Tc lymphocytes.
2. Determine the cause of ineffectiveness of Tc lymphocytes against the fetal cells.



Document 1

Moreover, the trophoblast cells carry on their surface and secrete in the medium a protein called HLA-G, a non-polymorphic molecule. A hypothesis assumes that this HLA-G protein prevents trophoblast cells from being recognized by the immune system as non-self-cells.

In order to validate this hypothesis, experiment 1 is performed. The conditions and the results of this experiment are presented in document 2.

Experiment 1:

Medium	A	B	C
Conditions	Immune cells of the mother	Immune cells of the mother	Immune cells of the mother
	Non-self cells	Trophoblast cells carrying HLA-G molecules	Trophoblast cells carrying HLA-G molecules blocked by a chemical substance
Results	Lysis of the non-self cells	No Lysis of trophoblast cells	Lysis of trophoblast cells

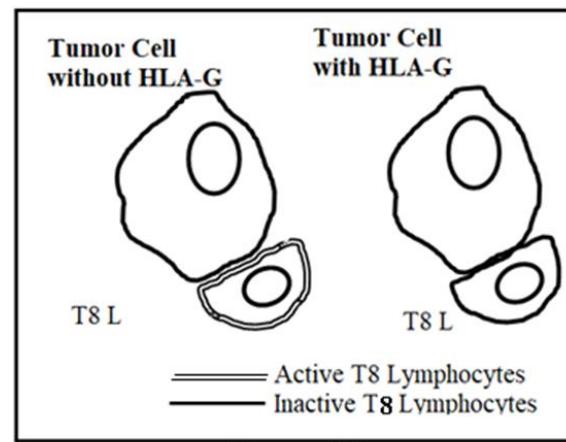
Document 2

3. Do the obtained results validate the tested hypothesis? Justify the answer.

Some cancer cells also produce HLA-G protein. In an attempt to find out if this molecule allows the cells to escape the action of the T lymphocytes, the following experiments, 2 and 3, are performed.

Experiment 2: Macrophages are placed in contact with the non-self cells carrying HLA-G. Their capacity to activate T4 lymphocytes becomes reduced.

Experiment 3: T8 lymphocytes are cultured in the presence of two types of cancer cells. The results are shown in document 3.



Document 3

4. Determine, by referring to each of the experiments 2 and 3, how the HLA-G contributes to making the specific immune response less effective.

Exercise 3 (5 points)

Maintaining Resting Potential

Nerve cells have a potential difference (pd) of -70 mV across the plasma membrane. This resting potential is correlated with differences in ion concentrations on either side of the plasma membrane, ECM and ICM, (Document 1).

1. 1.1. Compare the ionic composition of the two media (Document 1).

1.2. What can you draw out?

It is hypothesized that the plasma membrane is impermeable to ions. In order to test this hypothesis, experiments 1 and 2 are performed.

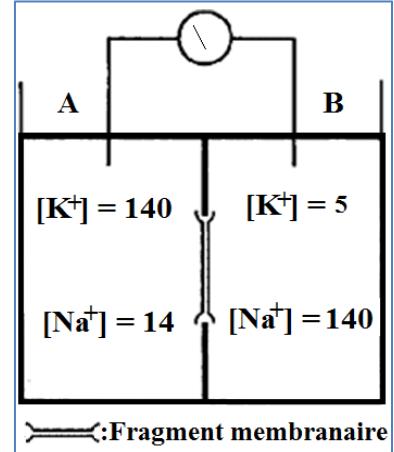
	Na^+ (mmol/L)	K^+ (mmol/L)
Extracellular medium(ECM)	140	5
Intracellular medium(ICM)	14	140

Document 1

Experiment 1: Two compartments A and B are separated by an impermeable teflon membrane pierced with a hole. This hole is covered with a fragment of plasma membrane (Document 2). Initially, the Na^+ ions in compartment B are radioactive. After time "t" a quantity of radioactive Na^+ "Q1" is detected in compartment A.

Experiment 2: The above experiment (experiment 1) is repeated but radioactive K^+ is initially placed in compartment A. After time "t" "Q2" of radioactive K^+ , is detected in compartment B, where Q2 is greater than Q1.

2. Is the tested hypothesis valid? Justify the answer.
3. Justify the direction of ion diffusion through this membrane.
4. Draw out the origin of membrane resting potential.



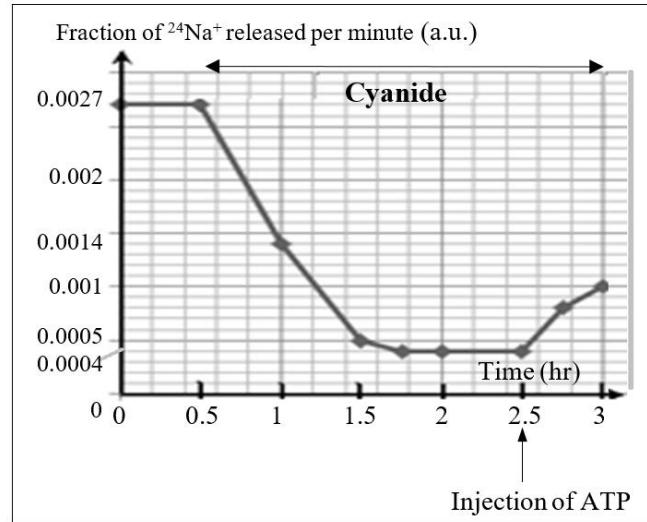
Document 2

If in the previous setup, the diffusion continues; the differences in ionic concentrations should disappear and the resting potential too. However, in living cells, the resting potential is maintained. In order to understand the mechanism that is responsible for maintaining this resting potential, experiments 3 and 4 are performed.

Experiment 3:

An axon is injected with radioactive $^{24}\text{Na}^+$ ions. Then, it is immersed in a solution containing cyanide, a poison that blocks all reactions that require energy (ATP) in the cell. At time 2.5hrs the axon is injected with ATP, energy molecule used by the cells. The level of $^{24}\text{Na}^+$ ions released by the axon is measured (Document 3).

Experiment 4: An axon is placed in a medium enriched with radioactive $^{40}\text{K}^+$ ions. Radioactivity appears rapidly in the cytoplasm of the axon. Later, this same experiment 4 is repeated in the presence of cyanide. Radioactivity is not detected in the cytoplasm of the axon.



Document 3

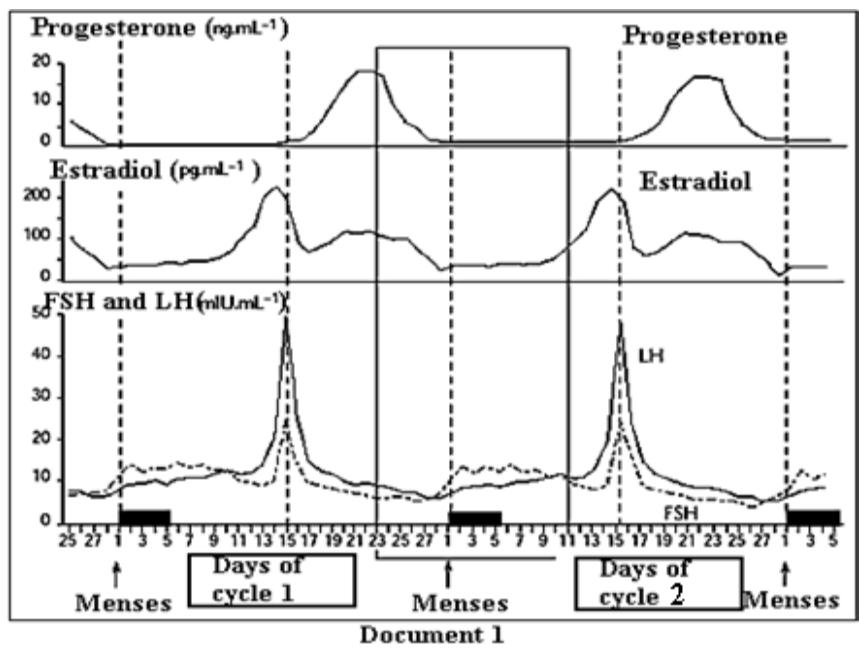
5. Construct a table showing the results of document 3.
6. Interpret the results of the experiments 3 and 4.
7. Name the protein molecule involved in the active transport of ions across the plasma membrane.

Exercise 4 (5 points) Hormonal Influence on the Renewal of Sexual Cycle

A research is performed to explain the hormonal mechanisms involved in the renewal of the ovarian cycle, on the first day of the menses.

In fact, in women, the beginning of each cycle is marked by the appearance of menses (document 1). This menses results from the sloughing off of the uterine mucosa that occurs if no embryo implantation takes place during the luteal phase (day 15-day 28) of the preceding cycle.

Document 1 shows the evolution of the secretion of the anterior pituitary hormones, FSH and LH, and ovarian hormones, estrogen and progesterone, during two consecutive cycles in the woman.



1.1 What are the characteristics of the luteal phase concerning hormone secretions, document 1?

1.2 Draw out the type of feedback control exerted by the ovarian hormones on the pituitary gland during the luteal phase.

In order to show the effects of progesterone during the luteal phase of the cycle, the following experiment is performed.

Few days before the end of the cycle, ewes in lots A and B are subjected to the ablation of their corpus luteum followed by the implantation of a capsule under the skin of each of them according to the following conditions:

- For each ewe in lot A, an empty capsule.
 - For each ewe in lot B, a capsule containing progesterone, releases continuously and slowly their content in the body.
- The concentrations of FSH and progesterone in the plasma are measured during the days following the implantation of the capsules (Document 2).

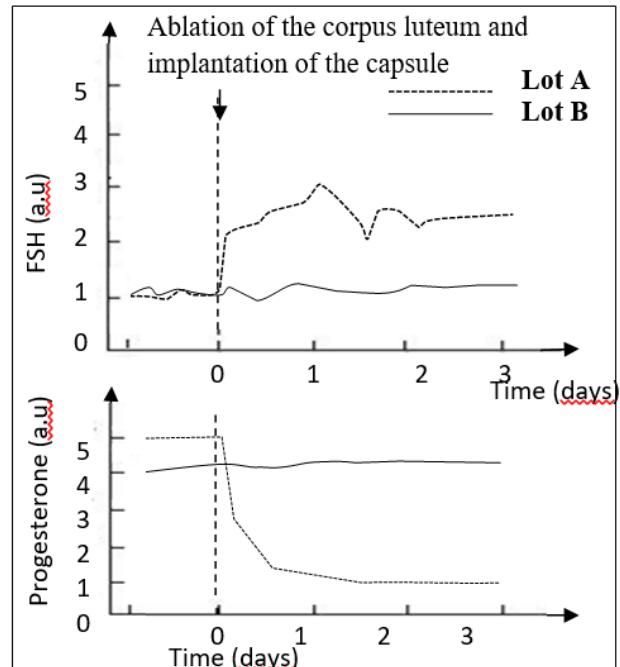
Note: The evolution of the plasma concentration of LH and FSH hormones is synchronized during the experiment.

2. What can you deduce from the obtained results, document 2?

3. Explain, by referring to the acquired knowledge, the mechanism that permits the renewal of an ovarian cycle.

The implantation of the capsule containing progesterone can be used as a contraceptive method.

4. Explain the mode of action of this contraceptive capsule.

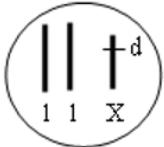
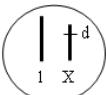


Document 2

المادة: علوم الحياة - لغة إنجليزية الشهادة: الثانوية العامة الفرع: علوم الحياة نموذج رقم ٢٠١٩/١ المدة: ثلاثة ساعات	الهيئة الأكاديمية المشتركة قسم : العلوم	
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أسس التصحيح:

Ex	Part	Exercise 1 (5 points) Case of Two Phenotypic Abnormalities	Mark
1	1	<p>The allele coding for color blindness is recessive since the normal parents II₃ and II₄ have a color blind boy III₁. Since this gene is located on the sex chromosome X and since this latter chromosome is inherited from his mother who is normal, thus the mother possesses the allele responsible for colorblindness which is masked by the allele responsible for normal vision.</p> <p>N: normal dominant allele d: recessive color blind allele</p>	0.5
	2	<p>The genotype of the father II₄ and that of the boy III₂ is : X^N//Y The genotype of the mother II₃ is : X^N//X^d The genotype of the color blind boy III₁ is: X^d//Y The genotype of the girl with normal vision III₃: X^N//X^N or X^N//X^d</p>	1
	3.1	<p>The mother II₃ is heterozygous, of genotype X^N//X^d, since she gave birth to a color blind boy III₁.</p> <p>The genotype of the father II₄ is X^N//Y. He transmits the sex chromosome Y to his sons and X^N to his daughters.</p> <p>Since all daughters inherit X^N from their father and since allele N is dominant over allele d, then all the girls will have normal vision. Hence, the risk for this couple to obtain a color blind girl is null.</p>	0.5
	3.2	<p>For this couple to obtain a color blind son having a genotype X^d//Y, this son should inherit X^d from his mother and Y from his father.</p> <p>Since the mother II₃ have the genotype X^N//X^d, thus she might transmit either X^N or X^d to her sons.</p> <p>The risk of the mother to transmit X^d to her son is ½. Therefore, the risk to have a color blind boy is ½.</p>	0.5
	4	<p>Color blindness is a recessive abnormality due to a gene carried on the sex chromosome X. So, a color blind female should have the genotype X^d //X^d (a recessive allele is only expressed phenotypically when it is present in two copies). Therefore, this girl should get X^d from her mother, II₃, and X^d from her father, II₄. Consequently, her father should have the genotype X^d //Y and should be colorblind; However, he has normal vision of genotype X^N //Y. Hence, this is why the birth of this color blind girl from this couple is unexpected.</p>	0.5
	5	<p>This karyotype shows the presence of only 1 sex chromosome instead of two. This sex chromosome is X. Therefore, the other abnormality revealed by this karyotype is monosomy X or Turner syndrome.</p>	0.5
	6.1	<p>The girl is affected with monosomy X and is color blind. Her unique sex chromosome X carries the allele d. Since, her father is of normal vision then he doesn't have X^d. Consequently, this girl has certainly inherited X^d from her mother. Hence, the origin of daltonism in this girl is maternal.</p>	0.5
	6.2	<p>Since the origin of the only sex chromosome X in this female is of maternal origin, this means that this female results from the fusion a female gamete having one X chromosome</p>	0.25

		and a male gamete lacking any sex chromosome. Hence, the origin of this abnormality (monosomy X) is paternal.	
	7.1	Karyotype of the color blind girl: 	0.25
	7.2	karyotypes of the parental gametes Maternal gamete Paternal gamete  	0.5

Ex	Part	Exercise 2 (5 points) Pregnancy and Immune Defense	Mark
2	1	The T cell receptor (TCR) on the membrane of Tc lymphocyte binds to HLA-I – nonself-peptide complex on the target cell membrane, tumor or infected cell. Then, Tc lymphocyte releases its perforin molecules which assemble into polymers that form a hollow channel through the target cell membrane. Then, the Tc releases granzymes that penetrate into the target cell through the polyperforin channels triggering an enzymatic chain reaction within the cell leading to DNA degradation. This causes cell death by apoptosis.	1.5
	2	The action of Tc lymphocyte on the target cell necessitates the double recognition of the nonself peptide associated to self HLA-I protein. The trophoblast isolates the fetus from the maternal immune system. The cells of this trophoblast do not express the self HLA- class I proteins; thus, these cells are not recognized by Tc lymphocyte. This makes Tc lymphocyte unable to reach and destroy the fetal cells.	1
	3	The hypothesis is validated since the mother's immune cells lyse the non-self cells (medium A) but not the trophoblast cells that carry HLA-G molecules (medium B). This shows that HLA-G prevents the action of maternal immune cells on trophoblast cells. This is also confirmed by the result obtained in medium C, where the trophoblast cells carrying blocked HLA-G molecules are lysed by maternal immune cells.	1
	4	Experiment 2 shows that the capacity of macrophages to activate T4 lymphocytes is reduced when placed in contact with the non-self cells carrying HLA-G. Furthermore, the activation of the T4 cells is a step necessary for the induction of the specific immune response, humoral and cellular mediated. Hence, this immune response becomes less effective. The results of experiment 3 show that T8 lymphocyte remains inactive when it binds to a tumor cell which carries HLA-G molecules. However, it becomes active if HLA-G molecules are absent. Thus, T8 lymphocyte are not activated by tumor cells carrying HLA-G molecules. Hence, the specific immune response triggered by lymphocytes is less effective.	1.5

Ex	Part	Exercise 3 (5 points) Maintaining Resting Potential	Mark															
3	1-1.	The extracellular medium is more concentrated in Na^+ ions than the intracellular medium ($140 \text{ mmol.L}^{-1} > 14 \text{ mmol.L}^{-1}$). However, the intracellular medium is more concentrated in K^+ ions than the extracellular medium ($140 \text{ mmol.L}^{-1} > 5 \text{ mmol.L}^{-1}$).	0.5															
	1-2.	Resting potential is due to an uneven distribution of Na^+ and K^+ ions on either side of the plasma membrane, where excess Na^+ ions are present towards the ECM with respect to ICM, and excess K^+ ions exists towards ICM with respect to ECM.	0.5															
	2	The formulated hypothesis is not valid because in experiment 1, the appearance of radioactivity in compartment A shows that an amount Q1 of radioactive Na^+ ions diffuses from compartment B to compartment A through the membrane fragment during time t. Similarly, the result of experiment 2 shows a diffusion of an amount Q2 of K^+ ions from compartment A to compartment B ($Q2 > Q1$) during the same time interval. This means that the plasma membrane is permeable to Na^+ and K^+ ions.	0.75															
	3	The diffusion of ions across the plasma membrane takes place with concentration gradient, from a medium of higher concentration to a medium of lower concentration for the same ions. Na^+ ions diffuse from compartment B to compartment A because the concentration of Na^+ ions in compartment B is 140 mmol.L^{-1} which is higher than that in compartment A (14 mmol.L^{-1}). Similarly, K^+ ions diffuse from compartment A to B because the concentration of K^+ ions in compartment A is 140 mmol.L^{-1} which is higher than that in compartment B (5 mmol.L^{-1}).	0.5															
	4	The origin of the resting potential is the selective permeability of the membrane, which is more permeable to K^+ ions than to Na^+ ions.	0.25															
	5	Title: The variation of the fraction of radioactive $^{24}\text{Na}^+$ released across the plasma membrane of the axon as function of time within different conditions. <table border="1"> <thead> <tr> <th>Time (hour)</th> <th>0</th> <th>0.5</th> <th>1</th> <th>1.5</th> <th>2</th> <th>2.5</th> <th>3</th> </tr> </thead> <tbody> <tr> <td>Fraction of $^{24}\text{Na}^+$ released /min (a.u)</td> <td>0.0027</td> <td>0.0027</td> <td>0.0014</td> <td>0.0005</td> <td>0.0004</td> <td>0.0004</td> <td>0.001</td> </tr> </tbody> </table> 	Time (hour)	0	0.5	1	1.5	2	2.5	3	Fraction of $^{24}\text{Na}^+$ released /min (a.u)	0.0027	0.0027	0.0014	0.0005	0.0004	0.0004	0.001
Time (hour)	0	0.5	1	1.5	2	2.5	3											
Fraction of $^{24}\text{Na}^+$ released /min (a.u)	0.0027	0.0027	0.0014	0.0005	0.0004	0.0004	0.001											
6	Between 0 and 0.5 hours, and in the presence of ATP, the fraction of $^{24}\text{Na}^+$ ions released by the axon is 0.0027 a.u. However, between 0.5 hrs and 2.5 hrs and in the presence of cyanide that inhibits ATP production, the fraction of released $^{24}\text{Na}^+$ ions decreases from 0.0027 to 0.0004 a.u. at 1.3 hours and then remains constant. Hence, the release of $^{24}\text{Na}^+$ from ICM to ECM requires energy.	1.25																

	<p>On the other hand, following ATP injection at 2.5 h and in the presence of cyanide, the release of $^{24}\text{Na}^+$ ions out of the axon is resumed and its rate increases from 0.0004 to 0.001 a.u. within half an hour. Therefore, the release of $^{24}\text{Na}^+$ ICM to ECM, against the concentration gradient, is an active mechanism that requires the supply of energy in the form of ATP.</p> <p>In Experiment 4, the radioactivity appears rapidly in the cytoplasm of an axon placed in a medium enriched in radioactive $^{40}\text{K}^+$ ions in the presence of ATP. However, it ceases only in the presence of cyanide that inhibits ATP production. Therefore, the movement of $^{40}\text{K}^+$ ions from ECM to ICM against the concentration gradient is an active mechanism too.</p>	
7	The protein molecule involved in the active transport of ions across the plasma membrane is the Sodium-Potassium pump (Na^+/K^+ Atpase pump)	0.25

Ex	Part	Exercise 4 (5 points) Hormonal Influence on the Renewal of Sexual Cycle	Mark
3	1.1	The luteal phase is characterized by an increase in the secretion of progesterone and estrogen but with higher level of progesterone compared to estrogen. However, this phase is characterized by a fall in the levels of FSH and LH.	1
	1.2	The high levels of ovarian hormones, progesterone and estradiol exert a negative feedback on the activity of the pituitary gland during the luteal phase.	0.5
	2	Before removal of the corpus luteum, the FSH level is about 1 au. in both lots ,A and B and that of progesterone is 5 a.u. for lot A and 4 a.u. for lot B. After the ablation of the corpus luteum and the implantation of capsule at day 0, the level of FSH increases in lot A having an empty capsule to 3 au at day 1 then fluctuated around 2a.u till day 3, but the level of progesterone decreases from 5 a.u. to 1a.u after 0.5 day and then remains constant till day 3. However, the level of FSH in Lot B where the implanted capsule contains progesterone, fluctuates around 1 a.u. till day 3 and that of progesterone remained constant at 4 a.u. during the same interval of time. Hence, progesterone inhibits the secretion of FSH.	1 1
	3	The renewal of an ovarian cycle is manifested by the initiation of menses- resuming the increase in production of estrogen hormone by the developing follicles(at the level of the theca interna and granulosa). At the end of the luteal phase of the previous cycle the corpus luteum degenerates leading to a drop in the levels of the ovarian hormones. This drop leads to an increase in the level of GnRH and pituitary hormones, mainly FSH. FSH stimulates the growth and development of the cavitary follicles as well as the secretion of estrogen by follicular cells.	1
	4	Progesterone capsules continually and slowly diffuse this hormone into the body. This hormone will block the pituitary function, thereby inhibiting the secretion of pituitary hormones LH and FSH and consequently ovulation.	0.5

الاسم:	مسابقة في مادة علوم الحياة
الرقم:	المدة: ثلاثة ساعات

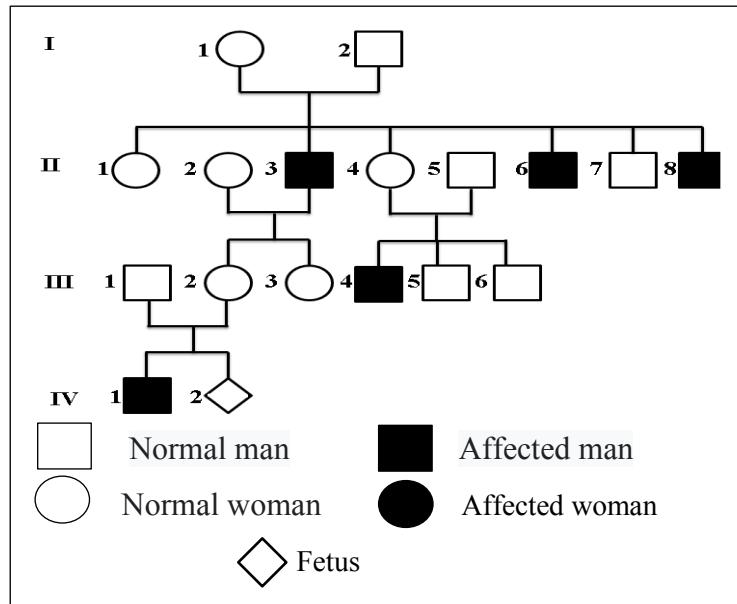
Exercise 1 (5 points)

Bruton Disease

Bruton disease is a genetic disease that affects one newborn in 200,000 birth. It is manifested by recurrent bacterial infections of the respiratory tract, starting from the age of six years. The disease predisposes patients to the risk of having chronic infections with viruses that attack particularly the digestive tract and the nervous system.

Document 1 represents the pedigree of a family, whose certain members are affected with Bruton disease.

- Indicate whether the allele responsible for Bruton disease is dominant or recessive. Justify the answer.
- Show that the gene responsible for the disease is localized on the non-homologous segment of chromosome X.
- Indicate the genotypes of individuals III₁, III₂ and IV₁.
- Determine the risk for the fetus IV₂ to be affected with this disease.



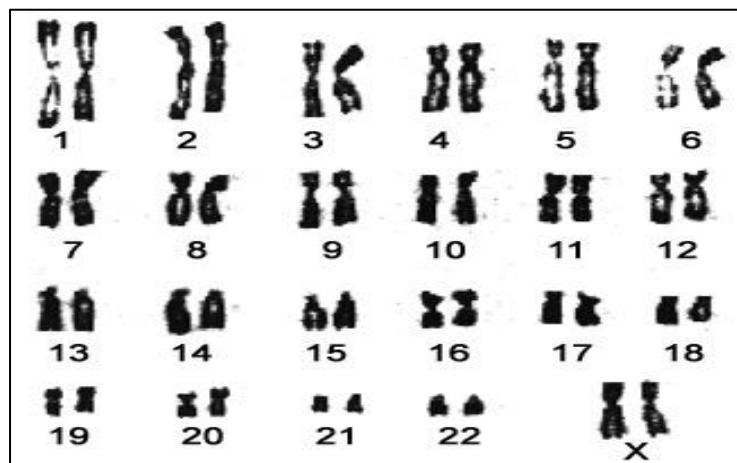
Document 1

To find out if the fetus IV₂ will be affected with Bruton disease, the doctor requests establishing the karyotype of the fetus. The obtained result is shown in document 2.

- Determine, referring to documents 1 and 2, if the fetus IV₂ will be affected by Bruton disease.

The doctor completes the diagnosis by performing DNA analysis using the Southern Blot technique. The used probe makes it possible to distinguish between the mutant allele and the normal allele of the gene involved in Bruton disease. The obtained results are shown in document 3.

- Specify the band that corresponds to the mutant allele.
- Draw out the genotype as well as the phenotype of the fetus IV₂.



Document 2

	III ₁	III ₂	IV ₁	Fetus IV ₂
A	—	—		—
B		—	—	—

Document 3

Exercise 2 (5 points)

Action of Antibodies and the Complement

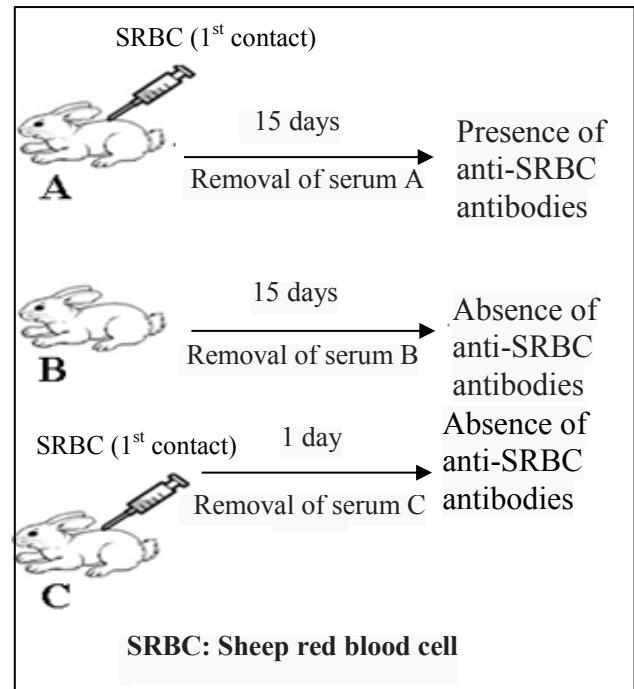
In order to study certain aspects of the action of antibodies, researchers performed experiments on three rabbits A, B and C. The experimental conditions and the obtained results are represented in document 1.

- 1- Show, by referring to document 1, that:
 - 1.1- The production of antibodies necessitates a prior contact with the concerned antigen.
 - 1.2- The production of antibodies necessitates a definite duration.
- 2- Explain the necessity of the 15 days, time delay to obtain the agglutination of SRBC by the serum of rabbits immunized against SRBC.

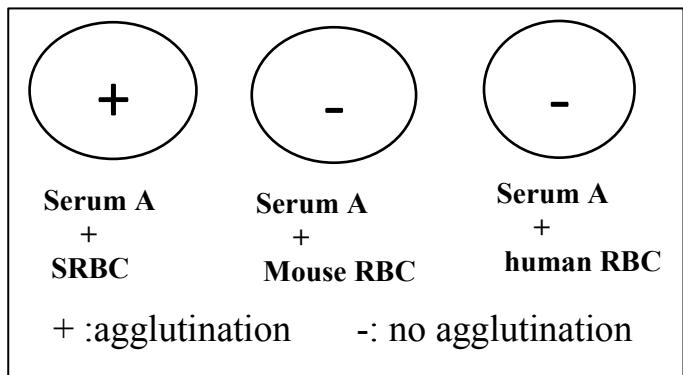
The serum taken from rabbit A, 15 days after being injected with SRBC, is placed in the presence of different substances. The conditions and the obtained results are shown in document 2.

- 3- Specify, referring to document 2, a characteristic of the humoral-mediated immune response.
- 4- Schematize the mechanism of agglutination of SRBC.

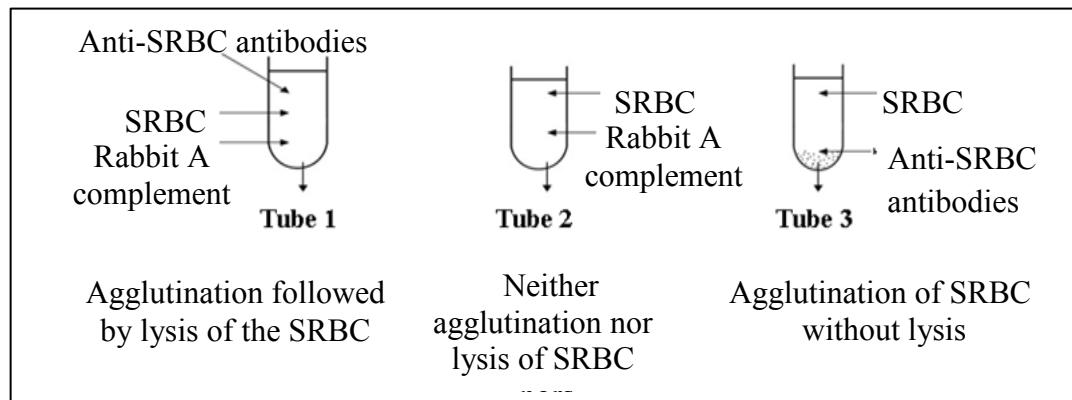
The complement is a set of plasma proteins. Researchers aim to show the role of the complement in the humoral immune response.



Document 1



Document 2



Document 3

- 5.1- Analyze the obtained results.
- 5.2- What can you conclude?

The experiment in tube 1 is repeated; the complement of rabbit A is replaced by the complement taken from a mouse. Agglutination of SRBC followed by their lysis is obtained.

- 6- Draw out a characteristic concerning the action of the complement.

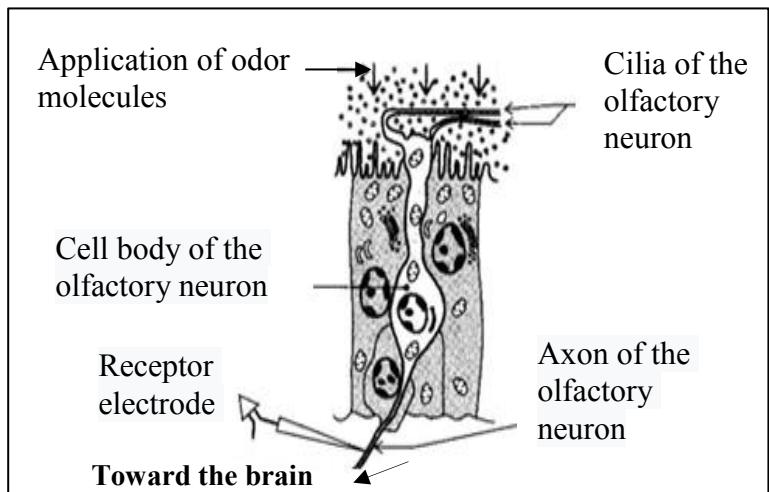
Exercise 3 (5 points)

Coding of the Olfactory Stimulus

Through olfaction (sense of smell), an organism is able to detect the presence of odor molecules in the air, estimate their concentration, and distinguish among different odors.

Odor molecules in the air bind to chemoreceptors found on the cilia of olfactory neurons. This results in a nerve message that propagates toward the brain (document 1).

- Pick out from the text, the localization of the olfactory chemoreceptors.



Document 1

Researchers expose cilia of olfactory neuron to increasing concentrations of an odor molecule. The frequency of the obtained AP is measured by a receptor micro-electrode placed on the axon of the olfactory neuron. Document 2 represents the obtained results.

- Represent, in a table, the results shown in document 2.
- Analyze the obtained results.
- What can you draw out concerning the coding of an olfactory stimulus?

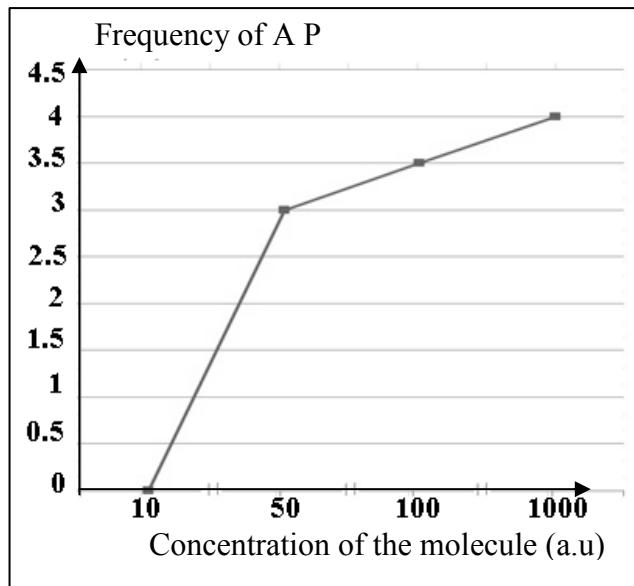
Researchers aim to study the action of three different odor molecules A, B and C on an olfactory neuron (document 3).

In the absence of any stimulus, the olfactory neurons show spontaneous activity that corresponds to a frequency of action potential of 100 a.u.

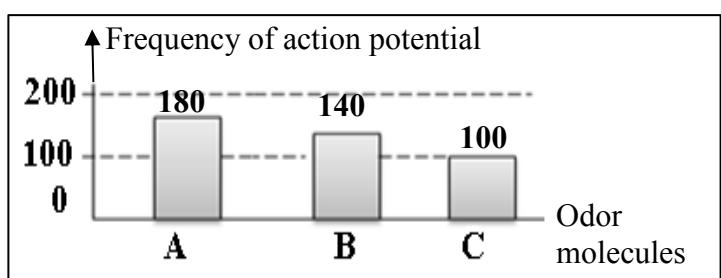
- Specify the molecule(s) which stimulate the olfactory neuron.

Document 4 represents the response of 4 different olfactory neurons in response to two odor molecules A and B.

- Show that the activity of the olfactory neuron depends on the nature of odor molecules.



Document 2



Document 3

Olfactory neurons		1	2	3	4
Frequency of A.P	Odor molecule A	200	100	150	100
	Odor molecule B	100	150	100	180

Document 4

Exercise 4 (5 points)**Hormonal Regulation**

In order to study the relations existing between the hypothalamus, pituitary gland and ovaries, researchers performed the following experiments on adult female mammals.

Experiments	Conditions	Results
1	Electric stimulation on a specific zone of the hypothalamus	Hyperactivity of the anterior pituitary with hypersecretion of pituitary hormones, FSH and LH
2	Hypophysectomy (ablation of the anterior pituitary)	Atrophy of the ovaries and the uterus with disappearance of the sexual cycles

Document 1

- 1- Draw out the role of:

1.1- the hypothalamus.

1.2- the anterior pituitary.

Experiment 3: researchers inject the ovarian hormones to ovariectomized adult female mammals and then they measure the variations of the levels of LH and FSH. The conditions and the results are shown in document 2.

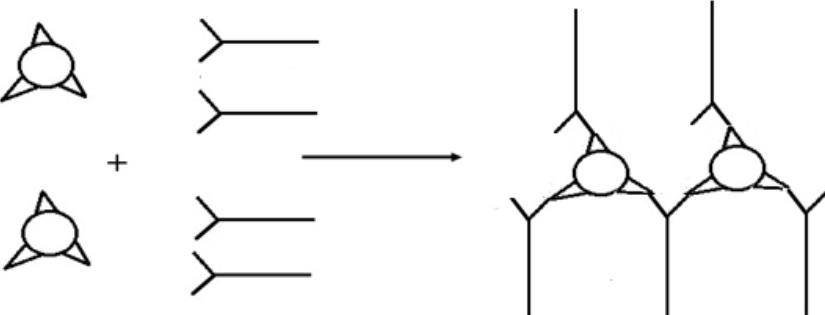
Experiment	Conditions	Results																			
3	Case A: Injection of a moderate dose of estrogen. Case B: Injection of a high dose of estrogen. Case C: Injection of high dose of estrogen and progesterone.	<table border="1"> <thead> <tr> <th></th> <th>Before the injection</th> <th>Case A</th> <th>Case B</th> <th>Case C</th> </tr> </thead> <tbody> <tr> <td>FSH (ng/mL)</td> <td>12</td> <td>3</td> <td>18</td> <td>3</td> </tr> <tr> <td>LH (ng/mL)</td> <td>40</td> <td>10</td> <td>65</td> <td>10</td> </tr> </tbody> </table>		Before the injection	Case A	Case B	Case C	FSH (ng/mL)	12	3	18	3	LH (ng/mL)	40	10	65	10				
	Before the injection	Case A	Case B	Case C																	
FSH (ng/mL)	12	3	18	3																	
LH (ng/mL)	40	10	65	10																	

Document 2

- 2- Draw a histogram that represents the data of experiment 3.
- 3- Indicate the types of the feedback revealed in experiment 3. Justify the answer.
- 4- Name the structures that secrete estrogen during a sexual cycle.
- 5- Establish, by referring to the preceded experiments, a functional diagram showing the relations between the ovaries, the anterior pituitary and the hypothalamus.

الاسم:	علوم الحياة
الرقم:	أسس التصحيح

Part of ex	Exercise 1 (5 points)	Bruton Disease	Grade
1	The allele responsible for the disease is recessive, since individuals 3, 6 and 8 are affected from healthy parents. This means that the mutant allele is present in the parents but masked by the normal allele (N).		0.5
2	There is discrimination of sex, only males are affected, so the allele of the disease is sex-linked. If the studied gene were localized on the non-homologous segment of chromosome Y, any affected boy should have an affected father, which is not the case (normal father I2 has affected boys). So the studied gene is localized on the non-homologous segment of chromosome X.		1
3	N: normal allele dominant m : affected allele recessive III ₁ : X ^N Y III ₂ : X ^N X ^m IV ₁ : X ^m Y		0.75
4	The fetus IV ₂ has a heterozygous mother (III ₂) of genotype X ^N X ^m . If the fetus were a boy, the risk of this fetus, in this case, to receive the chromosome Xm from his mother will be ½. He obligatory will receive chromosome Y from his father. If the fetus were a girl, the risk will be null since she will receive obligatory X ^N from her healthy father of genotype X ^N Y. Therefore, the fetus will be necessarily healthy regardless of the gamete received from her father, N is dominant.		0.75
5	The karyotype (document 2) shows that the fetus is a girl with two chromosomes X. Referring to the pedigree (document 1), the fetus has a healthy father of genotype X ^N Y. Thus, he must have obligatory received X ^N from his father and then the fetus will be a healthy girl since allele N is dominant, then the female is healthy		1
6	Band B corresponds to the mutated allele, because document 3 shows that male IV ₁ of genotype X ^m Y possesses a single band B.		0.5
7	Fetus: genotype X ^N X ^m Phenotype : Healthy [N]		0.5

Part of ex	Exercise 2 (5points) Mode of Action of Antibodies and the Complement	Grade
1.1	<p>Anti-SRBC antibodies are only found in the serum taken from rabbit A which is injecting with SRBC 15 days after the injection. However, no anti-SRBC antibodies are found in the serum taken from rabbit B, being not injected with SRBC.</p> <p>This shows that, the production of antibodies necessitates a prior contact with the concerned antigen.</p>	0.5
1.2	<p>Anti-SRBC antibodies are only found in serum of rabbit A 15 days being injected with SRBC but not after 1 day from the injection (rabbit B). Therefore, the production of antibodies necessitates a definite duration.</p>	0.5
2	<p>15 days is the time delay necessary for the induction of a humoral immune response during which :</p> <ul style="list-style-type: none"> activation of B lymphocytes by IL4 secreted by activated T4 lymphocytes and following their multiplication and differentiation to plasmocytes that secrete anti-SRBC antibodies. 	1
3	<p>The humoral immune response is specific, because agglutination occurs only when adding the serum of an immunized rabbit against the SRBC with the same antigen, SRBC.</p> <p>(no agglutination occurs neither with RBC of a mouse nor with RBC of a human).</p>	0.5
4	 <p style="text-align: center;">SRBC Anti-SRBC antibodies Immune complex (Agglutination)</p>	0.5
5.1	<p>There is agglutination with no lysis, when the SRBCs are mixed with only anti-SRBC antibodies (having no complement). However; there is neither agglutination nor lysis, when the SRBCs are mixed with complement in the absence of antibodies.</p> <p>On the other hand, there is agglutination followed by lysis when anti- SRBC antibodies are mixed with SRBC and the complement rabbit A.</p>	1
5.2	<p>Antibodies are responsible for the agglutination of the antigen but the complement is responsible for the lysis of only antigens previously agglutinated by antibodies.</p>	0.5
6	<p>The action of complement protein is not specific.</p>	0.5

Part of ex	Exercise 3 (5 points) Coding of the Olfactory Stimulus	Grade										
1	The cilia of the olfactory neuron.	0.5										
2	<table border="1"> <tr> <td>Concentration of the molecules (a.u.)</td> <td>10</td> <td>50</td> <td>100</td> <td>1000</td> </tr> <tr> <td>Frequency of AP (a.u.)</td> <td>0</td> <td>3</td> <td>3.5</td> <td>4</td> </tr> </table> <p>Variation of the frequency of AP (a.u.) as a function of the concentration of the molecules (a.u.)</p>	Concentration of the molecules (a.u.)	10	50	100	1000	Frequency of AP (a.u.)	0	3	3.5	4	1
Concentration of the molecules (a.u.)	10	50	100	1000								
Frequency of AP (a.u.)	0	3	3.5	4								
3.1	The frequency of AP increases sharply from 0 to 3 a.u. with the increase in concentration of the odor molecules from 0 to 50 a.u. Then, such variation shows slight increase from 3 to 4 a.u. when the concentration of the molecules increases from 50 to 1000 a.u.	1										
3.2	The intensity of the stimulation is modulated by frequency of AP.	0.5										
4	The frequency of AP for the odor molecule A (approximately 180 a.u.) is greater than that obtained by the odor molecule B (approximately 140 a.u.). However, the frequency of action potentials for odor molecule C is low (100 a.u) which corresponds to the spontaneous activity of the olfactory neuron. Thus, odor molecules A and B stimulate this olfactory neuron.	1										
5	Neurons 1 and 3 are excited by the odor molecule A with 200 AP and 150 AP respectively, but not excited by the odor molecule B, they show an activity (100 AP) similar to the spontaneous activity in the absence of any stimulation. However, neurons 2 and 4 are excited by the odor molecule B with 150 and 180 AP respectively; they are not excited by the odor molecule A with 100 AP. Thus, the activity of the olfactory neurons depends on the nature of the odor molecule.	1										

Part of ex	Exercise 4 (5points)	Hormonal Regulation	Grade															
1.1	The hypothalamus stimulates the secretion of the pituitary hormones FSH and LH.		0.5															
1.2	The anterior pituitary is responsible for the development of the ovaries and uterus, and for their cyclic activities.		0.5															
2	<p>The variation of FSH and LH as a function of the injected hormones.</p> <table border="1"> <caption>Data from Figure 2: Variation of FSH and LH levels (ng/mL)</caption> <thead> <tr> <th>Condition</th> <th>FSH (ng/mL)</th> <th>LH (ng/mL)</th> </tr> </thead> <tbody> <tr> <td>Before injection</td> <td>12</td> <td>40</td> </tr> <tr> <td>Case A</td> <td>2</td> <td>10</td> </tr> <tr> <td>Case B</td> <td>18</td> <td>65</td> </tr> <tr> <td>Case C</td> <td>2</td> <td>10</td> </tr> </tbody> </table>	Condition	FSH (ng/mL)	LH (ng/mL)	Before injection	12	40	Case A	2	10	Case B	18	65	Case C	2	10		1
Condition	FSH (ng/mL)	LH (ng/mL)																
Before injection	12	40																
Case A	2	10																
Case B	18	65																
Case C	2	10																
3	<p>Negative feedback: moderate dose of estrogen alone (case A) or the presence of high dose of estrogen coupled with progesterone (case C) decreases respectively the secretion of FSH and LH from 12 to 3 and from 40 to 10 mg/mL</p> <p>Positive feedback: High dose of estrogen (case B) increases respectively the secretion of FSH and LH from 12 to 18 and from 40 to 65 mg/mL.,</p>		1															
4	<p>Estrogen is secreted by:</p> <p>Follicular cells and those of the internal theca during the follicular phase.</p> <p>Luteal cells during the luteal phase.</p>		0.5															
5	<p>Functional diagram showing the relations between the ovaries, the anterior pituitary and the hypothalamus.</p> <p>(-): Negative feedback (+): Positive feedback</p>		1.5															

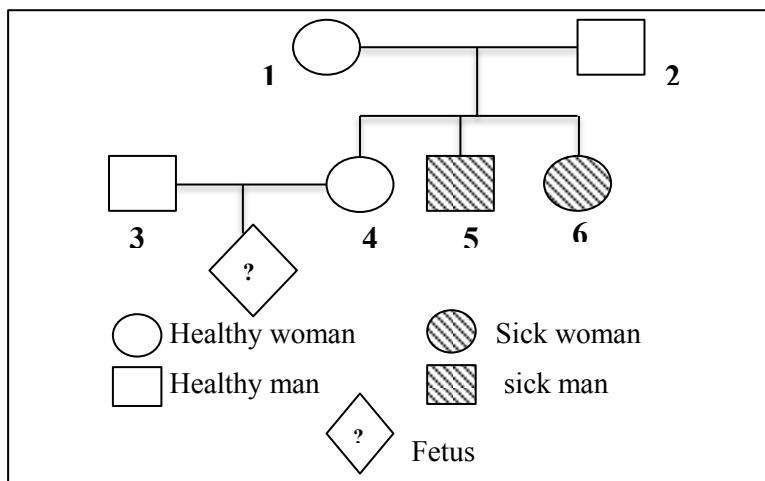
الاسم: _____ مسابقة في مادة علوم الحياة
الرقم: _____ المدة: ساعتان ونصف

Answer the following exercises.

Exercise 1 (4 points)

Transmission of a Hereditary Character

Fructosemia is a disease caused by deficiency of aldolase B enzyme. The following pedigree in document 1 shows the transmission of this disease in a family.



Document 1

- Indicate whether the allele responsible for the disease is dominant or recessive. Justify the answer.
- Determine the chromosomal localization of the gene responsible for this disease.
- Write the possible genotypes of each of the individuals 3 and 4.

Document 2 represents the results of the electrophoresis performed on the alleles of the studied gene in individuals 3, 4 and the fetus.

Allele \ Individual	3	4	Fetus
Normal	—	—	—
Mutant	—		

Document 2

- Specify, by referring to document 2:
 - the real genotype of each of individuals 3 and 4.
 - the phenotype of the fetus.
- What advantage does this technique provide to the determination of the genotype of an individual?

Exercise 2 (4 points)

Fructosemia

Congenital fructosemia is an intolerance to fructose, preventing the absorption of fructose and all sugars containing fructose. It is due to a deficiency in aldolase B, an enzyme located in the liver, small intestine and kidneys. Aldolase B enzyme is responsible for the cleavage of fructose -1- phosphate into two molecules: DHAP and glyceraldehyde. Children affected by this disease show a dysfunction of the liver and kidney weakness with abnormal high levels of sugar, amino acids and salts in the urine.

Document 1

- Pick out from document 1:

- the cause of fructosemia.
- the consequences of this disease.

Document 2 represents the partial sequence of the nucleotide of DNA in the normal and the mutant alleles of the gene determining the synthesis of the enzyme aldolase B.

Allele	Nucleotide sequence of the transcribed strand of DNA
Normal	↑↓ ↓24 TTA CCT GAC CAT GGA TAA CAA CTT
Mutant	↑↓ ↓18 TTA CCT GGA TAA CAA CTT

- Compare these two sequences (document 2).
- Indicate the type of the revealed mutation.
- Write, referring to documents 2 and 3:
 - the mRNA that corresponds to each allele.
 - the sequence of amino acids that corresponds to each allele.
- Explain how the modification of the nucleotide sequence of the allele leads to the appearance of fructosemia.

Document 2

		Nucleotides position 2					
		U	C	A	G		
Nucleotides position 1	U	UUU UUC UUA UUG } phenylalanine	UCU UCC UCA UCG } serine	UAU UAC UAA UAG } tyrosine	UGU UGC UGA UGG } cysteine	U C A G	Nucleotides position 3
	C	CUU CUC CUA CUG } leucine	CCU CCC CCA- CCG } proline	CAU CAC CAA CAG } histidine	CGU CGC CGA CGG } arginine	U C A G	
	A	AUU AUC AUA AUG } isoleucine methionine	ACU ACC ACA ACG } threonine	AAU AAC AAA AAG } asparagine	AGU AGC AGA AGG } serine	U C A G	
	G	GUU GUC GUA GUG } valine	GCU GCC GCA GCG } alanine	GAU GAC GAA GAG } aspartic acid	GGU GGC GGA GGG } glycine	U C A G	

A : Adenine U : Uracile G : Guanine C : Cytosine.

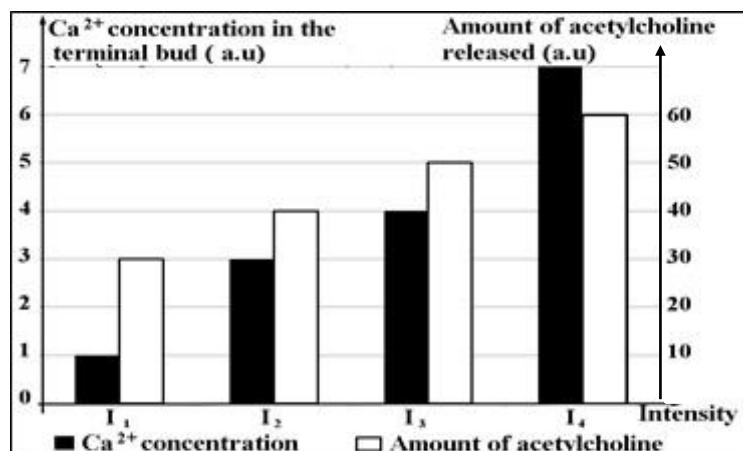
Document 3: Genetic Code

Exercise 3 (6 points)

Synaptic Transmission

Nerve messages are transmitted along the nerve fibers and across synapses. In the framework of studying the synaptic transmission of the nervous message, the following experiments are performed.

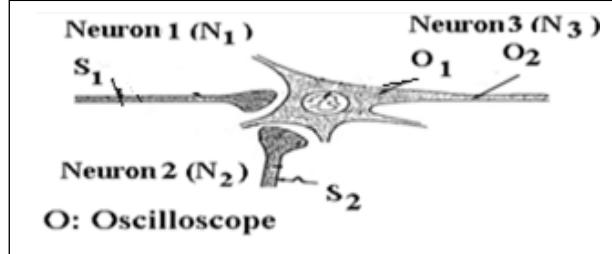
Experiment 1: In a physiological medium and using an experimental set up, four effective stimuli of increasing intensities ($I_1 < I_2 < I_3 < I_4$) are applied on a motor neuron innervating a skeletal muscle.



Document 1

The concentration of Ca²⁺ in the presynaptic terminal bud as well as the amount of acetylcholine released into the synaptic cleft are measured. The obtained results are represented in document 1.

1. List the steps of the transmission of the nervous message at the level of the synapse.
2. Analyze the obtained results.
3. What can you conclude concerning the coding of the nervous message revealed by document 1?



Document 2

Conditions	Recordings	
	O ₁	O ₂
Stimulation S ₁	+30 mV AP -70 mV	+30 mV AP -70 mV
Stimulation S ₂	-70 mV -72 mV	-70mV

Document 3

Conditions	Recordings	
	O ₁	O ₂
Stimulations S ₁ + S ₂	-68 mV -70 mV	-70mV

Document 4

Exercise 4 (6 points)**Uterus and Ovarian Hormones**

The uterus, an organ of the female reproductive system, is made up of 3 layers: an external layer (serous external), a muscle layer (myometrium) and an internal layer (endometrium). This endometrium undergoes cyclic development controlled by the ovarian hormones.

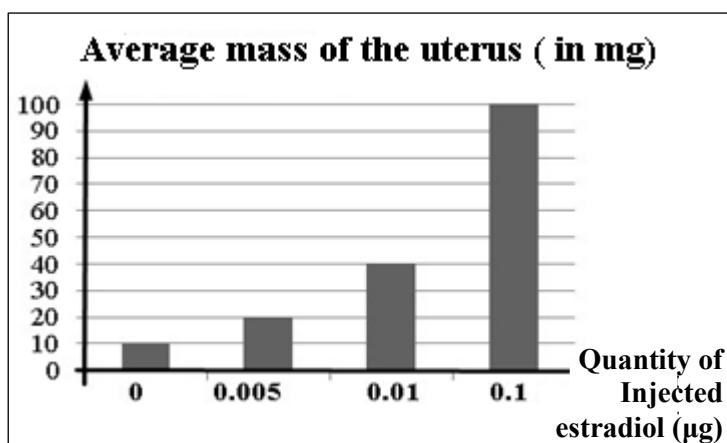
In the framework of studying the effect of the ovarian hormones on the uterus, the following experiments are performed.

Experiment 1: Estradiol of increasing quantities is injected to lots of ovariectomized mice at puberty. Document 1 shows the obtained results.

1. Name the ovarian structures that secrete estradiol during female sexual cycle.
2. Draw a table that represents the variation of the average mass of the uterus (mg) as a function of the quantity of the injected estradiol (μg).

3.1. Analyze the obtained results.

3.2. What can you conclude?



Document 1

Experiment 2: Estradiol and progesterone are injected to lots of ovariectomized mice at puberty. The conditions and the results are represented in document 2.

Lots Injections	Lot 1	Lot 2	Lot 3	Lot 4
Estradiol	-	+	-	+
Progesterone	-	-	+	+

(+): presence (-): absence

Results	No thickening of the endometrium. No uterine lace	Endometrial thickening. No uterine lace	No thickening of the endometrium. No uterine lace	Endometrial thickening with uterine lace
---------	--	--	--	--

Document 2

4. Specify, referring to document 2:

4.1. the role of estradiol.

4.2. the role of progesterone.

5. Show that the action of progesterone on the endometrium of the uterus necessitates the presence of estradiol.

الاسم: مسابقة في مادة علوم الحياة
الرقم: اسس التصحيح

Q.	Exercice 1: Transmission of a Hereditary Character Answer key	Note
1	<p>The fructosemia allele is recessive (1/4). Because, parents 1 and 2 of healthy phenotype have affected children 5 and 6 (1/4). These children inherited the disease allele from at least one of the parents. This parent has the morbid allele in the masked state. Let N be the symbol of the Normal, dominant allele. Let m be the symbol of the allele responsible for recessive fructosemia.</p>	1/2
2	<p>If the gene is localized on the non-homologous segment of Y chromosome, then none of the female should be affected. This is not the case, since female 6 is an affected one. Or father and son would be of the same phenotype because the boy inherits his Y from his father. Or, sons should have the same phenotypes as their fathers (they have inherited the Y chromosome from their father). (1/4).</p> <p>The affected male 5 would have as genotype $X//Y^m$, he has inherited Y^m from his father who would have as genotype $X//Y^m$. Possessing such genotype, the father should be affected, which is not. (1/4).</p> <p>If the gene is localized on the non-homologous segment of X chromosome, then the affected female 6 would have as genotype $X^m//X^m$, she has inherited X^m from his father 2 who would have as genotype $X^m//Y$. Possessing such genotype, the father should be affected, which is not. (1/4).</p> <p>If the gene is localized on the homologous segments of X and Y chromosomes, then the affected female 6 would have as genotype $X^m//X^m$, and similarly the affected male 5 would have as genotype $X^m//Y^m$, female 6 has inherited X^m from her father while male 5 has inherited Y^m from the same father. The father as such should have as genotype $X^m//Y^m$. Possessing such genotype, the father should be affected, which is not. Thus the gene is not gonosomal, therefore autosomal. (1/4).</p>	1
3	3 : $N//m$ or $N//m$ 4 : $N//m$ or $N//m$	1/2
4.1	<p>The electrophogram of individual 3 shows a band that corresponds to the normal allele and another that corresponds to the mutant allele (1/4). Consequently, the genotype of individual 3 is $N//m$ (1/4)..</p> <p>The genotype of individual 4 is $N//N$ (1/4) because he has one band that corresponds to the normal allele (1/4).</p>	1
4.2	The fetus has only the normal allele, so he has two copies of the allele N (1/4). He has a normal phenotype (1/4).	1/2
5	The electrophoresis permits determining the real genotype of the individual.	1/2

Q.	Exercise 2 Fructosemia Answer Key	Note
1.1	Deficiency of aldose B.	1/2
1.2	Children affected by this disease show a dysfunction of the liver and kidney weakness with abnormal high levels of sugar, amino acids and salts in the urine.	1/2
2	The number of nucleotides in the mutated allele is smaller than in the normal allele 18 < 24 (1/4) Nucleotides 7,8,9,10,11 and 12 (or 8, 9, 10, 11, 12 and 13) are absent in the mutated allele (1/4). However, the remaining nucleotides are identical (1/4). Or the first six nucleotides (or the first seven nucleotides) are identical in both sequences (1/4). However, the remaining nucleotides are different (1/4).	3/4
3	Mutation by deletion.	1/4
4.1	mRNA that corresponds to the normal allele: AAU GGA CUG GUA CCU AUU GUU GAA mRNA that corresponds to the mutant allele: AAU GGA CCU AUU GUU GAA	1/2
4.2	The amino acid sequence Asp - Gly - Leu - Val - Pro - Ile - Val - Glu. Diseased mRNA Amino acid sequences: Asp - Gly - Pro - Ile - Val - Glu	1/2
5	The mutation by deletion in DNA was transcribed at the level of the mRNA by the absence of codons which results in an absence of the two amino acids Leu and Val. This new amino acid sequence affects the three-dimensional form of the protein (Enzyme aldolase B) which becomes non-functional. As this enzyme is responsible for the cleavage of fructose 1 phosphate, the change in its function is manifested by fructose intolerance.	1

Q.	Exercise 3: Synaptic transmission Answer key	Note
1	<ul style="list-style-type: none"> - Arrival of an action potential to the presynaptic nerve ending. - Opening of calcium channels and entrance of Ca^{2+} ions into the presynaptic membrane. - Liberation of neurotransmitters by exocytosis into the synaptic cleft. - Fixation of neurotransmitters to postsynaptic receptors. - Opening of chemo-dependent channels. - Genesis of PSP in the postsynaptic element. <p>Recapture of neurotransmitters by the presynaptic neuron or its degradation.</p>	1 1/2
2	As the intensity of the stimulation increases from I_1 to I_4 , the concentration of Ca^{2+} in the presynaptic element increases from 1 to 7, similarly, the amount of released acetylcholine increases from 30 to 60 a.u.	1
3	The nerve message at the level of a synapse is coded by modulation in Ca^{2+} concentration and the neurotransmitter acetylcholine concentration as a function of the intensity of the stimulation.	1/2
4	<p>The synapse N-N3 is excitatory, because following the application of S_1, an AP is obtained at the level of O_1.</p> <p>The synapse N₂-N₃ is inhibitory, because following the application of S_2, a hyperpolarization is obtained at the level of O_1.</p>	2
5	<p>When S_1 and S_2 are applied simultaneously, an EPSP of amplitude +2mV less than threshold is generated at the level of O_1, but a RP at the level of O_2.</p> <p>In fact, the motor neuron plays an integrative role. It sums up algebraically the IPSP produced at the level of the synapse N2N3 and the EPSP produced at the level of the synapse N1N3. These PSPs are added (spatial summation), producing, thus, an EPSP of amplitude +2mV. Since this amplitude is less than threshold of depolarization, it remains incapable of generating a propagating AP at the level of N3 neuron.</p>	1

Q.	Exercice 4: Uterus and Ovarian Hormones Answer key	Note										
1	Estradiol is secreted by: the follicle during the follicular phase and by the corpus luteum during the luteal phase.	1										
2	<table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="padding: 5px;">The quantity of the injected estradiol (μg).</td> <td style="padding: 5px;">0</td> <td style="padding: 5px;">0.005</td> <td style="padding: 5px;">0.01</td> <td style="padding: 5px;">0.1</td> </tr> <tr> <td style="padding: 5px;">The average mass of the uterus (mg)</td> <td style="padding: 5px;">10</td> <td style="padding: 5px;">20</td> <td style="padding: 5px;">40</td> <td style="padding: 5px;">100</td> </tr> </table> <p>The variation of the average mass of the uterus (mg) as a function of the quantity of the injected estradiol (μg).</p>	The quantity of the injected estradiol (μg).	0	0.005	0.01	0.1	The average mass of the uterus (mg)	10	20	40	100	1 1/2
The quantity of the injected estradiol (μg).	0	0.005	0.01	0.1								
The average mass of the uterus (mg)	10	20	40	100								
3.1	The average mass of the uterus increases from 10 to 100 mg when the quantity of the injected estradiol increases from 0 to 0.1 μg .	1										
3.2	Thus, estradiol favors the development of the uterus.	1/2										
4.1	Estradiol is responsible for the thickening of the endometrium because following its injection (lot 2), thickening of the endometrium is observed.	1/2										
4.2	Progesterone is responsible for the formation of the uterine lace because following its injection with estradiol (lot 4), formation of the uterine lace and also thickening of the endometrium are observed.	1/2										
5	Since the injection of progesterone alone (lot 3) does not provoke the formation of uterine lace whereas with estradiol (lot 4) provokes the formation of uterine lace, then the action of progesterone on the endometrium necessitates the presence of estradiol.	1										

الاسم:	مسابقة في مادة علوم الحياة
الرقم:	المدة: ساعتان ونصف

Exercise 1 (6 points)

Huntington Disease

Huntington disease is a rare neurodegenerative disease of the central nervous system. It is characterized by uncoordinated and involuntary movements of great amplitude and by psychological problems. It is due to a mutation at the level of the gene coding for a protein called huntingtin which is essential for the survival of the neurons.

1- Pick out from the text:

Document 1

- 1.1- the origin of Huntington disease.
- 1.2- the symptoms of this disease.

A study is performed on individuals carrying the mutated allele responsible for this disease. Document 2 represents the variation of the percentage of individuals showing the symptoms of the disease as a function of their age.

Age (years)	10	30	40	60	70
Percentage of individuals showing the symptoms of the disease (%)	0	30	60	90	100

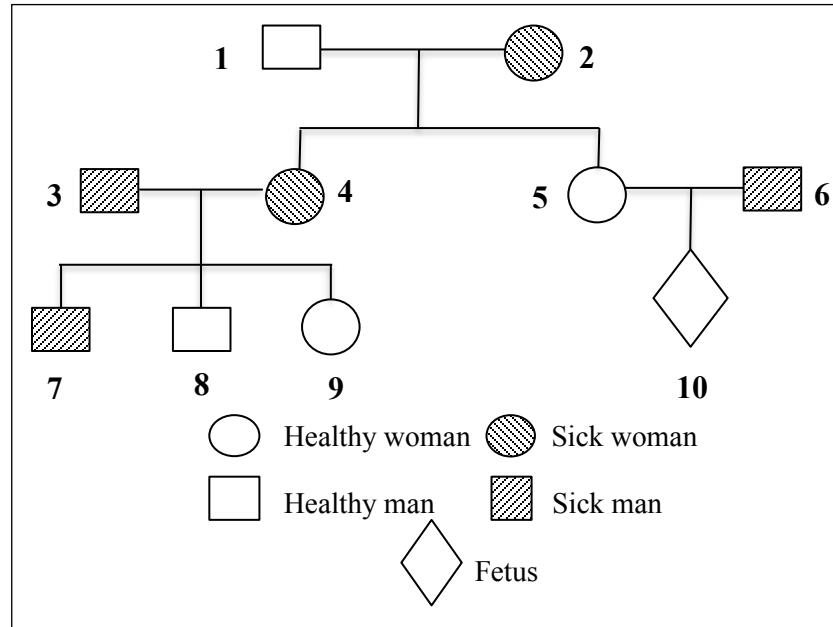
Document 2

2- Interpret the obtained results.

Document 3 shows the genealogical tree of a family which certain members are affected by the disease.

3- Indicate whether the allele of this disease is recessive or dominant. Justify the answer.

4- Determine the localization of the gene responsible for this disease.



Document 3

Bands	Individuals		
	5	6	Fetus
A	—	—	—
B		—	—

DNA analysis is performed on certain individuals of this family using the Southern blot method. The used probe permits to distinguish the mutated allele from the normal one of the studied gene. The obtained results are shown in document 4.

- 5- Specify the band which corresponds to the mutated allele.
- 6- Determine the genotype and the phenotype of the fetus.

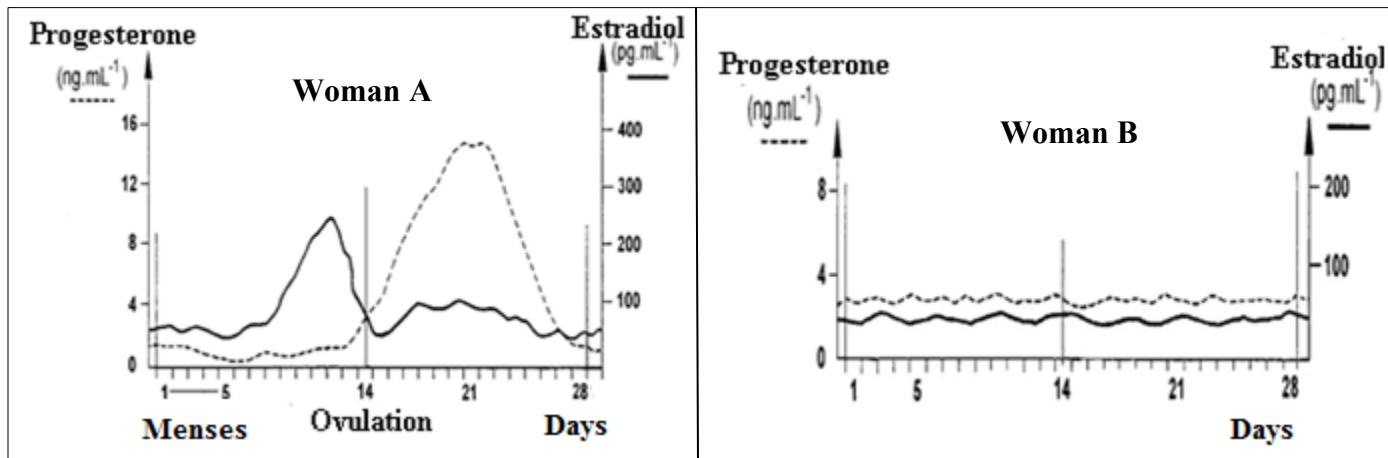
Document 4

Exercise 2 (6 points)

Evolution of the Ovarian Hormones with Age

Female fertility evolves with age; beyond 50 years old, the cyclic menses as well as ovulation disappear. Studies are done to explain the mechanisms at the origin of these modifications.

Study 1: During a period of 28 days, the levels of ovarian hormones are measured in two women: woman (A) whose age is 25 years old and woman (B) whose age is 50 years old, at menopause. The obtained results are presented in document 1.



Document 1

1- Compare in these two women:

1.1- the variations of estradiol levels.

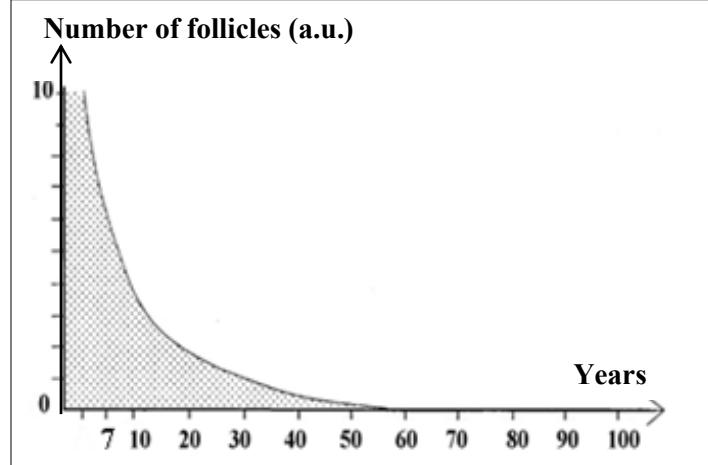
1.2- the variations of progesterone levels.

2- Draw out the effect of age on the ovarian activity.

3- Indicate a role of estradiol and a role of progesterone.

Study 2: The ovarian follicles which are responsible for the secretion of estradiol and progesterone are evaluated throughout the female life span. The obtained results are presented in document 2.

4- Deduce the cause of the variation of the ovarian hormones observed at menopause.



Document 2

In order to determine the cause of the disappearance of the ovarian follicles, two hypotheses are proposed:

Hypothesis 1: The disappearance is due to the aging of the ovary itself.

Hypothesis 2: The disappearance is due to the stoppage of the stimulation of the ovary by the anterior pituitary hormones.

Document 3 represents the evolution in the average plasma level of FSH (pituitary hormone) which stimulates the growth and maturation of ovarian follicles as a function of the age of a woman.

5- Determine which of the two hypotheses is valid.

Age (years)	20-29	34-39	48-54
FSH (in mg.L ⁻¹)	22	34	60

Document 3

Exercise 3 (4.5 points)

Cocaine

In the framework of studying the mode of action of cocaine at the level of the dopamine synapses, the following experiments are performed.

Experiment 1: Dopamine percentage is measured at the level of the synaptic clefts in two lots of rats: the rats of **lot 1** which are considered as control group, and those of **lot 2** which are injected with cocaine at time $t = 0$ minute.

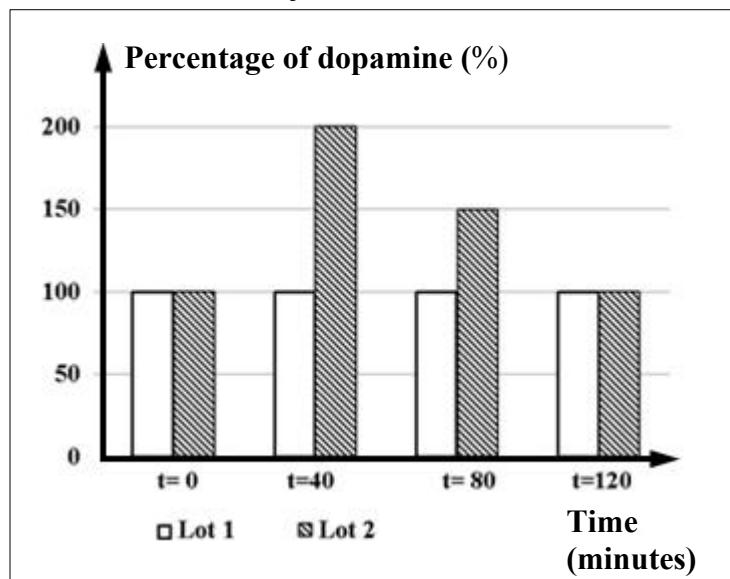
Document 1 represents the obtained results.

1- Represent in the same table, the variation of the percentage of dopamine in the two lots as a function of time.

2.1- Analyze the obtained results.

2.2- What can you conclude?

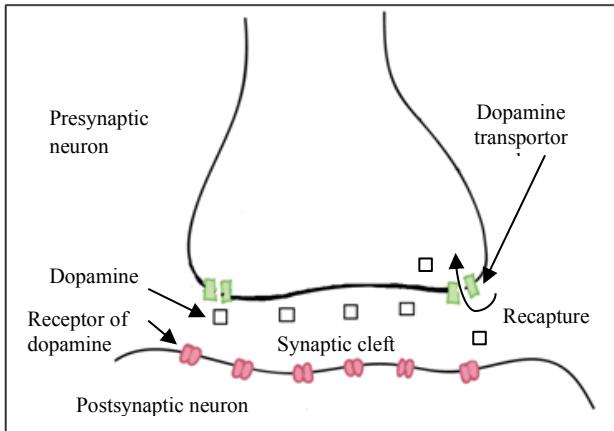
3- Propose two hypotheses explaining the mode of action of cocaine at the level of this synapse.



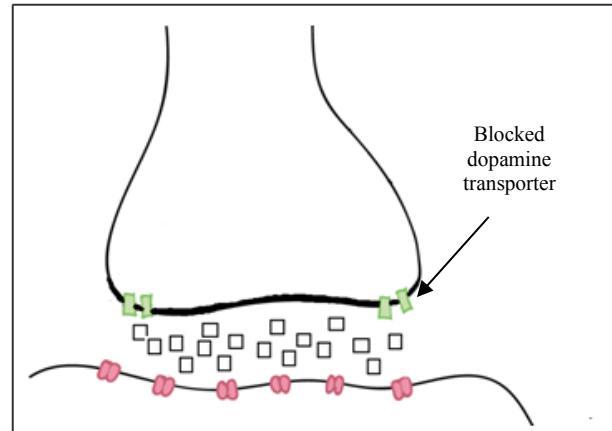
Document 1

Experiment 2: In order to determine the validity of the proposed hypotheses, cocaine is injected into the synaptic cleft of a dopamine synapse.

Document 2 shows the aspect of two synapses, synapse 1 not injected with cocaine (control) and synapse 2 injected with cocaine.



Synapse 1



Synapse 2

Document 2

N.B.: Dopamine transporters are responsible for the recapture of dopamine by the presynaptic neuron.

4- Which of the two proposed hypotheses is valid? Justify the answer.

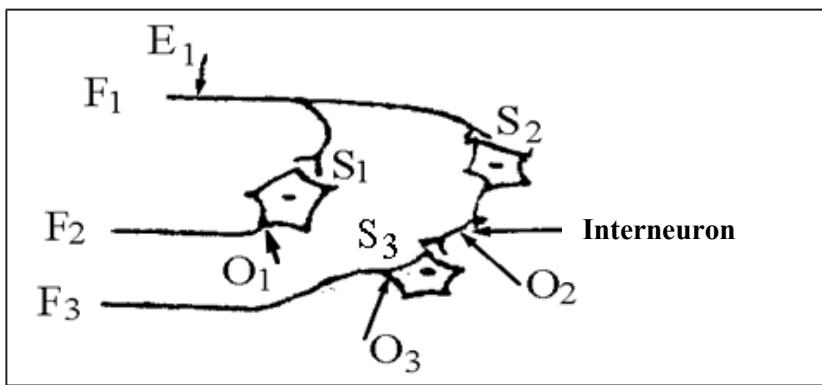
5- Explain the following statement: "Cocaine consumption induces a state of dependence and a state of tolerance".

Exercise 4 (3.5 points)

Synaptic Transmission

In order to study certain aspects of the synaptic transmission, the following experiments are performed using the experimental setup shown in document 1.

Experiment 1: Fiber F₁ is stimulated by an effective intensity (E₁). The results are recorded by oscilloscopes at the level of the three post-synaptic structures: the cone of implantation of the axon F₂ (O₁), the axon of the interneuron (O₂) and the cone of implantation of axon F₃ (O₃). The obtained results are shown in document 2.



Document 1

Oscilloscopes	Recordings
O ₁	Hypopolarization followed by an action potential
O ₂	Action Potential
O ₃	Hyperpolarization

Document 2

- 1- List the steps of the transmission of the nervous message at the level of a synapse.
- 2- Specify the nature of each of the synapses S₁, S₂ and S₃.

Experiment 2: Two types of neurotransmitters, acetylcholine and GABA, are injected into the synaptic clefts of S₁ and S₃. The experimental conditions and the results are presented in document 3.

Synapses	Injected Neurotransmitters	Oscilloscopes	
		O ₁	O ₃
S ₁	Acetylcholine	Hypopolarization	
	GABA	Resting potential	
S ₃	Acetylcholine	Resting potential	
	GABA	Hyperpolarization	

Document 3

- 3- Indicate the site of action (synapse(s) S₁ and/or S₃) for each of the utilized neurotransmitters. Justify the answer.

Q.	Exercise 1 : Huntington Disease Answer key (6 points)	Note
1.1	The origin of Huntington disease is a mutation at the level of the gene coding for a protein called huntingtin.	0.5
1.2	The symptoms of this disease are uncoordinated and involuntary movements of great amplitude and psychological problems.	0.5
2	The percentage of individuals showing the symptoms of the disease increases from 0 % to 100% as the age of these individuals increases from 10 to 70 years. Therefore, the expression of Huntington disease symptoms is enhanced with age.	1.5
3	The mutated allele is dominant over the normal allele because the affected parents (3 and 4) have healthy children (8 and 9). This means that the allele for normal is present in the parents but it is masked by the allele responsible for the disease. (D = allele responsible for Huntington disease; n = normal allele) D>n	0.5
4	<ul style="list-style-type: none"> - If the allele responsible for the disease is carried on the non –homologous segment of chromosome Y then, it should be transmitted from father to son; however the affected father (3) has a healthy son (8). Therefore, the allele is not carried by the non-homologous segment of chromosome Y. - If the allele of the disease is carried on the non-homologous segment of chromosome X, then the affected father (3) should transmit this dominant allele to all his daughters who should be all affected; however, his daughter (9) is healthy, thus the allele is not carried on the non-homologous segment of X chromosome. - If the allele is carried on the homologous segment of chromosome X and Y, then boy (8) who is normal should have the genotype $X^n Y^n$, inheriting X^n from his mother(4) and Y^n from his father (3). His sister (9) who is normal should have the genotype X^nX^n taking X^n from her father (3) and therefore he should have the genotype X^nY^n and should be healthy but it is not the case. Therefore, the allele is not carried by the homologous segment of chromosomes X and Y. <p>Hence the allele responsible for the disease is carried on an autosome.</p>	1
5	The band at the level of B corresponds to the mutant allele. This is because the DNA analysis of individual 5 who is healthy shows only a thick band at the level of A indicating that band A corresponds to the normal allele. On the other hand, individual 6 who is diseased shows 2 thin bands at the level of A and another at the level of B. Hence, that band at the level of B corresponds to the mutant allele which is responsible for the disease.	1
6	Since the fetus has two thin bands at the levels of A and B corresponding to the normal allele and mutant allele respectively, then the genotype of the fetus is D//n. Since the mutant allele D is dominant over the normal allele n, the phenotype of the fetus is [D].	1

Q.	Exercise 2: Evolution of the Ovarian Hormones with Age Answer key (6points)	Note				
1.1	In woman A, during a period of 28 days, the estradiol level shows two peaks: the first peak reaches approximately 250 pg/ml around day 12, and a second lower peak that reaches 100 pg/ml around day 21. On the other hand, Woman B shows constant variation of estradiol that fluctuates around 50 pg/ml during the same interval of time (from day 0 till day 28).	1				
1.2	In woman A, during a period of 28 days, the progesterone level shows only one peak that reaches approximately 15 ng/ml around day 21. On the other hand, Woman B shows constant variation of progesterone that fluctuates around 3ng/ml during the same interval of time (from day 0 till day 28).	1				
2	At menopause, the cyclic variation of ovarian hormones disappear indicating the cease of the ovarian activity.	0.5				
3	<table border="1"> <thead> <tr> <th>Estrogen (any of the following roles)</th><th>Progesterone (any of the following roles)</th></tr> </thead> <tbody> <tr> <td> <ul style="list-style-type: none"> - Proliferation of the uterine and vaginal mucosa - Development tube-like glands of the endometrium. - Development of the cervical glands - Growth of blood vessels. </td><td> <ul style="list-style-type: none"> - Stimulation of gland secretions in the uterine mucosa and the cervix. - Development of spiral arterioles. - Increase in the body temperature. - Inhibition of the uterine contractions. </td></tr> </tbody> </table>	Estrogen (any of the following roles)	Progesterone (any of the following roles)	<ul style="list-style-type: none"> - Proliferation of the uterine and vaginal mucosa - Development tube-like glands of the endometrium. - Development of the cervical glands - Growth of blood vessels. 	<ul style="list-style-type: none"> - Stimulation of gland secretions in the uterine mucosa and the cervix. - Development of spiral arterioles. - Increase in the body temperature. - Inhibition of the uterine contractions. 	1
Estrogen (any of the following roles)	Progesterone (any of the following roles)					
<ul style="list-style-type: none"> - Proliferation of the uterine and vaginal mucosa - Development tube-like glands of the endometrium. - Development of the cervical glands - Growth of blood vessels. 	<ul style="list-style-type: none"> - Stimulation of gland secretions in the uterine mucosa and the cervix. - Development of spiral arterioles. - Increase in the body temperature. - Inhibition of the uterine contractions. 					
4	The number of the ovarian follicles which are responsible for the secretion of estradiol and progesterone decreases from 10 a.u. at around 7 years old to reach 0 a.u. beyond 50 years old. Therefore, the constant level of ovarian hormones and the absence of their cyclic variation observed at menopause are due to the depletion of ovarian follicles.	1				
5	Given that FSH is responsible for the growth and maturation of ovarian follicles and since by referring to doc 3, the plasma level of FSH increases to 60 mg/l at menopause (48 -54 years old), then the cause of the disappearance of ovarian follicles is not due to the stoppage of the stimulation of the ovary by the anterior pituitary hormones (FSH), and therefore the first hypothesis (The disappearance of the follicles is due to the aging of the ovary) is the valid one.	1.5				

Q.	Exercise 3: Cocaine Answer key (4.5 points)	Note																	
1	<table border="1"> <thead> <tr> <th colspan="2">Time (minutes)</th> <th>0</th> <th>40</th> <th>80</th> <th>120</th> </tr> <tr> <th rowspan="2">Percentage of dopamine (%)</th> <th>Lot 1</th> <td>100</td> <td>100</td> <td>100</td> <td>100</td> </tr> </thead> <tbody> <tr> <th>Lot 2</th> <td>100</td> <td>200</td> <td>150</td> <td>100</td> </tr> </tbody> </table> <p>Variation of the percentage of dopamine (a.u.) as a function of time (minutes) in 2 lots of rats.</p>	Time (minutes)		0	40	80	120	Percentage of dopamine (%)	Lot 1	100	100	100	100	Lot 2	100	200	150	100	1
Time (minutes)		0	40	80	120														
Percentage of dopamine (%)	Lot 1	100	100	100	100														
	Lot 2	100	200	150	100														
2.1	The percentage of dopamine is 100% at time= 0 min in both lots of rats, lot 1, the control group, and lot 2 which are injected with cocaine. The % of dopamine remains constant(100%) during 120 min while it duplicates after 40 min in lot 2 to reach maximum of 200% then it decreases back to the initial level (100%) at 120 minutes.	0.5																	
2.2	Cocaine amplifies the level of dopamine in the synaptic cleft for a certain period of time.	0.5																	
3	Hypothesis1: Cocaine increases the release of dopamine into the synaptic cleft. Hypothesis2: Cocaine prevents or decreases the recapture of dopamine by the presynaptic neuron.	1																	
4	Hypothesis 2 is valid, because according to document2, dopamine transporters which are responsible for the recapture of dopamine after being released in to the synaptic cleft are blocked in the presence of cocaine and eventually it leads to excess dopamine in the synaptic cleft for a longer duration of time. .	0.5																	
5	The consumption of cocaine leads to a sensation of pleasure followed by a desire to continue its use. This leads to a state of dependence. The repetitive use of cocaine will lead to the adaptation of the body to the repeated doses of the product and loss of its effect. Consequently, the consumer tends to increase the dose in order to obtain the desired effect. This leads the consumer to a state of tolerance.	1																	

Q.	Exercise 4: Synaptic transmission Answer key (3.5 points)	Note
1	<ul style="list-style-type: none"> - The steps of the transmission of the nervous message at the level of a synapse are: - Arrival of action potential at the presynaptic terminal buds leads to the opening of calcium voltage gates. The inflow of Ca^{2+} ions into the presynaptic terminal bud causes the fusion of vesicles that contain neurotransmitters with the presynaptic membrane. - The release of neurotransmitters by exocytosis into the synaptic cleft. - The Binding of neurotransmitters to postsynaptic receptors allow the opening of chemical-dependent channels that modifies the membrane potential thus creating PSP at the level of the postsynaptic membrane. - Later, the neurotransmitters are rapidly destroyed by a specific enzyme or recaptured by the presynaptic neuron. 	1
2	<p>S_1 and S_2 are excitatory synapses because O_1 and O_2 connected to the fibers of the postsynaptic neurons recorded A.P after applying an effective intensity of stimulation, E_1, to the nerve fiber F_1 of the presynaptic neuron.</p> <p>S_3 is an inhibitory synapse because for the same effective stimulation E_1, hyperpolarization is recorded by O_3 connected to the post synaptic neuron F_3.</p>	1.5
3	<p>Acetylcholine acts at the level of the synapse S_1 only because it records a hyp polarization at the level of O_1 but not O_3 where it records a resting potential. On the other hand, GABA acts at the level of the synapse S_3 only because it records a hyperpolarization at the level of O_3 but not O_1 where it records a resting potential.</p>	1

الاسم: مسابقة في مادة علوم الحياة
الرقم: المدة: ساعتان ونصف

Exercise 1 (5 points)

Hemophilia B

Hemophilia B is characterized by the absence of blood clotting which may lead to significant bleeding. It is due to mutations in the F9 gene coding for the coagulation factor IX. The severity of the clinical manifestations depends on the severity of the factor IX deficiency: if the biological activity of the coagulation factor is less than 1%, hemophilia is severe. However, if it is between 5 and 40% hemophilia is minor.

1. Pick out from the text:

- 1.1. the cause of hemophilia B.
- 1.2. the consequence of hemophilia B.

Document 1 shows the genealogical tree of a family in which some members are affected with hemophilia B.

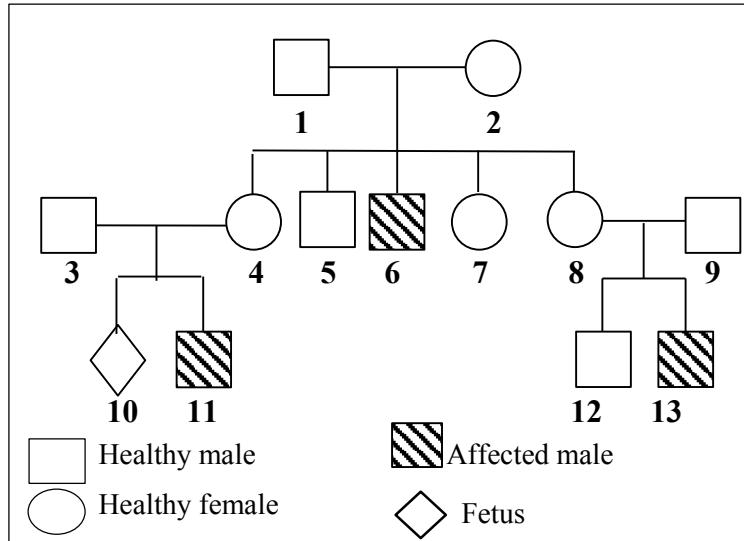
2. Specify whether the allele responsible for hemophilia B is dominant or recessive.
3. Show that the gene responsible for hemophilia B might be located on the non-homologous segment of the X chromosome.

Document 2 represents the number of alleles of the studied gene in males and females.

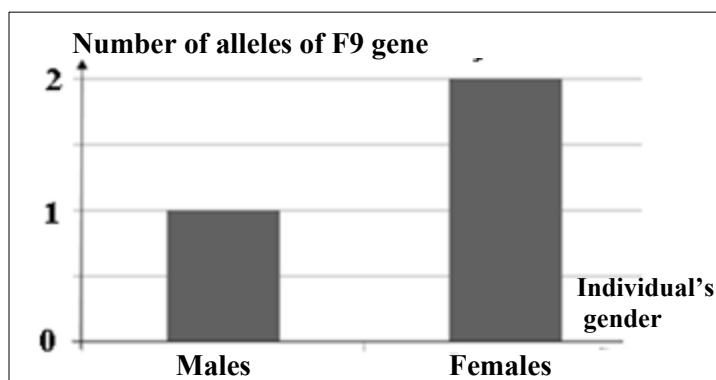
4. Do the results of document 2 confirm the localization of the F9 gene on the non-homologous segment of the X chromosome? Justify the answer.

Using a specialized technique, a DNA analysis of F9 gene in couple (3-4) and in the fetus 10 is carried out. Several DNA fragments are obtained, the size of which can be measured in kilobases (document 3).

5. Identify, from document 3, the fragment that corresponds to hemophilia B allele.
6. Draw out the phenotype of the fetus 10.



Document 1



Document 2

Individuals Fragments of DNA (kb)	3 Father	4 Mother	10 Fetus
1.8	—	—	
1.3		—	—

Document 3

Exercise 2 (5 points)

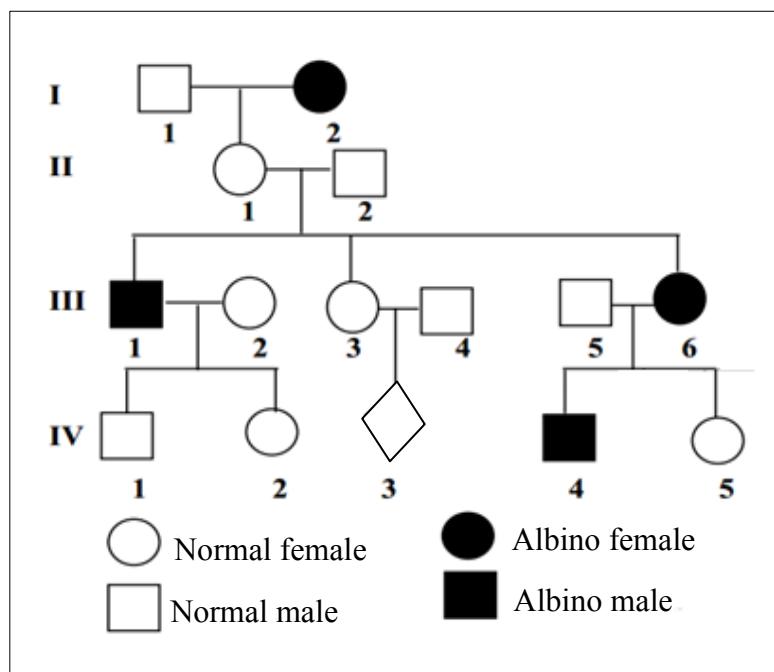
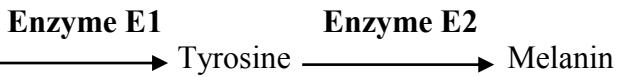
Albinism is a hereditary autosomal and recessive disease caused by the absence of melanin, a pigment responsible for hair color. Document 1 represents the pedigree of a family where some members are affected by the disease.

- Indicate the genotypes of individuals I-1, II-1, and III-6.

In the population where this family lives, among each 100 normal individuals 15 are heterozygous.

- Determine the genetic risk for the fetus IV-3 to be albino.

Researchers have identified gene E1 coding for enzyme E1 and gene E2 coding for enzyme E2. These enzymes are essential for the synthesis of melanin according to the following reactions:



Document 1

To find the exact origin of albinism in this family, the researchers determined the nucleotide sequences of specific parts of the non-transcribed DNA strand of gene E1 (document 2a) and gene E2 (document 2b) for a normal individual and an albino individual of this family.

Non-transcribed DNA strand of gene E1									
	1	2	3	4	5	6	7	8	9
Normal individualACG	AGG	CCT	ACG	GGC	TTA	TGG	GGC	GAA...
Albino individualACG	AGG	CCT	ACG	GGC	TTA	TGG	GGC	GAA...

Document 2a

Non-transcribed DNA strand of gene E2									
	1	2	3	4	5	6	7	8	9
Normal individual	...ATC	ATG	CGA	ACC	GGC	TGC	TCA	AAC	CCA...
Albino individual	...ATC	ATG	CGA	ACC	GGC	TGC	TGA	AAC	CCA...

Document 2b

		Second letter					
		U	C	A	G		
First letter	U	UUU Phe UUC UUA UUG	UCU Ser UCC UCA UCG	UAU Tyr UAC UAA UAG	UGU Cys UGC UGA STOP UGG Trp	U	C
	C	CUU Leu CUC CUA CUG	CCU Pro CCC CCA CCG	CAU His CAC CAA CAG	CGU Arg CGC CGA CGG	C	A
	A	AUU Ile AUC AUA AUG Met	ACU Thr ACC ACA ACG	AAU Asn AAC AAA AAG	AGU Ser AGC AGA AGG	A	G
	G	GUU Val GUC GUA GUG	GCU Ala GCC GCA GCG	GAU Asp GAC GAA GAG	GGU Gly GGC GGA GGG	G	U
		Third letter					

- Show that the gene responsible for albinism in this family is gene E2.
- Determine, using the genetic code (document 3), the amino acid sequence of enzyme E2 that corresponds to each of the two individuals, the normal and the albino.
- Explain how the modification in the nucleotide sequence of the allele coding for enzyme E2 leads to albinism in this family.

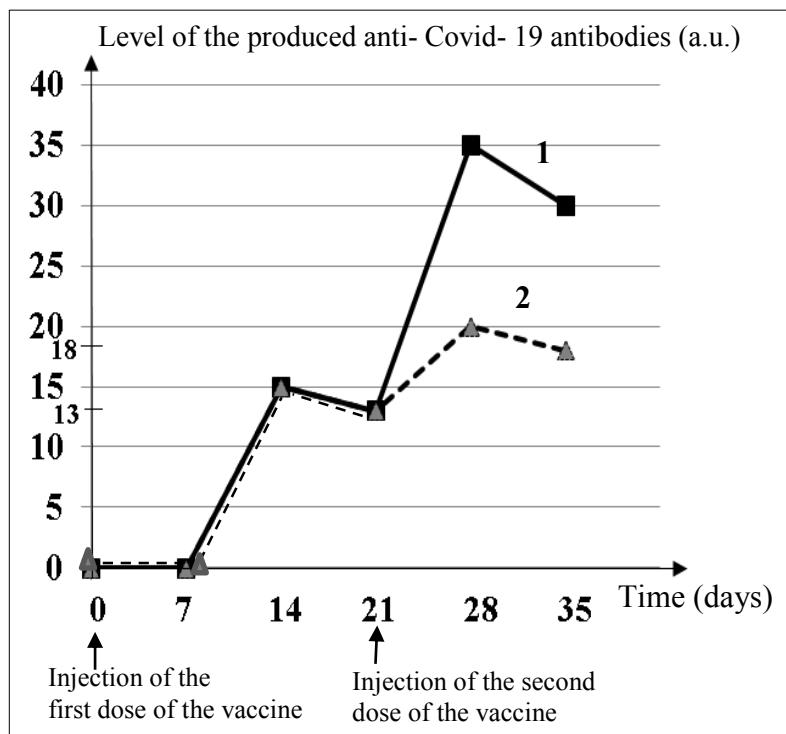
Document 3

Exercise 3 (6 points)**Effectiveness of Vaccines against Covid-19**

Faced with the “corona” pandemic caused by the Covid-19 virus, the world has experienced the largest vaccination campaign in history. Thus, nowadays several vaccines are in the market. Among these vaccines are vaccine X and vaccine Y. Researchers study the effectiveness of each of the two types of vaccines.

For this sake, two injections of the same vaccine, either X or Y, are introduced to healthy individuals who have never been affected by Corona. Then, researchers measure the variation of the antibody level as a function of time after each injection. Document 1 represents the results obtained in individuals vaccinated by X (curve 1) and in individuals vaccinated by Y (curve 2).

1. Indicate the type of specific immune response triggered by these vaccines. Justify the answer.
2. Name the cells that secrete antibodies.
3. Construct a table that represents the results of document 1.
4. Justify that the secondary immune response is:
 - 4.1. faster than the primary immune response.
 - 4.2. more amplified than the primary immune response.
5. Specify which vaccine is more effective against Covid-19.

**Document 1**

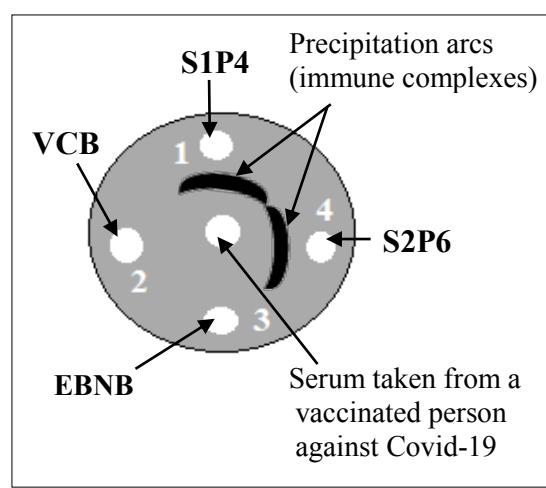
To identify some of the antigenic determinants of Covid-19 virus, an immunodiffusion in gel is performed.

We deposit:

- the serum taken from a vaccinated person against covid-19 virus in the central well.
- one antigenic substance S1P4, VCB, EBNB, or S2P6, in each of the wells; 1, 2, 3 and 4.

The results are shown in document 2.

6. Draw out the antigenic determinants of Covid-19 revealed by the obtained results.
7. Schematize the mechanism which leads to the formation of an immune complex.

**Document 2**

Exercise 4 (4 points)**Relationship between Hypothalamus, Pituitary and Ovaries**

We aim to study the relations among the hypothalamus, pituitary gland and ovaries.

Three experiments are performed on adult female mammals. Document 1 represents the experimental conditions and the obtained results.

1. Draw out from document 1:

- 1.1. the role of the hypothalamus.
- 1.2. the hypothalamic hormone.

2. Show, by referring to experiments 2 and 3, that the action of the hypothalamus on the ovaries is indirect.

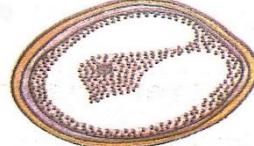
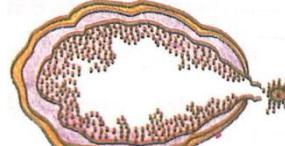
In experiment 4, an adult female mammal is injected with GnRH labeled with radioactive tritium. The autoradiography of pituitary and ovarian sections shows radioactivity only at the level of the pituitary.

Experiments	Conditions	Results
1	Lesion of the hypothalamus	Disappearance of ovarian cycle
2	Lesion of the hypothalamus followed by a discontinuous injections of hypothalamic extract containing GnRH	Restoration of the ovarian cycle
3	Lesion of the hypothalamus + Ablation of the pituitary gland + discontinuous injections of GnRH	Disappearance of ovarian cycle

Document 1

3. Interpret the results obtained in experiment 4.

Experiments 5 and 6 are performed on two lots of female mammals that did not reach puberty and whose ovaries have only primordial follicles. The conditions and results are represented in document 2.

Experiments	Conditions	Results : Microscopic Observation
5	Injections of FSH	Mature follicle 
6	Injections of FSH followed by injection of LH	Ovulation: Rupture of the mature follicle 

Document 2

4. Specify the role of each of the pituitary hormones FSH and LH.

الاسم:	علوم الحياة
الرقم:	أسس التصحيح

Parties	Exercise 1 (5 points)	Hemophilia B	Notes
1.1		It is caused by mutations in the F9 gene coding for the coagulation factor IX.	0.5
1.2		Hemophilia B is characterized by the absence of blood clotting which may lead to significant bleeding.	0.5
2		The allele responsible for hemophilia is recessive because the normal parents 1 and 2 gave birth to a diseased child 6. This means that the allele responsible for the disease (d) is present in the genotype of at least one of the 2 parents and it is masked by the normal dominant allele (N).	0.75
3		Since the disease affects only males, then the gene is sex-linked. Since the affected boy 6 has a normal father 1, then this gene is not located on the non-homologous segment of Y. Therefore it might be localized on the non-homologous segment of X.	0.75
4		Yes. If the gene is localized on homologous autosomes or on homologous segment of X and Y, then the number of alleles of F9 gene in males and females should be two, but it is not the case. If the gene is localized on the non homologous segment of Y, then the females should not have any allele for the gene, which is not the case. Since the number of alleles in the females. If the gene is localized on the non homologous segment of x, then the male should possess one allele of the gene while the female should possess two alleles of the gene.	1
5		The DNA analysis of father 3 who is normal and whose genotype is XNY shows one band at the level of 1.8 Kb, then this band corresponds to the normal allele. Mother 4 who is normal but has a diseased child 11 is of genotype XNXd. Her DNA analysis shows two bands at the level of 1.8 Kb, which corresponds to the normal allele and another band at the level of 1.3 Kb which corresponds to the allele of the disease.	1
6		The fetus 10 is affected with hemophilia B.	0.5

Exercise 2 (5 points)		Albinism
1	N; normal dominant a: affected recessive Genotype I-1 N//N or N//a Genotype II-1 N//a Genotype III-6. a//a	1
2	Genetic Risk= frequency of father to be hetero X frequency of mother to be hetero X probability of having an affected child frequency of father III-4 to be hetero = 15/100 (no family history) frequency of mother III-3 to be hetero = 2/3 (having affected brother or sister) probability of having an affected child = 1/4 Genetic Risk= $15/100 \times 2/3 \times 1/4 = 10/400 = 1/40$	1
3	Since mutation is observed only at the level of gene E2, where in the seventh triplet the second nitrogenous base C in the normal individual is substituted by T in the albino individual. Therefore, the gene responsible for albinism in this family is gene E2 only.	1
4	The transcribed strand of the normal individual TAG TAC GCT TGG CGG ACG AGT TTG GGT... The m RNA of the normal individual: AUC AUG CGA ACC GGC UGC UCA AAC CCA... The amino acid sequence of the normal individual: Ile – Met – Arg – Thr – Gly – Cys – Ser – Asn – Pro – The transcribed strand of the albino individual TAG TAC GCT TGG CGG ACG ACT TTG GGT... The m RNA of the albino individual: AUC AUG CGA ACC GGC UGC UGA AAC CCA... The amino acid sequence of the normal individual: Ile – Met – Arg – Thr – Gly – Cys	1
5	The mutation by substitution at the level of the seventh triplet where the second nitrogenous base C is substituted by G in the mutant allele leads to a stop codon in the sequence of nucleotide in the transcribed m RNA. The translation of this mRNA results in an incomplete/truncated/ peptide of abnormal 3D structure. The non-functional E2 enzyme is not able to transform tyrosine into melanin, thus leading to albinism.	1

Exercise 3 (6 points)		Effectiveness of Vaccines against Covid-19																												
1	The type of specific immune response induced by these vaccines is humoral, since such injection induces the production of antibodies which are the effector molecules of humoral immune response.		0.5																											
2	Plasma cells or plasmocytes		0.25																											
3	<p style="text-align: center;">Injection of the first dose of the vaccine ↓ Injection of the second dose of the vaccine ↓</p> <table border="1" style="margin-left: auto; margin-right: auto;"> <thead> <tr> <th>Time (days)</th><th>0</th><th>7</th><th>14</th><th>21</th><th>28</th><th>35</th></tr> </thead> <tbody> <tr> <td>Level of produced anti-Covid-19 antibodies (a.u.)</td><td>Vaccine X</td><td>0</td><td>0</td><td>15</td><td>13</td><td>35</td></tr> <tr> <td></td><td>Vaccine Y</td><td>0</td><td>0</td><td>15</td><td>13</td><td>20</td></tr> <tr> <td></td><td></td><td></td><td></td><td></td><td></td><td>18</td></tr> </tbody> </table> <p style="text-align: center;">Variation of level of the produced anti-Covid-19 antibodies (a.u) as a function of time</p>	Time (days)	0	7	14	21	28	35	Level of produced anti-Covid-19 antibodies (a.u.)	Vaccine X	0	0	15	13	35		Vaccine Y	0	0	15	13	20							18	1.75
Time (days)	0	7	14	21	28	35																								
Level of produced anti-Covid-19 antibodies (a.u.)	Vaccine X	0	0	15	13	35																								
	Vaccine Y	0	0	15	13	20																								
						18																								
4.1	The latency time for the production of antibody as a response of the second dose of vaccine X or Y (the secondary response), is almost null which is shorter than that in case of its primary response 7 days. Thus, the secondary immune response is faster.		0.75																											
4.2	<p>The maximum antibody level produced during the secondary response for the vaccine X is 35 a.u. that is greater than that in case of its primary response 15 a.u. Thus, the secondary immune response is more amplified.</p> <p>Or the maximum antibody level produced during the secondary response for the vaccine Y is 20 a.u. that is greater than that in case of its primary response 15 a.u. Thus, the secondary immune response is more amplified.</p>		0.75																											
5	The vaccine that is more effective against Covid-19 is vaccine X, since the maximum antibody level produced during the secondary response for vaccine X is 35 a.u. which is greater than that of the secondary response for the vaccine Y which is 20 a.u.		0.75																											
6	The antigenic determinants are S1P4 and S2P6		0.5																											
7	<p>The mechanism of the formation of an immune complex.</p>		0.75																											

Parties	Exercise 4 (4 points) Relationship between Hypothalamus, Pituitary and Ovaries	Notes
1.1	The hypothalamus is indispensable for the appearance of the ovarian cycles	0.5
1.2	The GnRH	0.5
2	The ovarian cycle disappears in the adult female mammals following the lesion of the hypothalamus and discontinuous injections of GnRH in the absence of the pituitary gland (experiment 3). However, such cycle is restored in the presence of the pituitary gland (experiment 2), thus the action of the hypothalamus on the ovaries is indirect.	1
3	Radioactive GnRH is detected only at the level of the section of the pituitary following the injection of GnRH labelled with radioactive tritium in the adult female mammals.. Thus reveals that GnRH receptors are located only at the level of the pituitary.(the pituitary is the target organ of GnRH)	1
4	FSH enhances the development of the primordial follicles into mature follicle, since in experiment 5, mature follicle is observed following the injections of FSH. LH provokes ovulation since in experiment 6, after the addition of the LH injection to the FSH injections, the result shows the rupture of mature follicle indicating ovulation.	1

الاسم: الرقم:	مسابقة في مادة علوم الحياة المدة: ساعتان ونصف
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Exercise 1 (5 points)

Hereditary Diseases

Duchenne muscular dystrophy and Huntington's chorea are two hereditary diseases one of which is observed in a family.

Document 1 summarizes the modes of transmission of these two diseases and document 2 represents the genealogical tree of this family.

- Show, referring to document 2, that the disease affecting the family is dominant.
- Draw out the disease present in the family.

Document 3 represents the result of electrophoresis of the DNA of the gene responsible for the disease affecting the family.

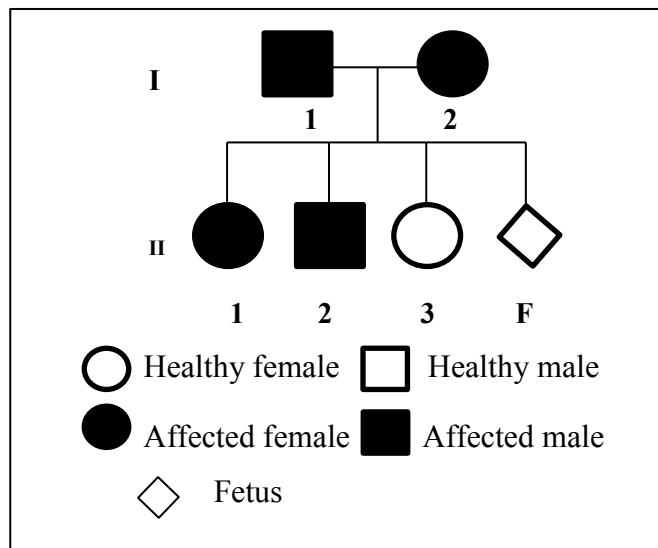
- Write the genotype of the fetus.
- Specify, if this fetus will be affected by the disease.

DNA analysis showed the presence of allele A1 in the two sisters of the fetus, II1 and II3.

- Identify which of the alleles, A1 or A2, is the one responsible for the disease.
- Write the genotypes of the brother and the two sisters of the fetus.

Modes of transmission of the diseases
Duchenne muscular dystrophy: A disease controlled by a recessive allele carried by the sex chromosome X.
Huntington's chorea: A disease controlled by a dominant allele carried by an autosome (non-sex chromosome)

Document 1



Document 2

Alleles of the studied gene	A1	Fetus
	■	■
	■	■

Document 3

Exercise 2 (6 points)**Cellular Cooperation**

A plasmocyte is an active differentiated B lymphocyte (BL). It performs its role in two steps: the proteosynthesis, where a plasmocyte synthesizes enormous number of the same clone of antibody that is protein in nature, and the secretion of these antibodies by exocytosis across its cytoplasmic membrane. When plasmocytes perform these steps, they consume a huge amount of energy and die afterward.

- 1- Pick out from the text the name of each of the two steps performed by plasmocytes.
- 2- Draw out the type of the considered specific immune response.
- 3- Indicate one structural modification that an inactive BL undergoes when being differentiated into plasmocyte.

In the framework of studying the mechanism of the activation of B lymphocyte into plasmocyte, many experiments are performed on three lots of mice. These lots are injected with different cells as shown in document 1.

- 4- Describe each of these experiments.
- 5- Interpret the obtained results.

Some mice of lot 2 are injected with interleukin 2 (IL_2) while others are injected with interleukin 4 (IL_4). These interleukins are molecules released by active T4 Lymphocytes (T4 L).

Ablation of the thymus then irradiation of the mice			
Lot 1	Sheep red blood cells (SRBC) + BL + macrophages + T4L	Sheep red blood cells (SRBC) + BL + macrophages + T4L	Presence of plasmocytes
Lot 2	SRBC + BL + macrophages	SRBC + BL + macrophages	Absence of plasmocytes
Lot 3	BL + macrophages + T4 L	BL + macrophages + T4 L	Absence of plasmocytes
SRBC: Antigen		: Injection	

Document 1

Only the B lymphocytes of the mice injected with IL_4 are transformed into plasmocytes.

- 6- What do these results reveal?

To study the mechanism of the elimination of the antigen, SRBCs are added to the serum taken from mice of lot 1. The observation of the aspect suspension shows agglutination of the SRBC by the formation of immune complexes. These immune complexes are then separated in three different cultures. Many experiments are performed where the conditions and the results are shown in document 2.

Culture	Conditions	Results
Culture 1	Immune complex	Persistence of agglutinated SRBC
Culture 2	Macrophages Immune complex	Disappearance of immune complex
Culture 3	Complement proteins Immune complex	Disappearance of immune complex

Document 2

- 7- Explain the results obtained in cultures 2 and 3.

Exercise 3 (5 points)**Ovaries-Hypophysis Interaction**

The ovaries, by the intermediate of their hormones, interact with the anterior pituitary gland to induce ovulation.

- 1- Define ovulation.
- 2- Name the structure(s) that secrete each of the ovarian hormones: estrogen and progesterone.

In the framework of studying the aspects of the interaction between the ovaries and the hypophysis, two series of experiments are performed on adult female monkeys.

First series of experiments:

Five different lots of adult female monkeys that are previously hypophysectomized (undergoing ablation of the hypophysis) are subjected to different manipulations. The conditions and the obtained results are summarized in document 1.

	Conditions		Results		
	FSH	LH	Secretion of estrogen	Secretion of progesterone	Ovulation
Lot 1	No perfusion	No injection	No	No	No
Lot 2	Continuous perfusion for 10 days	No injection	Yes	No	No
Lot 3	Continuous perfusion for 10 days	Injection of high dose at day 12	Yes	Yes	Yes
Lot 4	Continuous perfusion for 10 days	Injection of low dose at day 12	Yes	No	No
Lot 5	No perfusion	Injection of high dose at day 12	No	No	No

Document 1

- 3- Indicate the importance of the ablation of the hypophysis in the adult female monkeys before carrying out the experiments.
- 4- Draw out:
 - 4.1 the role of FSH
 - 4.2 the conditions of ovulation
- 5- Explain this statement: “Progesterone will not be secreted unless ovulation has taken place”.

Second series of experiments:**Experiment 1:**

Female monkeys are subjected to bilateral ovariectomy; the average LH level reaches 25.1 ng/ml.

Experiment 2:

The ovariectomized female monkeys are injected with moderate dose of estrogen; the average level of LH reaches 5.3 ng/ml.

Experiment 3:

The ovariectomized female monkeys are injected with high dose of estrogen; the average level of LH reaches 35.2 ng/ml.

- 6- Indicate the type of feedback control revealed by each of the experiments 2 and 3. Justify the answer.

Exercise 4 (4 points)**Detection of Genetic Polymorphism**

In the framework of studying the genetic polymorphism, a study is performed on a gene having two alleles: one is normal and the other is mutant.

Document 1 shows the partial sequences of the two alleles of this gene.

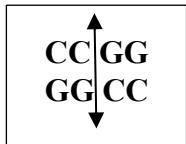
	Normal allele	Mutant allele
Non-transcribed strand	GGAACCGGAT	GGAACAGGAT
Transcribed strand	CCTTGGCCTA	CCTTGTCCCTA

Document 1

1.1- Compare the two nucleotide sequences.

1.2- Draw out the type of the mutation.

Document 2 represents the restriction site of an enzyme X.

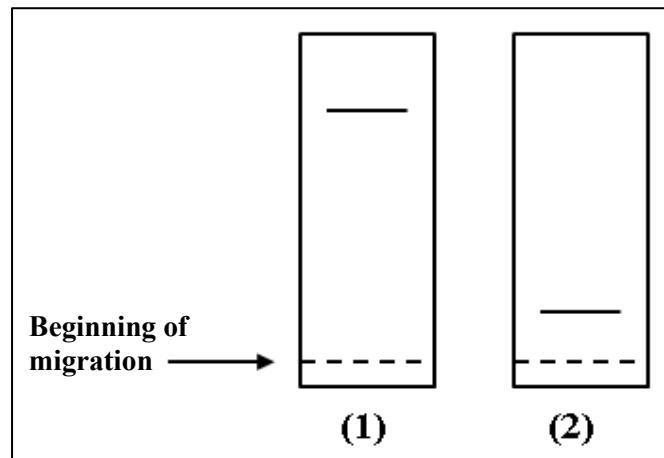
**Document 2**

- 2-** Determine, for each of the two sequences, the number and the nucleotide sequence of the fragments obtained after cutting the DNA by enzyme X.

To visualize these fragments, we use a radioactive molecular probe of sequence **GAAC**.

The obtained electrophoreograms are shown in document 3.

- 3-** Match each electrophoreogram to one of the alleles of the studied gene. Justify the answer.

**Document 3**

الاسم:	علوم الحياة
الرقم:	أسس التصحيح

Parts	Exercise 1 (5 points) Heredity Diseases	Notes
1	The two affected parents I1 and I2 have a healthy daughter II3. This means that at least one of the parents has the healthy allele that is being masked by the affected one. Therefore, the healthy allele is recessive and allele of the disease is dominant.	0.75
2	The family is affected by Duchenne muscular dystrophy.	0.75
3.1	The genotype of the fetus is A1//A2	0.5
3.2	The fetus is heterozygous, having both alleles A1 and A2 of the gene. The allele of the disease exists in the genotype of the fetus F. This allele is dominant and will necessarily express itself. The fetus will therefore be sick	1
4	Daughter II3 has a healthy recessive phenotype, thus, she is obligatory homozygous of genotype A1//A1. Therefore, allele A1 is the healthy allele. Or Allele A1 exists in the genotype of the two daughters II1 and II3 and these two daughters are of different phenotypes This allele cannot therefore be dominant and, consequently, cannot be responsible for the disease The allele of the disease is therefore A2.	1
5	The genotype of his brother II2: A2//A1 or A2//A2 The genotype of his sister II1: A2//A1 The genotype of his sister II3: A1//A1	1

Parts	Exercise 2 Cellular Cooperation	Notes
1	The first role is proteosynthesis, The second the secretion (of the antibodies)	0.5
2	The type of the considered specific immune response is humoral.	0.25
3	Developed RER Increase in the cytoplasmic volume Disappearance of the membranous antibodies Rich in mitochondria	0.25
4	Three lots of mice are previously thymectomized and exposed to irradiation. These mice are then separated in three lots. Lot 1 mice are injected with sheep red blood cell, BL, macrophages and T4L. It shows presence of plasmocytes. Lot 2 mice are injected with sheep red blood cell, BL and macrophages. It shows absence of plasmocytes. Lot 3 mice are injected with BL, macrophages and T4L. It shows absence of plasmocytes.	1.5
5	Presence of plasmocytes are noticed only in lot 1 mice injected with SRBC, BL, macrophages and T4 L. However, no plasmocytes are noticed in lot 2 mice injected with SRBC, BL and macrophages and in lot 3 mice (injected with BL, macrophage and T4L). This indicates that, for B lymphocyte to be differentiated into plasmocyte, it necessitates the presence the antigen along with BL, macrophages, and T4 L .	1.75
6	Out of the two interleukins secreted by active T4 lymphocytes , only IL4 is indispensable for the differentiation of BL into plasmocyte.,	0.75
7	The disappearance of the immune complex in culture 2, can be explained by opsonization: the phagocytosis of the immune complex by the macrophages is facilitated by the antibody that fixes the antigen (SRBC). The disappearance of the immune complex in culture 3, can be explained by the lysis of the cellular antigen (SRBCs) after the activation of the complement proteins by the antibody of the immune complex.	0.5 0.5

Parts	Exercise 3 Ovaries-Hypophysis Interaction	Notes
1	Ovulation is the process by which the oocyte II is released out of the ruptured ovarian mature follicle to the oviduct.	0.5
2	Estrogen is secreted by: the ovarian follicle and the yellow body (corpus luteum) Progesterone is secreted by: the yellow body	0.5
3	The ablation of the hypophysis before experimenting on the mice prevents the production of the hypophysarian endogenous hormones FSH and LH. This will ensure that the results, in terms of the secretion of estrogen and progesterone as well as the occurrence of ovulation. is due only to the injected exogenous hormones FSH and LH. .	0.75
4.1	FSH favors the secretion of the estrogen hormone by the ovaries.	0.75
4.2	The conditions of ovulation are the continuous perfusion of FSH for 10 days and the injection of high dose of LH at day 12	0.75
5	Progesterone is an ovarian hormone secreted by the corpus luteum. This latter is formed out of the transformation of De Graafian ovarian follicle (mature follicle). Since this transformation does not occur except after ovulation, thus as a consequence, progesterone will not be secreted unless ovulation has taken place.	0.75
6	<p>The results of experiment 2 reveals a negative feedback. In fact, the average LH level in the ovariectomied female monkeys decreases from 25.1 ng/ml.to 5.3 ng/ml following the injection of a moderate dose of estrogen.</p> <p>The results of experiment 3 reveals a positive feedback. In fact, the average LH level in the same ovariectomied female monkeys increases from 25.1 ng/ml.to 35.2 ng/ml following the injection of a high dose of estrogen.</p>	1

Parts	Exercise 4 Detection of Genetic Polymorphism	Notes						
1.1	Each of the two partial sequences includes 10 pairs of nucleotides. All these pairs are identical except the 5 th pair of nucleotides: TA in the normal allele is substituted by AT in the mutant allele.	1						
1.2	It is a point mutation by substitution.	0.5						
2	<p>In the nucleotide sequence of the normal allele, there is only one restriction site CCGG. This sequence is cut into two fragments:</p> <table style="margin-left: 80px;"> <tr> <td>GGAACC</td> <td>and</td> <td>GGAT</td> </tr> <tr> <td>CCTTGG</td> <td></td> <td>CCTA</td> </tr> </table> <p>In the mutant gene, the sequence recognized by the enzyme doesn't exist, there is no cutting. We obtain only one fragment of sequence.</p> <p>GGAACACAGGAT CCTT GTCCTA</p>	GGAACC	and	GGAT	CCTTGG		CCTA	1.5
GGAACC	and	GGAT						
CCTTGG		CCTA						
3	<p>The radioactive molecular probe GAAC is complementary to the sequence CTTG. This sequence is present in the DNA fragment that corresponds to the mutant allele and in one of the two fragments of the normal allele.</p> <p>The electro-phoregram (2) corresponds to the mutant allele. In fact, the mutation causes the disappearance of the restriction site of enzyme X. Consequently, the mutant allele is not subjected to any cut. The mutant fragment is hence longer and migrates on a smaller distance.</p> <p>The electro-phoregram (1) corresponds to the normal allele because the only visualised fragment is the one hybridized by the probe which is shorter than the original sequence before the cutting. The normal fragment is hence shorter and migrates on a longer distance.</p>	1						

الاسم: الرقم:	مسابقة في مادة علوم الحياة المدة: ساعتان ونصف
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Exercise 1 (5 points)

Neurofibromatosis Type 1

Neurofibromatosis type 1 is an autosomal hereditary disease which is manifested by the appearance of lightly pigmented spots at the level of the skin and malformations at the level of skeleton.

Neurofibromatosis type 1 is related to a protein called Neurofibromin1, symbolized by NF1. This protein is indispensable for the regulation of cell division.

The synthesis of NF1 protein is controlled by a gene called NF1 which exists in two allelic forms.
A research is performed to determine the genetic origin of this disease.

Document 1 presents a fragment of the transcribed strand of the normal allele of gene NF1 in a healthy individual and that of the abnormal allele in an individual affected by neurofibromatosis type 1.

- 1.1- Compare the two fragments.
- 1.2- Draw out the position and the type of mutation that took place.

Number of triplet :	1	2	3	4	5	6
Normal allele:	AAA	ACG	AAA	CTG	TAG	GAA
Abnormal allele:	AAA	ACG	AAC	TGT	AGG	AA

→
Reading direction

Document 2 presents a part of the genetic code table.

- 2- Write, based on documents 1 and 2, the mRNA and the amino acid sequences corresponding to each of the normal allele and the abnormal one.
- 3- Explain how the modification of the nucleotide sequence of the normal allele leads to the appearance of the symptoms of neurofibromatosis type 1.

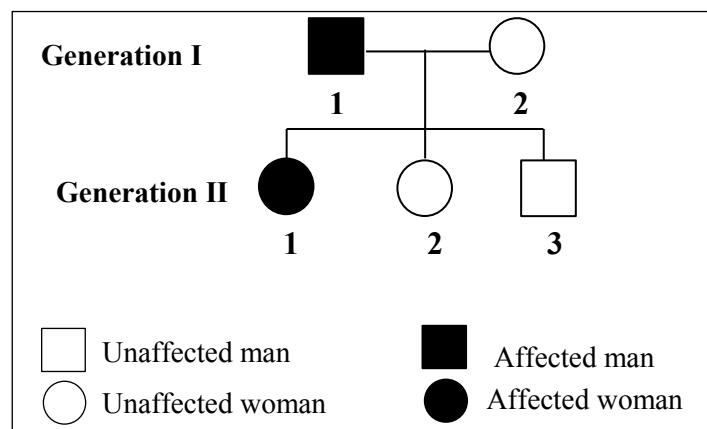
Codons	UAA UAG	UCU UCC	ACA ACG	AUU AUC	GAU GAC	CUU UUG	UGU UGC	UUU UUC
Amino acid	(Stop)	Ser	Thr	Ile	Asp	Leu	Cys	Phe

Document 2

Document 3 presents the pedigree of a family where some members are affected by neurofibromatosis type 1.

It is given that individual I₂ is homozygous.

- 4- Show that the allele responsible for the disease is dominant.
- 5- Determine the probability of couple I₁ - I₂ to have an affected child.



Document 3

Exercise 2 (5 points)

Familial Hypercholesterolemia

Familial hypercholesterolemia or HF is an autosomal dominant hereditary disease characterized by a high level of LDL (carrier of cholesterol) circulating in the blood. This hypercholesterolemia is due to a mutation of a gene responsible for the synthesis of a protein which plays the role of an LDL membrane receptor at the level of liver cells.

Let D represents the symbol of the mutant allele and n the symbol of the normal allele.

Document 1 presents the pedigree of a family where some members are affected by HF.

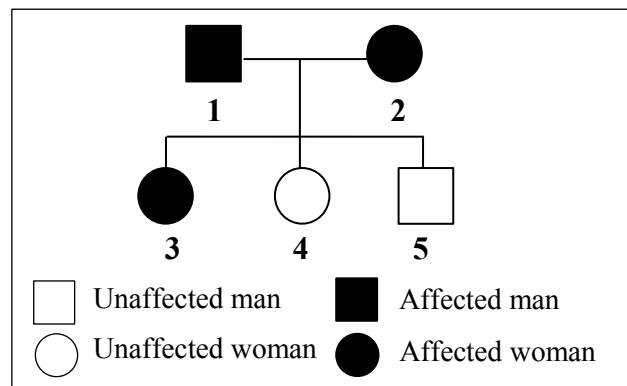
1- Choose the correct answer. Justify the choice.

1.1- the genotype of individual 2 is:

- a- D//n
- b- D//D

1.2- the genotype of individual 4 is:

- a- D//D
- b- n//n



Document 1

Familial hypercholesterolemia or HF has two origins:

Origin 1: A mutation leading to the production of a limited number of LDL receptors at the level of the liver.

Origin 2: A mutation leading to the production of abnormal LDL receptors at the level of the liver.

In order to determine which of these two origins is responsible for HF in a family A, the LDL concentration and the number of LDL receptors in liver cells are measured in normal and affected individuals in family A.

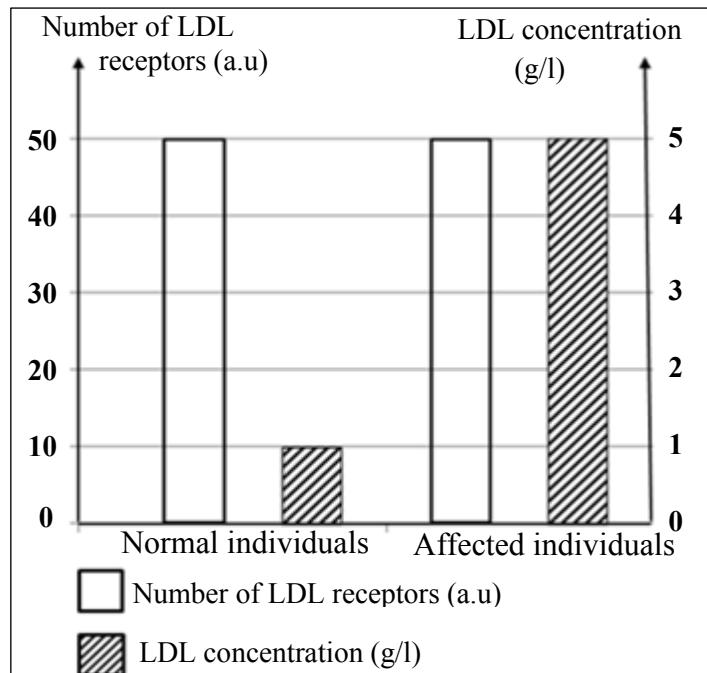
Note that the fixation of LDL on its receptor leads to a decrease in its concentration in the blood.

The obtained results are presented in document 2.

2- Construct a table representing the results of document 2.

3.1- Compare the obtained results.

3.2- Conclude the origin of HF in family A.



Document 2

The results of the same measurements in individuals affected by hypercholesterolemia belonging to another family B show that these individuals have an LDL concentration of 5g/l and a number of LDL receptors in liver cells of 3 a.u.

4- Draw out the origin of HF in family B.

Exercise 3 (5 points)

Immune Response Against Bacteria

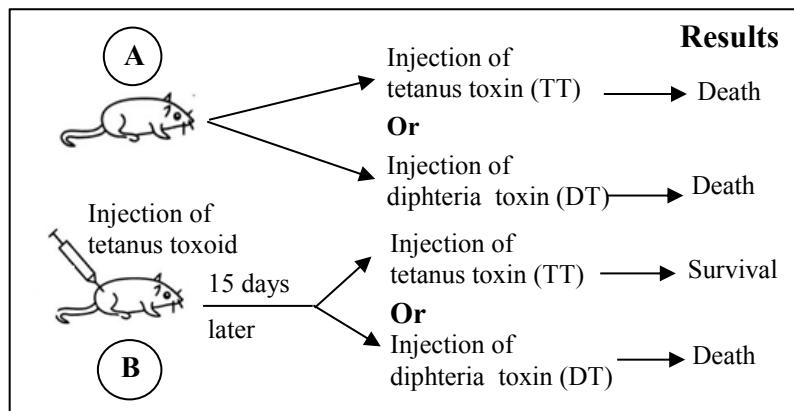
Tetanus and diphtheria are two diseases caused by the invasion of the organism by bacteria. These bacteria release tetanus toxin and diphtheria toxin respectively in the body.

In order to study the immune response against these bacteria, several experiments are carried out.

Experiment 1:

An experiment is carried out on two guinea pigs A and B. The experimental conditions and the results are given in document 1.

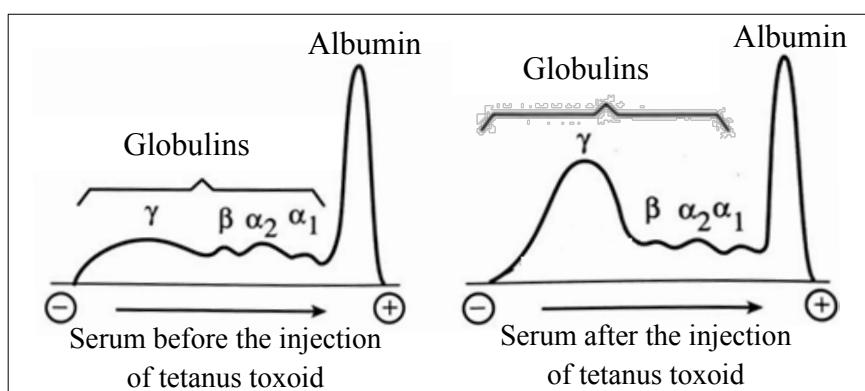
- 1- Interpret the obtained results.
- 2- Draw out a characteristic of the immune response.



Experiment 2:

Serum from guinea pig B is taken before and after the injection of tetanus toxoid. The analysis of the proteins found in this serum is done. The results are shown in document 2.

- 3- Specify the nature of the immune response triggered against tetanus toxin.

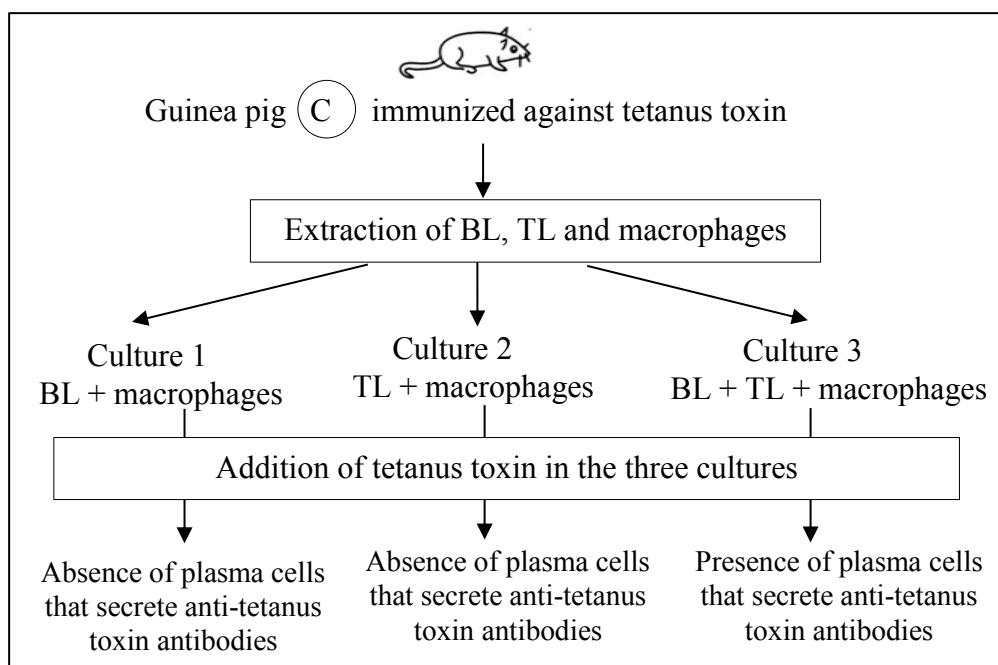


Document 2

Experiment 3:

Scientists seek to identify the cells involved in the immune response against tetanus toxin. Document 3 presents the experimental conditions as well as the results obtained.

- 4- Deduce the necessary condition for the production of anti-tetanus toxin antibodies.
- 5- Name the molecule indispensable for the differentiation of BL into plasma cells.



Document 3

Exercise 4 (5 points)

Immune Response against a Virus

In order to determine the nature of the immune response against an infection by a virus X, three experiments were carried out.

Experiment 1: In mice infected with virus X, we study the evolution of the blood concentration of virus X and that of the number of LT8 specific for virus X as a function of time. Document 1 presents the obtained results.

Time (days)	1	4	6	7
Blood concentration of virus X (a.u.)	10^7	10^6	10^4	0
Number of LT8 specific for virus X ($\times 10^3$)	0	100	300	120

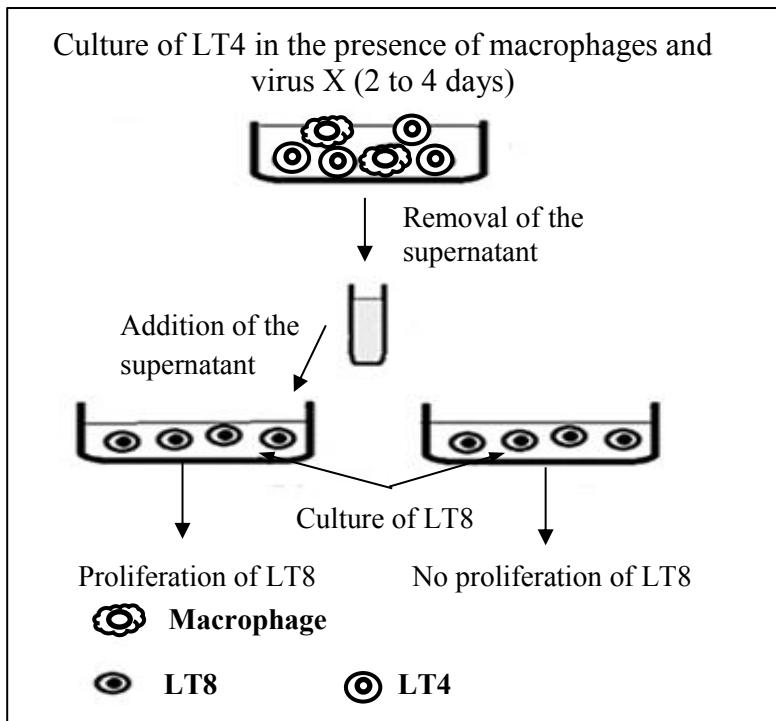
Document 1

- 1- Analyze the obtained results.
- 2- Name the type of the specific immune response triggered against virus X.

Experiment 2: LT4 are cultured in the presence of macrophages and virus X. The experimental conditions as well as the results are represented in document 2.

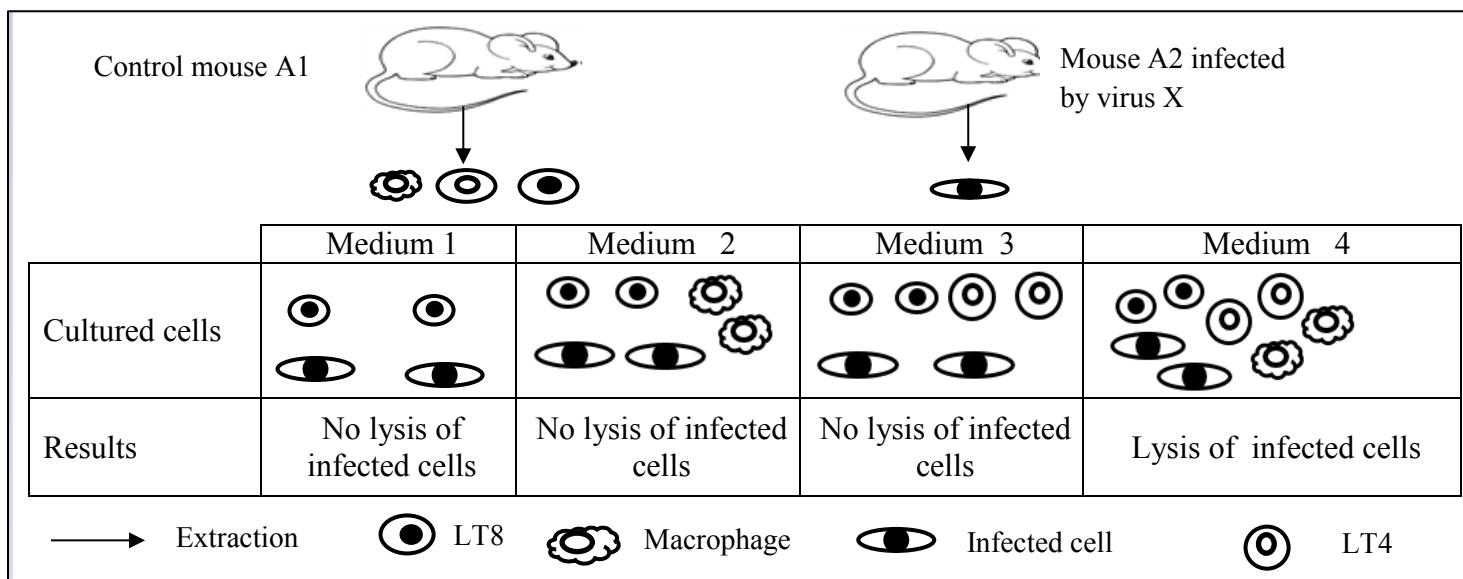
- 3.1- Analyze the results of experiment 2 (document 2).
- 3.2- Draw out the role and the mode of action of the involved LT4.

Experiment 3: An experiment is carried out on two mice of the same strain. Cells infected by virus X are extracted from mouse A2.



Document 2

Then, cells are added to the culture medium containing different immune cells extracted from the control mouse A1. The experimental conditions as well as the results are shown in document 3.



Document 3

- 4- Determine from document 3 the condition necessary for the lysis of the infected cells.
- 5- Explain, referring to acquired knowledge, the mechanism of lysis of infected cells by LT8.

الاسم:	علوم الحياة
الرقم:	أسس التصحيح

1	Exercise 1 (5 points) Neurofibromatosis Type 1	Grade
1.1	The first two triplets are identical in both alleles, but the number of total triplets in the fragment of the abnormal allele is 5 triplets in addition to 2 nucleotides (or 17 nucleotides) which is less than the number of triplets in the fragment of the normal allele which is 6 (or 18 nucleotides).	0.5
1.2	The mutation is at the level of one of the nucleotide (A) in the third triplet It is a point mutation by deletion.	0.5
2	Normal allele: Transcribed strand AAA ACG AAA CTG TAG GAA mRNA UUU UGC UUU GAC AUC CUU Amino Acids Phe - Cys - Phe - Asp - Ile - Leu Abnormal allele: Transcribed strand AAA ACG AAC TGT AGG AA mRNA UUU UGC UUG ACA UCC UU Amino Acids Phe - Cys - Leu - Thr - Ser	1
3	The mutation by deletion of the nucleotide A at the level of the third triplet will cause a modification at level of transcribed mRNA leading to a change at the level of the translated amino acid sequence. This new amino acid sequence affects the three-dimensional form of NF1 which becomes inactive (non-functional). As this protein is indispensable for the regulation of cell division, the symptoms of the disease, lightly pigmented spots at the level of the skin and malformations at the level of the skeleton, will appear.	1
4	Woman I2 is healthy and homozygous which means that she can only transmit the healthy allele to her children but her daughter II 1 is affected and inherits the normal allele which is not expressed and masked by the allele determining the disease. OR If the allele determining a normal phenotype was dominant, then woman I2 who is homozygous would be of genotype NN thus obligatory transmitting allele N to all her children who would be unaffected but this is not the case.	1
5	Mother I2 is unaffected of a recessive phenotype, her genotype is thus. Mother I2 of genotype n/n can only transmit one allele n. Thus, the phenotype of the child depends on the allele inherited from his father. Since the father is heterozygous, the probability to produce a gamete carrying the allele D is $\frac{1}{2}$. Father I1 of genotype Dn can transmit two alleles, allele D and $\frac{1}{2}$ allele n. Thus, the probability of the couple I1 and I2 to have an affected child = $\frac{1}{2}$.	1

Part	Exercise 2 (5 points) Familial Hypercholesterolemia	Grade									
1.1	Genotype of individual 2 is D//n, since the disease is autosomal dominant and woman 2 is affected but has 2 unaffected children 4 and 5 who must have inherited the normal allele which is masked by the mutant one. Thus woman 2 is heterozygous.	0.5									
1.2	Genotype of individual 4 is n//n, since she is an unaffected girl and the normal allele is recessive thus it is only expressed in the homozygote state.	0.5									
2	<table border="1"> <tr> <td>Individual</td> <td>Normal</td> <td>Affected</td> </tr> <tr> <td>Number of LDL receptors (a.u.)</td> <td>50</td> <td>50</td> </tr> <tr> <td>LDL Concentration (g/L)</td> <td>1</td> <td>5</td> </tr> </table> <p style="text-align: center;"><i>Variation in LDL receptor number (a.u.) and LDL concentration (g/L) in normal and affected individuals</i></p>	Individual	Normal	Affected	Number of LDL receptors (a.u.)	50	50	LDL Concentration (g/L)	1	5	2
Individual	Normal	Affected									
Number of LDL receptors (a.u.)	50	50									
LDL Concentration (g/L)	1	5									
3.1	The number of LDL receptors is the same and equal to 50 a.u. in both normal and affected individuals. However, the concentration of LDL in affected individuals is 5 g/l which is greater than that in normal individuals equals to 1 g/l.	1									
3.2	The origin of HF in family A is a mutation leading to the production of abnormal LDL receptors at the level of the liver.	0.5									
4	The origin of HF in family B is a mutation leading to the production of a limited number of LDL receptors at the level of the liver.	0.5									

Part	Exercise 3 (5 pts) Immune Response against Bacteria	Grade
1	<p>The death of guinea pig A is due to its injection by TT or by DT, this indicates that TT and DT are fatal.</p> <p>On the other hand, the injection of tetanus toxoid in the guinea pig B before 15 days of its injection by TT causes its survival or the injection of the guinea pig B by DT causes its death. This indicates that tetanus toxoid protects the guinea pig against TT and not against DT.</p>	1.5
2	The immune response against tetanus toxin is specific.	0.5
3	The immune response is a specific humoral immune response since antibodies are the effector molecules of the humoral response and following the injection of guinea pig B with tetanus toxoid, the concentration of δ globulins which are antibodies increases in the serum.	1
4	Since only in medium 3 containing LB, LT and macrophages, the presence of plasma cells secreting anti-tetanus toxin antibodies is obtained after the addition of tetanus toxin, hence the cooperation between macrophage, LB and LT is necessary for the production of anti-tetanus toxin antibodies.	1.5
5	Interleukin 4 (IL4).	0.5

Part	Exercise 4 (5 pts)	Specific Immune Response	Grade
1	From day 1 to day 7, following the infection of mice by virus X, the number of LT8 specific to virus X increases from 0 to 300×10^3 on d=6, then decreases to 120×10^3 at day 7; however the blood concentration of virus X decreases from 10^7 a.u. to null.	1	
2	The immune response is a specific cell-mediated immune response.	0.5	
3.1	There is proliferation of LT8 only upon the addition of the supernatant liquid taken from a culture of LT4 in the presence of macrophage and virus X. However, there is no proliferation of LT8 in the absence of the supernatant.	1	
3.2	LT4 stimulate the multiplication/proliferation of the LT8 lymphocytes by the secretion of a chemical substance/chemical messengers. (Interleukins)	0.5	
4	No lysis of infected cells is obtained in medium 1,2, and 3 despite the presence of LT8 in medium 1 or LT8 with macrophages in medium 2 or LT8 and LT4 in medium 3. On the other hand, the presence of macrophages, LT4 and LT8 in medium 4 causes the lysis of infected cells. This indicates that the cooperation between macrophages and LT4 is necessary to activate LT8 thus inducing the lysis of infected cells.	1	
5	The TCR of LT8 performs double recognition by binding to the MHC Class I - viral peptide complex on the target cell. It then secretes perforin which form a polyperforin channel on the plasma membrane of the target cell, then it releases granzymes which cross the polyperforin channel and enter the target cell, causing the degradation of its DNA and subsequently leading to its destruction by apoptosis.	1	

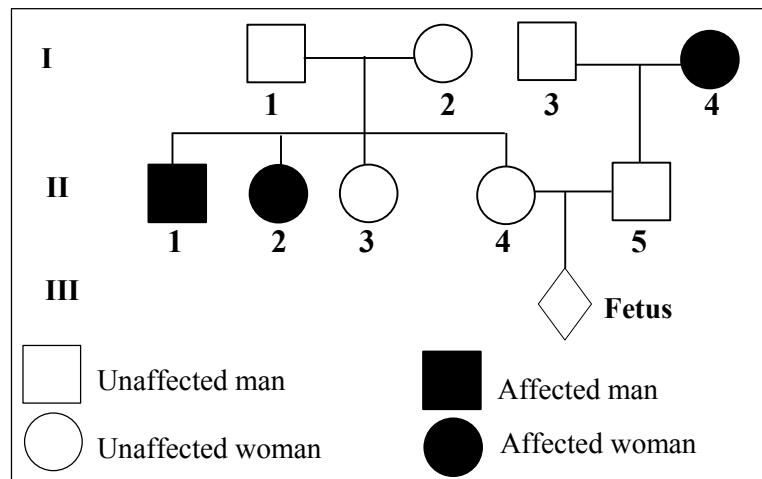
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Exercise 1 (5 points)

Transmission of a Hereditary Disease

Document 1 shows the genealogical tree of a family of which some members shown in black are affected by a hereditary disease.

- 1- Is the allele responsible for the disease dominant or recessive? Justify the answer.
- 2- Determine the chromosomal localization of the gene responsible for the disease.
- 3- Indicate the genotypes of individuals I₁ and II₁. Justify the answer.
- 4- Determine the risk of the fetus to be affected by the disease.



Document 1

Document 2 shows a partial sequence of the two alleles (allele 1 and allele 2) of the studied gene and the restriction fragments obtained by the cleavage of these sequences with the help of a restriction enzyme.

- 5- Specify, referring to document 2, the type of mutation at the origin of the studied disease.

Document 3 shows the results of the electrophoresis realized in certain members of the family of document 1.

Allele 1	
Nucleotide sequence	5' ... GGC ACG TTC ... 3' 3' ... CCG TGC AAG ... 5'
Restriction fragment	347 bp
Allele 2	
Nucleotide sequence	5' ... GGC ATG TTC ... 3' 3' ... CCG TAC AAG ... 5'
Restriction fragments	158 bp 189 bp

Document 2

Size of fragments	Individuals		
	II2	II3	Fetus
347 bp		—	—
189 bp	—	—	
158 bp	—	—	

Document 3

- 6- Identify which of the alleles, 1 or 2, is the one responsible for the disease.
- 7- Draw out the phenotype of the fetus.

Exercise 2 (5 points)

Steinert Myotonia

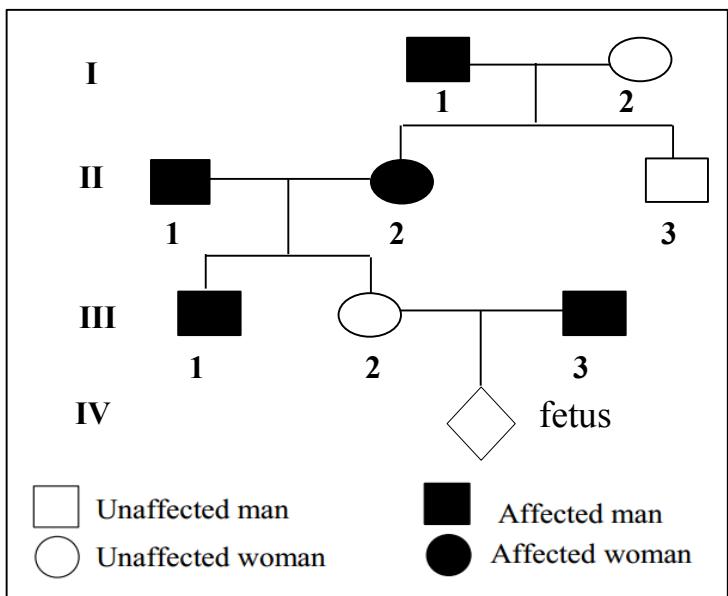
Steinert myotonia is an autosomal dominant genetic disease.

This disease is characterized by damaged muscles leading to a difficulty in relaxation after contraction and progressive muscles weakness with a decrease in muscles volume.

Document 1 shows the genealogical tree of a family belonging to an isolated community in which some members are affected by Steinert myotonia.

A study carried out on 1200 children belonging to this community that permits to identify 30 heterozygous children for Steinert myotonia.

- 1- Calculate the proportion of heterozygous children in this community.



Document 1

Woman III-2 is pregnant and wants to know if her unborn child (fetus) will be affected with Steinert myotonia.

- 2- Determine the probability of the fetus to be normal.

The gene (DMPK) responsible for Steinert myotonia is characterized by CTG repetition in which the number of repetition, in the normal state, varies from 5 to 35 repeats but in the mutated state varies from 50 to more than 3000 repeats.

- 3- Draw out the genetic cause of Steinert myotonia.

Document 2 shows the variation in the age of the appearance of the first symptoms of Steinert myotonia (in years) as a function of the number of triplets (CTG) of the studied gene.

Number of triplets (CTG)	50	500	800	1000
Age of the appearance of the first symptoms (in years)	80	50	30	10

Document 2

- 4- Construct a graph that shows the obtained results.

- 5.1- Analyze the obtained results.

- 5.2- What do you conclude?

Exercise 3 (5 points)**Cancer and Immune Response**

In the framework of studying the immune response against cancer cells, the following two experiments are realized.

Experiment 1:

Two lots of rats A and B are exposed to UV rays for a period of time. Lot A is the control one, whereas lot B is constituted of rats having an immune deficiency (insufficiency).

Document 1 represents the obtained results.

1.1- Analyze the obtained results.

1.2- What do you conclude?

Time after the exposure (in months)	Cancer frequency in lot A (a.u)	Cancer frequency in lot B (a.u)
0	0	0
2	0.5	0.75
4	1	2
6	1	4
8	1	6

Document 1

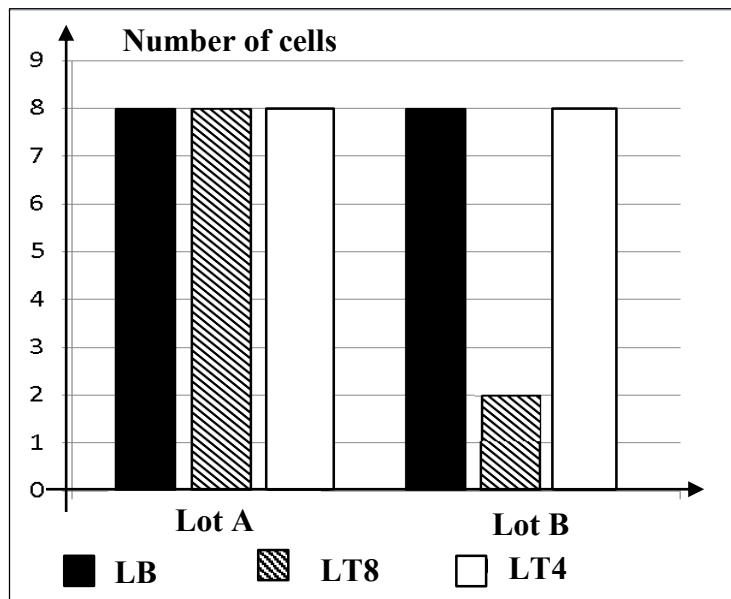
The number of the different types of immune cells LB, LT8 and LT4 is measured in the two lots A and B. The results are shown in document 2.

2- Represent in a table the obtained results.

3- Specify the type of immune response against cancer.

Experiment 2:

Lot B rats are injected with interleukin 2. The conditions and the results of the experiment are shown in document 3.

**Document 2**

Rats	Conditions	Number of cells		
		LT4	LT8	LB
Lot B	UV radiation + Injection of IL2	8	8	8

Document 3

4- Deduce the origin of the immune deficiency of the rats of lot B.

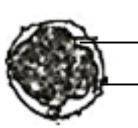
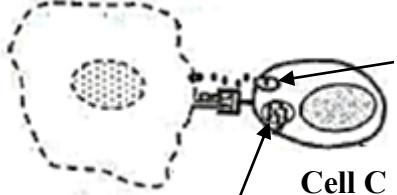
Exercise 4 (5 points)

Immune Responses Against a Virus

In the framework of studying the immune responses against a virus X, two experiments have been realized.

Experiment 1:

A mouse was injected with a virus X. Few days later, we observe the immune cells A, B and C, that are involved in the immune response against virus X (document 1).

Observation	
At the level of the spleen	At the level of the site of infection
 <p>Cell A</p> <p>Nucleus Rough endoplasmic reticulum</p> <p>Cell B (obtained out of the transformation of cell A)</p>	<p>Infected cell by virus X</p>  <p>Cell C</p> <p>Perforin vesicles Granzyme vesicles</p>

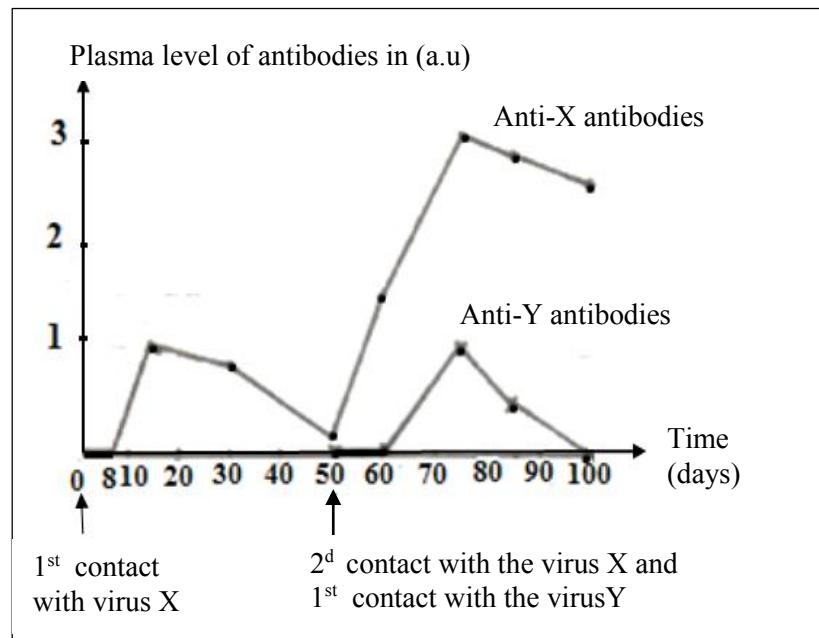
Document 1

- 1- Identify the cells B and C.
- 2- Specify the types of immune response triggered against virus X.
- 3- Indicate the role of each of the substances contained in the vesicles of cell C.

Experiment 2:

Another mouse is injected with virus X at time t_0 . Then, 50 days later, the same mouse is injected with the same virus X and another virus Y. The plasma level of antibodies directed against each virus X and Y is measured. The results are shown in document 2.

- 4- Compare the latency time and the maximum level of anti-X antibodies secreted during the first and the second contact with virus X.
- 5- What information gives the variation of the level of the anti-Y antibodies?



Document 2

Question	Exercise 1 Transmission of a Hereditary Disease	Note
1	<p>The allele of the disease is recessive.</p> <p>Because, parents I₁ and I₂ of healthy phenotype had sick children II₁ and II₂. These children have inherited the disease allele from at least one parent. This parent possesses the mutant allele in a masked state.</p> <p>Let N be the symbol of the normal, dominant allele.</p> <p>Let d be the symbol of the mutated allele responsible for albinism.</p>	0.75
2	<ul style="list-style-type: none"> If the gene is located on the proper part of the Y chromosome: The disease would be present only in men since girls do not have the Y gonosome. But, the pedigree shows girl III₂ sick. Or: Father and son would be of the same phenotype because the boy inherits his Y gonosome from his father. But, the boy II₁ is sick with genotype X//Y^d, his father should be sick with obligatory genotype X//Y^d, which is not the case. The gene is not carried by the proper part of the Y chromosome. If the gene is carried by the proper part of X chromosome: The genotype of the sick girl II₂ would be X^d//X^d with an X^d of paternal origin. The father I₂ would be sick with genotype X^d//Y. However, he is of normal phenotype. This is not the case. If the gene is located on the common part of X and Y. The boy II₁ would be of genotype X^d//Y^d with Y^d of paternal origin, and his sister II₂ of genotype X^d//X^d, one of them inherited from the same father. Their father I₁ would be of X^d//Y^d genotype and of diseased phenotype. But, this is not the case. <p>Hence, the mutant allele is autosomal.</p>	1.25
3	<p>I₁: N//d</p> <p>Justification: he is [N] and has a dominant allele N. He possesses the recessive allele d since he has 2 affected children, II₁ and II₂.</p> <p>II₁: d//d</p> <p>Justification: The allele of the disease is recessive, and only expressed in homozygous state.</p>	1
4	<p>For this fetus to be affected, his healthy parents should be heterozygous N//d</p> <p>The frequency for II₄ to be heterozygous is 2/3: since she has a sick brother (II₁) which indicates that her parents are heterozygous. The probability to be heterozygous.</p> <p>The frequency for the father II₅ to be heterozygous is 1: He is healthy and has inherited the affected allele d from his affected homozygous mother I₄.</p> <p>The probability for this couple to have affected child is $\frac{1}{4}$ (punnett square)</p> <p>Risk = $\frac{2}{3} \times 1 \times \frac{1}{4} = \frac{1}{6}$</p>	0.5

5	Mutation by substitution. The 2 fragments are identical except for the pair of nucleotide n°5 represented in this fragment where CG is replaced by TA	0.75
6	II2 has genotype d/d (pure race). She has 2 thick bands (each two overlapping fragments) at the level of 189 and 158 bp (doc.2) which correspond to allele 2, so allele 2 corresponds to allele of the disease.	0.5
7	Normal	0.25

Question	Exercise 2 Steinert Myotonia	Note										
1	Proportion of heterozygotes = $30/1200 = 1/40$	0.5										
2	III2 is normal with genotype n/n (homozygous) III3 is sick, the proportion of being heterozygous = $1/40$. The probability to produce normal gametes is $\frac{1}{2}$ Thus the probability of the fetus to be normal = $1/40 \times \frac{1}{2} \times 1 = 1/80$	1										
3	The increase in the number of triplet CTG greater than 50.	0.5										
4	<p>The variation of the age of the appearance of the first symptoms as a function of CTG triplets</p> <table border="1"> <caption>Data points from the graph</caption> <thead> <tr> <th>Number of triplets (CTG)</th> <th>Age of the appearance of first symptoms (years)</th> </tr> </thead> <tbody> <tr> <td>50</td> <td>80</td> </tr> <tr> <td>500</td> <td>50</td> </tr> <tr> <td>800</td> <td>30</td> </tr> <tr> <td>1000</td> <td>10</td> </tr> </tbody> </table>	Number of triplets (CTG)	Age of the appearance of first symptoms (years)	50	80	500	50	800	30	1000	10	1.5
Number of triplets (CTG)	Age of the appearance of first symptoms (years)											
50	80											
500	50											
800	30											
1000	10											
5.1	The age of the appearance of first symptoms decreases from 80 to 10 years when the number of CTG triplets increases from 50 to 1000.	1										
5.2	We conclude that high number of repetition of CTG favors earlier appearance of the symptoms of the disease.	0.5										

Question	Exercise 3 Cancer and Immune Response	Note															
1.1	<p>At t= 0, the frequency of cancer in the 2 lots A and B is null.</p> <p>However, after the exposure to UV, this frequency in both lots increases to be 0.75 a.u in lot B (having immune deficiency) which is greater than that of lot A (control) 0.5 a.u.</p> <p>After that, the frequency of cancer increases remarkably in lot B to be 6 a.u at the 8th month, while that in lot A it decrease to 1 a.u and remains constant between 4 to 8 months.</p>	1															
1.2	The immune deficiency favors the development of cancer.	1															
2	<table border="1"> <thead> <tr> <th rowspan="2"></th> <th colspan="3">Number of cells</th> </tr> <tr> <th>LB</th> <th>LT8</th> <th>LT4</th> </tr> </thead> <tbody> <tr> <td>lot A</td> <td>8</td> <td>8</td> <td>8</td> </tr> <tr> <td>lot B</td> <td>8</td> <td>2</td> <td>8</td> </tr> </tbody> </table> <p>The variation of number of cells in the 2 lots A and B</p>		Number of cells			LB	LT8	LT4	lot A	8	8	8	lot B	8	2	8	1
	Number of cells																
	LB	LT8	LT4														
lot A	8	8	8														
lot B	8	2	8														
3	Cell-mediated specific immune response, because it is observed that the LTc deficiency in lot B provokes the development of cancer.	1															
4	Since the injection of IL ₂ into lot B provokes the increase in the number of only LT 8 from 2 to 8. Thus, the origin of the immune deficiency of the rats of lot B is an IL ₂ deficiency.	1															

Question	Exercise 4 Immune Responses Against a Virus	Note
1	<p>Cell B: Plasma cell since it shows numerous and more developed rough endoplasmic reticulum, voluminous, condensed nucleus, developed golgi bodies, numerous mitochondria (anyone is enough)</p> <p>Cell C : LTc due to the presence of perforin vesicles and granzymes vesicles.</p>	1
2	<ul style="list-style-type: none"> • Humoral specific immune response since plasma cells are observed in the spleen following the infection of virus X. (the effectors of the HSIR) • Cell-mediated specific immune response since LTc (effector of this response) destroys the infected cell. 	1
3	<p>Perforin: Perforates the membrane of the infected cell forming a polyperforin channels.</p> <p>Granzymes : destroy the DNA of the infected cell.</p>	1
4	<p>The latency time of the secondary response 8 days which is greater compared to that of the primary immune response 1 day.</p> <p>The maximum level of anti-X antibodies in the secondary response is 3a.u higher than that of the primary response 1a.u.</p>	1.5
5	The immune response against the virus is specific.	0.5



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