وزارة التربية والتعليم العالي
المديرية العامة للتربية
دائرة الامتحانات

امتحانات الشهادة الثانوية العامة
فرع علوم الحياة

دورة سنة 2011 العادية

مسابقة في مادة علوم الحياة
المدة: ثلاث ساعات

الاسم:
الرقم:

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 ↓ 408 ↓ …CTG GCC ATC GAG CGG TAC…	131 ↓ 136 ↓ …Leu-Ala-Ile-Glu-Arg-Tyr…
Affected with retinitis pigmentosa	391 ↓ 408 ↓ …CTG GCC ATC GAG CTT TAC…	131 ↓ 136 ↓ …Leu-Ala-Ile-Glu-Leu-Tyr…
Leu = leucine, Ala = alanine, Ile = isoleucine, Glu = glutamic acid, Arg = arginine, Tyr = tyrosine.		

Document 1

1- Pick out from the text the cause of retinitis pigmentosa.

2- Compare the two nucleotides sequences and the two amino acids sequences presented in document 1. Draw out the origin of this disease.

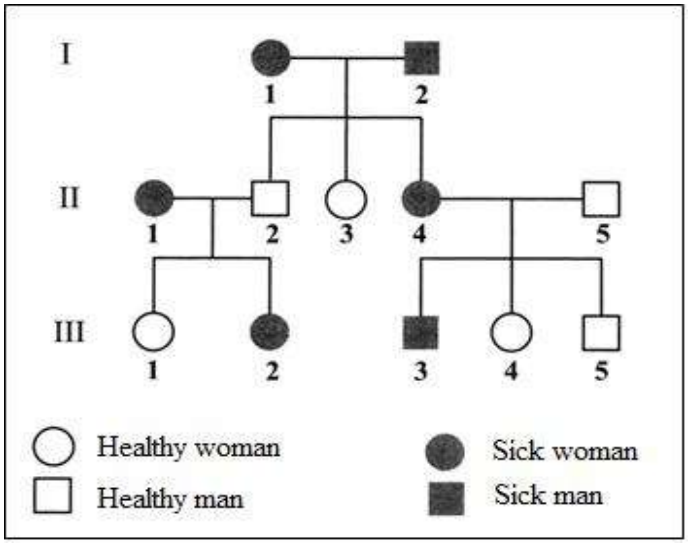
3- Explain how the modifications in the nucleotides sequence of the allele (doc.1) lead to the appearance of the previously mentioned symptoms of retinitis pigmentosa.

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

4- Specify if the allele responsible for the disease is dominant or recessive and indicate its chromosomal location. Justify both answers.

5- Determine the genotypes of individuals II3 and II4.

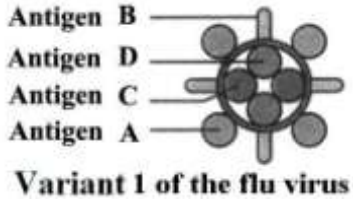
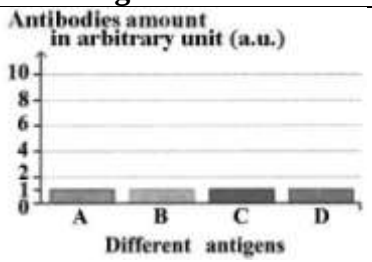
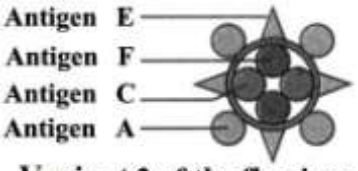
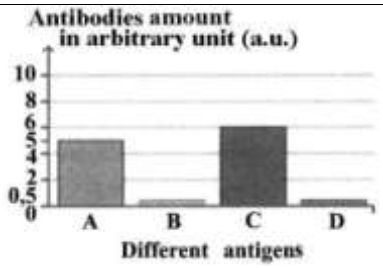
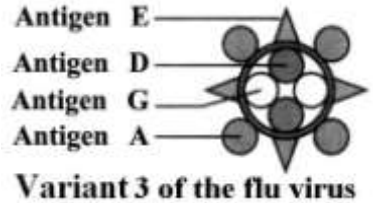
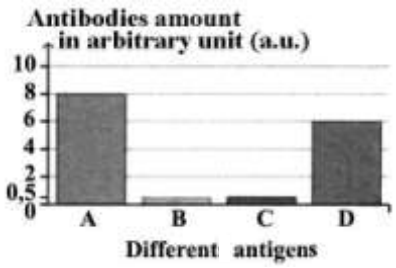
6- Woman III2 married her cousin III3; determine the risk for this couple to have children with retinitis pigmentosa.



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	
The same individual at the age of five years in contact with variant 2 of the flu virus	 Variant 2 of the flu virus	
The same individual at the age of 20 years in contact with variant 3 of the flu virus	 Variant 3 of the flu virus	

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.

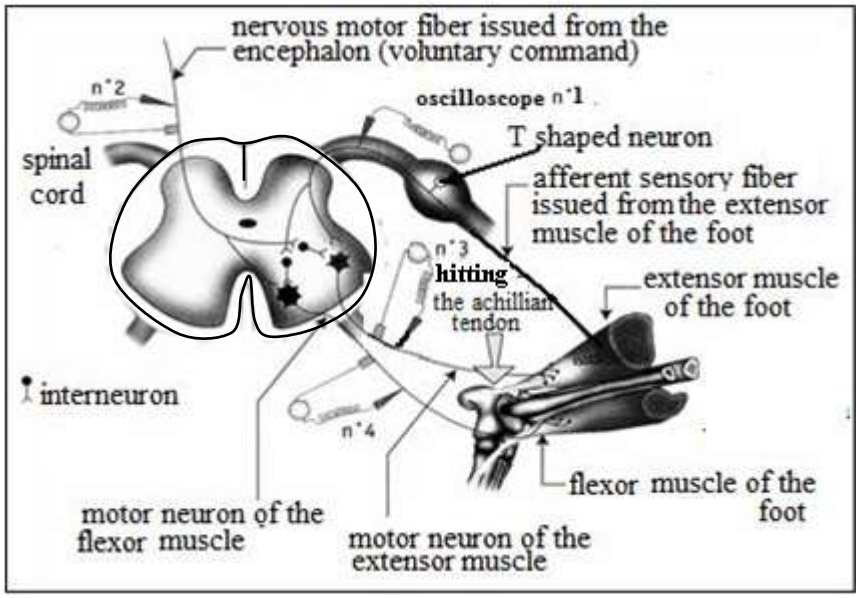
3- Name two cells implicated in the immune response triggered against variant 1 of the flu virus and specify the role of each cell.
4- Explain how the secreted antibodies contribute to the destruction of the flu virus.
5- Specify if the revealed immune response is capable alone to eliminate cells infected 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

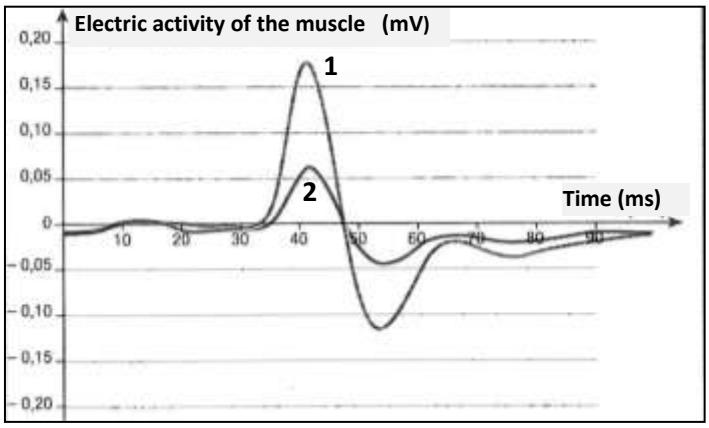
1- Interpret the results of document 2.

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.

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.

3- 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

4- 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

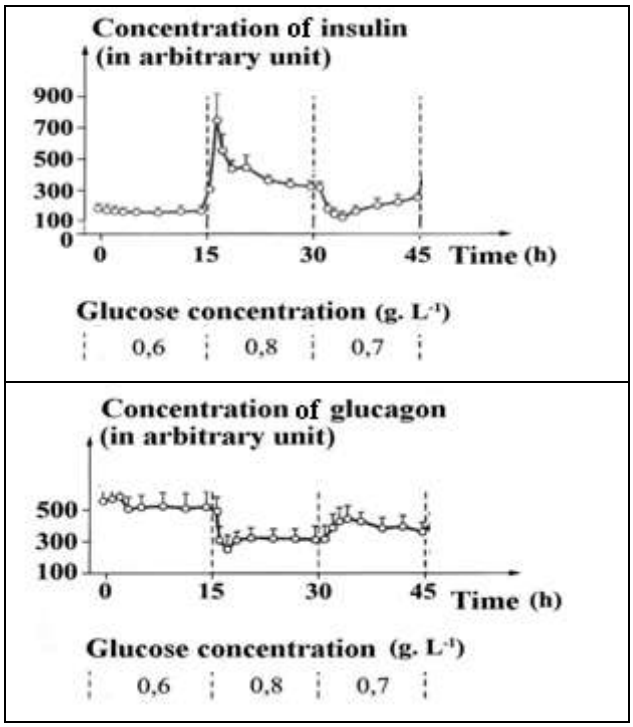
Document 1

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.



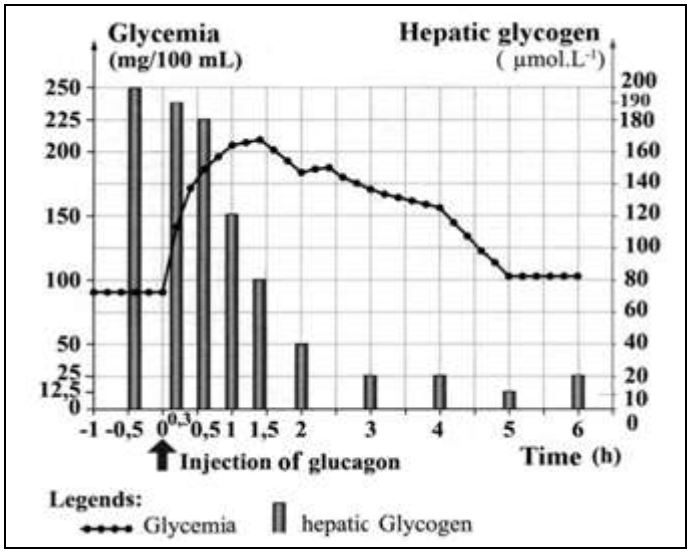
Document 2

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.



Document 3

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

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	It is caused by progressive degeneration of rod cells which are photoreceptor cells of the retina containing the protein rhodopsin.	**0.25**
2	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.	**1**
3	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.	**0.75**
4	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.	**1.25**
5	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.	**1**
6	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.	**0.75**

	A heterozygous couple have ¾ of its children affected. Thus the risk of this couple to have an affected child is 1x1x3/4=3/4 **Or** the child will be sick if he inherit: D from the father and D from the mother (1/2 x1/2 = 1/4) or D from the father and n from the mother (1/2 x1/2 = 1/4) or n from the father and D from the mother (1/2 x1/2 = 1/4) 1/4 +1/4+1/4=3/4 children with retinitis. **Or** the child will be normal if he inherit n from the father and n from the mother (1/2 x1/2 = 1/4) thus the risk to have a sick child is 1-1/4=3/4 **Or** refer to a table of cross	

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	a- During the first contact with the variant 1of 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(> 1a.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. 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 (> 1a.u) and 6 a.u (> 1a.u) thus 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 since the amount of antibodies has increased to 6 a.u(> 1a.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.	**2.25**
3	- 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 phagocyte 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	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. 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. OR 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.	**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	Oscilloscope no. 1:  Because the same stimulation at the level of achillian tendon records the same frequency of AP in the sensory fiber. Oscilloscope no.2:  (Any drawing showing a frequency > 3 AP is accepted) 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. Oscilloscope no. 3:  (Any drawing showing a frequency < 3 AP is accepted) 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. Oscilloscope no. 4:  (Any drawing showing a frequency > 3 AP is accepted) 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.	**2**

<table>
<tr><th>Part of the Ex</th><th>Exercise 4 (5 points)</th><th>Mark</th></tr>
<tr><td>1</td><td>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.</td><td>0.75</td></tr>
<tr><td>2</td><td>⇓ Injection of glucagon
<table><tr><td>Time (h)</td><td>-0,5</td><td>0</td><td>0,3</td><td>0,5</td><td>1</td><td>1,5</td><td>2</td><td>3</td><td>4</td><td>5</td><td>6</td></tr><tr><td>Hepatic glycogen (µmol.L^{-1})</td><td>200</td><td>-</td><td>190</td><td>180</td><td>120</td><td>80</td><td>40</td><td>20</td><td>20</td><td>10</td><td>20</td></tr></table>Variation of the hepatic glycogen in function of time before and after the injection of glucagon</td><td>1.5</td></tr>
<tr><td>3</td><td>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.
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.

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}.
This shows that the action of glucagon is temporary.</td><td>1.5</td></tr>
<tr><td>4</td><td>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).
This indicates that insulin causes an increase in the absorption of blood glucose by the muscle and its storage as glycogen.</td><td>0.75</td></tr>
<tr><td>5</td><td>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</td><td>0.5</td></tr>
</table>