1. DNA helicase and sliding DNA clamp are ring-shaped proteins participating in DNA replication.
   (1) Describe the functions of these proteins and the importance of their ring structures in DNA replication. (6 points)
   (2) Briefly describe how these proteins are loaded onto DNA in *E. coli*. (6 points)
   (3) During PCR (polymerase chain reaction), it is not necessary to add DNA helicase and sliding DNA clamp to the reaction. Explain why not. (4 points)

2. Compare the initiation of DNA replication in prokaryotes and in eukaryotes. (10 points)

3. (1) In eukaryotes, what are two mechanisms for repairing DNA double-strand breaks (DSBs)? (2 points)
   (2) During the S phase, which one is the major repairing system for DSBs? (2 points) Please describe details of the mechanism. (6 points)

4. Describe the physiological roles of histone modifications (8 points) and how to monitor the modification states of a specific histone. (6 points)

5. Compare the following terms: (20 points)
   (1) Monocistronic mRNA and polycistronic mRNA
   (2) 5'cap-dependent translation and 5'cap-independent translation
   (3) N and Q proteins of lambda phage
   (4) Exonic splicing enhancers (ESEs) and SR (serine-arginine-rich) proteins
   (5) Pri-miRNA and pre-miRNA

6. Describe the significance of the sequence 5'-CCA-3' at the 3' terminus of every tRNA. (6 points)

7. Describe the importance of eIF2 phosphorylation on translational control in eukaryotes. (6 points)

8. CRISPR-Cas9 system emerged as a powerful technology for genome editing. What are the CRIPSR and its mechanism? (8 points)

9. The following is the summary of one research article published in 2015. List two novel and important findings and give a title according to this summary. (10 points)

SUMMARY

Rett syndrome (RTT) is a neurodevelopmental disorder caused by *MECP2* mutations. Although emerging evidence suggests that MeCP2 deficiency is associated with dysregulation of mechanistic target of rapamycin (mTOR), which functions as a hub for various signaling pathways, the mechanism underlying this association and the molecular pathophysiology of RTT remain elusive. We show here that MeCP2 promotes the posttranscriptional processing of particular microRNAs (miRNAs) as a component of the microprocessor Drosha complex. Among the MeCP2-regulated miRNAs, we found that miR-199a positively controls mTOR signaling by targeting inhibitors for mTOR signaling. miR-199a and its targets have opposite effects on mTOR activity, ameliorating and inducing RTT neuronal phenotypes, respectively. Furthermore, genetic deletion of *miR-199a-2* led to a reduction of mTOR activity in the brain and recapitulated numerous RTT phenotypes in mice. Together, these findings establish miR-199a as a critical downstream target of MeCP2 in RTT pathogenesis by linking MeCP2 with mTOR signaling.

試題隨卷繳回