

## Lec 2

1. What are the essential components of the RNAPol?
2. What are their functions?
3. What are the steps for transcription?
4. What are the 4 different types of prokaryotic promoters?
5. How can you identify promoters in three ways?
6. Describe the steps for electrophoresis analysis
7. Describe the steps involving in footprinting analysis
8. What can't be determined by the electrophoresis analysis and foot printing?
9. How do you analyse the promoter sequence in vivo?
10. What is used to treat the DNA in footprinting analysis?
11. Whether ds/ss DNA is used in electrophoresis/footprinting analysis
12. How do you label the dna in footprinting analysis
13. Is TrpR a dimer
14. Is trp R a repressive/dominant and positive/negative regulator?
15. What is the features of TrpR operator?
16. What's the difference between electrophoresis gel mobility assay and footprinting?
17. If you need purified protein for electrophoresis gel mobility assay?

## Lec3

1. Describe the phenotypes of lacI mutants and whether they are dominant/recessive.
2. Refer the mutations to the 3D structure of the lacR.
3. How do the DNA binding proteins work?
4. Describe the phenotypes of mutation of DNA operators.
5. Describe the relationship between glucose cAMP, and CAP
6. What is the phenotype for CYA<sup>-</sup>? why?
7. Describe the two classes of Lac<sup>-</sup> in the promoter region.
8. Which promoters have dyad symmetry?  
Lac I operator, and trp R binding sites, Cap
9. What is the relative position for lacI binding site compared to promoter?

## Lec 4,

1. What are the evidences for CAP and RNA pol interaction?
2. What are the 4 things showed in biochemical studies for the CAP-RNA-P interaction
3. Which part of the RNAP does cap interact with?
4. What are the three classes of RNAPol-CAP interaction?
5. Describe the positive regulation of Ara operon?
6. What is the difference between arac and cap?
7. Describe MerT, what is its regulator's feature?
8. Draw down the basic structure of two component sensing mechanisms
9. What does kinase/phosphatase do?
10. What is the signal received by porins?
11. What are porins for?
12. What are the names for the receptor and the inner component for porins?
13. How do Nitrogen level control the signal?

trp R	<ul style="list-style-type: none"> <li>- Negative</li> <li>- Corepressed by tryptophan</li> <li>- Operator has a dyad symmetry-&gt; tryp R binds as a dimer</li> <li>- Regulate 5 different promoters</li> <li>- Sequences are similar but not identical, all have dyad symmetry</li> <li>- All operators overlap promoter but the position relative to the -10 and -35 sequence differs</li> </ul>
lacI <sup>-</sup>	constitutive, recessive to lacI <sup>+</sup>
lacI <sup>s</sup>	non inducible, dominant
lacI <sup>D</sup>	constitutive , dominant. have normal ability to bind inducer, and bind monomers but cant bind DNA and it poisons other subunit
O1 <sup>-</sup> , O2 <sup>-</sup> O3 <sup>-</sup>	O1 overlap with the RNA pol binding site loss of 2/3, 2-4x increase in basal expression loss of 2 n 3, 100x increase in basal expression O1 plus a second operator are essential for repression
CAP-	lac- positive regulator, inducible by camp Dimer, binds to camp and DNA consensus, <b>bends DNA</b> , directly interacts with RNAP to activate transcription
CYA-	lac-. CYA convert ATP to Camp
Lac-( class 1 and 2)of cap	Class one within cap binding site(L8) Class two within RNA pol binding site(L157)