



EXAMEN DU BACCALAUREAT

MINISTÈRE DE L'ÉDUCATION NATIONALE
DU PRÉSCOLAIRE ET DES SPORTS

1075: J

Série / Option :

COMPOSITION DE :

S.V.T

RESERVE ACADEMIE

65232

Note Globale

En chiffres

En lettres

19,00
20

Dix neuf
vingt

Appréciation de la note chiffrée

Nom du correcteur et signature :

Wajae Benhardouze BWajae

Exercice 1. (6)

1. By exploiting Data of Document 1.
a. Similarities: Both individuals (the healthy and the sick) have the same number of mitochondria and the presence of pyruvate and lactate in different concentration. Also the same activities of glycolytic enzymes.

+ Differences: In the healthy individual, we have the presence of pyruvate in an average concentration (about 0,03 mmol/L) and the same for lactate (about 1 mmol/L).

• In the sick individual the presence of a high concentration of pyruvate (about 0,121 mmol/L) and lactate (about 2,25 mmol/L).
* The presence of pyruvate is approximately the same in both individuals.

b. In the sick individual we have a high activity of the glycolytic enzymes → the degradation of glucose by way of the glycolysis metabolic pathway → the content of glucose into pyruvate → the product of pyruvate in high concentration.

- In the sick individual we have the very low activity of mitochondrial → the non use of the respiratory metabolic pathway (to continue the degradation of pyruvate) → the use of fermented (lactic fermentation) → product of lactate in high concentration (due to the presence of pyruvate in high concentration).

2. By exploiting the data of document 2,

+ In medium 1: we have the high consumption of O_2 (0,179 AU) and the high activity of the ATP synthase (0,301 AU) the high product of ATP.

+ In medium 2: we have the low consumption of O_2 (0,021 AU) and the low activity of the ATP synthase (0,030 AU) the low product of ATP.

01/24

→ therefore the ~~main~~ Mitochondria in the abnormal person is the responsible in the ~~prob~~ appearance of the defect / observed / the problem is in the mitochondria.

3. By exploiting the data of Document 2, we observe that in the sick individual we have the low activity of the following reaction: the reduction of $NADH, H^+$ and the activity of the ATP synthase and the ADP phosphorylation:

1/

→ Since in the sick individual we have the low activity of the reduction reaction (product of $NADH, H^+$) → low frequency of electrons → the low activity of the complexes transformers of electrons (due to the small amount of electron and the inactive enzymes) → the low synthesis of ATP (due to the inactive ATP synthase "enzymes") → inactive electron transport chain → the low consumption of O_2 .

2/

4. Based on the previous data: we in the person with NARP (sick individual) / inactive electron transport chain → inactive mitochondria → the non-use of respiratory pathway to do the complete degradation of glucose and produce high amount of ATP → the use of lactic fermentation → the incomplete degradation of glucose (pyruvate) → the production of low amount of ATP.

Exercice 2. 215

01/

1. Based on document 1.
+ In the healthy person we have: the LDL quantity on the cell surface is decreasing with time / ~~LDL~~ LDL quantity inside the cells is increasing with time / the increase of LDL on the inside is compared by the decrease of LDL in the surface.
+ In the sick individual: LDL quantity in the cell surface is remaining constant (about 10 μM) / ~~LDL~~ ^{LDL} and the quantity of the LDL quantity inside the cells is very low and remains constant (about 2 μM).

01/

2. Since in the sick individual we have the more entry of LDL into the cells / remains constant in high quantity on the cell surface, due to the inactivity of LDL receptors →

amachic proteins → the high concentration of the cholesterol in the blood (higher than normal) → the appearance of hypercholesterolemia.

3. For the normal individual:
+ the untranscribed strand of the normal LDLR allele:
AGA - AAC - GAG - TTC - CAG - TGC - CAA
+ the transcribed strand of the abnormal LDLR allele:
TCT - TTG - CTC - AAG - GTC - ACG - GTT
+ the mRNA strand:
AGA - AAC - GAG - UUC - CAG - UGC - CAA ✓
+ the sequence of amino acids:
Arg - Asn - Glu - Phe - Gln - Cys - Gln ✓

• For the abnormal individual:
+ the untranscribed strand of the abnormal LDLR allele:
AGA - AAC - GAG - TTC - TAG - TGC - CAA
+ the transcribed strand of the abnormal LDLR allele:
TCT - TTG - CTC - AAG - ATC - ACG - GTT
+ the mRNA strand:
AGA - AAC - GAG - UUC - UAG - UGC - CAA ✓
+ the sequence of amino acids:
Arg - Asn - Glu - Phe ✓

* the genetic origin of FH disease:
We have mutation by substitution in the triplet number 33 of the nontranscribed strand of the abnormal allele (substitution of C by T) if substitution of G by A in the transcribed strand → the appearance of a stop codon in the amino acid sequence corresponding to the fragment of the normal allele (stop of the amino acid sequence) → non functional protein → the appearance of the abnormal LDL receptors (inactive receptors) → the non transfer of LDL to the cells → the high concentration of LDL in the blood → the appearance of hypercholesterolemia (FH disease)

Exercice 3. (2,5)

1. Dihybridism case: We study the transmission of two hereditary traits "Coat color" and "Hair size".
Cross 1: Between parent of pure lines.
We obtained F₁ generation composed of two phenotypes:
50% ♂ with an orange coat and short hair ✓
and 50% ♀ with bicolor coat and short hair ✓

امتحان شهادة البكالوريا

النقطة الإجمالية	
بالأرقام	بالحروف
التقدير المفسر للنقطة	

الشعبة / المسلك :
 مادة :

خاص بالأكاديمية

اسم المصحح وتوقيعه (ها) :

* For the hair traits we obtained a homogeneous generation all individuals are with short hair so the first law of Mendel is verified. The hair allele responsible for the short hair is dominant "R" and the allele responsible for the long hair is recessive "r".

* For the hair "coat color" we have 4 males of F₁ generation have the same color as their mother (the genes responsible for the hair is carried on the X chromosome) and the females of the F₂ generation have an intermediate color. This for us is a dominant case because the allele responsible for the black color "N" and the allele responsible for the gray color "n" the we get an intermediate character = bicolor "Nn".

2: since we have the gene responsible for the coat color is carried on the X chromosome and the gene responsible for the hair is carried on the autosome thus the two genes are independent.

Thus Hypothesis number 2 is correct.

3. Chromosomal interpretation of cross 2.

cross 2: (reciprocal cross)

phenotype ♂ [N ; R] × ♀ [n ; r]

genotype $\frac{X^N}{Y} ; \frac{R}{R} \times \frac{X^n}{X^n} ; \frac{r}{r}$

gametes 50% $\frac{X^N}{Y} ; \frac{R}{R}$ × 50% $\frac{X^n}{X^n} ; \frac{r}{r}$

50% $\frac{X^N}{Y} ; \frac{R}{R}$ 50% $\frac{X^n}{X^n} ; \frac{r}{r}$

- تبيينه: يمنع على المترشح(ة) أن يوقع أو يضع أية علامة في ورقته(ها) تبين موطنه(ها).



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Note Globale	
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Nom du correcteur et signature :

Fertilizant. Pur et Squash

$\begin{array}{c} \text{♀} \\ \text{X} \frac{0}{1} \end{array}$	$\begin{array}{c} \text{♂} \\ \text{X} \frac{N}{1} \end{array}$	$\begin{array}{c} \text{♂} \\ \text{X} \frac{R}{1} \end{array}$
$\begin{array}{c} \text{X} \frac{0}{1} \\ \text{80\%} \end{array}$	$\begin{array}{c} \text{X} \frac{N}{1} \\ \text{85\%} \end{array}$	$\begin{array}{c} \text{X} \frac{R}{1} \\ \text{85\%} \end{array}$
$\begin{array}{c} \text{X} \frac{0}{1} \\ \text{50\%} \end{array}$	$\begin{array}{c} \text{X} \frac{N}{1} \\ \text{25\%} \end{array}$	$\begin{array}{c} \text{X} \frac{R}{1} \\ \text{25\%} \end{array}$

W = Obtenir 50% ♂^P [0, R] and 50% ♀ [0N, R]

H₀ = l'écoulement - expérimental stable au soleil

Exercise 4. 4/5

1. From document 1:

the two tectonic clues of this collision are
Fold \rightarrow } this tectonic clues signifies that a strong
Fault \rightarrow overlap } compressive forces has been applied on the
region

0,5

2. From the data of Document 2,

+ during the passage from Rock R₁ to R₂ we have
the absence of Chlorite and the appearance of Biotite and
Muscovite and the ~~remains~~^{0,5} of Andalusite

+ during the passage from R₂ to R₃:

the absence of Andalusite and Muscovite and the appearance
of Sillimanite and Potassium Feldspar and the remains of
Biotite ^{0,5}

1

3. • the passage from R₁ to R₂

during the passage from R₁ to R₂ we have the change in domain
of presence of the minerals of R₁ (Chlor and And) (low pressure
(0,2 GPa) and low temperature (350°C) into the domain
of the presence of the minerals of R₂ (Bio - Mus - And) (average
temperature (450°C) and average pressure (0,35 GPa))
 \rightarrow the transformation from P₁ to P₂

• the passage from R₂ to R₃

during the passage from R₂ to R₃ we have the change in domain
of presence of the minerals of R₂ (Bio - And - Mus) (average
temp = 500°C) and average pressure (0,3 GPa) into
the domain of presence of the minerals of R₃ (Bio - Sill - FK)
(high temperature (670°C) and average pressure (0,4 GPa))

1,5

4. Based on document 4 we have that the gneiss rock R₄
are existed near to the gneiss (State of minerals) and
R₅ migmatite are existed in the gneiss state of minerals
and R₄ & R₅ are the 1st migmatite comes from gneiss there for
the migmatite is a result of the fusion of gneiss and its
collig \rightarrow after the fusion of gneiss the anatectic magma
is collig in the both to give the migmatite Rock R₄
with two structures (granular and foliated)

2

5. Based on document 4 and 5 we can observe that the pressure and temperature conditions under which each of the rocks (R1, R2 and R3) are found are (at a high pressure and high temperature) thus this region has been subjected to a collision because the conditions of temperature and pressure are as the case of collision geology.

0,5

Exercice 1

4,5

I.

1. Eutrochical 1,25

3. Qishata 1,25

2. Global warming 1,25

4. renewal of energy 1,25

1

II.

1. true x

2. true 1,25

3. False x

4. true 1,25

0,5

III. 1. a 1,25

3. a 1,25

2. d 1,25

4. c 1,25

2

IV (1. e)

(2. c)

(3. a)

(4. b)

1,25

1,25

1,25

1,25

1