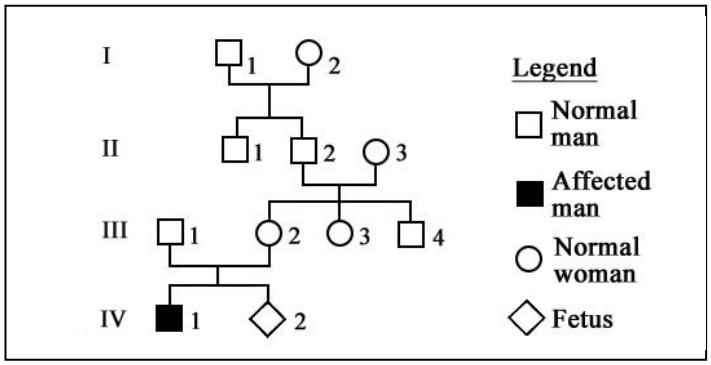
وزارة التربية والتعليم العالي المديرية العامة للتربية دائرة الامتحانات	امتحانات الشهادة الثانوية العامة فرع علوم الحياة	دورة سنة 2013 العادية
الخميس 27 حزيران 2013	مسابقة في مادة علوم الحياة المدة: ثلاث ساعات	الاسم: الرقم:

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 III_1- III_2 (document 1), who already had an affected child, expects another one and would like to know if it will be affected or not.

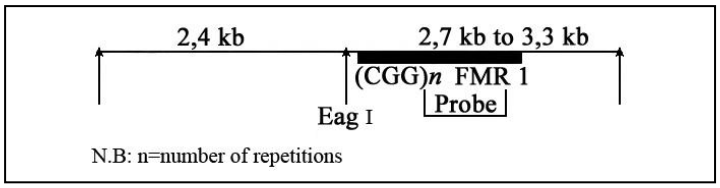


Document 1

1. Justify that the gene is localized on X chromosome.

2. 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.



Document 2

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.

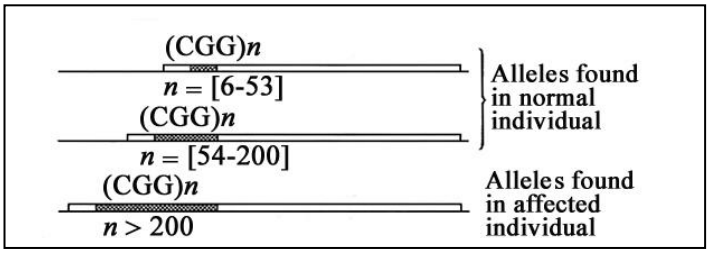
Individuals	II3	III1	III2	IV1	IV2
5,8 kb				—	
3,2 kb			—		
2,8 kb	—	—	—		—

Document 3

3. Identify the band(s) corresponding to the alleles of the disease and those corresponding to the normal alleles.

4. Determine whether the fetus IV2 will be affected or not by the fragile X syndrome.

5. Pose the problem that arises from the study of document 3 concerning the origin of the disease in IV1.



Document 4

Document 4 shows the position and the number of repetitions of CGG triplet for the allele of FMRI 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.

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

Exercise 2 (5 points)

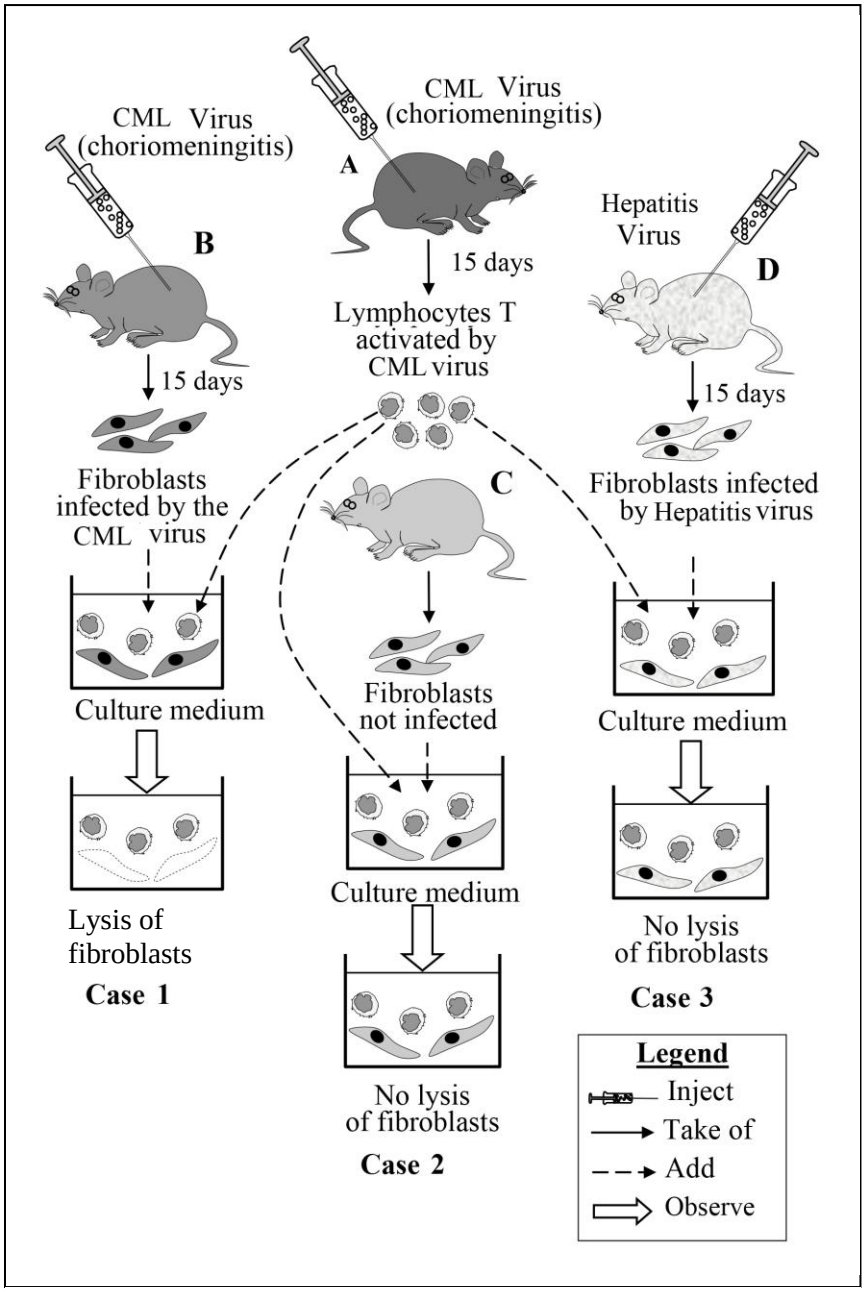
Cell Lysis

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.

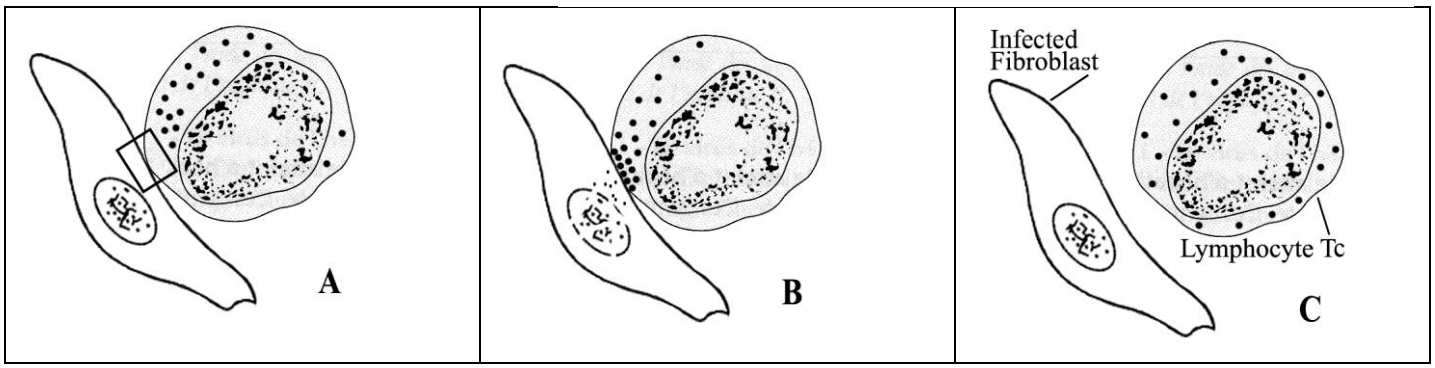
To better understand the immune mechanisms responsible for the lysis of infected cells, a set of experiments are performed on mice of the same line (document1).

1. Pick out from the text the means of contamination of humans by CML virus.
2. Describe the experiments schematized in document 1.
3. 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

4. Arrange, in chronological order, the schematic representations of document 2. Justify the answer.
5. 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:

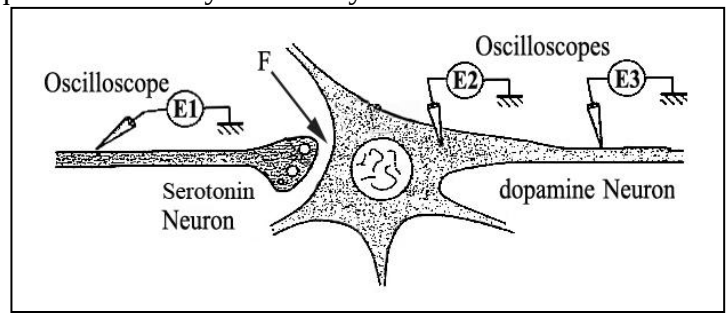
« …if the quantity of the consumed ecstasy is limited, the consumer becomes euphoric, very talkative, and feels extreme happiness. This phase can last 2 to 4 hours depending on the dose and the individual's sensibility. It is followed by a "descent" period marked by exhaustion and even a strong depressive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



Document 1

The obtained results are shown in document 2.

Conditions	Recordings of E1	Recordings of E2	Recordings of E3
2 stimulations seperated by a long time interval	+ 30 AP - 70	Threshold of depolarization - 60 - 70	-70
2 stimulations seperated by a short time interval	+ 30 AP - 70	+ 30 AP - 70	+ 30 AP - 70

Document 2

2. Determine if the synapse F is excitatory or inhibitory.

3. Indicate, at the level of dopamine-releasing neuron, the type of summation revealed by this experiment. Justify the answer.

4. 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				

5- 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:

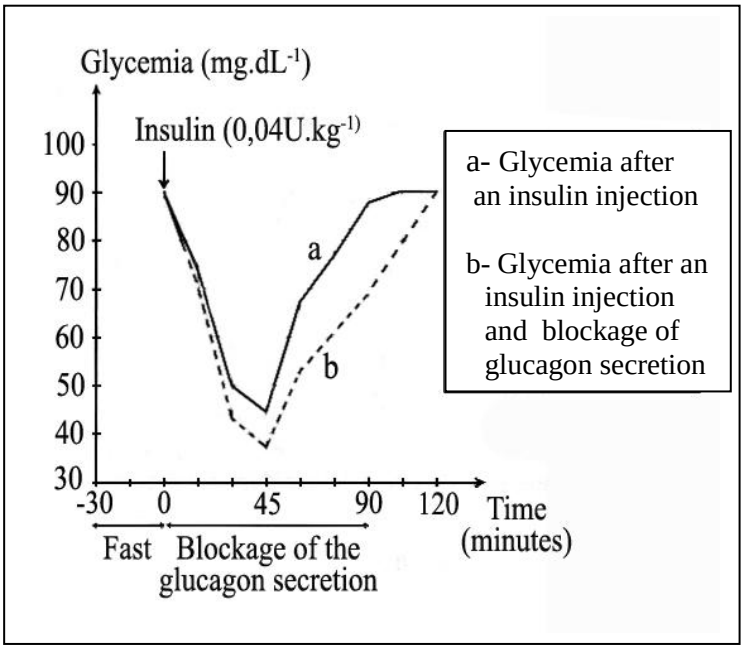
- first an intravenous injection of insulin; the obtained results are represented by curve **a**.
- later they receive an intravenous injection of insulin accompanied by a blockage of glucagon secretion from the moment of insulin injection till 90 minutes; the obtained results are represented by curve **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	Grade
	Exercise 1	
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	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). 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. Or 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. The same reasoning for the normal male III1 indicating that the band which length is 2.8kb corresponds to the normal allele. 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.	1,5
4	Doc 3 shows that the fetus has only one band of 2,8kb length same as his normal father III1 thus he is normal. Or 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.	0,75
5	How come that the disease appeared in child IV1 although both of his parents carry only normal alleles? Or Both parents of IV1 have only the normal alleles, so where does the allele of the disease of child IV1 come from?	0,75
6	The origin of the disease in child IV1 is due to an abnormality that occured 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	Grade
	Exercise 2	
1	The means of human contamination by the CML virus are: food and dust contaminated by infected mice.	**0,5**
2	Mouse A is injected with CML virus (choriomeningitis). 15 days later T lymphocytes activated by CML of this mouse are take of and added to three culture media. 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. 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. 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.	**1,5**
3	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.	**1**
4	1- The order is: C A B (0,25) The first scheme C shows near the infected fibroblast one T lymphocyte with vesicles that are spread in its cytoplasm. In the second scheme A, the T lymphocyte is in contact with the membrane of the infected fibroblast, what corresponds to the double recognition. In the third scheme B, we notice that the granules are in contact with the infected fibroblast and destroy its nucleus. (0,75 pt)	**1**
5	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 perforine to form polyperforine channels through the membrane of the infected cell. After that the TcL releases granzymes that penetrates into the infected cell through the polyperforine channels leading to the degradation of its DNA, thus causing lysis of the infected cell.	**1**

Part	Answer key	Grade
	Exercise 3	
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 persistant 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 serotonine 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 serotonine, 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	The pancreas has a hypoglycemic role since we observe hyperglycemia following pancreas ablation. (½ pt) 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) 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)	**1,5**
2	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.	**1**
3	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 ahypoglycemic hormone. (0.75 pt) 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)	**1,5**
4	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.	**1**