1.

HARDY-WEINBERG CALCULATIONS

		A1A1	A1A2	A2A2	Total
O		35	19	3	57
p(A1)=	0.7807	=(35+19/2)/57			
p(A2)=	0.2193	=(3+19/2)/57			
Sum(check)	1				
Genotypic frequencies		0.6095	0.3424	0.0481	
E		34.7412	19.5175	2.7412	57
X^2		0.0019	0.0137	0.0244	0.0401

a) p(a2) = 0.2193, p(a1) = 0.7807 0.5 mark
b) expected numbers, chi-square, etc. see E above. 1 mark
c) 0.9>P>0.5 0.5 mark
d) no evidence for deviation from Hardy-Weinberg. The result is highly probable under the null hypothesis. 0.5 mark

If anyone rounded to whole numbers in the calculations of EXPECTED NUMBERS, subtract 0.5 marks. In the lecture and the lecture notes (look for "calculator notes") I tell them to store multiple decimal places in their calculator memories, and that Expected Numbers can be fractional: for example, what is the expected number of sixes I should get in 1 throw of the dice? The answer is 1/6, but it would be 0 if we rounded to a whole number.

2. a) a fraction of 10^{-4} newborns affected. Assuming random mating, this is the square of the frequency of the gene. The frequency must therefore be about 10^{-2}. So the frequency of heterozygous carriers should be about $2\times0.01\times0.99 \approx 0.02$. 1 mark

b) Assuming the equilibrium has been reached, $s = \mu/q^2 = 2\times10^{-6}/10^{-4} = 0.02$. No, this does not seem sensible, because in the model, s=1 implies 100% mortality (or lack of reproduction). The question said that phenylketonuria is a form of "severe" mental retardation, that is s~1. Something must be wrong if it is severe, perhaps the mutation rate is higher? Or perhaps it is due to genetic drift in European populations? Perhaps most likely, the high frequency of the gene is due to some heterozygous advantage we don't know about, as with sickle-cell anaemia among West Africans, which is also effectively a recessive lethal (s=1). 0.5 mark for correct calculation, 0.5 mark for sensible interpretation.

3. 2pq(1-F) = Het. So F = 1 – (Het/2pq), where Het is the fraction of heterozygotes. Het is in fact the Observed frequency, and 2pq is the Expected frequency of hets. You could also use the values of Obs/Exp heterozygote numbers for the fraction Het/2pq: top and bottom lines differ by a factor of the total, 57. From Problem 1:

$$F = 1 - \frac{19(\times57)}{19.5175(\times57)}$$
$$=1-0.9735 = 0.0265$$

1 mark total for rearranging the equation and calculating correctly; 0.5 off if they calculate incorrectly (but don't penalize them *again* if they rounded in q.1 so expected values = observed: they would get F=0!).

The inbreeding coefficient F can vary between 0 and 1; so 0.03 is not a lot of inbreeding. Since we know from (1) that it isn't significant anyway, this isn't surprising.

4 a) Frequency of gene = 0.05; therefore; under Hardy-Weinberg assumptions, the genotype frequency is $(1/20)^2 = (1/400) = 0.0025$ 0.5 mark

b) $\Delta p = spq^2/(1-sq^2)$ was the formula for rate of allele frequency change given in the lecture "selection and the single gene"(Find in page: "How fast"), and the approxmation for rare recessive genes was: $\Delta p = sq^2$. In the formula, it must be realized that the recessive allele is has frequency q, and the dominant allele is p, but unfortunately, I in part **d** I said "p=0.05" when I meant "q=0.05"! Some students realized, this, but many seemed flummoxed. In any case, we really want the change in the recessive allele frequencies, so the appropriate equation is: $\Delta q = -spq^2/(1-sq^2)$, because $\Delta q = -\Delta p$ (as explained in the question).. Suppose the gene is a recessive lethal, then s=1, and the "true value" of Δq=-0.002381 to 4 sig. figs. 0.5 marks

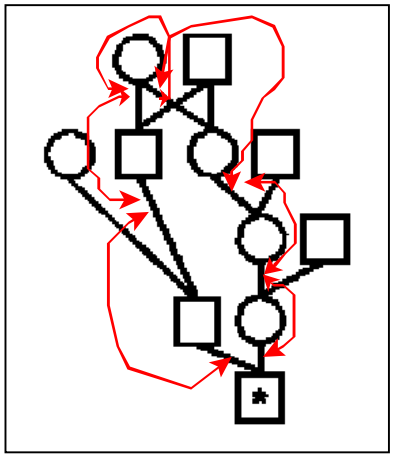
c) New gene frequency q= 0.0497619. New genotype frequency, $q^2 = 0.00227$. 0.5 marks

d) The appropriate approximation is: $\Delta q = -sq^2$ This gives: $\Delta q = -0.002500$. So 0.0024 to 0.0025, pretty similar to me! Yes, it is a good approximation. One could use any value of s to check this, of course, but the obvious value to use is 1, since this represents the factor change due to the approximation best, whatever the s. 0.5 mark

e) The major point of this question is to show students that draconian eugenic measures to prevent people with homozygous deleterious mutation from breeding are only going to have a very small effect. The gene frequency only changes at 5% of its value per generation. Not a great vote-getter, perhaps, since the elimination will take approximately 20 generations, 500 years, to have a major effect: a rather longer time than the wait till the next general election! One could perhaps argue that one should prevent heterozygotes from breeding as well: this would have a much more rapid elimination effect (proportional to q, not q^2, since by eliminating heterozygotes as well as homozygotes you effectively make the mutation dominant). This would be a good idea were it not for the fact that most of us have about one lethal or deleterious lethal equivalent somewhere in our genomes! So not a very popular choice for a government to make, I think.

So the answer is: probably not such a good idea. Too many ethical problems for too little gain. 0.5 marks

5a) See at right for arrows connecting the two gametes that formed me through my "grandmother+great grandmother". There are 6 links, and each link has probability ½; thus the overall probability of identity through "her" is $(½)^6$. The only other possible path of identity by descent is through my male ancestor of the same kind; again, this has probability $(½)^6$; so the overall probability of identity by descent is $2(½)^6 = 1/32$, or 0.03125. 1.5 marks



b) inbreeding this much isn't usually so bad. Darwin had plenty of inbreeding, and he bred with his first cousin Emma Wedgwood. 0.5 mark.