MEGA Solution Series

Volume IV

Biotechnology Biochemistry Microbiology

covers following

- **1000 Analytical Problems and Solutions in Biology**

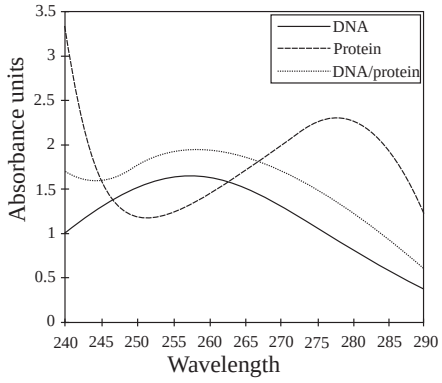
Editor
Kar Debasish

CHAPTER

01

Analytical Problems and Solutions Life Science-01

1. Following figure shows absorbance profiles of DNA and protein samples. The absorbance of purified (.....) plasmid DNA (80 mg/ml); (.....) 3 mg/ml aqueous bovine serum albumin (BSA) solution; or a (.....) 10:1 (w/w) DNA to protein mixture in aqueous solution was determined in 1 nm increments from 240 nm to 290 nm. Also in an another experimental setup, BSA has an extinction coefficient value of 0.7 for a 1 mg/ml solution at 280 nm, while streptavidin, on the other hand, with an extinction coefficient of 3.4 absorbs almost five times as much light at 280 nm at the same concentration. Wavelengths are measured using a Perkin Elmer Lambda 3B spectrophotometer. Which of the following statements is true for this setup of experiment?



A. A260 : A280 ratio is 0.6 and signifies DNA : RNA concentration

B. A260 : A280 ratio is 0.6 and signifies RNA : DNA concentration

C. A protein with a very high content of amino acids with aromatic side chains would in turn have a higher extinction coefficient than a protein with very few

(*a*) A and C (*b*) B and C

(*c*) Only A (*d*) Only C

2. Human hexokinase amino-acid sequence begins with Met-Trp-Lys-Trp-Trp-Met. But the protein made from your plasmid begins with Met-Trp-Met-Trp-Trp-Met. A mutation must have occurred. The RNA sequence was 5′- AUG-UGG-AAG-UGG-UGG-AUG 3′

There is only one codon for Met: AUG and two possible codons for Lys: AAA and AAG. Trp is encoded by UGG. Perhaps you could salvage your recombinant protein by treating it with a mutagen in order to get a reversion mutation that restores the correct amino-acid sequence. Following are mutagens and their effects on the nucleic acid.

1. Ethidium bromide (makes one-base insertions or deletions)
2. Aminopurine (causes A → G substitutions)
3. Imidazolecarboxamide (causes A → T substitutions)

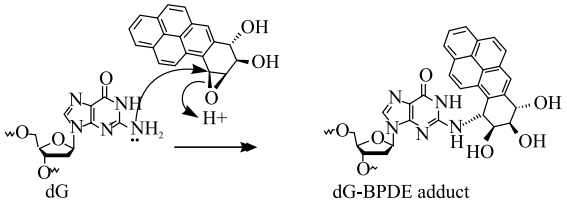
Which one of following could be the strategy that can be used for reversion of mutation?

(*a*) 1 and 2 (*b*) 2 and 3

(*c*) Only 2 (*d*) Only 3

3. Benzo[a]pyrene (BaP) is a widespread potent carcinogen found in food, coal tar, cigarette smoke, and industrial smoke. Benzo[*a*]pyrene is biologically activated to a dihydrodial epoxide during normal metabolic processes. The activated (+)benzo[*a*] pyrene-7, 8-dihydrodiol-9,10-epoxide can form a DNA adduct with guanine residues. If left unrepaired, during DNA replication an adenine will usually be placed across from the lesion in the daughter molecule.

There is no other described repair pathway for bulky O^6-alkylguanine lesions in humans. The production of adduct is shown below.



Which of the following verdict is true about the above DNA damage?

(*a*) Subsequent repair of the adduct will result in the replacement of the damaged guanine base with a thymine, causing a G → T transversion mutation.

(*b*) Nonhomologous end joining (NHEJ)

(*c*) Homologous recombination repair (HRR)

(*d*) None of the above

4. A primer was designed in order to clone a DNA fragment from the genome of the organism. It was a responsibility of the student to order a primer. She designed it and placed the order. Her lab mate asked her whether she calculated the expected Tm for the sequence. She quickly calculated it and responded. How much do you think she has got the Tm value for the following DNA sequence?

CTCTATCAGCTCTGTACCG

(*a*) 64.5 (*b*) 64.1

(*c*) 64.9 (*d*) 64.0

5. Thermal denaturation studies of DNA have revealed that the melting temperature, T_m of a DNA double helix depends on strand length, strand concentration, base sequence and ionic strength of added salt. Such studies indicate that double helix stability can be predicted in terms of the standard free energy change, $\Delta G° = \Delta H° - T\Delta S°$, if one knows the standard enthalpy and entropy changes ($\Delta H°$ and $\Delta S°$) for the melting of each nearest-neighbor doublet of base pairs in DNA. The melting temperature (T_m) for a particular DNA was found to be 52°C and enthalpy measured at this T_m was 0.039 kJ. The entropy change would be:

(*a*) 0.0012KJ (*b*) 0.00012KJ

(*c*) 0.0012J (*d*) 0.00012J

6. Blood and Saliva samples were collected from 20 non-related volunteers after informed consent. A total of 5 ml of blood was collected using sterile syringes and stored in sterile Ethylenediamine tetraacetic acid (EDTA) vials till further use. Saliva was collected by asking the subjects to spit in a sterile disposable Petri dish, 2 ml of this saliva was transferred from Petri dish to sterile vials, using a sterile pipette, and stored at − 20°C until further use. DNA extraction from blood and saliva was carried out by salting out method using the phenol-chloroform. DNA was quantified by measuring the optical density (OD) at 260 nm. 5 μl of stock genomic DNA was taken and 995 μl of water was added (Dilution factor = 200). Average OD at 260 nm for blood and saliva was found to be 1.8 and 1.1 resp. Which of the two samples do you think have yielded more amount of DNA for the further testing experiments?

(*a*) Blood

(*b*) Saliva

(*c*) Similar amount in both samples

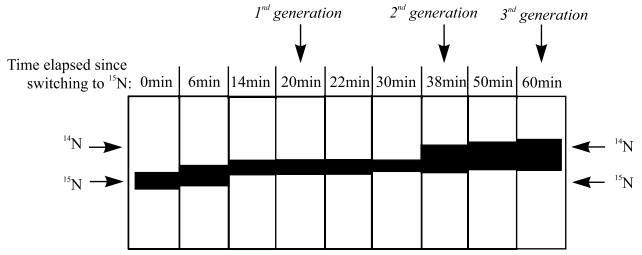
(*d*) None of the above

7. You have isolated a plasmid from a bacterial strain which you further want to transform into mycobacterium for your experimental setup. If you have a 40ng of that plasmid and its size is 5.2 Kb, How many copies of plasmids do you have?

(*a*) 5.2×10^9 (*b*) 4×10^9

(*c*) 5×10^9 (*d*) 7×10^9

8. A large batch of *E. coli* bacteria was grown in heavy nitrogen (^{15}N) and then the bacteria was switched to a media that contained only regular nitrogen (^{14}N). They were grown for 3 generations. *E. coli* DNA over multiple generations was isolated and was subjected to density gradient centrifugation. *E. coli* grown in ^{15}N DNA were switched to ^{14}N and then harvested at nine different time points. The DNA was centrifuged resulting in the banding pattern shown here.

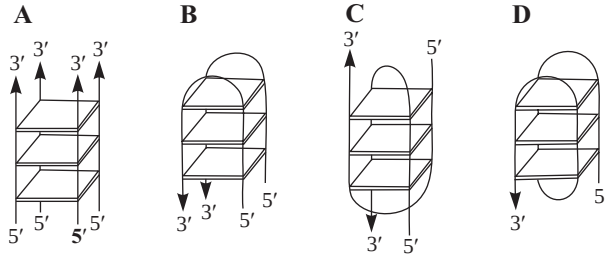


Which mode of DNA replication is shown in this figure?

(*a*) Conservative (*b*) semi-conservative

(*c*) dispersive (*d*) cannot say

9. The DNA strands are held together by pairing the Watson-Crick edge of each guanine with the Hoogsteen edge of an adjacent guanine, creating a cyclic arrangement of four guanines into G-tetrads. G-quartets can be formed from the association of one, two, or four G-rich DNA strands with various topologies. Topologies constructed from four parallel strands. The precise length of each telomere controls the cell's ability to replicate, suggesting a regulatory role for their G-quadruplex structures. Which of the following is possible topology for G-quartet structures?



(*a*) Only (A) (*b*) (A) and (B)

(*c*) Only (C) (*d*) All of the above

Which of the statements are true about the above experimental data?

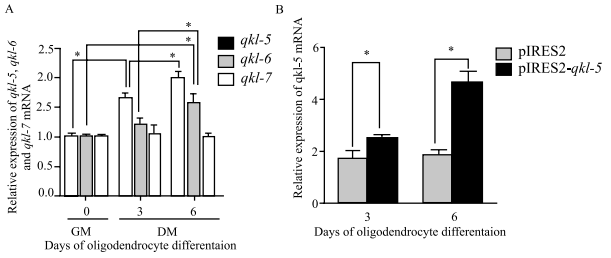
(*a*) IFN-γ–mediated p19 mRNA inhibition may be regulated at the posttranscriptional level

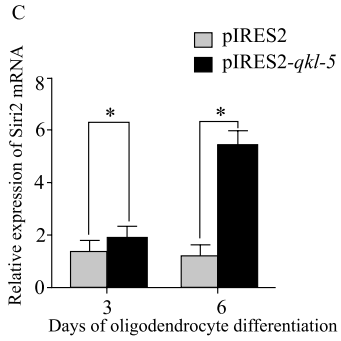
(*b*) IFN-γ–mediated p19 mRNA inhibition may be regulated at the transcriptional level

(*c*) IFN-γ–mediated p19 mRNA inhibition may be regulated at the posttranslational level

(*d*) IFN-γ–mediated p19 mRNA inhibition may be regulated at the translational level

46. Following figure A shows expression profiles of different qkI variants during differentiation shows qkI-5 mRNA levels. To further evaluate the role of QKI-5 in regulating Sirt2 expression, CG4-OL cells were transfected with a pIRES2 vector containing a qkI-5 cDNA under differentiation conditions. Transfection with pIRES2-qkI-5 changed expression of qkI-5 mRNA (*b*) Results of over expression of qkI-5 (*c*)shows differentiation on days 3 and 6 after expression of SIRT2 protein





Which of the following can be interpreted from above data?

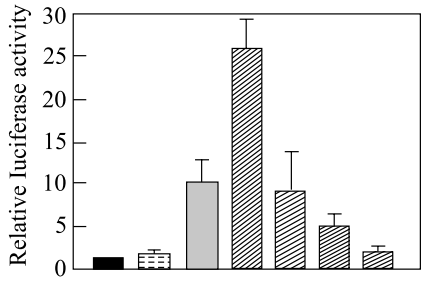
(*a*) Overexpression of qkI-5 promotes the expression of Sirt2 mRNA but not SIRT2 protein

(*b*) Overexpression of qkI-5 inhibits the expression of Sirt2 mRNA but not SIRT2 protein

(*c*) underexpression of qkI-5 promotes the expression of Sirt2 mRNA but not SIRT2 protein

(*d*) underexpression of qkI-5 inhibits the expression of Sirt2 mRNA but not SIRT2 protein

47. M12 cells were transiently cotransfected with pGL3-VE/1–4GG alone (black bar), 2 mg pCATCH-Bob-1/OCA-B (dotted bar), or pCG-Oct2RR alone (gray bar), or in combination with either 6 mg of pCG-Oct2RR or various alanine mutants of Oct-2RR (hatched bars). Enhancer reporter activities were measured 23 hr after electroporation in the presence of 10 mg/ml LPS.



pGL3-VE/1-4GG	+	+	+	+	+	+	+
OCA-B		+		+	+	+	+
Oct-2^{RR}			+	+			
Oct-2RR-T302A					+		
Oct-2RR-S303A						+	
Oct-2RR-T302A/S303A							+

Coexpression of Oct-2RR with OCA-B stimulated the reporter activity by more than 25-fold Equal amounts of total nuclear proteins, as compared to that with OCA-B alone.

Which of the following statement is true?

(*a*) OCA-B coactivates with Oct-2

(*b*) OCA-B activates OCT-2

(*c*) OCA-B coprecipitates OCT-2

(*d*) OCA-B cotranscipts OCT-2

48. To determine the effects of 15d-PGJ2 on the expression of COX-2 gene, we performed Northern blot analysis using RNA derived from the differentiated U937 cells. Figure A shows macrophage-like differentiated U937 cells were treated for 5 h with LPS in the presence or absence of the indicated concentrations of 15d-PGJ2. Total RNA (10 mg) was isolated from the U937 cells and subjected to Northern blot analysis using specific COX-2 and GAPDH cDNA probes. Figure B shows time course of COX-2 mRNA expression in the U937 cells treated with LPS in the presence or absence of 10 mM 15d-PGJ2. The relative amount of COX-2 mRNA was measured by an image analyzer after normalization with that of GAPDH. Values represent the means 6 standard deviations of three separate dishes. Figure C shows PGE2 in the culture medium was measured by enzyme immunoassays after treatment of the cells with LPS (10 mg/ml) and/or DEX (100 nM) or 15d-PGJ2 (10 mM) for 12 h.

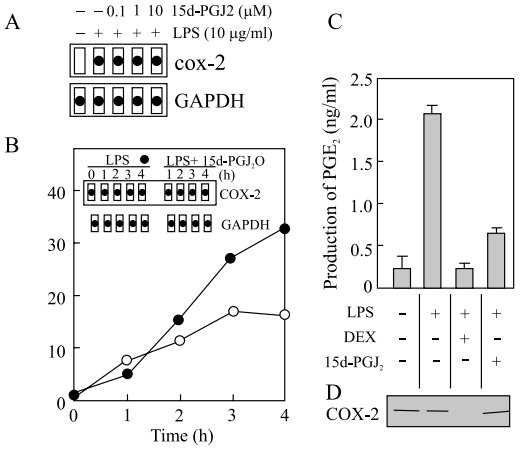
Following inferences were made.

A. LPS-induced the expression of COX-2 mRNA

B. Production of PGE2 were suppressed by 15d-PGJ2

C. The suppressive effect of 15d-PGJ2 was dose-dependent

D. The suppressive effect of 15d-PGJ2 was milder than that of DEX

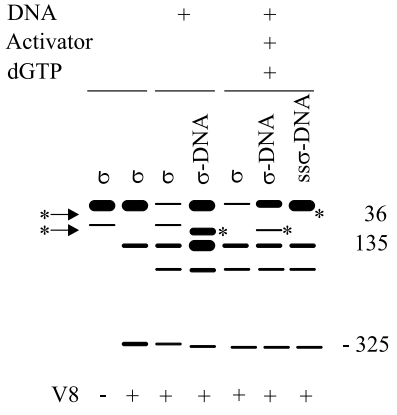


Which of the above inference are correct for the above set of experiment?

(*a*) A, B (*b*) B, C

(*c*) C, D (*d*) all of them

49. V8 protease treated samples were run on native gels and ^{32}P end-labeled σ^{54} was isolated from the gel either as free protein (σ) or as a part of a DNA complex (σ-DNA or ssσ-DNA), eluted and run on a denaturing SDS gel. DNA was unlabelled. Cut sites (marked with arrows) were assigned based on markers and previous sigma protein footprints. Significant differences are marked with asterisks. Horizontal bars indicate complexes isolated from same lane.



Which of the following is the inference of the SDS image?

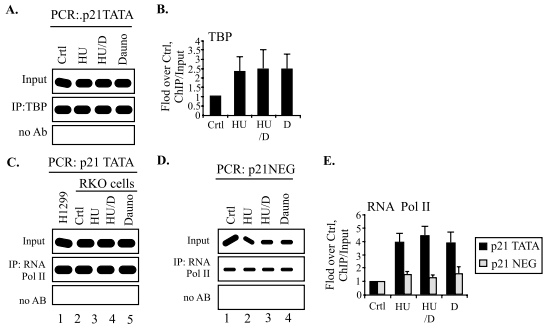
(*a*) σ sequences are involved in binding to the early melted DNA.

(*b*) σ sequences are involved in binding to mRNA

(*c*) σ sequences are involved in binding to circular plasmid DNA

(*d*) σ sequences are involved in binding to double stranded DNA

50. TATA-binding protein and RNA polymerase II are recruited to the p21 promoter following HU treatment. ChIP assays were performed using lysates with a polyclonal TBP antibody to immunoprecipitate TBP/chromatin complexes. Approximately 0.2% of input chromatin and 4% of ChIP-DNA was used as a template in the PCRs including [α-32P]dCTP in the reaction mix.Graphical representation of the data shown in panel A, C to E is data for ChIP assays that were performed using a polyclonal antibody against the large subunit of RNA Pol II. (E) Graphical representation of the data shown in panels C and D.



Which of the following is not true from above experimental data.

(*a*) Basal binding by TBP is negligible

(*b*) No difference in TBP binding in cells treated with HU or daunorubicin

(*c*) Recruitment of polymerase to the p21 promoter TATA box region occurred in a stress- and p53-dependent manner

(*d*) RNA Pol II poised to initiate transcription to similar extents in cells that have been treated with HU, dauno, or a combination of both drugs.

51. Yanofsky's lab isolated two different mutants with mutations in the *trp* leader-attenuator region. The mutants are shown below as they would affect the *trp* leader RNA. Mutation #1 is a base substitution mutant and mutant #2 is a deletion mutation. The effects of these mutations on expression of the *trpE* structural gene are shown in the table below.

Yanofsky's lab also isolated "revertants" of mutants #1 and #2. The *trpE* expression in these mutants is also shown in the table below. Some revertants, in the case of mutant #1, were true revertants in that the wild type base was restored. However, several mutants were "pseudorevertants" in that they still retained the #1 or #2 mutations but also contained a new mutation. Assuming that the pseudorevertants are base substitutions mutations, how do they affect *trp* expression at the molecular level?

Strain	***trpE* Activity**
trpRtrpL+ (parent)	1.0
trpRtrpL #1	0.1
trpRtrpL #2	0.1
trpRtrpL #1 pseudorevertants	2.5 – 4.0
trpRtrpL #2 pseudorevertants	2.5 – 4.0

(*a*) base substitution in attenuator region

(*b*) base substitution in operator region

(*c*) base substitution in regulator region

(*d*) base substitution in leader sequence

52. A partially diploid bacterial cell or merozygote (or merodiploid) is constructed through conjugation. The F+ cells transfer the fertility factor, a plasmid, into an F- cell. F′ are F plasmids which can also carry parts of the chromosome. An F′ can carry parts of the lac operon. Through this process, an E. coli cell can carry two separate and distinct mutations of the lac operon. Now, one has to determine which mutant is dominant to decide whether the operon is inducible or repressed.

$F^- I^+O^+Z^+Y^+$ (constitutive) X $F^1 I^+O^+Z^+Y^+$ (inducible)

$$\frac{F^- I O^+Z^+Y^+}{F^1 I^+O^+Z^+Y^+} = \text{CONSTITUTIVE}$$

Which are the proteins that are constitutive in the progeny of this merozygote?

(*a*) Z, Y (*b*) I, O, Z, Y

(*c*) I, Z, Y (*d*) O, Z, Y

53. A partially diploid bacterial cell or merozygote (or merodiploid) is constructed through conjugation. The F+ cells transfer the fertility factor, a plasmid, into an F- cell. F′ are F plasmids which can also carry parts of the chromosome. An F′ can carry parts of the lac operon. Through this process, an E. coli cell can carry two separate and distinct mutations of the lac operon. Now, one has to determine which mutant is dominant to decide whether the operon is inducible or repressed.

To start the decision-making process, one must decide whether the mutation deals with a diffusible gene product or not. That is, is the mutation in a gene that produces a gene product or is the mutation in a regulatory DNA sequence such as the operator region or not.

$F^- I^+O^+Z^+Y^+$ (inducible) × $F' I^+O^cZ^+Y^+$ (constitutive)

$$\frac{F^- I^+O^+Z^+Y^+}{F' I^+O^cZ^+Y^+} = \text{CONSTITUTIVE}$$

Which of the following statement is true about the above information?

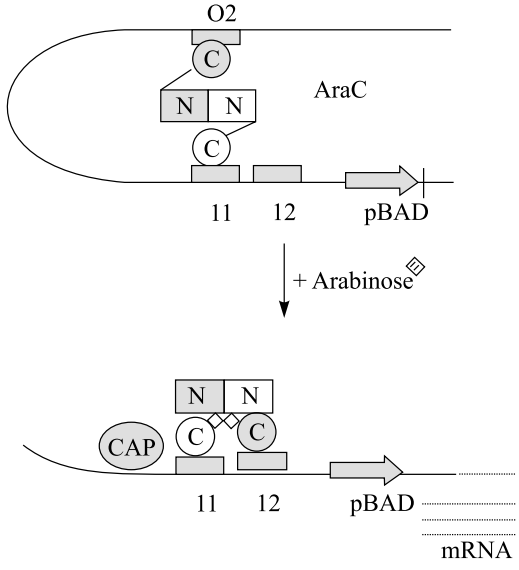
(*a*) Merozygote is cis dominant, producing enzymes all the time

(*b*) Merozygote progeny will be inducible

(*c*) The merozygote will have constitutive Z but not Y

(*d*) Merozygyote will produce enzyme on repression.

54. Control of the PBAD promoter is shown in the following figure. When arabinose binds to the araC, it releases the O2 site and binds to the I2 site. This relaxes the DNA loop and transcription starts. The cAMP activator protein (CAP) stimulates the binding of araC to I1 and I2. N and C respectively designate the N-terminus and the C-terminus of the araC. Which of the following are true?



(*a*) araC is the repressor

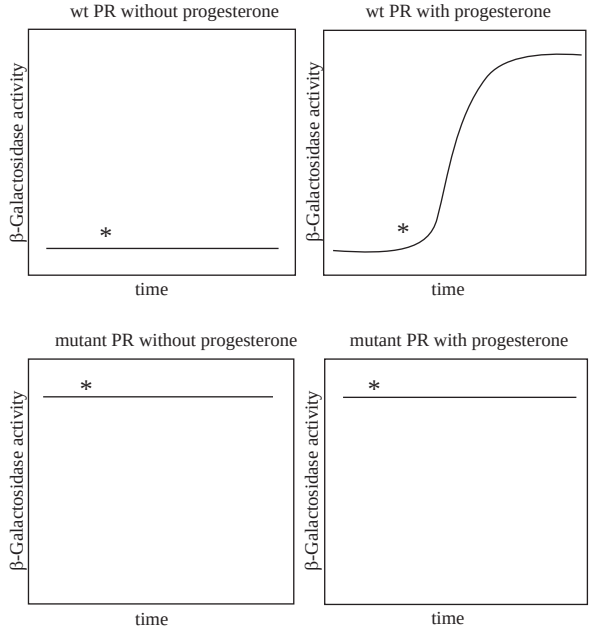
(*b*) arabinose is the inducer

(*c*) both positively and negatively regulated

(*d*) All of the above

55. The lacZ gene can be used as a reporter to study transcriptional activation, because its protein product (β-galactosidase) can be easily measured by a simple enzyme assay. The wildtype and a mutant PR were studied using the DNA construct shown below. Progesterone Response Element (PRE) refers to the DNA sequence that is bound by the activated PR.

The following results were obtained, where the asterisk indicates when progesterone was (or was not) added to the cells. Which of the below said statement would be true about the experiment?



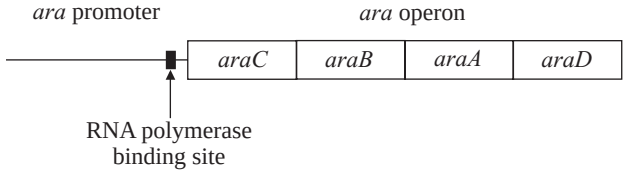
(*a*) Progesterone is inducible

(*b*) Progesterone is repressible

(*c*) Progesterone is constitutive

(*d*) none of the above

56. In the absence of glucose, E. coli can proliferate using the pentose sugar arabinose. The ability of E. coli to utilize the sugar arabinose is regulated via the arabinose operon, depicted in the figure below. The araA, araB, and araD genes encode enzymes for the metabolism of arabinose. The araC gene encodes a gene regulatory protein that binds adjacent to the promoter of the arabinose operon. To understand the regulatory properties of the AraC protein, you engineer a mutant bacterium in which the araC gene has been deleted and look at the effect of the presence or absence of the AraC protein on the AraA enzyme.



Your findings from the experiment are summarized in the following table:

Genotype	araA RNA Levels	
	in the absence of arabinose	in the presence of arabinose
araC+ (normal cells)	low	high
araC– (mutant cells)	low	low

Do the results indicate that the AraC protein regulates arabinose metabolism by acting as a gene repressor or a gene activator?

(*a*) The results are consistent with AraC acting as a gene activator for the arabinose operon

(*b*) The results are consistent with AraC acting as a gene promotor for the arabinose operon

(*c*) The results are consistent with AraC acting as a gene regulator for the arabinose operon

(*d*) The results are consistent with AraC acting as a gene repressor for the arabinose operon

57. You are studying the regulation of the lac operon in E. coli and perform a merodiploid (partial diploids) analysis with various regulatory and structural gene variants that you isolated. Your first results are shown below (in units of β-galactosidase activity).

Experiment	Genotype	βgal activity (+inducer)	βgal activity (–inducer)
1.	$LacI^{+}O^{+}Z+$	100	0.1
2.	$LacI^{-}O^{c}Z^{+}$	100	100
3.	$LacI^{-}O^{c}Z^{-}$	1	0.1
4.	$LacI^{-}O^{+}Z^{+}$/ $LacI^{+}O^{+}Z^{+}$	200	0.1

lacI encodes the lac repressor and is active in the absence of inducer (i.e., lac repressor binds to the lac operator and inhibits transcription from the lac promoter). Addition of inducer inhibits the repressor and stimulates transcription from the lac promoter. lacO is the operator, the region to which lac repressor binds, and lacOc mutations are constitutive operator mutations, causing lacZ expression in the presence of repressor and absence of inducer. lacZ encodes β-galactosidase, an enzyme that functions as a tetramer.

Why is the induced activity in experiment 4 twice that of 1?

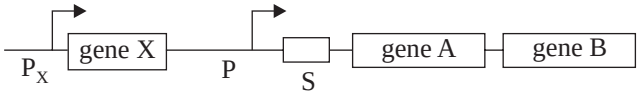
(*a*) In expt 4 LacZ is induible and in expt 1 it's not.

(*b*) In expt 4 LacZ is constitutive and not in expt 1

(*c*) There are two copies of lacZ in expt 4, and only one copy in expt 1.

(*d*) None of the above

58. In E. coli, the fictitious AB operon is induced by the presence of Compound W. A diagram of the operon, its regulatory proteins and regulatory sites is shown below:



P_X promoter for the regulatory protein
X gene for the regulatory protein of the AB operon
P promoter for the AB genes
S sequence shown to be important for regulation by W
A structural gene for enzyme A
B structural gene for enzyme B

The following table shows the genotypes of different E. coli strains with a wild-type AB operon and various mutant AB operons, and the number of molecules of proteins A and B per cell in the absence or presence of Compound W (–W or +W, respectively). The symbol "+" indicates that the gene or control element is functional (wt) and "–" indicates that the gene or control element is non-functional. Assume the genes not listed are wild type.

Strain	X	P	S	A	–W	+W	Expression
wt	+	+	+	+	0	200	inducible
m1	–	+	+	+	200	200	constitutive
m2	+	–	+	+	0	0	uninducible
m3	+	+	–	+	200	200	constitutive
m4	+	+	+	–	0	0	uninducible

Based on the data shown above, what is the observation?

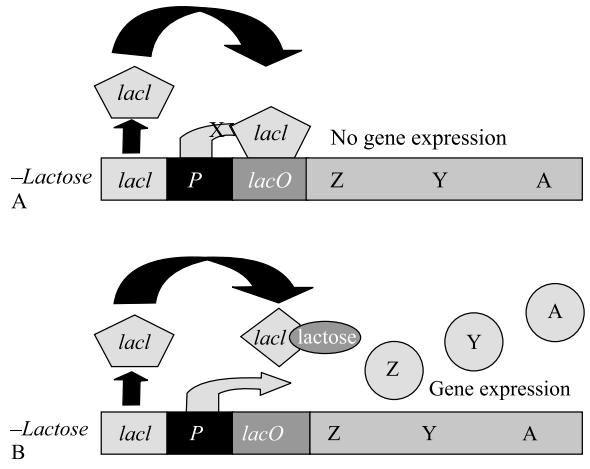
(*a*) protein X act as a repressor

(*b*) protein X act as a activator

(*c*) protein X act as a operator

(*d*) none of the above

59. In *E. coli*, partial diploids can be created by adding an extrachromosomal ring of DNA called a plasmid that has an origin and terminator regions so it is replicated and maintained throughout each cell division. The plasmid can be modified in the *lab* to contain different genes such as all the genes in the *lac* operon. When the plasmid is introduced into the bacteria, there are two copies of each of the genes in the lac operon, and this is denoted as I+P+O+Z+Y+/I+P+O+Y+Z+ where the genes before the "/" are chromosomal and those genes after the "/" are on the plasmid. The following partial diploid, I−O+Z+Y−/I−O+Z+Y+, has β-galactosidase activity and lactose permease activity with or without an inducer. Why?



(*a*) Mutation in repressor

(*b*) Mutation in operator

(*c*) Mutation in co repressor

(*d*) (A), (B)

60. You have isolated two mutations in the Lac operon that cause constitutive expression of Lac genes. You designate these mutants $Lac1^-$ and $Lac2^-$. Making use of an F' that carries the Lac operon with the LacY gene mutated, you construct strains that you test for both β-galactosidase activity and Lac permease activity with results shown below.

	β-galactosidase activity		Lac permease activity	
	– IPTG	+ IPTG	– IPTG	+ IPTG
$Lac\ 1^-\ lac\ Z^-/F'\ Lac\ Y^-$	–	+	+	+
$Lac\ 2^-\ lac\ Z^-/F'\ Lac\ Y^-$	+	+	+	+

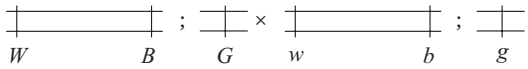
Which of the following statements will be true in above case of experiment?

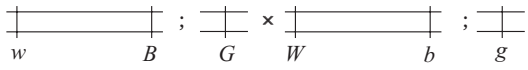
(*a*) Lac 1^- is recessive (*b*) Lac 2^- is dominant

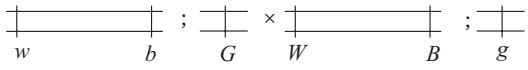
(*c*) Lac 1^- is dominant (*d*) Lac 2^- is recessive

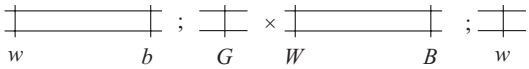
61. You have isolated two mutations that show decreased expression of the Lac operon. However, unlike like the promoter mutations described in part (*b*) these mutations don't respond to the inducer IPTG. These

What are the chromosomes of the parents?

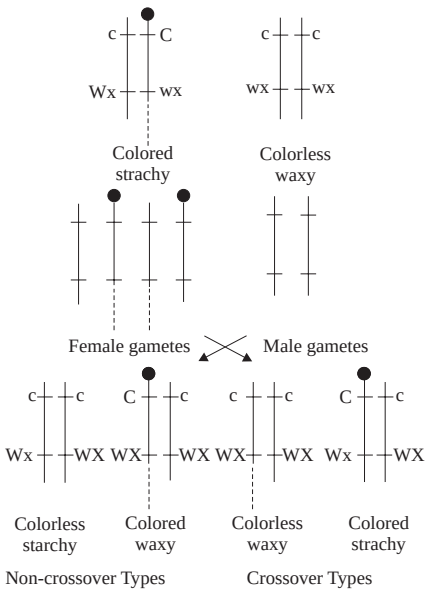
(*a*) 

(*b*) 

(*c*) 

(*d*) 

79. Knobbed chromosome carried the genes for color (C) and waxy (wx) endosperm -The knobless #9 carries colorless (c) and starchy (Wx) alleles Genes like knob and C (color) Much like genetic map of Drosophila. This was done by crossing Knob (C-wx)/knobless (c-Wx) with double knobless (c-wx). Following are the gametes and crosses observed.



What is the percentage of progenies from cross between coloured waxy and colourless waxy generated in F2 generation?

(*a*) 50% colored waxy and 50% colorless waxy

(*b*) 70% colored waxy and 30% colorless waxy

(*c*) 50% colored starchy and 50% colorless starchy

(*d*) 25% colored starchy and 75% colorless starchy

80. A three-point testcross was made in corn. The results and a recombination analysis are shown in the display below, which is typical of three-point testcrosses (*p* = purple leaves, + = green; *v* = virus-resistant seedlings, + = sensitive; *b* = brown midriff to seed, + = plain).

The cross between P +/+ · +/+ · +/+ × *p*/*p* · *v*/*v* · B/*b* gives rise to gametes + · + · + *p* · *v* · *b*

Which genes are linked and are what distance apart?

Class	Progeny phenotypes	F_1 gametes	Numbers	Recombinant for		
				p–b	p–v	v–b
1.	gre sen pla	+ · + · +	3,210			
2.	pur res bro	*p* · *v* · *b*	3,222			
3.	gre sen pla	+ · *v* · +	1,024		R	R
4.	pur res bro	*p* · + · *b*	1,044		R	R
5.	pur sen pla	*p* · *v* · +	690	R		R
6.	gre res bro	+ · + · *b*	678	R		R
7.	gre res bro	+ · *v* · *b*	72	R	R	
8.	pur sen pla	*p* · + · +	60	R	R	
		Total	10,000	1,500	2,200	3,436

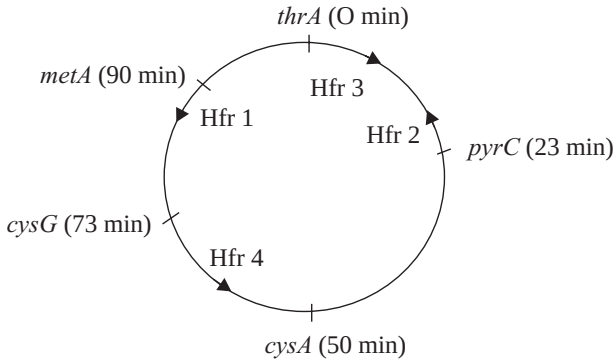
(*a*) *v p b*; *v* to *p*-22 m.u, *p* to *b*-15 m.u.

(*b*) *v p b*; *v* to *p*-15m.u, *p* to *b*-22 m.u.

(*c*) *v b p*; *v* to *p*-22 m.u, *p* to *b*-15 m.u.

(*d*) *v b p*; *v* to *p*-15m.u, *p* to *b*-22 m.u

81. A high-frequency recombination cell (Hfr cell) (also called an Hfr strain) is a bacterium with a conjugative plasmid (for example, the F-factor) integrated into its chromosomal DNA. The integration of the plasmid into the cell's chromosome is through homologous recombination. A new mutant was isolated that is Str^R and unable to use acetate as a carbon source (ace). To determine where the mutation maps, it was mated with the four different Str^S *ace*$^+$ Hfr donor strains shown below. [Arrowheads indicate the location and direction of transfer from each different Hfr.]



Given the results in the following table, where does the ace mutation map?

Donor strain	Ace$^+$ colonies
Hfr 1	1000
Hfr 2	5
Hfr 3	1000
Hfr 4	80

Answer Keys

Chapter-1 : Analytical Problems and Solutions Life Science-01

1. (*a*)	**2.** (*d*)	**3.** (*a*)	**4.** (*a*)	**5.** (*b*)	**6.** (*a*)	**7.** (*d*)	**8.** (*b*)	**9.** (*d*)	**10.** (*a*)
11. (*c*)	**12.** (*a*)	**13.** (*a*)	**14.** (*d*)	**15.** (*d*)	**16.** (*a*)	**17.** (*a*)	**18.** (*d*)	**19.** (*c*)	**20.** (*a*)
21. (*a*)	**22.** (*a*)	**23.** (*b*)	**24.** (*d*)	**25.** (*d*)	**26.** (*a*)	**27.** (*a*)	**28.** (*a*)	**29.** (*a*)	**30.** (*b*)
31. (*a*)	**32.** (*a*)	**33.** (*a*)	**34.** (*a*)	**35.** (*b*)	**36.** (*a*)	**37.** (*d*)	**38.** (*a*)	**39.** (*c*)	**40.** (*b*)
41. (*a*)	**42.** (*a*)	**43.** (*d*)	**44.** (*d*)	**45.** (*a*)	**46.** (*a*)	**47.** (*a*)	**48.** (*d*)	**49.** (*a*)	**50.** (*a*)
51. (*d*)	**52.** (*d*)	**53.** (*a*)	**54.** (*d*)	**55.** (*c*)	**56.** (*a*)	**57.** (*c*)	**58.** (*a*)	**59.** (*d*)	**60.** (*b*)
61. (*c*)	**62.** (*a*)	**63.** (*a*)	**64.** (*a*)	**65.** (*a*)	**66.** (*a*)	**67.** (*a*)	**68.** (*a*)	**69.** (*a*)	**70.** (*d*)
71. (*a*)	**72.** (*a*)	**73.** (*a*)	**74.** (*a*)	**75.** (*a*)	**76.** (*a*)	**77.** (*a*)	**78.** (*a*)	**79.** (*a*)	**80.** (*a*)
81. (*a*)	**82.** (*a*)	**83.** (*a*)	**84.** (*a*)	**85.** (*a*)	**86.** (*a*)	**87.** (*a*)	**88.** (*a*)	**89.** (*a*)	**90.** (*a*)
91. (*a*)	**92.** (*a*)	**93.** (*a*)	**94.** (*a*)	**95.** (*a*)	**96.** (*a*)	**97.** (*a*)	**98.** (*a*)	**99.** (*a*)	**100.** (*a*)

Chapter-2 : Analytical Problems and Solutions Life Science-02

1. (*d*)	**2.** (*c*)	**3.** (*b*)	**4.** (*d*)	**5.** (*d*)	**6.** (*b*)	**7.** (*a*)	**8.** (*d*)	**9.** (*b*)	**10.** (*a*)
11. (*a*)	**12.** (*a*)	**13.** (*a*)	**14.** (*c*)	**15.** (*c*)	**16.** (*d*)	**17.** (*d*)	**18.** (*b*)	**19.** (*b*)	**20.** (*b*)
21. (*a*)	**22.** (*d*)	**23.** (*a*)	**24.** (*a*)	**25.** (*a*)	**26.** (*d*)	**27.** (*c*)	**28.** (*d*)	**29.** (*a*)	**30.** (*a*)
31. (*c*)	**32.** (*a*)	**33.** (*a*)	**34.** (*a*)	**35.** (*a*)	**36.** (*a*)	**37.** (*a*)	**38.** (*a*)	**39.** (*a*)	**40.** (*a*)
41. (*a*)	**42.** (*d*)	**43.** (*a*)	**44.** (*c*)	**45.** (*c*)	**46.** (*d*)	**47.** (*a*)	**48.** (*d*)	**49.** (*a*)	**50.** (*d*)
51. (*c*)	**52.** (*c*)	**53.** (*c*)	**54.** (*a*)	**55.** (*c*)	**56.** (*c*)	**57.** (*d*)	**58.** (*c*)	**59.** (*a*)	**60.** (*c*)
61. (*a*)	**62.** (*a*)	**63.** (*a*)	**64.** (*d*)	**65.** (*d*)	**66.** (*a*)	**67.** (*d*)	**68.** (*d*)	**69.** (*a*)	**70.** (*a*)
71. (*c*)	**72.** (*a*)	**73.** (*c*)	**74.** (*b*)	**75.** (*a*)	**76.** (*a*)	**77.** (*a*)	**78.** (*c*)	**79.** (*a*)	**80.** (*a*)
81. (*a*)	**82.** (*a*)	**83.** (*a*)	**84.** (*d*)	**85.** (*a*)	**86.** (*d*)	**87.** (*a*)	**88.** (*c*)	**89.** (*d*)	**90.** (*b*)
91. (*a*)	**92.** (*b*)	**93.** (*b*)	**94.** (*a*)	**95.** (*c*)	**96.** (*c*)	**97.** (*b*)	**98.** (*d*)	**99.** (*c*)	**100.** (*d*)

Chapter-3 : Analytical Problems and Solutions Life Science-03

1. (*a*)	**2.** (*a*)	**3.** (*b*)	**4.** (*d*)	**5.** (*b*)	**6.** (*d*)	**7.** (*b*)	**8.** (*b*)	**9.** (*c*)	**10.** (*a*)
11. (*a*)	**12.** (*d*)	**13.** (*a*)	**14.** (*a*)	**15.** (*d*)	**16.** (*d*)	**17.** (*d*)	**18.** (*a*)	**19.** (*a*)	**20.** (*d*)
21. (*b*)	**22.** (*d*)	**23.** (*c*)	**24.** (*b*)	**25.** (*c*)	**26.** (*c*)	**27.** (*a*)	**28.** (*b*)	**29.** (*d*)	**30.** (*c*)
31. (*b*)	**32.** (*d*)	**33.** (*b*)	**34.** (*c*)	**35.** (*d*)	**36.** (*d*)	**37.** (*d*)	**38.** (*c*)	**39.** (*d*)	**40.** (*c*)
41. (*b*)	**42.** (*d*)	**43.** (*d*)	**44.** (*d*)	**45.** (*b*)	**46.** (*d*)	**47.** (*d*)	**48.** (*d*)	**49.** (*a*)	**50.** (*c*)
51. (*a*)	**52.** (*c*)	**53.** (*b*)	**54.** (*d*)	**55.** (*d*)	**56.** (*a*)	**57.** (*a*)	**58.** (*d*)	**59.** (*c*)	**60.** (*c*)
61. (*d*)	**62.** (*c*)	**63.** (*b*)	**64.** (*d*)	**65.** (*d*)	**66.** (*b*)	**67.** (*d*)	**68.** (*b*)	**69.** (*d*)	**70.** (*d*)
71. (*d*)	**72.** (*a*)	**73.** (*b*)	**74.** (*c*)	**75.** (*d*)	**76.** (*d*)	**77.** (*a*)	**78.** (*d*)	**79.** (*a*)	**80.** (*c*)
81. (*c*)	**82.** (*b*)	**83.** (*d*)	**84.** (*b*)	**85.** (*d*)	**86.** (*b*)	**87.** (*d*)	**88.** (*b*)	**89.** (*d*)	**90.** (*d*)

Explanations

Chapter-1 : Analytical Problems and Solutions Life Science-01

1. (*a*) Nucleic acids absorbs at 260 hence absorbance of DNA is measured at 260, also presence of aromatic amino acids decides the absorbance for proteins.

2. (*d*) This could work because it could change the Met codon (TAC on the template strand) to TTC, which would be AAG (Lys) in the mRNA. In the mutated version, Lys is changed to Met (AUG). AAG could change to AUG with only one mutation, while AAA would need two mutations to change to AUG. So it's most likely that the Lys codon was AAG.

3. (*a*) Nucleotide excision repair is a repair pathway that removes a variety of DNA damages, including UV- and benzo[a]pyrene (BaP)-induced DNA damages. BaP, a widespread carcinogen, is the major cause of lung cancer. BPDE preferentially forms bulky covalent DNA adducts at N2 position of guanines and causes mutations if these BPDE-deoxyguanosines (BPDE-dGs) are not efficiently eliminated by nucleotide excision repair

4. (*a*) For sequences longer than 13 nucleotides, the equation used is

$T_m = 64.9 + (yG + zC - 16.4)/(wA + xT + yG + zC)$

5. (*b*) Entropy = Enthalpy/T_m.

Enthalpy = 0.039 kJ = 39 J

T_m = 52°C or 325 K

Entropy = 39/325 = 0.12 J = 0.00012 kJ

6. (*a*) DNA concentration (μg/ml)

$$= \frac{\text{OD at 260 * DIlution factor * 50}}{1000}$$

A_{260} of 1.0 = 50 μg/ml pure dsDNA

7. (*d*) Number of copies = (40 × 6.022 × 1023)/(5, 200 × 1 × 109 × 650) 6.022 $\times 10^{23}$ = Avogadro's number. Length is the length of your DNA fragment in base pairs. Just multiply by 1000 if you are working in kb. We multiply by 1×10^9 to convert our answer to nanograms. The average weight of a single DNA base pair (bp) is 650 daltons. This can also be written as 650 g/mol

8. (*b*) Presence of a new and an old strand in the 2nd generation

9. (*d*) Parallel and antiparallel are both the possible typesof G-quadruplex structures

10. (*a*) The hyperchromic effect is the striking increase in absorbance of DNA upon denaturation. The two strands of DNA are bound together mainly by the stacking interactions, hydrogen bonds and hydrophobic effect between the complementary bases.

11. (*c*) Glucose levels are constantly up hence the person is hyperglycemic

12. (*a*) After a high carbohydrate meal, insulin level will shoot up and during fasting glucagon will rise

13. (*a*) Glucagon is a peptide hormone, produced by alpha cells of the pancreas. It works to raise the concentration of glucose and fatty acids in the bloodstream, and is considered to be the main catabolic hormone of the body.

14. (*d*) In liver disease, the glucose tolerance curve is diabetic but the fasting plasma glucose level is low. In functional hypoglycemia, the plasma glucose rise is normal after a test dose of glucose, but the subsequent fall overshoots to hypoglycemic levels

15. (*d*) A disorder in which the adrenal glands don't produce enough hormones. Specifically, the adrenal glands produce insufficient amounts of the hormone cortisol and sometimes aldosterone, too. See that glucose levels is increased on epinephrine injection.

16. (*a*) Addison's disease is a disorder in which the adrenal glands don't produce enough hormones Epinephrine (adrenaline) is released from nerve endings and the adrenals, and acts directly on the liver to promote sugar production (via glycogenolysis). Epinephrine also promotes the breakdown and release of fat nutrients that travel to the liver and that are converted into sugar and ketones.

17. (*a*) Insulin helps your body turn blood sugar (glucose) into energy. It also helps your body store it in your muscles, fat cells, and liver to use later, when your body needs it.

18. (*d*) Galactosemia is an inherited disorders that impair the body's ability to process and produce energy from a sugar called galactose. When people with galactosemiainjest foods or liquids containing galactose, undigested sugars build up in the blood.

19. (*c*) In overall comparisons, glucose lowering in animals treated with GCGR ASO 148359 and GCGR ASO 180475 was significantly different compared individually with saline- and control ASO–treated animals.

20. (*a*) Glycogen resynthesis in muscle studies bar graph increases with time hence X is glycogen

21. (*a*) Chaperons regulates proteolysis and they regulates homeostasis in stress conditions

22. (*a*) Similar growth patterns in p33 : p92 and p33 : p33

23. (*b*) In high levels of sucrose ribosomes stall, hence it's a repressor.

24. (*d*) The phoP mutant displayed the highest ATP levels. ATP levels are inversely proportional to translation efficiency.

25. (*d*) Ψ promotes incorporation of additional nucleotide, so that limiting concentrations of aa-tRNA can be compensated. This is the role of unusual nucleotides.

26. (*a*) eIF4G, eIF4A and eIF4E are transcription

27. (*a*) IL24 induces apoptosis through phosphorylation of eIF2a and thereby inhibiting translation initiation

28. (*a*)

(*a*) UUU was read as ΨUU and valine was incorporated

(*b*) UUU was mutated to ΨUU and valine was incorporated

(*c*) ΨAA was read by mRNA and valine was incorporated

(*d*) ΨAA was mutated to ΨUU and valine was incorporated

UUU or ΨUU in the A site in the presence lead to valine incorporation as Ψ was read as U.

29. (*a*) The interaction of eRF1 with the uORF2 peptidyl-tRNA may stabilize a termination intermediate that prevents puromycin from gaining access to the peptidyl-tRNA ester bond. In such a case, removal of eRF1 from the stalled ribosome complex should convert the uORF2 peptidyl-tRNA to a puromycin-sensitive form

30. (*b*) See the thickness of band to measure the quantity. Absence of Kozak sequence cannot induce transcription as seen by 0 IPTG well.

31. (*a*) Condensins are large protein complexes that play a central role in chromosome assembly and segregation during mitosis and meiosis.

32. (*a*) Aneuploidy is the presence of an abnormal number of chromosomes in a cell, for example a human cell having 45 or 47 chromosomes instead of the usual 46. It does not include a difference of one or more complete sets of chromosomes. A cell with any number of complete chromosome sets is called a euploid cell.

33. (*a*) Rules for standard nomenclature for karyotyping

34. (*a*) Patau syndrome (trisomy 13)

35. (*b*) Writhe is a number of a time DNA double helix is crossed. DNA is closed circular thus writhe is 0

Linkage number = Twist + Writhe

Twist = (5200 base pairs)/(bp/turn) = 490 turns

36. (*a*) For circular, closed, double stranded DNA, writhe is 0

Linking number = 5300 bp/(10.6 bp/turn) + 0

Linking number = 500 turns

37. (*d*) The integrated F factor occasionally leaves the chromosome of an Hfr cell and moves back to the cytoplasm, in some rare cases carrying a few host chromosomal genes along with. This modified F, called F′ (pronounced "F prime").

Whole lac^+ gene following the origin of replication gave rise to F′ strain

38. (*a*) P element also encodes a suppressor of transposition, which accumulates in the cytoplasm during the development of cells.

39. (*c*) *Electrophoretic mobility* of a protein-nucleic acid complex is typically less than that of the free nucleic acid

40. (*b*) Shorter telomere lengths leads to an aging process.

41. (*a*) In yeast two hybrid system, the protein fused to the BD may be referred to as the bait protein, and is typically a known protein the investigator is using to identify new binding partners. The protein fused to the AD may be referred to as the prey protein

42. (*a*) Immunoprecipitation refers to the interaction of proteins so while pulling down one protein the interacting partner also gets precipitated showing the band

43. (*d*) When a co-inducer (oval) is bound, it drastically increases the RNA polymerase binding capacity. Hence its playing the role of positive regulator of transcription

44. (*d*) Log –8 to –6 gives 1000 folds, i.e. 1mM concentration. arsM and arsC2 shows similar levels.

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