UNIVERSITY OF OSLO
Faculty of Mathematics and Natural Sciences

Exam in: MBV4160/ MBV9160 Advanced Cancer Biology
Day of exam: Friday 27th of February 2015
Exam hours: 10.00 – 13.00
This examination paper consists of 2 pages
Appendices: None
Permitted materials: None

You can achieve a maximum of 38 points in this written exam. Each module will be awarded the following points: I: 3, II: 6, III: 10, IV: 9, V: 7 and VI: 3.

Make sure that your copy of this examination paper is complete before answering

I Important definitions (3 points)

1) Within cancer research several important terms are used. Please define the following:

A) Cancer
B) Incidence
C) Prevalence
D) Prognostic marker
E) Predictive marker
F) Biomarker

II Tumor suppressors (6 points)

2) What is a tumor suppressor gene?

3) There are 6 mechanisms for how tumor suppressor genes can be inactivated. Shortly describe each of them.

4) TP53 is the 'superstar' of the tumor suppressors. Give an example of a *different* tumor suppressor gene and shortly describe how it contributes to cancer development.

III The cell cycle (10 points)

5) Describe the role of the E2F transcription factors in the cell cycle, and explain how their activity is regulated.

6) TP53 is activated by different types of cellular stress. Give four examples of cellular stress that may activate the TP53 protein

7) Describe the role of MDM2 and ARF ($p14^{ARF}$) in regulation of TP53, and briefly explain how dysregulation of MDM2 and ARF may contribute to cancer development.

8) Explain in brief why yeast -at the molecular level- is a suitable model organism for studying the cell cycle and its regulation.

IV Colorectal cancer: (9 points)

9) Approximately how many new cases of colorectal cancer do we have in Norway every year?

10) CpG island methylator phenotype (CIMP) is one of the three subtypes of colorectal cancer. Briefly explain what characterizes these tumors at the molecular level, and approximately how common this subtype is among colorectal cancer patients.

11) CIMP was recently suggested to be caused by a genetic event. Explain in detail the main 'cascade' leading to CIMP positive colorectal cancers.

12) CIMP tumors often have microsatellite instability (MSI). Briefly explain the following:

A) What is causing the MSI phenotype?
B) What is characterizing the MSI tumors at the molecular level?
C) How can these changes provide the tumor cells with a potential selective advantage?

V Lymphoma: (7 points)

13) According to the World Health Organization (WHO) there are at least 60 different types of lymphoma. What is the biological explanation for this vast number of molecularly and clinically different lymphoma types?

14) Approximately how many new cases of lymphoma do we have in Norway every year (when all types are combined)?

15) Where are the lymphomas located in the body?

16) Rituximab is an antigen-based therapy used for certain groups of lymphoma patients.

A) Which patients will respond to this treatment?
B) What does Rituximab target?
C) Will patients be cured by this treatment?
D) Is the drug specific for cancer cells?

17) Lymphomas are frequently caused by chromosomal translocations. The result of this on the protein level can be either qualitative or quantitative. Briefly explain the difference between these.

VI Personalized medicine and cancer treatment (3 points)

18) Cetuximab (also called erbitux, panitumumab and vectibix) is an EGFR inhibitor used to treat certain cancer patients.

A) How does this drug work?
B) At the molecular level, what characterizes the tumors of the patients that respond the best to this treatment?