وزارة التربية والتعليم العالي
المديرية العامة للتربية
دائرة الامتحانات

امتحانات الشهادة الثانوية العامة
فرع علوم الحياة

دورة سنة 2011 الاستثنائية

مسابقة في مادة "علوم الحياة"
المدة ثلاث ساعات

الاسم :
الرقم :

Answer the following exercises:

Exercise 1 (5 points) **Immune responses against a virus**

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.

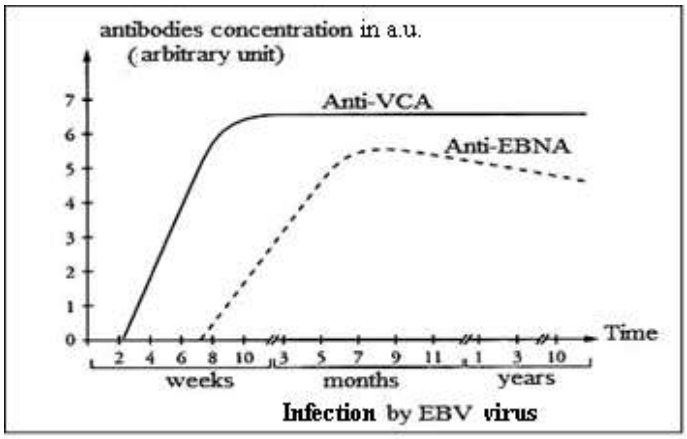
1- Determine by referring to document 1, how the EBV virus persists and is produced in the body.

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

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.

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



Document 2

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.

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

Experiment	Experimental conditions	Results
1	T L of an individual infected by EBV + B L 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)

DNA alteration

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).

NUCLEOTIDE POSITION 1	NUCLEOTIDE POSITION 2 U	NUCLEOTIDE POSITION 2 C	NUCLEOTIDE POSITION 2 A	NUCLEOTIDE POSITION 2 G	NUCLEOTIDE POSITION 3
U	UUU, UUC: phenylalanine; UUA, UUG: leucine	UCU, UCC, UCA, UCG: serine	UAU, UAC: tyrosine; UAA, UAG: stop	UGU, UGC: cysteine; UGA: stop; UGG: tryptophane	U C A G
C	CUU, CUC, CUA, CUG: leucine	CCU, CCC, CCA, CCG: proline	CAU, CAC: histidine; CAA, CAG: glutamine	CGU, CGC, CGA, CGG: arginine	U C A G
A	AUU, AUC, AUA: isoleucine; AUG: methionine	ACU, ACC, ACA, ACG: threonine	AAU, AAC: asparagine; AAA, AAG: lysine	AGU, AGC: serine; AGA, AGG: arginine	U C A G
G	GUU, GUC, GUA, GUG: valine	GCU, GCC, GCA, GCG: alanine	GAU, GAC: asparatic acid; GAA, GAG: glutamic acid	GGU, GGC, GGA, GGG: glycine	U C A G

A: Adenine U: Uracile G: Guanine C: Cytosine

Document 1

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

Document 2

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

Document 3

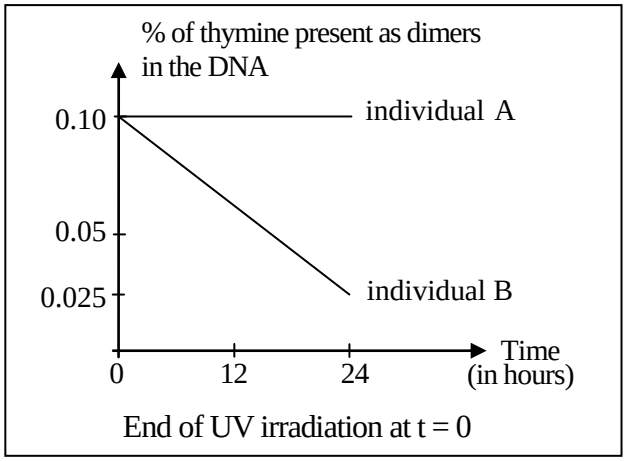
1- Determine using the genetic code table (doc.1) the amino acid sequence of the portion of each of the enzymes ERCC3 coded by the allele G1 and by the allele G2.

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.



Document 4

4- Analyze the obtained results in document 4.

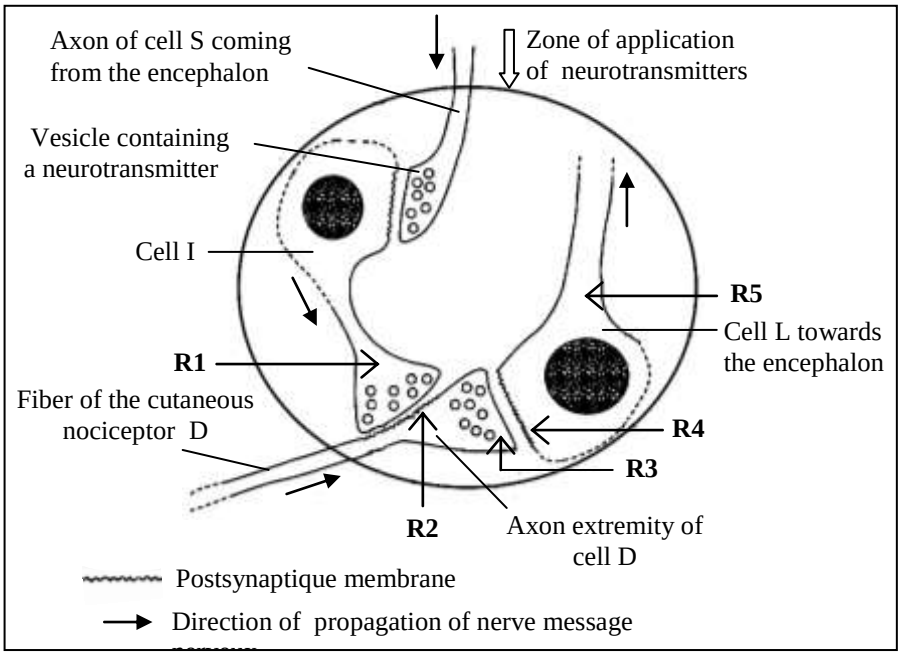
5- Based only on the previous given:

5-1- Explain the results of document 4.

5-2- Specify the factors that determine the development of the studied disease. Justify the answer.

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 frame work 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 document1.
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

50 mV / 2 ms	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.

50 mV / 2 ms	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 consequencesof these injections on three types of testicular cells are presented in document 1.

Pituitary hormones / Testicular cells	Injection of LH	Injection of FSH
Spermatogonia	inactive	activated
Sertoli cells	not developped	developped
Leydig cells	activated	inactive

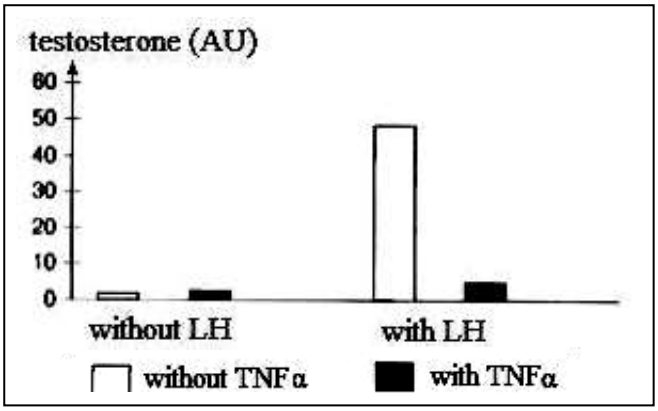
Document 1

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 TNFα, to the culture medium and we measure, at the same time, the production of testosterone. TNFα 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.



Document 2

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 / Activity of pituitary cells	Medium 1 Pituitary cells only	Medium 2 Pituitary cells + kidney cells or spleen cells	Medium 3 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.

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اسس التصحيح
مادة "علوم الحياة"

الاسم :
الرقم :

Part of the Ex	Answer key	Mark
	Exercise1 (5 points)	
1	The virus persists in the body because it remains in the dormant state in the memory BL**(0.25pt)** The virus is produced by naive B lymphocytes that are once infected and by the memory BL once reactivated.**(0.25pt)**	**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	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 than7 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)** 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)**	**1.5**
4	Experiment 1: Lymphocytes TL of an individual infected with the virus EBV are added into the medium containing BL infected with EBV, we obtain100% of lysis LB. 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. 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. 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 . 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 .	**1.25 (5 x0.25)**
5	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). In experiment 2, non-infected BL do not present non-self peptides this is why we do not observe any lysis. 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. In experience 4, BL infected by another virus present another non-self peptides. They are not identified by Tc and they are not lysed. 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.	**1.25 (5 x0.25)**

Part of the EX	Answer key	Mark
	Exercise 2 (5 points)	
1	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 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. 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	**0.75**
2	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)** 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)** 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**)	**1.5**
3	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)**.	**0.75**
4	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.	**0.5**
5-1	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)**	**0.5**
5-2	Two factors determine the development of Xeroderma pigmentosum: - 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	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)** 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)**	**2**
2	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)**	**0.75**
3	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)**	**1.5**
4	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.	**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 TNFα, 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 TNFα 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**