Assessment Schedule
Scholarship Biology – 2007

Question One

<table>
<tr><th colspan="2">Significant factors contributing to the fundamental niche of Balanus and Chthalamus</th></tr>
<tr><td colspan="2"><u>Upper limits</u>

BFU:CFU $_E$ (both species compared)
• Balanus has a lower physiological tolerance to dehydration/desiccation so can't survive in the upper tidal zone of the shore and Chthalamus has a wider physiological tolerance to dehydration/desiccation so can occupy the upper tidal area of the shore

FU
▪ Both species found below the high tide mark because they need access to water for: filter feeding /reproduction/gas exchange/avoiding desiccation

<u>Lower limits</u>

FL
Both species found above low tide mark because:
• competition (for space) with algae/seaweed FL$_c$
• predation by starfish FL$_p$
• can't tolerate total submersion FL</td></tr>
<tr><th colspan="2">Significant factors contributing to the realised niche of Balanus and Chthalamus</th></tr>
<tr><th>Balanus</th><th>Chthalamus</th></tr>
<tr><td>The realised niche of Balanus is the same as its fundamental niche

Lower limits: as for fundamental niche.

Upper limits: as for fundamental niche but no comparison with other species. BU
Eg, Balanus has a low physiological tolerance to dehydration/ desiccation so can't survive higher up the shore.</td><td>The realised niche of Chthalamus is reduced from its fundamental niche and it is limited to the upper tidal zone

Lower limits
• Out-competed by Balanus for space on rocks/ Balanus larvae settle earlier and so occupy space on rocks CRL$_E$
• Weakness in either concept CRL$_O$
• Predators such as whelks inhabit the lower intertidal zone and may preferentially predate Chthamalus, excluding them from this area. CRL

Upper limits: as for fundamental niche but no comparison with other species. CU

Eg, Chthalamus has a high physiological tolerance to dehydration/desiccation so can survive in upper tidal zone.</td></tr>
</table>

G_E Gause's/competitive exclusion principle: defined and used to explain why Chthalamus can't occupy its entire fundamental niche

G_D Gause's/competitive exclusion principle: defined but does not clearly explain why Chthalamus can't occupy its entire fundamental niche.

Judgement Statement

Mark	Judgement Minimum or equivalent required
8	3 main ideas explained ($_E$) plus 2FL Logical, coherent answer, with minimal irrelevant information and no significant errors
7	
6	2 main ideas explained And 1 other factor
5	1 main idea explained and 2 other factors
4	1 main idea described ($_D$) and 2 other factors
3	1 main idea described OR two other factors
2	Describes abiotic or biotic factors relating to niche of either species
1	Some correct biological ideas
0	Answer not attempted/ No correct evidence

Main ideas:

G

BFU:CFU

CRL

Other factors

FU, FL, CRL , BU. CU,

Question Two

Aspect	Described	Explained or Justified (J)	Integrated (I)
Genetics	• Gene is located on an autosome / autosomal (non-sex chromosome). **G1**	• is not sex-linked / not on sex chromosome. **G1J**	
	• Shows (complete) **dominance**, **G2**	• Is a **dominant allele** so will be **expressed** whenever present. • Only require **one copy** of the mutant allele for the person to get HD. • Disease expressed in heterozygote. • **50%** of the offspring will be affected if a heterozygote mated with a normal parent. • All the offspring of a **homozygous** individual will be affected. **G2J**	• Shows **complete dominance** which is usually is **indistinguishable between homozygotes and heterozygotes**, but does show **phenotype difference** (more rapid in homozygotes) in progression. **G2I**

Factors	Describes	Explained or Justified (J)	Integrates
Number of CAGs (Table 1)	• **The more CAG repeats** , the **more likely** an individual/ offspring is to **develop** HD. **F1**	• If an individual has **40 or more** CAG repeats, the individual **will** develop HD; whereas, **below 26** CAG repeats, individuals will **not develop** HD **and** will **not pass it on** to offspring. **J1**	
Age at onset (Table 2)	• Age of onset **decreases** with an **increase** in the number of CAG repeats (inversely proportional). **F2**	• For example: the **median** age of onset of HD for an individual with **39** CAG triplet repeats is **66 years old** whereas individuals with 50 CAG triplet repeats will **show onset much earlier** at a **median age of 27 years.** **J2**	• Individuals with low numbers of CAG repeats will have later onset and are more likely to pass HD onto their children because they are more likely to have reproduced before they realise they have the disease. **I2**

Genotype versus Age at onset (Table 3)	• Genotype does **not** affect the age at **onset** of symptoms • Median age of onset does **not show statistical difference** between **heterozygotes and homozygotes**, for HD individuals with similar number of CAG repeats. • For example: the median age for a homozygote is 51.5 years but could be anywhere from 48.6–54.0 years whereas a heterozygote with a median age of 48.6 could lie between 44.7–52.5 years, so statistically there is no difference. **F2/3**		

Factors	Describes	Justifies	Integrates
Genotype and disease progression: (Fig 1)	• **Heterozygotes survive** longer than homozygotes, from the time of onset. **OR** • Homozygotes **progress** through all stages **faste**r than heterozygotes **Not** : more severe. **F3**	• **The average duration for** homozygotes from **Stage I to V is 15.5 years whereas for heterozygotes is 40.5 years.** • Mean duration for homozygous individuals in Stage II is 2.5 yrs (SD of 1–4 years) **whereas** for heterozygote individuals, it is 8.5 yrs (SD of 5.5–12.5 years). • Homozygous individuals are at Stage V for an average of 8 years **whereas** heterozygotes are at Stage V for an average of 18.5 years. **J3**	
	• Mortality is about the **same** for homozygotes and heterozygotes. **F3**	• Approximately **50%** of homozygotes and heterozygotes died between stages III and IV. **J3**	

<table>
<tr><th>Factors</th><th>Describes</th><th>Justifies J</th><th>Integrates I</th></tr>
<tr><td>Offspring gender and CAG repeats (Table 4)</td><td>• Approximately 75% of offspring show a change in the number of CAG repeats compared to their father (25% show no change).
• Female offspring are more likely to have a decrease in the number of CAGs when compared to their father
• Male offspring are more likely to have an increase in the number of CAGs when compared to their father.
F4</td><td>• Female offspring of a male carrying mutated allele are less likely to develop HD as there is a greater chance of the number of CAG repeats decreasing rather than increasing.
• Male offspring of a male carrying mutated allele are more likely to develop HD as there is a greater chance of the number of CAG repeats increasing rather than decreasing.
J4</td><td>• Male children of a father with 30- 40 CAG repeats have a higher risk of developing HD due to the increased chance of expansion.
• Male offspring are more likely to have an earlier onset of HD whereas female offspring are more likely to have a later onset because males are more likely to have an increase in number of CAGs.
I4</td></tr>
<tr><td rowspan="2">Other Ideas</td><td rowspan="2"></td><td>Explanation for the faster progression in homozygotes:
• The presence of 2, rather than 1 mutated protein products may cause more of the mutated protein to form which may result in a greater toxic effect.
J_0</td><td rowspan="2"></td></tr>
<tr><td>Explanation for the difference in increase in the number of CAG repeats in male offspring compared to female offspring:
• Sex-influenced effect; possible protective function of the X chromosome.
• Issues with experimental design - Was sex of the parent important; repeat experiment using GE mother.
• Small sample size; query validity of data.
J_0</td></tr>
</table>

Judgement Statement

Mark	Judgement: Minimum or equivalent required
8	6 G/F and 4 J including G_2J or $F_{2/3}$ Logical, coherent answer with minimal irrelevant information and no significant errors
7	
6	5G/F and 3J
5	
4	3G/F and 1J
3	3 ideas described
2	2 ideas described
1	Some correct biological ideas.
0	Answer not attempted / No correct evidence

G = Genetics
F = Factors affecting HD
J = Justification/ explanation

Question Three

Application	Definition	Evidence of impact of gene pool Statement of the impact of a named application on the gene pool.	Further detail Further explanation / justification of the impact of application on the gene pool
Genetic Testing			
Pre-birth	PIGD is a technique used to identify genetic defects in embryos. **A1**	• The **frequency** of the allele for the genetic defect will **reduce** in the **gene pool** if embryos are **not used.** **NOT** reduce size of gene pool. **G1**	• Inequality of access to technology eg lack of money to pay for technology / not available in area or country. So will have different effect on gene pool in different countries/ parts of the community. **D1**
Amniocentesis	Amniocentesis is a technique used to **identify genetic defects** in foetus. **A1**	• The **frequency** of the allele for the genetic defect will **reduce** in the **gene pool** if foetuses are **aborted.** **G1**	• Inequality of access to technology eg lack of money to pay for technology / not available in area or country. So will have different effect on gene pool in different countries/ parts of the community. **D1**
Adult	This a technique used to **identify genetic defects** in adults. **A1**	• If adults are tested and are positive, they then have a choice as to having children or not which could mean either: o The frequency of the allele for the genetic disease will **reduce** in the gene pool because decide not to breed, OR o The frequency of the allele for the genetic disease will **increase/maintain** in the gene pool because they get treatment and then breed. **G1**	• Inequality of access to technology eg lack of money to pay for technology / not available in area or country. So will have different effect on gene pool in different countries/ parts of the community. **D1**

Application	Definition	Evidence of impact of gene pool	Further detail
Gene therapy	**Adding a gene** or group of genes to a cell in order **to treat or correct a genetic disorder**. **A2**	• Gene therapy targets **non germ line** cells, ie somatic cells, so will **not directly affect gene pool**. • The frequency of the allele for the genetic disease will **increase/maintain** in the **gene pool** because affected people get treatment and then can **reproduce.** **G2**	• Gene therapy technology as a whole has yet to be proven and it's meeting a number of stumbling blocks, so may have little impact (low success rate). • Should the viral vector invade non-target cells, especially the germ line cells, and integrate its DNA with the individual's DNA, then the possibility of the development of onco-genes in the gametes is increased, which could then be inherited by offspring. • To impact on **gene pool** would require gene therapy to affect **gonads** or to alter a zygote early enough in development that the gene was incorporated into **all** cells. **D2**
Stem Cell	Taking **undifferentiated cells and getting them to differentiate** into a specialist cells / tissue **A3**	• Stem cell therapy could enable individuals to **survive** and contribute their **alleles** to the **gene pool** who might **not** have already done so thus **increasing genetic diversity.** • Stem cells could be used in the treatment of **genetic diseases**, which could result in an increase in the **frequency of the defective allele** within the gene pool as individuals are able to **survive** and **reproduce** with the stem cell treatment **G3**	• Most defects / diseases treated by stem cell therapy are not **genetic** in origin so the therapy will have **no impact** on **gene pool** in terms of change in frequency of defective alleles. • As stem cell therapy treats defects / diseases of **somatic cells / organs** it will have **no impact** on gene pool other than the possible impact of longer survival of affected people. **D3**
Xeno-transplantation	Organs / tissues are **transplanted from one species to another.** **A4**	• Xeno-transplantation could enable individuals to **survive** and contribute their **alleles** to the **gene pool** who might not have already done so thus **increasing genetic diversity.** • Xeno-transplantation could be used in the treatment of **genetic diseases**, which could result in an increase in the **frequency of the defective allele** within the gene pool as individuals are able to survive and **reproduce** with the treatment. **G4**	• Most defects / diseases treated by xeno-transplanation are not **genetic** in origin so the therapy will have **no impact** on **gene pool** in terms of change in frequency of defective alleles. • As Xeno transplantation involves the transplanting of organs / tissues that do not affect the gametes, it will have no impact on gene pool other than the impact of longer survival of affected people. • **NOT** genes from other species entering human gene pool. **D4**
A5/6…	**Other applications** eg cloning, transgenesis (used in gene therapy) clearly defined, recognises that still in research stage and must alter germ line to have impact on gene pool.		

Biological evolution	**Discussion focuses on evolutionary impact on populations rather than impact of on gene pools** **Evolution: change in allele / gene frequencies of populations over generations as a result of selection pressures.**
B	• **The human population is very large,** so correcting genetic defects in a small number of people **isn't going to change** allele frequencies significantly • **Changes in allele frequency** due to impact of technologies may occur in **small populations** if **gene flow is restricted**. • **Significant changes in allele frequency unlikely because of gene flow** – migration of people between populations • Technologies that enable **people to survive longer than they would otherwise, will result in variation** being maintained in the gene pool, which is **advantageous** to *Homo sapiens* in their future evolution as it **provides more material** for **natural selection** to work on. • Genetic testing could result in people choosing to abort or not implant embryos, which could reduce variation in gene pool. This leads to **reduction in genetic diversity** / phenotypes for **natural selection** to work on. • Through these technologies we are **maintaining detrimental alleles** in the gene pool that **previously** would have been **eliminated** through **natural selection** – how will this affect future evolution? • A consequence of genetic testing may be the removal of alleles from gene pool. Due to possible pleiotropic effects of removed alleles (eg sickle cell anaemia) this may have **unforseen consequences** by **removing useful variation** for **natural selection** to work on. • How much impact **will changing allele frequencies** have on human evolution, when technology is **minimising** many **environmental selection pressures**?

Judgement Statement

Mark	Judgement – minimum or equivalent required
8	4 AG and 2B or 2D Logical, coherent answer, with minimal irrelevant information and no significant errors.
7	
6	4AG
5	3AG
4	2AG
3	3A
2	2A
1	Some correct biological ideas
0	Answer not attempted / No correct evidence

A = Application
G = Impact of application on Gene pool
D = Further detail
B = Impact on Biological evolution of humans.