

UNIVERSITY COLLEGE LONDON

University of London

EXAMINATION FOR INTERNAL STUDENTS

For the following qualifications :-

B.Sc. (Intercal)

Surgery 2: Cell Biology of Neoplasia

COURSE CODE : **SURG0002**

UNIT VALUE : **0.50**

DATE : **03-MAY-02**

TIME : **14.30**

TIME ALLOWED : **3 hours**

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UNIVERSITY OF LONDON
(University College London)

BSc Degree 2002

TUMOUR BIOLOGY SURG 0002 : CELL BIOLOGY OF NEOPLASIA

3rd May 2002: 14.30 to 17.30

Answer both Sections A and B: 5 questions from 2 sections

Please answer each Section in a separate answer book

You should allow about 1 hour for Section A.

One third of marks allocated to Section A

SECTION A Answer **ONE** of the following three questions

A1 What characteristics do cancer cells share with normal stem cells? Review the evidence which suggests that signalling paths controlling stem cell fate and tissue patterning in the embryo are also important for cancer development.

A2 Discuss whether apoptosis or cell proliferation is more important in tumorigenesis. Illustrate your reasoning with examples.

A3 Outline diagrammatically **one** named signal transduction path, from cell surface to the genome. Then

Either: Choose **two** cellular proto-oncogenes in this signalling path, then explain their normal functions and how they are altered in human cancers

or: choose **two** molecules from this signalling path, then explain why and how they are being targeted for cancer therapy.

SECTION B Answer **FOUR** of the following seven questions

B1 It is stated that the p53 gene is mutated in over 50% of human cancers. Why should this genetic change be so common?

B2 What is Knudson's hypothesis? How has it furthered our understanding of familial and sporadic carcinogenesis and what do you think are its limitations? Illustrate your answer with reference to specific cancer types.

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SURG 0002 SECTION B *continued*

B3 Explain the mechanism of action of retinoids. Why does *all-trans* retinoic acid produce remission in only some promyelocytic leukaemia patients?

B4 Summarise how you would make a transgenic knockout mouse. Give one example of the use of a knockout mouse in cancer research.

B5 What is TGF-beta? Outline its mechanism of action and give examples of its role in cancer.

B6 Discuss the molecular mechanism of action of a named DNA virus in relation to the aetiology of a specific human cancer.

B7 Starting with normal human cells in culture, you wish to produce a model of human carcinogenesis. Suggest:

- a) the essential genetic alterations you would make to the cells and why?
- b) how you would check at the completion of the study that you had made cancer cells

(no details of methodologies required for this question)

END OF PAPER