Question 1.

(1) Answer: False. Expression of the trpA-E genes will always be turned off. Because even when tryptophan levels are low, the ribosome no longer pauses while translating the leader mRNA as the tryptophan codons have been mutated (there is no more waiting by the ribosome for the trp-tRNA), thus causing the constitutive formation of the transcription terminator.

(2) Answer: True.

(3) Answer: False. The terminator also contains an A/T-rich sequence that lies immediately 3' to the G/C-rich palindromic sequence. Since the A/T-rich sequence does not have any rotational symmetry, the entire terminator is not symmetric and thus can only be read from one correct direction.

(4) Answer: False. The phosphorylation of I-κBα leads to its interaction with a specific E3 ubiquitin ligase and subsequent degradation by the proteosome, which exposes an existing nuclear localization signal in NF-κB that is previously masked by I-κBα.

(5) Answer: True.

(6) Answer: False. The zinc atom stabilizes the overall structure of the finger through interacting with two Cys and two His residues, which themselves do not participate in the interaction with the base pairs. Other amino acids in the α-helix portion of the zinc finger make specific contacts within the DNA major groove.

(7) Answer: False. Drosha processes pri-miRNAs into pre-miRNAs in the nucleus, whereas Dicer processes pre-miRNAs into 21–25 bp mature miRNAs in the cytoplasm.

(8) Answer: True.

(9) Answer: True.

(10) Answer: False. Although Tat is required to stimulate Pol II elongation along the proviral DNA template, the synthesis of the first Tat molecule, which kicks in a positive feedback loop, depends on the production of the full-length HIV mRNA by Pol II, which can finish elongation once a while without Tat albeit at extremely low frequency.

(11) Answer: False: The CG-rich sequences are more rigid and bound by histone octamers more weakly than CG-poor sequences and thus are nucleosome-free and more accessible to binding by general transcription factors and RNA Pol II, which set up the PIC complex at the promoters.

(12) Answer: False. Only the adenosine residue that is surrounded by several ribonucleotides matching the consensus sequences that can base pair with U2 snRNA and it must be separated by a polypyrimidine tract at a proper distance from the 3' splice site to be used as the branch site adenosine. (Needed both points for full credit)

Question 2.
Answer: Both recruit histone modification complexes but result in different transcriptional outcomes. Using the examples discussed in class, a UAS sequence can recruit a multi-subunit complex containing the histone acetyltransferase (HAT) activity with GCN5 being the catalytic subunit (HAT must be mentioned here but GCN5 is not absolutely necessary), which results in creation of open chromatin structure and activation of transcription of a nearby gene.

In contrast, a URS sequence recruits a histone deacetylase complex (HDAC) that contains Rpd3 as the catalytic subunit (HDAC must be mentioned here). The causes removal of acetyl groups from histone tails and promotes heterochromatin formation and gene silencing.

Question 3.

Answer:

NtrC bound to upstream enhancers contacts and activates sigma^{54}-polymerase at the core promoter region via a loop, leading to the activation of the *glnA* gene transcription.

The Lac repressor tetramer uses its two dimers to bind to the primary and secondary operators, respectively. This forms a loop between the two operators to inhibit transcription of the *lac* operon.

Question 4.

Case #1:
Answer: The E. coli Rho hexamer has RNA helicase/ATPase activity. It uses this activity to translocate along nascent RNA in a 5' to 3' direction. When it reaches the transcription bubble, it disrupts the RNA/DNA heteroduplex to terminate transcription.

Case #2:
Answer: The eukaryotic general transcription factor TFIIH complex contains both 5' to 3' and 3' to 5' helicase/ATPase activities (it's OK to leave out the directions). The complex uses these activities to unwind DNA around the transcription start site to facilitate the formation of an open complex (these activities are also important for TFIIH-mediated, transcription-coupled nucleotide excision repair. Three extra points for mentioning this second function).

Case #3:
Answer: All eukaryotic chromatin-remodeling complexes contain a helicase/ATPase component that is required to disrupt interactions between base-paired nucleic acids or/and between nucleic acids and histones (It is OK to just name one specific example of such complexes; e.g. hSwi/Snf). This activity allows the remodeling complexes to move nucleosomes out of the way of important regions such as the core promoter region for the establishment of PIC and transcriptional initiation.

Case #4:
Answer: Several non-snRNP proteins (it's OK to just say some splicing factors) present in the spliceosome have helicase/ATPase activity, which helps the rearrangements of base pairing in snRNAs

during the splicing cycle. These rearrangements are essential to convert the spliceosome into a catalytically active form.

Alternative Case #5
Answer: During nonsense-mediated decay (NMD), mRNAs harboring premature termination (nonsense) codons (PTC) are degraded by the NMD pathway that operates inside the P-bodies. Recruitment of the PTC-bearing mRNAs into P-bodies requires UPF1 (it's OK to just say a factor in the NMD pathway if the name is forgotten) that is an ATPase/RNA helicase.

Question 5.

Answer: Both FL Z and ΔZ can bind the DNA probe as a dimer. The FL Z-ΔZ heterodimer can also bind the DNA probe. The three retarded bands in lane 3, from top to bottom, contain the labeled DNA fragment associated with (1) a homodimer of FL Z protein produced from the wild-type chromosomal copy of the Z gene; (2) a heterodimer containing one molecule of FL Z and one molecule of the truncated ΔZ produced from the mutant copy of the Z gene; and (3) a homodimer of the truncated ΔZ, respectively.

Since the antibody (Ab) recognizes only the N-terminal 20 amino acids of FL Z, which are missing in ΔZ, the three retarded bands in lane 4, from top down, are Ab-FL Z (Ab bound to FL Z) x 2 + DNA; Ab-FL Z + ΔZ + DNA; and ΔZ x 2 + DNA.