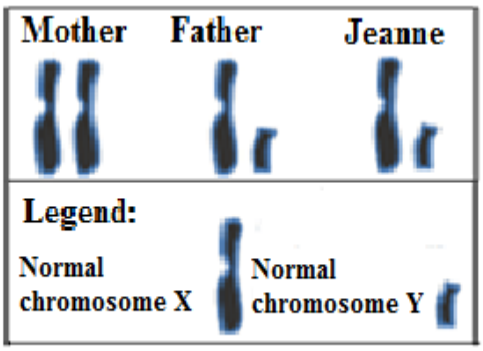
المركز التربوي للبحوث والإنماء	الهيئة الأكاديميّة المشتركة قسم : العلوم	المادة: علوم الحياة الشهادة: الثانوية الفرع: علوم الحياة نموذج رقم -١- المدّة : ٣ ساعات

نموذج مسابقة (يراعي تعليق الدروس والتوصيف المعدّل للعام الدراسي ٢٠١٨-٢٠١٩وحتى صدور المناهج المطوّرة)

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.

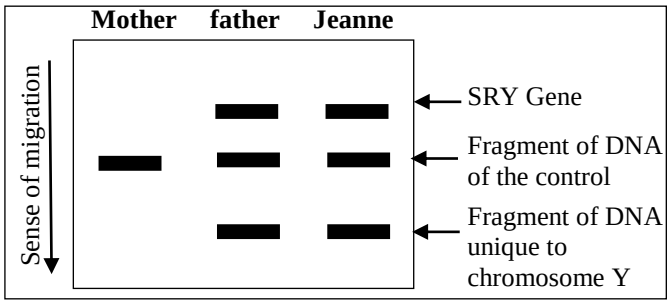


Document 1

1. 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.

2. Show that Jeanne's anomaly is not due to the absence of the SRY gene.



Document 2

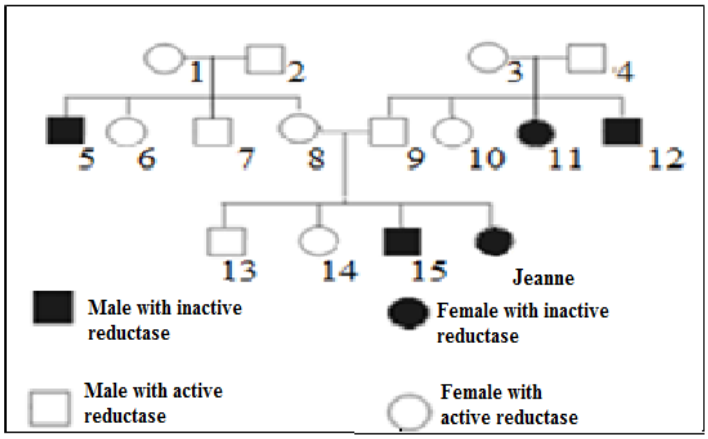
SRY gene codes for "TDF protein" which activates testosterone during embryonic life leading to the development of testicles in an embryo of karyotype XY.
Document 3 shows the partial sequences of amino acids of a functional TDF protein (A), a non-functional TDF protein (B) and a TDF protein (C) of Jeanne.

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

3. 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 5α reductase enzyme. Individuals 5, 12 and 15 show feminine phenotype before the age of 12. Janne's mother 8 and the woman 11 have similar karyotypes.

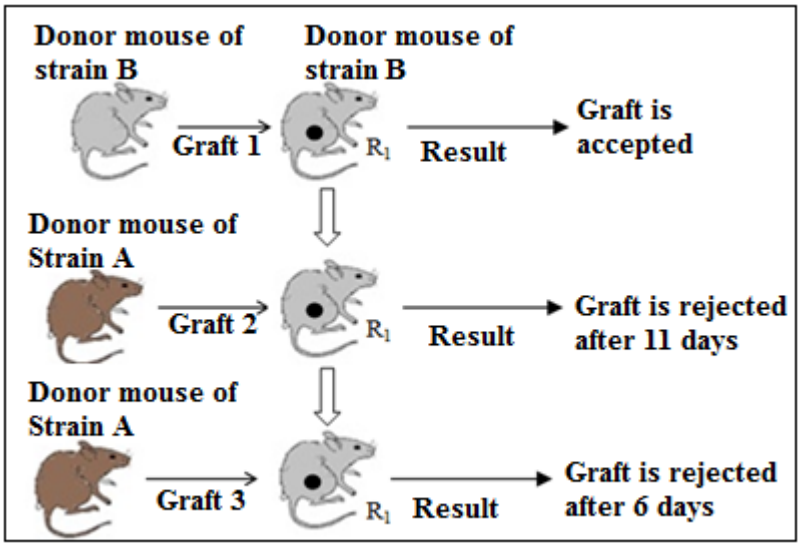


Document 4

4.1. Specify if the allele that determines the inactive form of 5α reductase is dominant or recessive.
4.2. Determine the chromosomal location of the gene responsible for the synthesis of 5 α reductase enzyme.
5. Explain why Jeanne who is born with a feminine phenotype becomes a boy at the age of 12.

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.



Document 1

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) on one hand and 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

Document 3

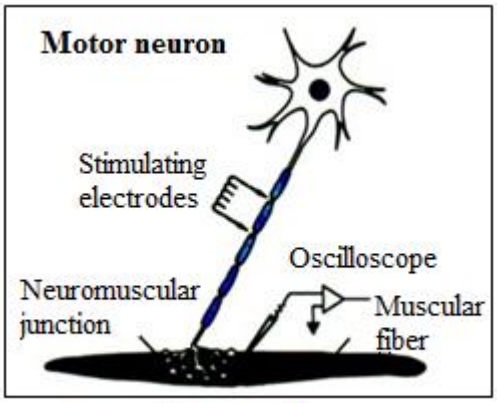
Exercise 3 (5 points) **Anesthesia and curare**

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.

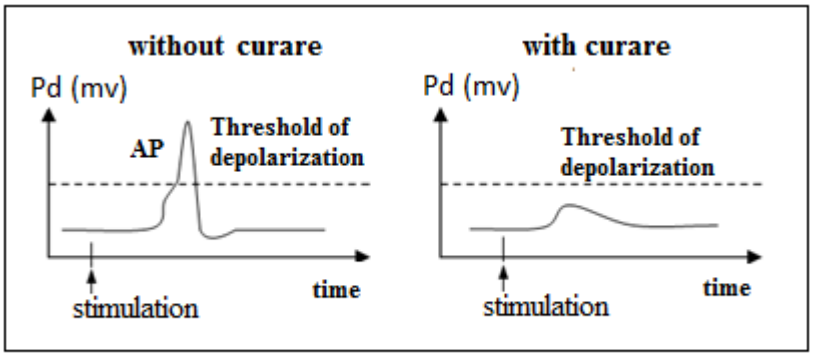
In order to explain the role and the mode of action of D-tubocurarine in cosmetic surgery, the following experiments are performed.

Experiment 1:

The axon of a motor neuron is effectively stimulated in the absence and then in the presence of curare injected in the neuro-muscular 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.



Document 1



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.

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

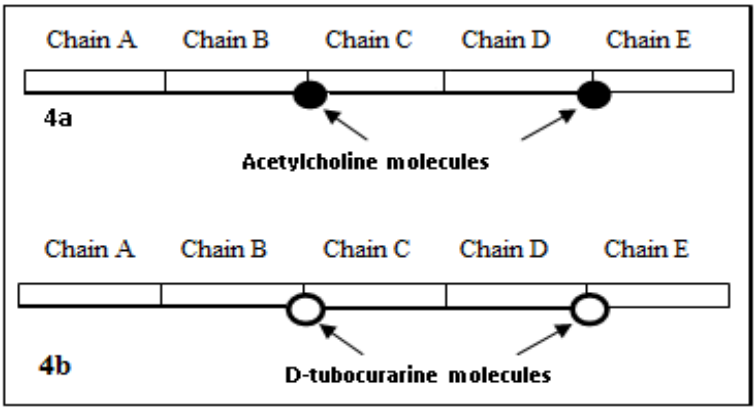
3. Construct, on the same graph, the curves that show the variation of the amplitude of muscular contraction as a function of acetylcholine concentration, with and without D-tubocurarine.

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**).



Document 4

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.

Exercise 4 (5 points) **Type 2 Diabetes**

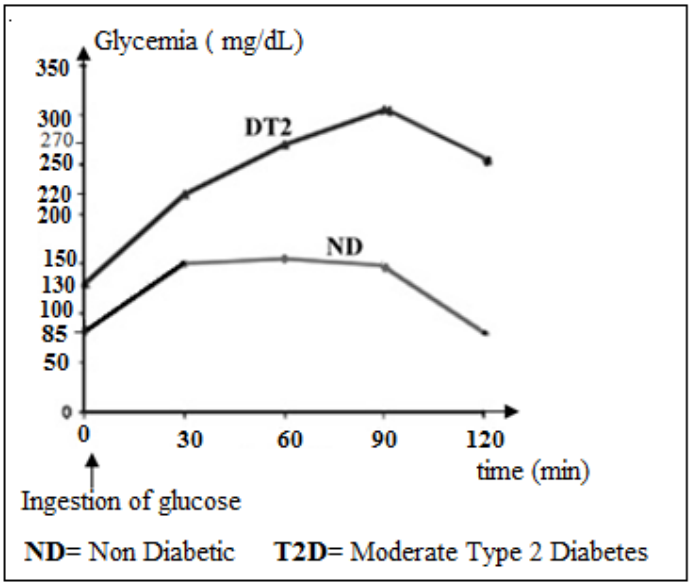
وقف العمل بهذا المحور (التعميم رقم ٢٨/م/٢٠١٨ تاريخ ٢٠١٨/٥/٢١).

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.



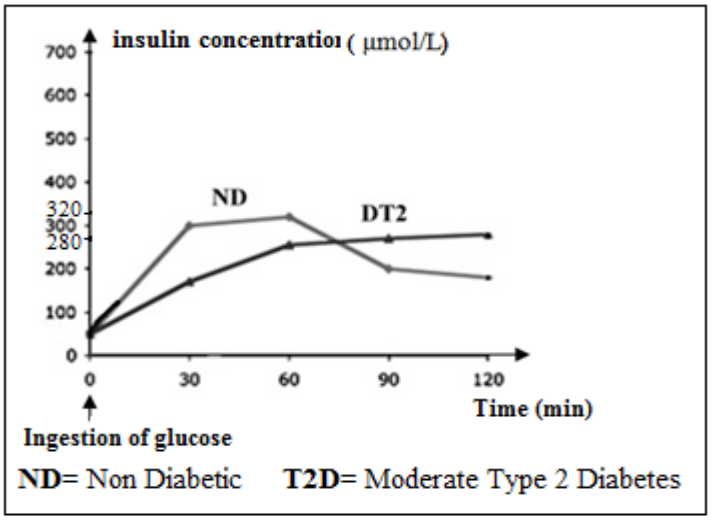
Document 1

1. Interpret the obtained results.

2. Formulated two hypotheses concerning the origin of type 2 diabetes.

Document 2 shows the results of measurement of insulin concentration in the blood of these two groups of individuals

3. Show, by referring to document 2, that treating individuals with T2D with insulin is not effective.



Document 2

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.

Quantity of glucose absorbed by the muscle cells ($nmol.mg^{-1}$ of tissue)	Normal Mice	Obese Mice
	5	3

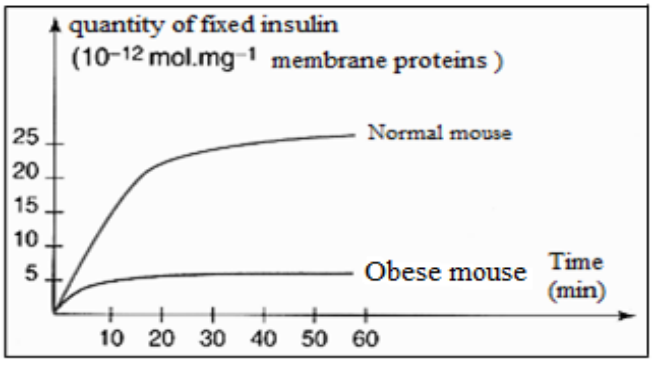
Document 3

4. 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.

5. Determine, by referring to document 4, the origin of type 2 diabetes.



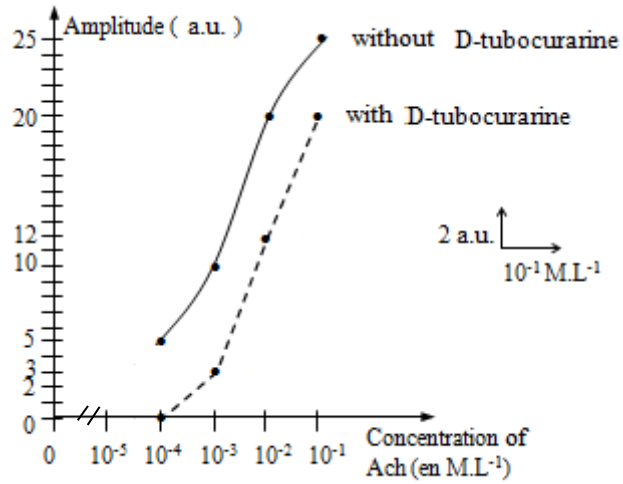
Document 4

المركز التربوي للبحوث والإنماء	الهيئة الأكاديميّة المشتركة قسم : العلوم	المادة: علوم الحياة الشهادة: الثانوية الفرع: علوم الحياة نموذج رقم -١- المدّة : ٣ ساعات

أسس التصحيح (تراعي تعليق الدروس والتوصيف المعدّل للعام الدراسي ٢٠١٨-٢٠١٩ وحتى صدور المناهج المطوّرة)

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 αreductase 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 11of 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^dY^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^dX^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	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. 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.	**1.25**
Parts of ex	**Exercise 2 (5 points)**	**Mark**
1	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). This shows that the graft is only accepted between individuals of the same strain. 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. This shows that the immune response responsible for graft rejection is much faster during the second contact with the same antigen.	**1.5**
2	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. This means that, the serum of hyper-immunized strain B has no effect in the rejection of the graft. Therefore, the hypothesis, which states that mice B have antibodies which are at the origin of graft rejection, is invalid. 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. This means that the lymphoid cells are responsible for triggering response against the antigen. Hence, the hypothesis which states that mice B possess immune memory cells which are at the origin of graft rejection is valid.	**1.5**
3	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. 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.	**1**

4	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. 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. 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.	**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	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 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.	**0.75**
3	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.  	**1.75**
4.1	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.uin 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.	**0.5**
4.2	We can conclude that D-Tubocurarine attenuates the action of acetylcholine on the muscle fibers.	**0.25**

5	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. Therefore, D-Tubocurarine replaces acetylcholine on postsynaptic receptors at the level of the muscle fiber and prevents the effect of acetylcholine.	**0.5**
6	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.	**0.75**
Parts of ex	**وقف العمل بهذا المحور (التعميم رقم ٢٨/م/٢٠١٨ تاريخ ٢٠١٨/٥/٢١).** **Exercise 4 (5 points)**	**Mark**
1	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. 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. 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. This shows that only the non-diabetic subject has a functional hypoglycemic regulation system. 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.	**1.25**
2	Hypothesis 1: Type 2 diabetes is due to a lack of insulin. Hypothesis 2: Type 2 diabetes is due to a lack of insulin receptors at the level of target cells.	**1**
3	The level of insulin in blood increases to 280 µmol / L in the diabetic individual during a period of 120 minutes, slightly lower than the maximum insulin level of 320 µmol / L reached in the non-diabetic individual, during a shorter duration of time, 60 minutes. This shows that the individual DT2 secretes an almost sufficient quantity of insulin but with a delay of time of 60 min. 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.	**1**
4	In obese mice, the amount of glucose absorbed by the muscle cells is 3 $nmol.mg^{-1}$of tissue, smaller than that absorbed by the muscle cells of the normal mouse which is 5 nmol.mg-1. It can be deduced that muscle cells in obese mice are less sensitive to insulin than those in normal mice.	**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 $25x10^{-12}$ $mol.mg^{-1}$ at t =60 min; whereas in obese mice, it increases to$5x10^{-12}$ 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**