3F08

BIOL 2500 FALL 2008

EXAMINATION 3

NAME [redacted]

PLEASE MAKE SURE THAT YOU WRITE YOUR NAME ON THE TOP OF EVERY PAGE
THIS PAGE IS RESERVED FOR GRADING. THERE ARE 13 QUESTIONS AND 10 PAGES. YOU MAY USE THE REVERSE SIDE OF EACH PAGE AS SCRATCH PAPER. A GENETIC CODE TABLE IS PROVIDED IN THE BACK OF YOUR EXAM.

Page 2 10 (max 10) ✓

Page 3 15 (max 15) ✓

Page 4 6 (max 18) +6

Page 5 7 (max 9) +1

Page 6 6 (max 6) ✓

Page 7 10 (max 12) +1

Page 8 10 (max 12) +1

Page 9 10 (max 10) ✓

Page 10 5 (max 8) +1.5

TOTAL: 79 +10,5 ⇒ ✓ 89.5%

Name: A

10

1. The assignment of amino acids to particular codons was done by several techniques, including analysis of proteins synthesized using cell-free translation of RNA copolymers of random sequence and RNA copolymers of known sequence. (10 pts).

a. What amino acids would be expected and in what ratios if the copolymer translated were a repeating copolymer of the dinucleotide UC?

b. What amino acids would be expected and in what ratios if a mixed copolymer consisting of 25% uracil and 75% cytosine in random sequence were translated?

a. UCUCUCUC = Ser
UCUCUCUC = Leu

Serine and Leucine would be produced from the repeating copolymer. Since it is a repeating copolymer with equal ratios of U and C and since Serine and Leucine alternate coding (as in a chain of ~Ser-Leu-Ser-Leu~ where ~ are more amino acids before and after continuing the pattern to the N and C termini), and therefore would be equally likely to be present. So:

Serine: 50%
Leucine: 50%

b. 25% U / 75% C

1U : 3C

UUU - $\frac{1}{4} \times \frac{1}{4} \times \frac{1}{4} = \frac{1}{64}$ - Phe

UUC, UCU, CUU - $\frac{1}{4} \times \frac{1}{4} \times \frac{3}{4}$ - $\frac{3}{64}$ - Phe, $\frac{3}{64}$ - Ser, $\frac{3}{64}$ - Leu

UCC, CUC, CCU - $\frac{1}{4} \times \frac{3}{4} \times \frac{3}{4}$ - $\frac{9}{64}$ - Ser, $\frac{9}{64}$ - Leu, $\frac{9}{64}$ - Pro

CCC - $\frac{3}{4} \times \frac{3}{4} \times \frac{3}{4} = \frac{27}{64}$ - Pro

Proline: $\left(\frac{27}{64} + \frac{9}{64}\right) \times 100 = 56.25\%$
Leucine: $\left(\frac{9}{64} + \frac{3}{64}\right) \times 100 = 18.75\%$
Serine: $\left(\frac{9}{64} + \frac{3}{64}\right) \times 100 = 18.75\%$
Phenylalanine: $\left(\frac{3}{64} + \frac{1}{64}\right) \times 100 = 6.25\%$

Name: _____ 15

aa bb
aa Bb
aa BB
Aa bb
Aa Bb
Aa BB

AA bb
AA Bb
AA BB

2. In the wood lily, most plants have orange flowers and are true-breeding, but true-breeding red and yellow flowered varieties also exist. Crosses with true-breeding strains yielded the following results:

O x R → O x O → ¾ O, ¼ R
O x Y → O x O → ¾ O, ¼ Y
R x Y → O x O → 9/16 O, 4/16 Y, 3/16 R

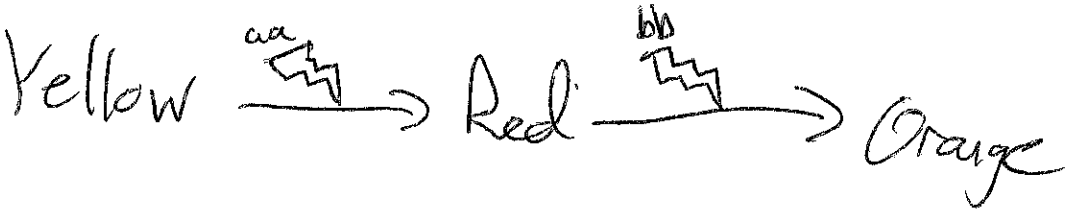
P_1	F_1	F_2
Orange x red	All orange	¾ orange, ¼ red
Orange x yellow	All orange	¾ orange, ¼ yellow
Red x yellow	All orange	9/16 orange, 4/16 yellow, 3/16 red

5

Yellow (bb) x Red (aa) → Orange

Propose a biochemical pathway that accounts for these results. (5 points)

If the colors are represented by the genotypes as follows – Orange A_ B_, yellow aa_ _, red A_ bb. Some gene A is required to create red, and both A and B are required for orange



3. Genetic mutants of the bread mold *Neurospora crassa* have been used to elucidate metabolic pathways, including the pathway for biosynthesis of the amino acid methionine.

a. Using the data provided in the table below, show the pathway for methionine biosynthesis, including all intermediates and the step blocked in each of the mutants. (8 pts). 10

Mutant	homocysteine	cysteine	cysteinethione	methioninine
met-1	-	-	-	+
met-2	+	-	+	+
met-3	+	+	+	+
met-4	+	-	-	+

	C	ct	h	M
1	–	–	–	+
4	–	–	+	+
2	–	+	+	+
3	+	+	+	+

precursor → met-3 → cysteine → met-2 → cystathionine → met-4 → homocysteine → met-1 → methionine

met-2 blocked in; met-4 blocked; met-1 blocked

b. What intermediate would accumulate in a *met-1, met-4* double mutant? (2 points)

Cystathionine would accumulate in this double mutant as met-4 mutant cannot make homocysteine from cystathionine, and therefore the met-1 mutation is not seen (=failure to make methionine from homocysteine).

Name: _____ 6

4a. MicroRNAs of the *let-7* family regulate *ras* expression. Explain how increased expression of *let-7* would affect expression of *ras* and the mechanism for this effect? (3 points)

Increased expression of let-7 would decrease expression of ras as microRNAs bind to mRNAs inhibiting them or targeting them for degradation. Therefore

3

b. How would increased expression of *let-7* mRNA affect a normal (non-cancer) cell? (3 points)

Increase expression of let-7 mRNA would allow a normal cell to continue through the cell-cycle without being destroyed by apoptosis.

c. Would you expect *let-7* miRNAs to be proto-oncogenes or tumor suppressor genes? Explain. (3 points)

I would expect let-7 mRNAs to be proto-oncogenes as they are involved in regulating ras expression which has to do with the apoptosis pathway.

5

5. Human papilloma virus (HPV) is present in > 90% of cervical cancers. HPV encodes two proteins, E6 and E7 that are potent contributors to its tumorigenicity. E7 is known to disable pRB (the product of the *RB1* gene); E6 binds to p53 and targets it for degradation.

a. Explain how these two HPV proteins might contribute to cancer development in HPV-infected cells. (6 points)

3

p53 is typically degraded in a normal cell, if it is not, it is due to a defect mutation, and leads to pathways that stop or halt the cell cycle by way of inhibiting cyclin D1 expression. If E6 is able to always degrade p53, it will never stop the cell cycle leading to uncontrolled growth (tumor formation) and likely increased mutation rates as the repair machinery cannot keep up. pRB also has a role in cancer in that when the protein is over-expressed it leads to the development of cancer. Therefore, if E7 is unable to disable the product of pRB, development of cancer would occur.

b. Do you think that siRNAs that target the E6 and E7 genes would be an effective therapy for prevention of cervical cancer in infected women? Why or why not. (3 points)

0

siRNAs inhibits their RNAs (and often destroy them) that code for gene products. It is clear that to inhibit them would be detrimental as E6 is required for normal function and growth of a cell. One would not want a cell meant to grow to remain in arrest forever.

viral protein?

Name: ✓ 7

6. Oocytes from the African clawed frog *Xenopus laevis* can be directly injected with mRNAs. These mRNAs can be translated into proteins in the Xenopus occyte cytoplasm. A microbiologist wants to use Xenopus oocytes to express *E. coli* protein X. Prior to injecting the protein X mRNA into the *Xenopu* soocyte, the microbiologist has ensured that his mRNA preparation has an intact Shine-Dalgarno sequence upstream of the coding region. However after numerous attempts, none of the injected oocytes expressed any protein X. Explain the reason for these negative data and suggest an alternative approach that would alleviate the problem. (4 points)

It is likely that the mRNA does not contain the proper promoter sequence meaning that the Xenopus oocytes translation machinery cannot properly recognize the sequence and convert it into a functioning protein. One should engineer the mRNA to contain Xenopu promoter and initiation sequences attached to the desired E coli gene.

7. In a microarray experiment, mRNA from a strain (Strain 1) of antibiotic resistant *E. coli* was used as a template for cDNA synthesis with red fluorescent nucleotides. The mRNA from a non-antibiotic resistant bacterial strain (Strain 2) was used as a template for cDNA synthesis using green fluorescent nucleotides. The cDNAs from the resistant and non-resistant cells were mixed and hybridized to a chip containing spots of DNA from *E. coli* genes 1 through 25. The results are shown below (N = no color; R = red; G = green; Y = yellow).

implies transcription is happening...

N 1	R 2	Y 3	G 4	Y 5
Y 6	Y 7	Y 8	Y 9	Y 10
Y 11	Y 12	N 13	N 14	Y 15
Y 16	G 17	Y 18	Y 19	Y 20
N 21	G 22	Y 23	R 24	Y 25

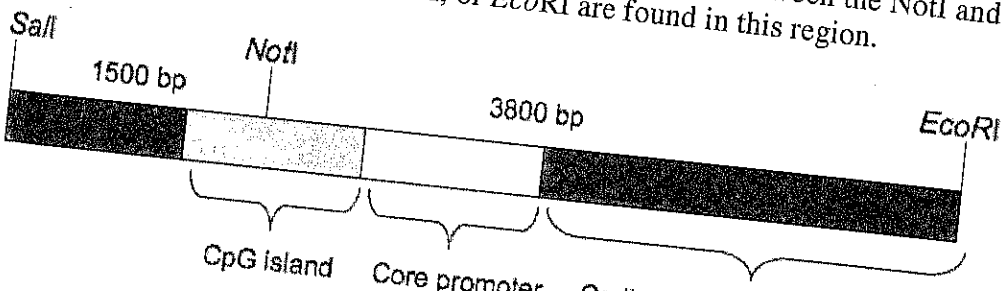
Which genes do you think are most likely to contribute to the antibiotic resistance? Explain (5 points).

5

Genes 2, 17, 22, and 24 are likely involved. In the antibiotic resistant E. coli, compared to the non-resistant, genes 2 and 24 are upregulated whereas genes 17 and 22 are downregulated. ✓

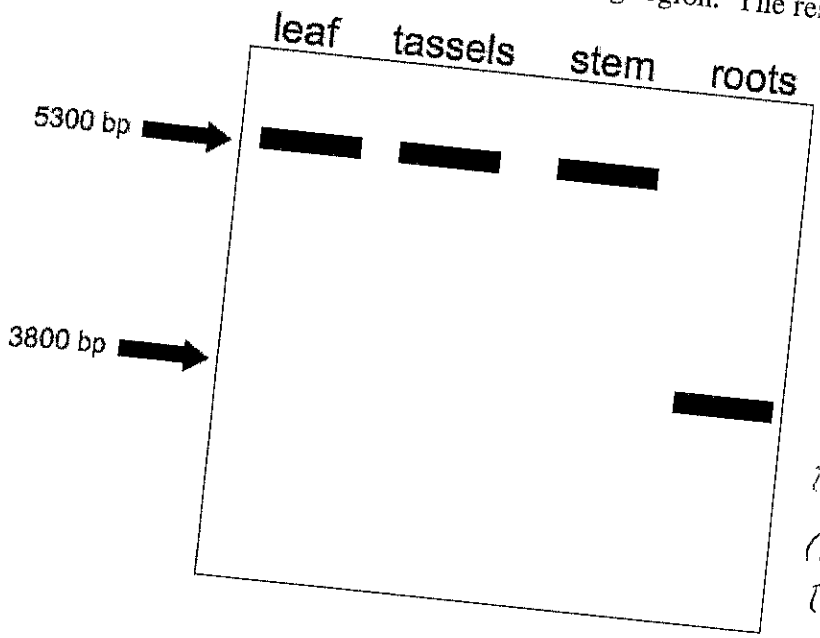
Name: _____ 6

8. The restriction enzyme *NotI* has the recognition sequence 5'-GCGGCCGC-3'. However, if the cytosines have been methylated, NotI is unable to cleave the DNA. A researcher is studying a gene, *T*, found in corn which encodes a transporter involved in uptake of phosphate from the soil. A CpG island is located near the core promoter for gene *T*, and there is a single *NotI* recognition site within the CpG island. A *SalI* restriction site is located upstream of the CpG island, and an *Eco*RI site is located near the end of the coding region for gene T. The distance between the *SalI* and *NotI* sites is 1500 bp, and the distance between the NotI and EcoRI sites is 3800 bp. No other sites for *SalI*, *NotI*, or *Eco*RI are found in this region.



5300bp total

A researcher has isolated DNA samples from four different corn plant tissues: the leaf, the tassel, the stem, and the roots. The DNA was digested with all three restriction enzymes (*SalI*, *NotI*, and *Eco*RI), separated by agarose gel electrophorese, and analyzed by Southern blotting using a probe complementary to the *T* coding region. The results are shown below:



In which tissue(s) is the CpG island methylated? Does this make sense based on the function of the protein encoded by gene *T*? (6 points).

The CpG Island is methylated in the leaf, tassels, and stem as these DNA from these sites is 5300bp, meaning NotI could not cleave the site in the CpG Island meaning it was methylated. This makes sense because methylation silences genes and one would not need a phosphate uptake mechanism in leaves, stems, or tassels - just in the roots.

Name: _____

10

9. What advantage(s) does the whole-genome shotgun sequencing approach have compared with map-based clone-by-clone sequencing? When would a map-based approach be necessary? (4 points)

A whole genome Shotgun Sequencing approach is advantageous as it is more cost effective and less time consuming. With it, are restricts many fragments of DNA and then sequences the small fragments. Then, the similar sequences are compared and aligned to rebuild the genome. A map-based approach requiring many small restrictions and the placement of these into smaller and smaller vectors is time consuming, tedious, and expensive. However, it is necessary if the Shotgun approach yields a gap in the sequence that can't be resolved (as the Shotgun method requires overlapping sequences).

4

10. The following mutations were discovered in the *C. elegans let-60* gene encoding a ras homolog. The gene has 4 exons and encodes a protein of 184 amino acids. Indicate whether each mutation would cause a detectable change in the size or amount of mRNA and/or a change in the size or amount of the protein product. (a detectable size difference would be greater than 1% of normal). (2 points each).

6

explain size and amount mRNA / protein

a. promoter mutation. A promoter mutation would not allow transcription of mRNA so yes, there would be a detectable change, namely, no mRNA or protein.

b. deletion of the poly-A addition site.

0

c. Gly136Ser (changes amino acid 136 from glycine to serine) The mRNA size and amount would be the same and so would the protein product so no detectable change would be seen (however the protein may not function properly).

d. codon 43 changed from GAG to UAG.

GAG is Glutamate and UAG is a stop codon so the mRNA would be the same length but the protein product would be significantly shorter.

2

Name: _____ A-T G-C

11. The following nucleotide sequence is found on the template strand of DNA: 10

5'-TCA ATA TAG TTG ATT GCC GGT CAT-3'
3'-AGU UAU AUC AAC UAA CGG CCA GUA-5'

a) Give the sequence of the mRNA that would be transcribed from this template DNA. (2 points)

3'-AGU UAU AUC AAC UAA CGG CCA GUA-5' 2

b) Give the sequence of the polypeptide that would be translated from the mRNA. (2 points) 10.

Met-Thr-Gly-Asn-Gln-Leu-Tyr-Stop 2

c) Give the altered polypeptide sequence that would result from each of the following mutations (2 points each):

A transition at nucleotide 11: Met-Thr-Gly-Asn-Arg-Leu-Tyr-Stop 2

A transition at nucleotide 13. Met-Thr-Gly-Asn-Gln-Leu-Tyr-Stop 2

A deletion of nucleotide 7. Met-Thr-Gly-Asn-Gln-Leu-Ile (??) 1
(Here two are only 2 AAC codons before the new stop and no coding region could be Asp or Gln with one nucleotide)

A transversion at nucleotide 15. Met-Thr-Gly-His-Gln-Leu-Tyr-Stop 1

OR...

TCA ATA T AG TCG ATT GCC GGT CAT
AGU UAU A UC AGC UAA CGG CCA GUA

TCA ATA T AG TTG GTT GCC GGT CAT
CAA
Asn

TCA ATA AG TGA TTG CCG GTC AT
AGU UAU UC ACU AAC GGC CAG UA

TCA ATA TAG TTG ATG GCC GGT CAT
UAC
GOA

Name: _____ 10

12. The *mmm* operon, consists of sequences *A*, *B*, *C*, and *D*, and encodes enzymes 1 and 2. Mutations in A, B, C, and D have the following effects, where a (+) indicates that the enzyme was synthesized and a (-) indicates that no enzyme was synthesized.

Mutation in sequence	mmm absent		mmm present	
	Enzyme 1	Enzyme 2	Enzyme 1	Enzyme 2
none	+	+	-	-
A	-	+	-	-
B	+	+	+	+
C	+	-	-	-
D	-	-	-	-

a. Is the *mmm* operon inducible or repressible? (2 points) Repressible 2

b. Indicate which of the sequences corresponds to the each component of the operon (2 points each):

Regulator gene: B

Promoter: D

Structural gene for Enzyme 1 A 8

Structural gene for Enzyme 2 C

Name: __

13. A bacterial strain carries a null mutation in the gene encoding adenyl cyclase, rendering the enzyme completely non-functional. All other genes in this bacterial strain are wild-type. Fill in the table with a YES to or NO to indicate whether ß-galactosidase would be expressed, and briefly explain your reasoning. (8 points).

YES: *lac* operon expressed
NO: *lac* operon not expressed

5

Glucose Present in Medium	Lactose Present in Medium	ß-galactosidase expression?	Reason
No	No	No ✓	There is no lactose to bind to the regulator which would activate expression. ✓
Yes	No	No ✓	Glucose is a preferred substrate and there is no lactose to bind to the regulator causing it to detach. ✓
Yes	Yes	No ✓	Glucose is present. so even though lactose can bind to the regulator ATP is not produced in glycolysis from glucose. This activates enter protein which inhibits the expression of β-galactosidase.
No	Yes	Yes	There is no glucose meaning must be preferred substrate for glycolysis (glucose), but there is lactose. The lactose induces expression of the lac gene when lactose deactivates the regulator causing transcription occurs to breakdown lactose for energy.

First Position (5′ end)	Second Position: U	Second Position: C	Second Position: A	Second Position: G	Third Position (3′ end)
U	UUU Phe UUC UUA Leu UUG	UCU UCC UCA Ser UCG	UAU Tyr UAC UAA Stop UAG Stop	UGU Cys UGC UGA Stop UGG Trp	U C A G
C	CUU CUC CUA Leu CUG	CCU CCC CCA Pro CCG	CAU His CAC CAA Gln CAG	CGU CGC CGA Arg CGG	U C A G
A	AUU AUC Ile AUA AUG Met	ACU ACC ACA Thr ACG	AAU Asn AAC AAA Lys AAG	AGU Ser AGC AGA Arg AGG	U C A G
G	GUU GUC GUA Val GUG	GCU GCC GCA Ala GCG	GAU Asp GAC GAA Glu GAG	GGU GGC GGA Gly GGG	U C A G

Optional:

This test was _____ too short ___X___ too long _____ about right.

This test was _____ too easy _____ too hard ___X___ about right.

[redacted]

Genetics Test 3 Corrections Problem 4

4. I previously erred on this problem because I did not remember the role of *ras* in the cell and how it related to both cancer and methods of regulation. Without this information, it was difficult to answer the questions asked. Additionally, I did not quite understand the subtle difference in proto-oncogenes and tumor suppressors as was needed for part c of this question.

   a. Increased expression of *let-7* would decrease expression of *ras* as microRNAs bind to mRNAs inhibiting them or targeting them for degredation. This is achieved through the RISC pathway, and the gene may even be silenced by the RITS pathway.

   b. Increased expression of *let-7* mRNA would inhibit normal cell cycle processes as *ras* expression would be inhibited. *Ras* is essential in responding to external growth factors and transmitting these signals through protein phosphorylation cascades to the nucleus where transcription of essential gene products takes place – ultimately pushing the cell out of quiescence.

   c. I would expect *let-7* miRNAs to be tumor-suppressors as they regulate *ras* which is a proto-oncogene. This is because the effect of *let-7* product is to prevent the cell from continuing with the cell cycle, a clear role of tumor-suppressor genes. Therefore, it would be likely that if the cell were damaged or mutated, the expression of *let-7* would make the cell insensitive to growth factors and therefore halt the cell cycle, thereby preventing tumor formation.

[redacted]

Genetics Test 3 Corrections Problem 5

5. I previously erred on this question because I did not remember the exact effect of pRB and p53 in relation to cancer and therefore, was unable to properly explain how E6 and E7 would affect a normal cell. Additionally, I misread the original question and answered part b completely incorrectly because for some reason I thought that E6 and E7 were alteration of normal gene products rather than the results of HPV. Additionally, I was vague as to the exact role of siRNAs (especially the difference between them and miRNAs).
    a. The product of the *RB1* gene, pRB, is an essential tumor suppressor in cells that controls the G1/S cell-cycle checkpoint. pRB is always present in the nucleus of cells and when it is inactive, and non-phosphorylated, binds to transcription factors such as E2F, essentially inactivating them. When it is phosphorylated by the CDK4/cyclin D1 complex, it releases these transcription factors allowing the cell to progress in the cell cycle. Then, after this is accomplished, pRB is dephosphorylated and rebinds transcription factors. Therefore, if E7 disables pRB the protein will most likely be unable to bind transcription factors, meaning that they would be free to bind to DNA and promote transition through the cell cycle. Thus, E7 would prevent regulation of this checkpoint of the cell cycle and potentially allow the cell to become cancerous. The tumor suppressor p53 is normally rapidly degraded and present in cells at low levels. Additionally, it is normally bound to Mdm2, tagging p53 for degradation as well as sequestering the transcriptional activation domain of p53. Additionally, it prevents phosphorylation and acetylation of p53 that allow it to become active. When DNA is damaged, p53 is activated leading to the arrest of the cell cycle and then DNA repair, or apoptosis and cell death if it cannot be repaired. If E6 always binds to p53 and always targets it for degradation, then the cell can no longer halt the cell cycle or initiate apoptosis meaning that the cell will remain damaged if it becomes damaged and most likely accumulate more and more mutations as the cell can never slow down to repair the damage nor may it commit suicide. This would promote the development of cancer.

+3 pts for 2 (+1.5)

    b. It is possible that siRNAs could be an effective treatment for the prevention of cervical cancer in women as they would act through the RISC pathway and inhibit the expression of E6 and E7 by sequestering the mRNA that codes for them and targeting it for degradation. However, it is unlikely that it would be an effective treatment as it would be difficult to have the siRNAs in every single cell that could mutate to become cancerous (as it only takes one unregulated cell to form a tumor and possibly spread cancer throughout a patient's body) and it is likely that over time, the siRNAs would mutate or degrade rendering them useless.

+3 pts for 2 (+1.5)

- Therefore, the time and expense of the procedure would seem to make it unreliable enough to not be utilized on a large scale.

[redacted]

Genetics Test 3 Corrections Problem 6

6. I previously erred on this problem for two reasons. Firstly, for some reason I believed that transcription was occurring rather than just translation so I discussed the "promoter" sequence, and secondly, I did not recognize the difference between eukaryotic and prokaryotic translation of proteins.

   There are several reason why the *Xenopus* oocyte would not translate the *E. coli* mRNA as the first is eukaryotic whereas the second is prokaryotic and therefore, they each contain different recognition machinery (and in fact may have different amino acids coded for the same codon, but since this is generally highly conserved, I will assume that this is not a large issue, at least in terms of the inability to express the protein). One major problem is that although the *E. coli* mRNA by its nature is compact and does not contain introns, only what one might consider "exons" even though this term would not properly apply, the Shine-Dalgarmo sequence that was added to the mRNA is not recognized by eukaryotes. Eukaryotes also have a recognition sequence that surrounds the AUG start codon – the Kozak sequence. Therefore, it would be necessary to add this sequence over the Shine-Dalgarmo sequence that is utilized by prokaryotes. Additionally, eukaryotic DNA has a 7-methylguanosine residue present at maturation that is absent in prokaryotes but essential for efficient translation in eukaryotes as those lacking this 7-mG cap are poorly translated. Additionally, it is possible that the oocyte contains miRNAs or siRNAs that target foreign RNA sequences for degradation, especially if the sequence appears prokaryotic rather than eukaryotic. Therefore, in order to obtain gene product, all of these errors would need to be corrected.

+2 pts / 2 = (+1)

[redacted]

Genetics Test 3 Corrections Problem 10

10. I previously erred on this problem because I did not recall what a poly-A addition site was and therefore did not answer part b of the problem.
    a. A mutation of the promoter of *let-60* gene would not allow the expression of the gene whatsoever as the transcription complex would not be able to recognize and bind to the promoter regions disallowing any mRNA creation and therefore no protein would be created either.
    b. Deletion of the poly-A addition site leads to rapid degradation of the transcribed mRNA and therefore one could say that the RNA is transcribed and would be about the same size (somewhat shorter without the poly-A tail), but due to the lack of the tail and subsequent degradation, there would be much less mRNA and no protein would be created.
    c. A mutation which changes amino acid 136 from glycine to serine would not affect the size of the mRNA nor the amount, and it also would not produce a detectable change in the linear size of the protein nor the amount. However, it is possible that the function would be changed in the protein and due to the serine, it is possible to have more hydrogen boding or addition of acetyl, phosphoryl or other groups so as to change the overall folded size and shape of the protein.
    d. If codon 43 were change from GAG to UAG the size and amount of mRNA would not be affected. However, the size of the protein would be affected as GAG encodes glutamate and UAG is a stop codon, thereby making the prtein significantly shorter (only having about 40 amino acids as opposed to 184 amino acids.

+2 pts = (+1)

[redacted]

Genetics Test 3 Corrections Problem 11

11. I previously erred on this question because I was not sure of what would happen with a deletion if due to it there were only 2 amino acids encoding the final portion of the polypeptide which did not code for a stop codon. Additionally, I was not entirely sure of what happened with a transversion and therefore did not give a complete answer for that problem.
    a. Since DNA codes for an antiparallel mRNA with T coding for A, C for G, A for U, and G for C, the transcript would be as follows:
    3'-AGU UAU AUC AAC UAA CGG CCA GUA-5'
    b. Because mRNA is translated from 5' to 3', reading the above transcript that way, one finds that the polypeptide sequence is as follows, with N representing the N-terminus of the protein (with the C-terminus on the other end but not denoted so as to avoid confusion with the denoted STOP):

N-Methionine-Threonine-Glycine-Asparagine-Glutamine-Leucine-Tyrosine-STOP

    c. The alteration answers are as follows:
        i. A transition is where a pyrimidine replaces a pyrimidine or a purine replaces a purine, and therefore, in this case, since nucleotide 11 is a T for DNA and an A for RNA, this is converted to an C in DNA and a G in RNA because both T and C are pyrimidines. This changes codon 5 of the mRNA making amino acid 5 change to an Arginine rather than a Glutamine, making a missense mutation. It appears as follows:

N-Methionine-Threonine-Glycine-Asparagine-Arginine-Leucine-Tyrosine-STOP

        ii. A transition at nucleotide 13 has a similar effect as before, simply changing a single purine for a purine, or pyrimidine for a pyrimidine. In this case, it is a purine rather than a pyrimidine, so the A of DNA (and thus T of mRNA) is changed to a G in DNA (and a C in mRNA), thus ultimately having no effect on the amino acid composition – a silent mutation that appears as follows:

N-Methionine-Threonine-Glycine-Asparagine-Glutamine-Leucine-Tyrosine-STOP

        iii. A deletion at nucleotide 7 not only shortens the mRNA transcript, it alters the order of nucleotides and thus alters the protein. In this case, it disallows a STOP codon at the end as well. The translated polypeptide is the same until what should be amino acid 7 which changes from tyrosine

to an isoleucine. Additionally, the remaining DNA is a 5'-TC which codes for 3'-AG. GA is the beginning of the codons that translate into aspartate and glutamate. Therefore, the transcripts would be as follows based on the fact that the last amino acid is not as important as the first two and therefore, tRNA may still bind and recognize just 2 codons:

N-Methionine-Threonine-Glycine-Asparagine-Glutamine-Leucine-Isoleucine-Aspartate ✓

N-Methionine-Threonine-Glycine-Asparagine-Glutamine-Leucine-Isoleucine-Glutamate

iv. A transversion is when a purine replaces a pyrimidine or a pyrimidine replaces a purine. Therefore, a transversion at nucleotide 15 which is a T (a pyrimidine) would be replaced by either purine A or G. If replaced by G, the mRNA codes for a C and the altered polypeptide is different in amino acid four because CAU codes for histidine (as opposed to the original AAU coding for Asparagine. If the T becomes an A, the mRNA would have a U making the codon UAU coding for Tyrosine at that position. The polypeptides are as follows:

N-Methionine-Threonine-Glycine-Histidine-Glutamine-Leucine-Tyrosine-STOP

N-Methionine-Threonine-Glycine-Tyrosine-Glutamine-Leucine-Tyrosine-STOP

$\frac{+2 \text{ pts}}{2} = (+1)$

[redacted]

Genetics Test 3 Corrections Problem 13

13. I previously erred on this problem because I did not completely understand what would happen if lactose was present in the medium. Mainly, I did not understand why there would not be beta-galactosidase expression in this example.

Glucose Present in Medium	Lactose Present in Medium	Beta-galactosidase expression?
No	No	No
Yes	No	No
Yes	Yes	No
No	Yes	No

a. *Reason for no expression of Beta-galactosidase when glucose and lactose are not present:*
In this example there would be no expression of Beta-galactosidase because there is no lactose to bind to the operator, and therefore transcription of the gene coding for Beta-galactosidase does not occur.

b. *Reason for no expression of Beta-galactosidase when glucose is present and lactose is not present:*
In this example, there is no expression because glucose is the preferred substrate for glycolysis as well as the fact that there is no lactose to bind to the operator which, without this binding, prevents transcription of the genes encoding Beta-galactosidase mRNA.

c. *Reason for no expression of Beta-galactosidase when glucose and lactose are present:*
In this example there is no expression because even though lactose is present (thereby allowing the detachment of the operator), the CAP-cAMP complex which allows greater expression is not bound due to the failure of adenyl cyclase to make AMP. However, if the failure was not in place, the same effect would be noted as glucose is present and this mechanism is how the operon may be deactivated when both substrates are present as there is no point in utilizing lactose and wasting energy converting it to a usable form when glucose is present.

d. *Reason for no expression of Beta-galactosidase when glucose is not present and lactose is present:*
In this example, normally Beta-galactosidase would be expressed because lactose binds to the operator allowing the RNA polymerase to bind, and galactose is absent meaning that there would be a cAMP cascade allowed by adenyl cyclase which converts ATP to cAMP which would then bind to catabolite-activating

protein to form the CAP-cAMP complex which would bind to the promoter region and allow transcription to occur. In this example, since adenyl cyclase is faulty, there would not be the cAMP cascade and therefore the complex would not be form meaning that the RNA polymerase would seldom bind and there would not be beta-galactosidase expression.

✓

$\frac{+3 \text{ pts}}{2} =$ +1.5