GCA EXAM 2010

Place your name and student number on top of every page

1) How do Moolenaar *et al* (EMBO (2001) vol.20 pp 6140-6149) conclude, without purification of the proteins, that the mutant $UvrB_{Y95+Y96}$ and $UvrB_{Y101+F108}$ proteins are disturbed in the repair of UV damage in DNA but (probably) not in the early step of the mechanism? (25) → artic
2) At different stages of the transcription cycle the RNA Polymerase (RNAPolII) binds different partners to carry out functions specific for that stage. How is this achieved? Describe an example. (50) → CTD
3) In literature one speaks of the 'Histone code'. What is the nature and the function of this code? (25) → artic
4) Describe two lines of evidence (from two different organisms) that DNA methylation might be initiated by Histone H3-Lys9 methylation. (25) → artic
5) In two silencing mechanisms, deacetylation of histones in yeast and in histone H3lys9 methylation in *drosophila*, the same basic principle is operational for spreading of these marks for silencing. Describe both mechanisms and the common basic principle. What is/are the problem(s) associated with this basic principle? (50)
6) Histone acetylation is associated with active chromatin. How might acetylation affect chromatin structure? (25) → essay
7) DSIF is somehow involved in Transcription Coupled Repair. Explain the experiment that led to this discovery. (25) → dS; F artic
8) Explain the sliding mechanism for chromatin remodeling. (25)