B242 Exam. 2002 Some model answers

Section A
2. How does sexual selection differ from natural selection?
This question is not as easy as it looks. NOT an excuse to blurge out all known facts on sexual selection or natural selection! Need to keep argument focussed on the differences between the two. I can't see how this question could involve speciation, in spite of some students' answers. (Perhaps they felt that by including some general knowledge about evolution they could in some way obtain some marks!). Including some or all of the following would have gathered good marks:

a) Sexual selection is actually just a form of natural selection.
b) Natural selection is due to differential survival or reproduction, wheras sexual selection only acts on the probability of mating.
c) Sexual selection tends to act only on the sex that invests least and therefore has the most to gain from multiple matings – usually that is the male. Natural selection usually acts on both sexes.
d) Some kinds of sexual selection enhance traits that seem poorly adapted for survival, bright colours which might be a predation risk for example. Handicap? Or merely counteradaptive due to runaway sexual selection? Not clear. However, it is also possible to imagine similar tradeoffs between different kinds of natural selection unrelated to sex. For example, lifespan may be shortened to enhance current reproduction or fertility.
e) In natural selection, selection is due to the environment or other organisms, including members of one's own species. In sexual selection, itis due to a very specific type of other organism, members of one's own species due to fighting (within sexes) or indirect competition for mates (between sexes).
f) Intrasexual selection: fighting ability is most clearly like natural selection. Bigger, tougher "fitter" individuals win. Intersexual selection, due to female choice is more difficult to entertain as an example of ordinary natural selection, and many strange and wonderful theories (handicap, good genes, receiver bias, and runaway models, for example) have been erected.
g) Sexual selection acts only during mating; natural selection acts throughout the rest of the life cycle as well.

Section B
4) "Mimicry is a marvellous example of natural selection." Discuss with reference to natural examples.

Pretty straightforward non-tricky qestion. But must try to discuss why mimicry gives many examples of more general evolutionary processes such as frequency-dependent selection, parasitism, coevolution (or rather not!), genetic architecture under selection, epistasis, linkage disequilibria and so on. Also, the adaptation in mimicry is visible and easily appreciated, as opposed to physiological. Most people just splurged out their knowledge of mimicry without answering the question properly. See also mimicry lecture notes.

6) "No-one has ever witnessed speciation". Is this true? And what other evidence might we use to understand speciation?

Basically, the answer is no; the apple maggot was given in the Speciation lectures as an example. You could argue it isn't speciation, but if so, then this must be argued! Again, high marks were *not* awarded for blurging out all the author's knowledge of speciation while not answering the question!

Other evidence we might use is comparative method to understand speciation by seeing whether speciation is altered by key adaptations, for instance (see Coevolution lecture) or to study "species in the making", or species which still hybridize, and see what keeps them apart – very likely this will tell us what is involved in the early stages of speciation. Or what about looking in the fossil record? No-one seems to have thought about that!

Whether species can be identified to have occurred also depends on species concepts, but I didn't really want lots of discussion about concepts here, and not many marks were awarded for such.

7) Describe and discuss three ways in which genetic drift might be an important component of evolution in nature.

Many of the processes in which drift has been implicated are controversial (such as speciation via the founder effect, and in the shifting balance). I don't mind whose side the student takes, so long as they know something about what is supposed to occur, and maybe something about the pros and cons of the processes. Here are four more or less distinct examples of "ways in which genetic drift might be an important component of evolution in nature":

In causing extinction or severe fitness loss in rare species with small population size. (Genetic drift lecture)

In neutral molecular evolution, slow evolution in large population sizes over very large periods of time. (Molecular evolution lecture).

In the shifting balance mode of evolution. (Various places: chromosomal evolution, warning colour and mimicry, and speciation lectures).

In founder effect speciation. (Speciation lecture).

Section C
Question 9

			d-other/ d-other	d-141/ d-other	d-141/ d-141	Total	
	O		11	43	0	54	
a)	q(d-141)=	0.3981					*3 marks*
	p(d-other)=	0.6019					
	Sum(check)	1					
b)	Exp Genot freqs		0.3622	0.4793	0.1585		
	E		19.5602	25.8796	8.5602	54	*3 marks*
c)	X^2		3.7462	11.3258	8.5602	23.6322	total chi^2

Yes, there is strong evidence for a deviation from Hardy-Weinberg. Here, X^2=23.63, for 1 d.f. (subtract 1 d.f. from the 3 data cells for the total, and 1 d.f. because we are also estimating the frequency from the data itself, leaving a total of 1 d.f.). Thus from tables, P<0.001.

The most likely cause here is the very strong selection (?lethality) against the homozygote d-141/d-141. *5 marks*

d)

O/E	0.5624	1.6615	0.0000
Relative fitnesses, standardized	0.3385	1	0.0000
Selection coefficients	s=		t=
	0.6615		1.0000

see calculations above. There is strong selection against homozygotes d-141/d-141 estimated to be t=1.00. Since the fitness w = 1-t for these homozygotes, the estimated fitness is 0, or lethal. This is a fairly trivial result, since it obviously depends on the complete absence of the genotype in the sample, presumably due to early death. *3 marks*

Slightly more interesting is the strong estimated selection selection against $PMM2^{d\text{-}other}$ /$PMM2^{d\text{-}other}$ "homozygotes". It must be remembered that *all* the individuals in this sample are affected by this highly debilitating disease to some extent, and are homozygotes for defective PMM2 genes, even the "heterozygotes", and the d-other/d-other "homozygotes" are mostly not homozygotes for exactly the same defecive allele, since "other" includes 23 other alleles. Thus *on the face of it* this result implies that d-141/d-other "heterozygotes" are in some way fitter than either the d-other/d-other "homozygotes" or than d-141/d-141 genotypes. I might expect a few students at this stage to understand what is going on here.

I don't really expect many students to realize that the apparent hetorozygote advantage in this question is due to the difficulty of calculating the expected genotypes when there is very strong selection, unless they are really thinking!

Doing the calculation as above, the normal way, assumes that the frequency in our sample is the same as the gene frequency in the overall population from which the sample is drawn, which is clearly not true if some genotypes are lethal. Supposing all other PMM2$^{d-?}$ genotypes are equally fit (or rather, unfit), but that d-141/d-141 genotypes always caused spontaneous abortion so that they never produced sufferers, then we could calculate gene frequencies a different way, which assumes that the d-other/d-other : d-other/d-141 ratio is correct, but all d-141/d-141 genotypes disappear.

Using *p* and *q* as defined in (a) above, we expect

The d-other/d-other : d-other/d-141 ratio = p^2:2pq = p:2q
Thus p/2q = 11/43 = 0.2558, so p/q = p/(1-p) = 0.5116,

and p = 0.511628/(1+ 0.511628) = 0.3385
q = 1-p = 0.6615

This gives expected genotypic frequencies:

d-other/d-other: p^2 = 0.1146
d-other/d-141: 2pq = 0.4478
d-141/d-141 (before death) : 0.4376

Or after lethality of d-141/d-141 what is actually observed, i.e.:

d-other/d-other: 0.1146/(0.4478+0.1146) = 0.1146/0.4478 = 0.2037
d-other/d-141: 1- 0.2037 = 0.7963
d-141/d-141: 0

If this seems complicated, it isn't really. To understand what is going on students would have to think about the assumptions of the estimation of selection pressures via deviations from Hardy-Weinberg. Any evidence that this is going on will produce high marks on this section.
2 marks

e) i) Probably not, if they are recessive and still highly deleterious, as here. Clearly, ability to treat genetic diseases will ultimately reduce the strength of natural selection against such diseases, and therefore the frequencies of the diseases ought to rise. However, if the diseases are recessive, the response time is extremely slow.
3 marks

ii) One could point out to carriers or disease sufferers that their offspring are likely to be either carriers or sufferers themselves, and suggest that prenatal diagnosis be undertaken to avoid the gene being incorporated into their offspring. This might prove popular because people who would not necessarily be happy about suffering in order to ameliorate the genetic stock of the

entire human population would probably be happy to undergo invasive treatment (such as abortion) when it is the health of their own offspring that is at stake.

3 marks

iii) Inbreeding, provided it is not too close, is not fatal, and therefore the existing taboos against inbreeding are probably valuable in preventing some examples of recessive genetic disease. Whether a society prohibits or allows genetic disease is an ethical decision, and scientists can only advise about the physical consequences. Or at least, that is my view of the situation! Students may have other views, but they get higher marks if they argue their cases by showing that they are aware of the consequences of their decisions, and the differences between genetics and ethics. *3 marks*