Questions lecture 3: Gene regulatory networks

1) Explain the take-home message of the seminal King and Wilson paper?
2) Explain the lego principle.
3) Provide a basic definition of gene regulatory networks.
4) Explain how the 10X chromium technology works enabling massive scRNA-seq?
5) Explain the principle of Bing Ren's promoter identification experiment (explain also the ChIP-seq assay). How did results from this experiment change our thinking about the architecture of promoters?
6) Describe nucleosomes. Is their genomic distribution: a) random versus non-random? b) static or dynamic? Please clarify. c) What is their genomic function?
7) How can we find out which regions in the genome tend to be nucleosome-free → describe an assay that allows us to directly identify these regions and describe how this assay works.
8) What do we know about histone modification enrichment dynamics, also compared to RNA pol II? What is meant with the "chromatin state"? Explain how defining this state helps in identifying regulatory elements, plus know active versus repressive histone modifications?
9) Explain the principle of p300-based enhancer mapping.
10) Define enhancers
11) Explain the famous Takahasha and Yamanaka experiment that led to the Nobel prize in physiology and medicine.
12) What is colinearity?
13) Explain using the mouse vs. chicken vs. python skeletal structure that new body designs do not necessarily require the involvement of new genes, what is main conclusion in terms of the requirement if genes to form new body morphologies?
14) Provide the typical protein structure of a transcription factor (TF) and provide a few examples.
15) Why is, in contrast to the first, the second or regulatory code considered "degenerate"?
16) Define the KD, what is the KD of a typical TF-DNA interaction?
17) Understand the principle of how developmental timing is mediated by DNA binding affinity.
18) Explain the chromatin conformation capture (3C) technique.
19) Explain how 3C was used to elucidate the dynamic chromatin architecture underlying the expression of globin genes.
20) Using a high-throughput version of 3C, Hi-C (please explain), it was discovered that the genome appears to be partitioned in topologically associated domains. Explain the underlying concept and how this impacts transcriptional organization.
21) It was found that the nuclear organization of the genome is not random. Provide examples to back-up this statement (going from a whole genome perspective, to the chromosome, chromosomal domains, and gene level)
22) Is there a link between chromatin conformation / structure and disease? Please explain, providing a possible mechanism

23) What is the principal TF-centered protein-DNA interaction detection method? Describe how it works.
24) What are a couple of the principal conclusions from the TF ChIP-seq experiments so far?
25) Provide an explanation as to why not every binding event may lead to a change in the expression of the respective target gene.
26) List at least three of the most overrepresented network motifs and their regulatory significance / function.