

II. For each of the propositions numbered from 1 to 4, there is only one correct suggestion in each set. Copy down these pairs (1;..), (2;..), (3;..), (4;..), and match each number with its corresponding letter. (2pts)

2. The turner syndrome appears in: 1. The individual carrying a balanced a- men with chromosomal formula chromosomal translocation shows: 2n+1=22AA+XYY; a- an abnormal structure of chromosomes and a b- women with chromosomal formula normal phenotype; b- an abnormal structure of chromosomes and an 2n-1=22AA+X; c- men with chromosomal formula abnormal phenotype; c- a normal structure of chromosomes and 2n-1=22AA+Y; d- women with chromosomal formula normal phenotype; d- a normal structure of chromosomes and an 2n+1=22AA+XXX. abnormal phenotype. 3. The reduction of chromosomal formula 4. A polysome designate a structure composed of a molecule of: take place during: a- RNA bound to several RNA polymerase; a- the anaphase I of meiosis; b- DNA bound to several DNA polymerase; **b**- the prophase I of meiosis; c- RNA bound to several ribosomes; c- the anaphase II of meiosis; d- the prophase II of meiosis. d- DNA bound to several ribosomes.

III. Copy down on your answer sheet the letter of each of the following propositions, and write whether the statements are « true » or « false »: (1pt)

- a. In Humans, the diploid cell contains two chromosomal batches of maternal origin.
- **b.** The fertilization amplifies the chromosomal mixing that occurs during the meiosis.
- c. The plasmids are the circular RNA molecules used as genetic engineering tool.
- **d.** The karyotype is a representation of relative location of genes on a chromosome.

IV. Match each element of Group 1 to the corresponding definition Group 2. Copy down these pairs (1;..), (2;..), (3;..), (4;..) and match each number to its corresponding letter. (1pt)

الصفحة	NS 32E
2	NO 32E
6	

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Group 1
1. Tetrad
2. Crossing over
3. Chromosomal
abnormality
4. Interchromosomal
recombination

Group	2
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- **a.** mixing of the alleles due to random separation of homologous chromosomes during the anaphase I.
- **b.** change in the number and /or the structure of chromosomes or in both.
- **c.** structure formed by homologous chromosomes pairing during prophase I.
- **d.** exchange of chromosomal fragments between homologous chromosomes during prophase I.

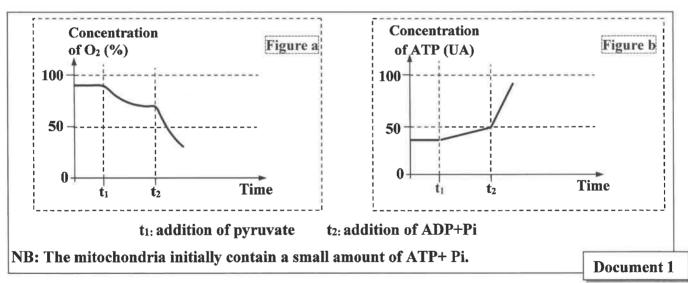
Section II: Scientific reasoning and communication in graphic and written modes (15 pts)

Exercise 1 (5.5 pts)

On the night of Wednesday, August 12, 2015, two terrible explosions shook the industrial zone of the port of the city of Tianjin in China causing more than a hundred dead and more than 700 injured. Many dangerous chemical substances are usually stored in this area including sodium cyanide the source of a very toxic gas, hydrocyanic acid (HCN) leading to death by cell and tissue asphyxiation. In order to understand the effect of hydrocyanic acid on respiratory metabolism and its relationship to asphyxiation, the following data are proposed:

• Data 1:

We place a mitochondrial suspension in a suitable rich-oxygen medium, then we follow the evolution of the concentration of O_2 and ATP in the medium. The document 1 shows the experimental conditions and obtained results.



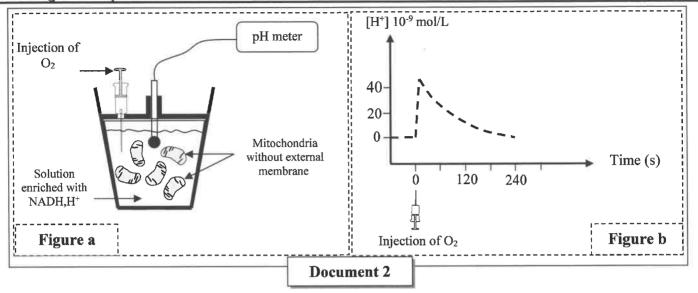
1. Based on the document 1, **describe** the variation of concentrations of O_2 and ATP in the medium, then **deduce** the effect of adding pyruvate and ADP + Pi on mitochondrial respiratory metabolism. (1.5pts)

• Data 2:

Mitochondria, deprived of their external membranes, are placed in a solution without oxygen and enriched with electron donors (NADH, H⁺). The variation in the concentration of H⁺ protons in the solution is measured before and after the injection of a limited amount of oxygen. **Figures a** and **b** of document 2 present respectively the conditions and results of this experiment.



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2. Based on the data in document 2, **describe** the evolution of the concentration of protons (H⁺) in the solution, then **deduce** the effect of oxygen injection on the movement of protons (H⁺) through mitochondrial inner membrane. (1pt)

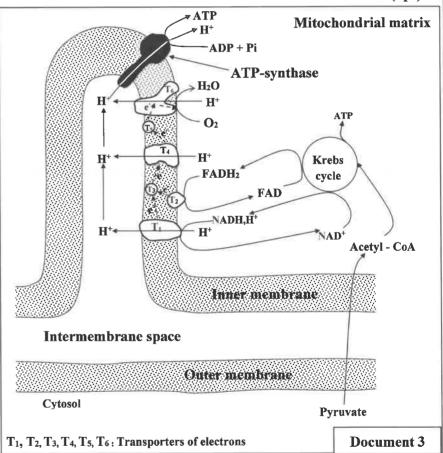
• Data 3:

The scheme in document 3 summarizes the reactions of mitochondrial respiratory metabolism and the relationship between pyruvate degradation and ATP synthesis.

3. By exploiting the document 3, explain the variation of concentrations of O₂, protons (H⁺) and ATP registered in experiments of documents 1 and 2. (2pts)

• Data 4:

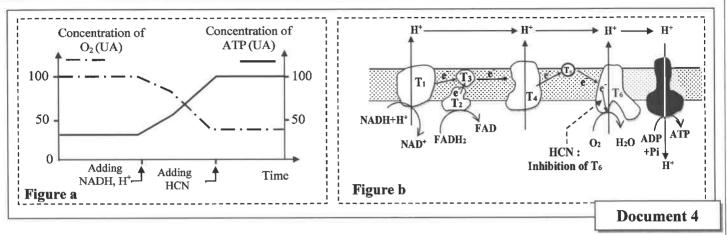
In order to understand the relationship between the exposure to hydrocyanic acid (HCN) and the asphyxiation states recorded following the explosions in the industrial zone in the port of the Tianjin city, the data in Document 4 is proposed.



The **figure a** in document 4 shows the evolution of concentrations of O_2 and ATP in a mitochondrial suspension placed in a suitable medium rich in O_2 and ADP + Pi following the addition of NADH,H⁺ and HCN. The **figure b** of the same document represents the mechanism of oxidative phosphorylation at the mitochondrion and the site of action of HCN.



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4. By exploiting data of document 4, explain the asphyxiation related to the exposure to hydrocyanic acid. (1 pt)

Exercise 2 (6.5 pts)

Tay-Sachs disease is a hereditary neurodegenerative disease which one of the forms occurs around the age of 2 to 3 years. Among its main symptoms: loss of motor skills, epileptic crisis, balance disorders, hypersensitivity to noise, mental retardation and sometimes decreased vision. Children affected by this disease usually die around the age of 5 years. In order to understand the genetic origin of this disease, the following data is proposed:

• Data 1:

Research has linked this disease to the lack of Hexosaminidase A (HEX-A) enzyme activity in cytoplasmic vesicles called lysosomes. In the normal case, this enzyme ensures the degradation of a substance called Ganglioside (GM2). In the abnormal case the accumulation of GM2 in the lysosomes is toxic for the nerve cells causing their degeneration. The document 1 represents the future of Ganglioside GM2 in nerve cells and the appearance of these cells in a healthy individual and in an affected individual.

Molecule level	Cell level (nerve cell)	Individual level
Ganglioside GMA GM2 Gunctional HEX-A Ganglioside GNA Complex	Nucleous Lysosome	Healthy individual
Non-functional HEX-A Ganglioside GM2 Accumulation of Ganglioside GM2	Nucleous Giant lysosome	Affected individual by Tay-Sachs Document 1

1. Based on document 1, show the protein-trait relationship.

(0.75pt)

الصفحة NS 32E

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• Data 2:

The synthesis of **HEX-A** enzyme is controlled by a gene **HEX-A** which exists in two allelic forms: The normal allele responsible for functional **HEX-A** enzyme synthesis and the abnormal allele responsible for non-functional **HEX-A** enzyme synthesis. The document 2 present a fragment of untranscribed strand of DNA for each of the two alleles. The document 3 presents the table of the genetic code.

Numbers of nucleotide	1270 ↓		Read	ing dire	ection		1290 ↓)	
Fragment of normal allele	ĊGT	ATA	TCC	TAT	GCC	CCT	GAĊ		
Fragment of abnormal allele	CGT	ATA	TCT	ATC	CTA	TGC	CCC	TGA C	

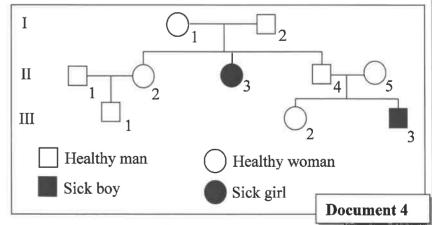
Document 2 2nd letter 3rdletter \mathbf{U} G 1st letter UUU UCU UAU UGU Phe Tyr Cys UUC UAC UGC UCC \mathbf{C} \mathbf{U} UUA Ser STOP **UCA UAA UGA** A STOP Leu UUG UGG UCG **UAG** Trp G CUU CCU CAU **CGU** U His CUC CCC CAC CGC \mathbf{C} Arg \mathbf{C} Leu Pro CUA CAA CCA **CGA** A Gln **CUG** CCG CAG CGG G AUU ACU AAU AGU U Asn Ser **AUC** ACC AAC AGC C Ile A Thr AUA ACA AAA AGA Lys Arg AUG Met ACG AAG AGG \mathbf{G} **GUU GCU** GAU GGU \mathbf{U} Ac.asp GUC GAC **GCC** GGC \mathbf{C} G Val Ala Gly **GUA GCA** GAA GGA A **Document 3** Ac.glu **GUG GCG GAG** GGG

2. Based on documents 2 and 3, determine mRNA and the amino acids sequences corresponding to each of the two alleles, then explain the genetic origin of the disease. (1.5pts)

• Data 3

The document 4 presents a pedigree of a family whose members are affected by Tay-Sachs disease.

- 3. Based on the pedigree of document 4, determine the mode of transmission of this disease. (1pt)
- 4. a. Give by justifying your answer, genotypes of individuals I₂, II₂ and III₃. (1pt)



(Use the symbols N and n for the two alleles of the studied gene)

b. The couple II₄ and II₅ wish to have a third child, **determine** the probability that this couple will give birth to a healthy child. **Justify** your answer by Punnet square. (0.75pt)

الصفحة 6 NS 32E

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• Data 4

The Tay-Sachs disease is a rare hereditary disease, however in some North American populations it affects one child in 3600.

- 5. Based on the previous data and knowing that these populations are in the Hardy-Weinberg equilibrium:
 - a. Calculate the frequency of the two allele N and n in these populations.

(1.pt)

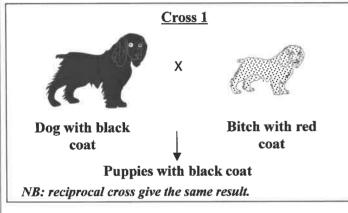
b. deduce the frequency of healthy carrier individuals in these populations.

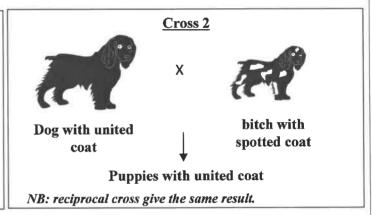
(0.5pt)

N.B: Give only four digits after the decimal point in numerical applications.

Exercise 3 (3 pts)

To study the transmission of two hereditary traits in cocker dog: the color (black or red) and type (united or spotted) of coat, we suggest the following crosses:





1. What do you deduce from the results of the two crosses 1 and 2?

(1pt)

- Cross 3: The cross between dog with united and black coat and bitch with spotted and red coat yielded the following results:
 - 25% puppies with black and united coat;
 - 25% puppies with red and spotted coat;
 - 25% puppies with black and spotted coat;
 - 25% puppies with red and united coat.
- 2. Determine, by justifying the answer if the two studied genes are linked or independents. (0.5pt)
- Cross 4: The cross between dog with black and united coat and bitch with red and united coat yielded the following results:
 - 3 puppies with black and united coat;
 - 3 puppies with red and united coat;
 - 1 puppy with black and spotted coat;
 - 1 puppy with red and spotted coat.
- 3. a. Determine the genotype of each of the parents in cross 4. Justify your answer

(0.5pt)

b. Use Punnett square to Interpret the results obtained in cross 4.

(1pt)

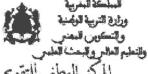
Use the following symbols: -R and r for alleles responsible for the color of coat; -B and b for alleles responsible for the type of coat.

-End-



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0.5

SSSSSSSSSSSSSSSS

NR 32E

المركز الوطني للتقويم والامتحانات

علوم الحياة والأرض 3h مدة الإنجاز المادة شعبة العلوم التجريبية مسلك علوم الحياة والأرض (خيار أنجليزية) 7 المعامل الشعبة أو المسلك

	Key and marking scale	
Questions	Response elements	Scores
	Section I : Knowledge Retrieval (5 pts)	
	definition (accept any correct definitions)	
I	a- Mitosis: Cell division that allows to obtain, from a mother cell, two genetically identical daughter cells carrying the same genetic information as the mother cell.	0.5
	b- Restriction enzyme: enzyme that allows of cutting the DNA in specific locations.	0.5
II	(1, a); (2, b); (3, a); (4, c)	0.5x4
Ш	1- false 2- true 3- false 4- false	0.25x4
IV	(1, c); (2, d); (3, b); (4, a)	0.25x4
Sect	ion II: Scientific reasoning and communication in graphic and written modes (15	pts)
	Exercise 1 (5.5 pts)	
	Description:	
1	+Variation in the concentration of O ₂	0.5
	-before adding pyruvate, the concentration of O ₂ is constant at about 90%.	
	-after adding pyruvate (t_1) , the concentration of O_2 decreases then stabilizes at 70%.	
	-after adding ADP +Pi (t ₂), the concentration of O ₂ decrease to reach 30%.	
	+Variation in the concentration of ATP	0.5
	-before adding pyruvate, the concentration of ATP is constant at about 37 UA.	
	-after adding pyruvate (t ₁), the concentration of ATP increases to reach 50 UA.	
	-after adding ADP +Pi (t ₂), the concentration of ATP increase to reach more than	
	90UA.	
	N.B: accept values close to that of the response elements	
	Deduction	0.5
	The pyruvate and ATP + Pi activate the consumption of O ₂ and the ATP production	
	at the mitochondrion level.	
	(Accept: the pyruvate and ADP +Pi activate the mitochondrial respiration).	
	Description:	0.5
2	-Before O ₂ injection, the concentration of H ⁺ is zero in the medium.	
	-Just after the injection of O ₂ , the concentration of H ⁺ increase to reach maximal	
		1

value (more than 40.10⁻⁹mol/L) and decreases to reach its initial value after 240s. Deduction of the effect of O2 injection on the direction of movement of protons

The O₂ activates the exit of H⁺ from the matrix to external medium through the

H⁺:

mitochondrial inner membrane.

Reference Part P	الصفحة	r		_				
- Adding pyruvate to mitochondrial suspension → degradation of pyruvate in the matrix → reduction of electron and proton transporters	2	NR 32E	الامتحان الوطني الموحد للبكالوريا - الدورة العادية 2021 - عناصر الإجابة - مادة: علوم الحياة والأرض- شعبة العلوم التجريبية مسلك علوم الحياة والأرض (خيار أنجليزية)					
H ⁺ gradient on either side of the mitochondrial inner membrane. (Figure b document 2) → return of H ⁺ protons to the matrix (decrease in the concentration of H ⁺ protons in the external medium) through the ATP synthase → phosphorylation of ADP and ATP synthesis (Figure b document 1). Explanation of asphyxiation due to exposure to the HCN: Exposure to hydrocyanic (HCN) inhibits the transporter T ₆ → the electrons do not arrive to final acceptor that is O₂ (no reduction of O₂) which explain the stop of consumption of O₂→ stop the oxidative phosphorylation which explain the stop of ATP synthesis ⇒ the cells are unable to use O₂ even in its presence from where asphyxiation. Exercise 2 (6.5 pts) Protein-trait relationship: in healthy individual: The enzyme (HEX-A) is functional → degradation of ganglioside GM2 in GM3 + GNA → no accumulation of GM2 in lysosomes of nerve cells→ normal nerve cell→ healthy individual: in affected individual: The enzyme (HEX-A) is non-functional → no degradation of ganglioside GM2 → accumulation of GM2 in lysosomes of nerve cells→ degeneration of nerve cell→ individual raffected by Tay-Sachs. The modification in the protein (enzyme HEX-A) causes a modification in the phenotype of individual (healthy or sick individual) from where the protein-trait relationship. mRNA and amino acids sequences corresponding to each two alleles: mARN: CGU - AUA- UCC- UAU- GCC- CCU- GAC Peptide: Arg - Ile - Ser - Tyr - Ala - Pro - Ac.asp - Fragment of normal allele: mARN: CGU - AUA- UCU- AUC- CUA- UGC- CCC - UGA- C Peptide: Arg - Ile - Ser - Ile - Leu - Cys - Pro The Genetic origin of disease: Mutation by addition of four untranscribed strand (DNA) has changed the reading	3	 Adding pyruvate to mitochondrial suspension → degradation of pyruvate in the matrix → reduction of electron and proton transporters						
the external medium) through the ATP synthase → phosphorylation of ADP and ATP synthesis (Figure b document 1)		H ⁺ gradient on either side of the mitochondrial inner membrane. (Figure b document 2) → return of H ⁺ protons to the matrix (decrease in the concentration of H ⁺ protons in						
Exposure to hydrocyanic (HCN) inhibits the transporter T ₆ → the electrons do not arrive to final acceptor that is O ₂ (no reduction of O ₂) which explain the stop of consumption of O ₂ → stop the oxidative phosphorylation which explain the stop of ATP synthesis ⇒ the cells are unable to use O ₂ even in its presence from where asphyxiation. Exercise 2 (6.5 pts) Protein-trait relationship: in healthy individual: The enzyme (HEX-A) is functional → degradation of ganglioside GM2 in GM3 + GNA → no accumulation of GM2 in lysosomes of nerve cells→ normal nerve cell → healthy individual: The enzyme (HEX-A) is non-functional → no degradation of ganglioside GM2 → accumulation of GM2 in lysosomes of nerve cells→ degeneration of nerve cell → individual raffected by Tay-Sachs. The modification in the protein (enzyme HEX-A) causes a modification in the phenotype of individual (healthy or sick individual) from where the protein-trait relationship. mRNA and amino acids sequences corresponding to each two alleles: "Fragment of normal allele: mARN: CGU - AUA- UCC- UAU- GCC- CCU- GAC Peptide: Arg - Ile - Ser - Tyr - Ala - Pro - Ac.asp - Fragment of abnormal allele: mARN: CGU - AUA- UCU- AUC- CUA- UGC- CCC - UGA- C Peptide: Arg - Ile - Ser - Ile - Leu - Cys - Pro The Genetic origin of disease: Mutation by addition of four untranscribed strand (DNA) has changed the reading		the A	e external medium) through the ATP synthase → phosphorylation of ADP and TP synthesis (Figure b document 1)	0.5				
Protein-trait relationship: in healthy individual: The enzyme (HEX-A) is functional → degradation of ganglioside GM2 in GM3 + GNA → no accumulation of GM2 in lysosomes of nerve cells→ normal nerve cell→ healthy individual: in affected individual: The enzyme (HEX-A) is non-functional → no degradation of ganglioside GM2 → accumulation of GM2 in lysosomes of nerve cells→ degeneration of nerve cell→ individual raffected by Tay-Sachs The modification in the protein (enzyme HEX-A) causes a modification in the phenotype of individual (healthy or sick individual) from where the protein-trait relationship. mRNA and amino acids sequences corresponding to each two alleles: Fragment of normal allele: mARN: CGU - AUA- UCC- UAU- GCC- CCU- GAC Peptide: Arg - Ile - Ser - Tyr - Ala - Pro - Ac.asp - Fragment of abnormal allele: mARN: CGU - AUA- UCU- AUC- CUA- UGC- CCC - UGA- C Peptide: Arg - Ile - Ser - Ile - Leu - Cys - Pro The Genetic origin of disease: Mutation by addition of four untranscribed strand (DNA) has changed the reading	4	Ex arr co.	sposure to hydrocyanic (HCN) inhibits the transporter $T_6 \rightarrow$ the electrons do not rive to final acceptor that is O_2 (no reduction of O_2) which explain the stop of nsumption of $O_2 \rightarrow$ stop the oxidative phosphorylation which explain the stop of TP synthesis	1				
Protein-trait relationship: in healthy individual: The enzyme (HEX-A) is functional → degradation of ganglioside GM2 in GM3 + GNA → no accumulation of GM2 in lysosomes of nerve cells→ normal nerve cell → healthy individual in affected individual: The enzyme (HEX-A) is non-functional → no degradation of ganglioside GM2 → accumulation of GM2 in lysosomes of nerve cells→ degeneration of nerve cell → individual raffected by Tay-Sachs								
mRNA and amino acids sequences corresponding to each two alleles:	1	- Th	in healthy individual: The enzyme (HEX-A) is functional → degradation of ganglioside GM2 in GM3 + GNA → no accumulation of GM2 in lysosomes of nerve cells→ normal nerve cell→ healthy individual in affected individual: The enzyme (HEX-A) is non-functional → no degradation of ganglioside GM2 → accumulation of GM2 in lysosomes of nerve cells→ degeneration of nerve cell→ individual raffected by Tay-Sachs the modification in the protein (enzyme HEX-A) causes a modification in the enotype of individual (healthy or sick individual) from where the protein-trait	0.25 0.25 0.25				
Mutation by addition of four untranscribed strand (DNA) has changed the reading	2	- n	RNA and amino acids sequences corresponding to each two alleles:	0.25x2 0.25x2				
normal→ synthesis abnormal protein →enzyme HEX-A non-functional →the Tay-		Ma fra no sac	utation by addition of four untranscribed strand (DNA) has changed the reading me → synthesis of RNAm modified include codon Stop compared to RNA rmal→ synthesis abnormal protein →enzyme HEX-A non-functional →the Taychs disease appear	0.5				

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4	العلوم التجريبية مسلك علوم الحياة والأرض (خيار أنجليزية)	- مادة: علوم الحياة والارض- شع						
	- Addition of TCTA betwen nucleotides 1275 and 1276 Addition of TATC betwen nucleotides 1273 and 1274 Addition of TATC betwen nucleotides 1277 and 1278 Addition of CTAT betwen nucleotides 1276 and 1277.							
	The mode of transmission of this diseases: (accept a							
	• The responsible allele for disease is recessive							
3	sick girl II3 (or a sick boy III3)	0.25						
	• The responsible gene for disease is carried by auto	0.20						
	Justification: the disease is dominant, the girl II ₃ is a healthy father I ₂	0.35						
	 a. Genotypes of individuals I2, II2 et III3 with justification: I2: N//n because the individual is healthy and gave birth to a sick girl. II2: N//n or N//N because she is healthy and descends from heterozygous parents III3: n//n because he is affected. b. The probability that the couple (II4 and II5) give birth to a healthy child 							
	with justification: [N] II4 x II5 [N]							
4	N//n N//n							
	½ N/; ½ n/ ½ N/; ½ n	0.25						
	Punnet square:							
	Gametes of parents ½ N/ ½ N/ ¼ N//N [N]	½ n/ 0.25						
	72 N/ 74 N//N [N]	1/4 N//n [N] 1/4 n//n [n]						
	The probability that the couple (II4 and II5) give birth to							
	a. The frequency of two alleles N and n in these population is in equilibrium f (so							
	- Abnormal allele frequency is: $f(N) = p = 1 - q = 0$ N.B : accept also numerical applic	0.5 0834						
5	- Normal allele frequency is: $f(n) = q = 0.0141$ - Abnormal allele frequency is: $f(N) = p = 1 - q = 0.9859$							
	b. deduction: the healthy carrier individuals are heterozygous of genotype $(N//n) \rightarrow$ the frequency of healthy carrier individuals in these populations is: $f(N//n) = 2pq = 2 \times 0.0166 \times 0.9834 \approx \textbf{0.0326}$ $N.B: accept \ also \ numerical \ applications:$ $f(N//n) = 2pq = 2 \times 0.0141 \times 0.9859 \approx \textbf{0.0278}$							
	Exercise 3 (3 pts)							
	According to the first and second cross, we deduce to the parents are from pure lineage in each of the two cross. The responsible allele for black coat is dominant (R) and the second cross, we deduce the parents are from pure lineage in each of the two cross.	sses 0.25						
1	coat is recessive (r)							

4	الصف 4 NR :	جابة نيار أنجليزية)	202' - عناصر الإ. لوم الحياة والأرض (ذ	ريا - الدورة العادية 1 رم التجريبية مسلك عا	الوطني الموحد للبكالق ة والأرض- شعبة العلو	الامتحان ا مادة: علوم الحياا				
		spotted is recessive (b)								
		study traits are not sex linked)								
	2	the two studied go because the cross phenotypes (with	3 is a test cross v	which gives four	different and equ	iprobable	0.5			
		a. genotypes of p + the parent with R //r B//b	U		nited coat) is hete	erozygous:				
Justification: the parent has descendants double-recessives with red and coat +the parent with red and united coat is homozygote for coat color but heterofor coat type: r//r B//b						0.25				
	Justification: the parent has a recessive phenotype for the color and it gave spotted descendants.					0.25				
b. Interpretation of result			of results:							
	3	Phenotypes:	_	., 10]	$\langle [r, B]$		0.25			
		Genotypes:	R //r B//b $r//r B//b$							
		Gametes:	Gametes: $\frac{1}{4} \frac{R}{B} = \frac{1}{4} \frac{r}{b}$							
		D	¹ / ₄ R/b/;	½ r/B/			0.25			
		Punnet square :	½ R/B/	½ r/b/	1/4 R/b/	1//D/				
		gametes	R //r B//B	r //r B//b	R //r B//b	1/4 r/B/ r //r B//B				
		½ r/B/	1 /8 [R, B]	1 //1 B//0 1 /8 [r, B]	1 /8 [R,B]	1 /8 [r, B]				
		721111	R //r B//b		R //r b//b	r //r B//b				
		½ r /b/	1 /8 [R, B]		1 /8 [R, b]	1 /8 [r, B]				
		Results: 3/8 [R,					0.25			
	The theoretical results are identical to experimental results						0.25			