ملاحظات عامّة

١- النّماذج المطروحة تراعي التّقليصات الّتي حصلت في المنهج خلال العام الدراسيّ الحاليّ.

٢- النّماذج المطروحة تراعي قرارات المركز التّربويّ من ناحية مدّة المسابقة وعدد الأسئلة.

٣- للاستفادة يُفَضّل حلّ النموذج أوّلًا ومن ثمَّ الذّهاب للاطّلاع على الباريم والعلامة الّتي ممكن أن تنالها.

٤- للاستفادة يُفَضّل أثناء حلّ النموذج الالتزام بالوقت المحدّد له.

٥- لا داعيَ للتّوتّر في حال عدم تمكّنك من حلّ بعض الأسئلة. يمكن مراجعة الباريم ومعرفة طريقة الإجابة. يمكن مراجعة الدّروس والأفكار على المنصّة عند الحاجة.

٦- هذا النّموذج هو نموذج تدريبيّ لمراجعة الأفكار، وليس المسابقة المقرّرة للامتحان الرّسميّ.

٧- يجري العمل على تنزيل أكثر من نموذج في معظم المواد. لذلك راجع المنصّة للتّأكّد من تنزيل نماذج إضافيّة.

٨- هذه النّماذج خاصّة بالمشتركين بمنصّة شاطر. لذلك يُمنع نشرها وتداولها تحت طائلة المسؤوليّة.

٩- احتمال ورود أخطاء مطبعيّة هو احتمال ضئيل نتيجة مراحل التّدقيق. أمّا في حال حصول ذلك فلا داعي للهلع، فقط يرجى إخبارنا بذلك والقيام بتفقّد النّموذج خلال 24 ساعة للتأكّد من تصحيح أي خطأ مطبعيّ أو توضيح إضافيّ لأيّ إجابة.

بالتّوفيق

نموذج 1 **2023**	**امتحانات الشّهادة الثّانويّة العامّة** الفرع: علوم الحياة	

مسابقة في مادة علوم الحياة
المدة: ساعتان ونصف

Exercise 1 (5 pts) Abnormal Hemoglobin

Hemoglobin is a protein made up of four polypeptide chains (2 alpha chains and 2 beta chains). A mutation in the beta chain of hemoglobin is known to be at the origin of abnormal hemoglobin, which precipitates in red blood cells and leads to sickle cell anemia.

Two mutations in the beta globin gene are being studied to determine the one responsible for sickle cell anemia. The sequence of the non-transcribed strand of each of the two mutant alleles of this gene, as well as that of the normal allele, is given in document 1. Document 2 represents the genetic code table.

Codon	1	2	3	4	5	6
Normal allele:	GTG-	CAC-	CTC-	ACT-	CCT-	GAG
Mutant allele 1:	GTG-	CAT-	CTC-	ACT-	CCT-	GAG
Mutant allele 2:	GTG-	CAC-	CTC-	ACT-	CCT-	GTG

Document 1

1. Specify the type of mutation at the origin of each of the two mutant alleles (1 and 2) in the beta globin gene.

2.1. Write the sequence of the mRNA corresponding to the normal allele of the beta globin gene.

2.2. Write the amino acid sequence corresponding to the given portion of the normal allele of the beta globin gene.

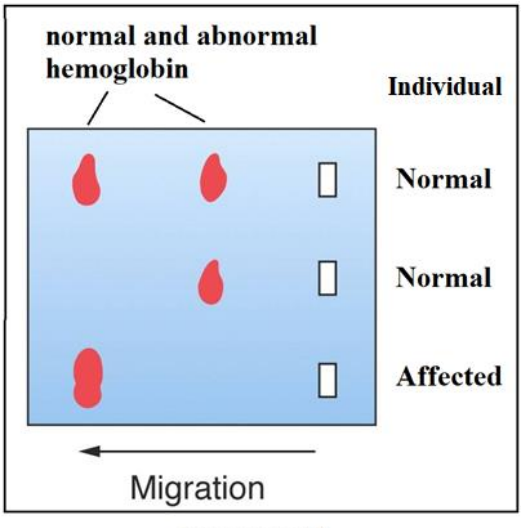
3. Determine which mutant alleles (1 or 2) is at the origin of sickle cell anemia.

First base	Second base: U	Second base: C	Second base: A	Second base: G	Third base
U	UUU, UUC: Phe UUA, UUG: Leu	UCU, UCC, UCA, UCG: Ser	UAU, UAC: Tyr UAA Stop, UAG Stop	UGU, UGC: Cys UGA Stop, UGG Trp	U C A G
C	CUU, CUC, CUA, CUG: Leu	CCU, CCC, CCA, CCG: Pro	CAU, CAC: His CAA, CAG: Gln	CGU, CGC, CGA, CGG: Arg	U C A G
A	AUU, AUC, AUA: Ile AUG Met start	ACU, ACC, ACA, ACG: Thr	AAU, AAC: Asn AAA, AAG: Lys	AGU, AGC: Ser AGA, AGG: Arg	U C A G
G	GUU, GUC, GUA, GUG: Val	GCU, GCC, GCA, GCG: Ala	GAU, GAC: Asp GAA, GAG: Glu	GGU, GGC, GGA, GGG: Gly	U C A G

Document 2

In order to determine the number of mutant alleles required for a person to be affected by sickle cell anemia, we decided to determine the dominant allele among the alleles of the beta globin gene.

Three persons are subjected to hemoglobin electrophoresis, and the results are shown in document 3. Moreover, the beta globin gene is located on chromosome 11.



Document 3

4. Draw out the posed problem.
5. Show that the normal allele of the beta globin gene is dominant.
6. Specify the number of mutant alleles required for a male to be affected by sickle cell anemia.

Exercise 2 (5 pts) Cooperation During Immune Response

One of the most important characteristics of the immune system is the cooperation that can occur between its components. This cooperation is performed through various modes of action and is done between the components of the specific or the non-specific immunity.
In this exercise, we aim to study the cooperation performed during the immune responses.

Experiment 1:
Many cell cultures are made using macrophages and T4 lymphocytes in the presence or absence of antigens. The results are shown in document 1.

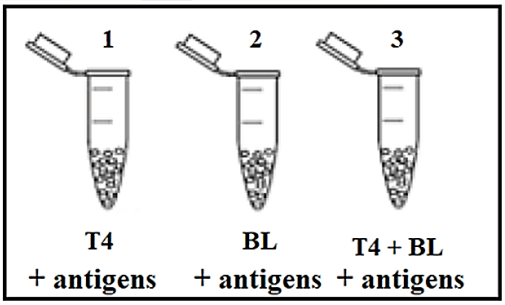
1. Name the process at the origin of the disappearance of antigens in culture 1.
2. Based on document 1, show that:
   2.1. Macrophages activate T4 lymphocytes.
   2.2. The activation of T4 lymphocytes by macrophages requires the presence of antigens.
3. Explain the mechanism leading to the proliferation of T4 lymphocytes in culture 3.

Culture	Conditions	Result
1	Macrophages + antigens	Disappearance of antigens
2	T4 lymphocytes + antigens	Persistence of antigens and no proliferation of T4 lymphocytes
3	Macrophages and T4 lymphocytes + antigens	Disappearance of antigens and proliferation of T4 lymphocytes
4	Macrophages and T4 lymphocytes	No proliferation of T4 lymphocytes

Document 1

Experiment 2:
We use the T4 lymphocytes that proliferated in experiment 1 to perform the experimental setup shown in document 2. A few days later, the content of each culture is mixed with antigens. Immune complexes are obtained only in culture 3.

4. Name the molecule responsible for the formation of immune complexes in culture 3.
5. Interpret the obtained results.



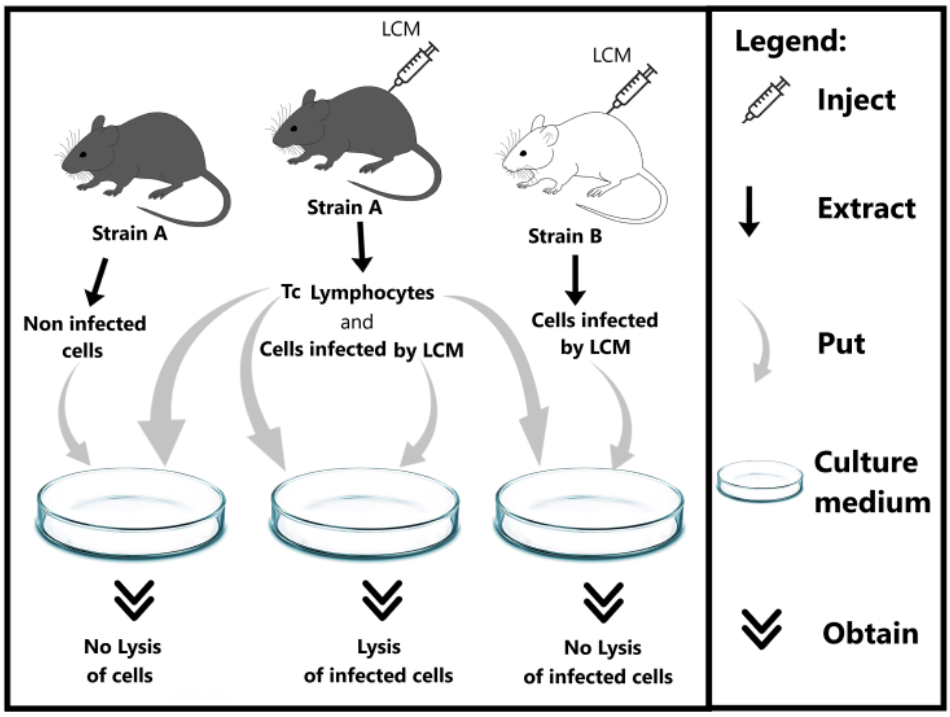
Document 2

Experiment 3:
Immune complexes are incubated alone or in the presence of macrophages. Disappearance of immune complexes is observed only in the presence of macrophages and this disappearance is faster than the disappearance of antigens observed in culture 1 in document 1.

6. Explain the mechanism by which immune complexes disappear in the presence of macrophages.
7. Based on all that precedes, show that there is cooperation between specific and non-specific immune responses.

Exercise 3 (5 pts) Immune Response Against a Virus

In order to study the characteristic of the immune response triggered against the infection by a virus, we inject the LCM (lymphatic choriomeningitis) virus into a mouse. After a few days, we find in the blood cytotoxic T lymphocytes that have destroyed the infected cells.
We use two strains of mice (A and B) and perform the experiment represented in document 1. We observe in the culture mediums, the action of cytotoxic T lymphocytes on the cells of mice A or B, whether infected or not by LCM.



Document 1

1. Pick out from the text one evidence that confirms that LCM is an intracellular intruder.
2. Describe the experiment shown in document 1.
3. Indicate the type of immune response revealed by the results in document 1.
4. Interpret the results of the experiment shown in document 1.

In order to study the mechanism by which Tc lymphocytes are able to destroy infected cells, we perform the experiment summarized in the following table (Document 2).

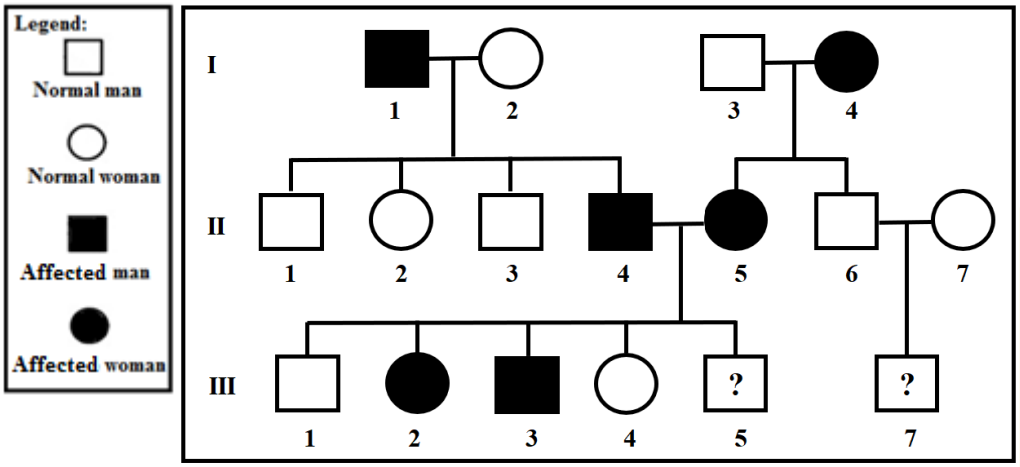
Culture	Tc lymphocytes of a mouse infected by LCM	Cells from the same mouse infected by LCM	Results		
			Binding of Tc to infected cells	Formation of pores in the membranes of infected cells	Degradation of DNA in infected cells
1	Normal Tc containing perforin and granzymes	LCM	Yes	Yes	Yes
2	Tc deprived of perforin	LCM	Yes	No	No
3	Tc deprived of granzymes	LCM	Yes	Yes	No
4	Normal Tc	Hepatitis	No	No	No

Document 2

5. Determine, based on document 2, the role of perforin and granzymes.
6. Explain the results obtained in culture 4.
7. Explain the mechanism by which Tc lymphocytes destroy infected cells.

Exercise 4 (5 pts) Charcot-Marie-Tooth Disease

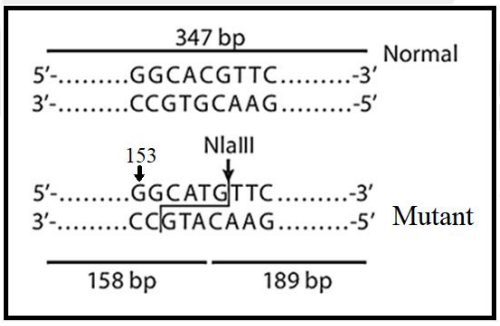
Charcot–Marie–Tooth disease (**CMT**) is one of the hereditary motor and sensory rare neuropathies, a group of varied inherited disorders of the peripheral nervous system characterized by progressive loss of muscle tissue and touch sensation across various parts of the body.
A Costa Rican family affected with Charcot-Marie-Tooth disease is represented in document 1. In this family, some individuals are asking medical help to know if they are at risk of being affected by this disease since they know that each affected individual surely has an affected parent.



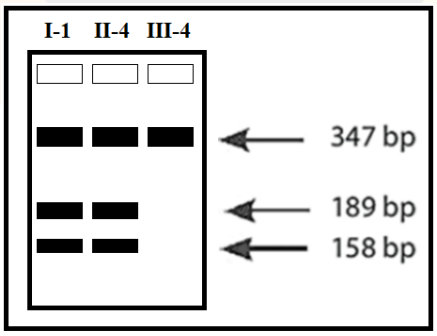
Document 1

1. Pick out from the text the information that indicates that this disease is dominant.
2. Show that the gene for this disease is autosomal.
3. Write the genotypes of the individuals: III-1, II-4, III-2.
4. Determine the risk for each of the individuals III-5 and III-7 to be affected by this disease.

Document 2 shows the part of the gene affected by a mutation leading to this disease. Document 3 shows the results of the electrophoresis performed on some individuals from the family mentioned in document 1 concerning the disease.



Document 2

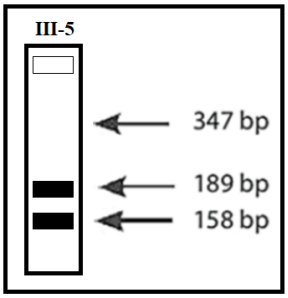


Document 3

5. Specify the type of mutation that leads to the mutant allele of this gene.
6. Explain, based on documents 1 and 2, the result of the electrophoresis of individual I-1 shown in document 3.

Individual III-5 has the electrophoresis result shown in document 4.

7. Specify the genotype and the phenotype of III-5.



Document 4

Answer key

Part	**Exercise 1 (5 pts) Abnormal Hemoglobin**	Mark
1.	The mutation leading to the mutant allele 1 is a point substitution mutation since all the nucleotides are the same as the normal allele, except for the third nucleotide of codon 2, which is C in the normal allele but is replaced with T in the mutant allele 1. The mutation leading to the mutant allele 2 is a point substitution mutation since all the nucleotides are the same as the normal allele, except for the second nucleotide of codon 6, which is A in the normal allele but is replaced with T in the mutant allele 2.	1
2.1.	mRNA: GUG-CAC-CUC-ACU-CCU-GAG	0.5
2.2.	Amino acid sequence : Val-His-Leu-Thr-Pro-Glu	0.5
3.	The mutation in codon 2 of the mutant allele 1 does not change the amino acid His (silent mutation), so hemoglobin remains normal, while the mutation in codon 6 of the mutant allele 2 changes the amino acid Glu to Val, leading to abnormal hemoglobin. So the mutant allele 2 is at the origin of sickle cell anemia.	1
4.	How many mutant alleles are required for a person to be affected by sickle cell anemia?	0.5
5.	Document 3 shows that one of the normal persons has two different types of hemoglobin, one of which is the abnormal type. This means that the normal phenotype is expressed in the presence of the mutant allele of beta globin. So the normal allele is dominant.	1
6.	The studied gene is autosomal and is carried by chromosome 11. Since the mutant allele is recessive, it is expressed only in homozygous individuals. So in order to be affected by sickle cell anemia, a person requires two mutant alleles of beta globin gene.	0.5

Part	**Exercise 2 (5 pts) Cooperation During Immune Response**	Mark
1.	Phagocytosis.	0.25
2.1	When T4 lymphocytes are put with antigens in culture 2, no proliferation is obtained; while when they are put with macrophages and antigens in culture 3, we obtain proliferation of lymphocytes. This means that macrophages activate T4 lymphocytes proliferation.	0.5
2.2.	In culture 3, the presence of T4 lymphocytes and macrophages in presence of antigens resulted in the proliferation of T4 lymphocytes; while in culture 4 the same cells in the absence of antigens resulted in the absence of lymphocytes proliferation. This means that the activation of T4 lymphocytes by macrophages requires the presence of antigens.	0.5
3.	Macrophage makes phagocytosis of antigens, digest them and associate their peptides to the HLA-II. Specific T4 cells make double recognition of the HLA-II non-self-peptide complex by their TCR and get activated, then proliferate. This is the induction of the specific immune response.	1
4.	Antibodies.	0.25
5.	Proliferated T4 lymphocytes cultured alone with the antigen do not show formation of immune complexes same as the BL cultured in presence of the antigen, while T4 and BL cultured together in the presence of the antigen show formation of immune complexes. This means that there is a cooperation between T4 and BL in order to obtain the formation of immune complexes.	1
6.	When the antibodies bind to the antigen by their variable regions, they become able to bind on the membrane receptors of macrophages by their constant regions. This binding facilitates the adhesion of the immune complexes to macrophage, this is called opsonization. Then macrophages phagocyte the antibody bound antigens and digest them, this leads to the elimination of the immune complexes.	1
7.	Macrophage is a component of the non-specific immune response, it cooperates with T4 lymphocytes (components of the specific immune response) in order to induce their proliferation, then they cooperate with antibodies (components of the specific humoral immune response) in order to eliminate antigens.	0.5

Part	**Exercise 3 (5 pts) Cooperation During Immune Response**	Mark
1.	LCM is a virus. T lymphocytes have destroyed the infected cells.	0.25
2.	Non-infected cells are extracted from a mouse of strain A. Cells infected by LCM and Tc lymphocytes are extracted from a mouse of strain A injected by LCM and cells infected by LCM are extracted from a mouse of strain B injected by LCM. We put each of the non-infected cells, the cells infected by LCM from the mouse of strain A and the cells infected by the LCM from the mouse of strain B, in a culture medium and we put with all of them the Tc lymphocytes extracted from the mouse of strain A injected by LCM. We obtain non-lysis of non-infected cells from the mouse of strain A, lysis of the infected cells from the mouse of strain A, and non-lysis of cells from the mouse of strain B infected by LCM.	1.5
3.	Specific cell mediated immune response.	0.25
4.	Tc lymphocytes of a mouse of strain A injected by LCM and cultured with non-infected cells of the same strain show no lysis of these cells; while when they are cultured with infected cells of the same strain they resulted in lysis of the infected cells. This means that Tc lymphocytes make lysis only of infected cells. Tc lymphocytes of a mouse of strain A injected by LCM and cultured with cells of the same strain infected by the same virus show lysis of these cells; while when they are cultured with the infected cells of the another strain B they resulted in no lysis of the infected cells. This means that Tc lymphocytes of a certain strain make lysis of only the infected cells of the same strain.	1
5.	In presence of normal Tc containing perforin and granzymes, we observe the formation of pores in the membranes of infected cells and degradation of their DNA; while when Tc are deprived of perforin, there is no formation of pores in the membranes of infected cells neither degradation of their DNA. But when the Tc are deprived of granzymes, there is formation of pores in the membranes of infected cells, but no degradation of DNA. This indicates that perforin is responsible for the formation of pores in the membranes of the infected cells, and the granzymes are responsible for the degradation of DNA and the presence of perforin is necessary for the action of granzymes.	1
6.	The cells infected by hepatitis have on their cell membranes HLA-I associated with non-self-peptides of the hepatitis virus. Tc lymphocytes have TCR specific for HLA-I and the non-self-peptides of LCM. So they are not able to recognize the cells infected by the hepatitis virus and thus they are not able to release perforin in order to make pores in their membranes neither to release granzymes in order to degrade their DNA.	0.5
7.	Tc recognizes the target cell by double recognition and binds by its TCR on the HLA-I, non-self-peptide complex on its surface, then it releases perforin that makes polyperforin channels through the cell membrane of the infected cell. Then it releases granzymes, which enter by the polyperforin channels and trigger inside the infected cell an enzymatic chain reaction that leads to the degradation of the DNA of the infected cell and leads to its death.	0.5

Part	**Exercise 4 (5 pts) Charcot-Marie-Tooth Disease**	Mark
1.	Each affected individual surely has an affected parent. (Affected: D, normal: n).	0.5
2.	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 father I-1 has healthy sons II-1 and II-3. Thus, the gene is not carried by the non-homologous segment of the chromosome Y. If the gene is carried on the non-homologous segment of the chromosome X, the healthy girl III-4 must be homozygous for the genotype Xn//Xn; she should have inherited the normal allele from her father II-4, who should be healthy with the genotype Xn//Y. But her father is affected, so the gene is not carried on the non-homologous segment of X. If the gene is carried on the homologous segments of X and Y, the healthy girl III-4 with the genotype Xn//Xn should have inherited Xn from her father II-4, and the healthy boy III-1 with the genotype Xn//Yn should have inherited Yn from his father II-4. Father II-4 should be healthy with the genotype Xn//Yn which is not the case (II-4 is affected). Thus, the gene is not carried on the homologous segments of X and Y. Therefore, the gene is carried by an autosome.	1
3.	III-1: n//n II-4: D//n III-2: D//D or D//n	0.75
4.	The parents of III-5 are heterozygous D//n. In order to be normal, the child III-5 must take n from each parent. The probability for each parent to give n is ½. The probability of the child being of genotype n//n and normal is ½ × ½ = ¼. So the risk for child III-5 to be affected is 1 – ¼ = ¾. The two parents of the child III-7 are normal with genotype n//n. Each parent will surely give n to the child, resulting in the child having a genotype n//n and he will be normal. So the risk for child III-7 to be affected is null.	1
5.	The pair of nucleotides C-G number 157 of the sequence shown in the normal allele in document 1 is replaced by the pair T-A in the mutant allele. So the mutation is a point mutation by substitution.	0.5
6.	The normal allele is not cut, it gives one single fragment of 347 bp. The mutant allele is cut by the enzyme NIaIII into two fragments of sizes 158 bp and 189 bp. Individual I-1 is with the genotype D//n, has the normal allele that corresponds to the fragment 347 bp and the mutant allele that corresponds for the fragments 189 bp and 158 bp that are shown by the electrophoresis.	0.75
7.	III-5 has the fragments 189 bp and 158 bp, corresponding only to two mutant alleles, he has the genotype D//D, so he is affected.	0.5

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