UNIVERSITY COLLEGE LONDON

University of London

EXAMINATION FOR INTERNAL STUDENTS

For The Following Qualification:-

B.Sc. (Intercal)

Surgery 2: Cell Biology of Neoplasia

COURSE CODE	:	**SURG0002**
UNIT VALUE	:	**0.50**
DATE	:	**04-MAY-04**
TIME	:	**14.30**
TIME ALLOWED	:	**3 Hours**

TURN OVER

UNIVERSITY OF LONDON
(University College London)

BSc Degree 2004

TUMOUR BIOLOGY SURG 0002 : CELL BIOLOGY OF NEOPLASIA

04 May 2004: 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 and 2 hours for Section B

One third of marks allocated to Section A

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

A1 Why is dysregulation of the wnt signalling path important in the development of most colorectal polyps? Explain the underlying molecular mechanisms involved and the cellular outcomes.

A2 Describe a signal transduction pathway which is utilised by a number of growth factor families to promote tumour growth and/or progression. For one growth factor family, discuss how alterations in ligands, receptors or downstream molecules have been implicated in growth and progression of named human cancers.

A3 What is apoptosis? Describe the apoptotic pathway and how it may escape from normal control in cancer. Why do you think loss of the ability to undergo apoptosis is undesirable for the patient in terms of (1) tumour progression and (2) response to assault by chemotherapeutic agents?

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

B1 What are autocrine and paracrine stimulation? Give examples of how these two types of stimulation contribute to tumour growth and progression.

B2 What are the principal effects of retinoids on epithelial cells and tissues? Outline how these effects are achieved at the molecular level.

B3 Describe the proposed molecular mechanisms of action of a **named** DNA virus in the aetiology of a **named** human cancer. What is the main difference between the way in which the virus operates at the molecular level and the ways in which oncogenic RNA viruses exert their effects in non-human species?

B4 How does the retinoblastoma protein interact with the cell cycle in normal cells and how do the actions of retinoblastoma become subverted in cancer? What is the fundamental genetic difference between the development of inherited and sporadic retinoblastoma?

B5 What signal transduction pathway does TGFbeta use? How does the growth factor oppose cell proliferation? Name two human cancers where loss of TGFbeta action is thought to contribute to oncogenesis.

B6 Write short notes on **two** of the following:
(a) sonic hedgehog
(b) HGF
(c) tumour suppressor genes predisposing to early onset breast cancer
(d) MDM2

B7 Explain why **two** of the following mechanisms are considered important in the progression of human breast carcinomas:
(a) gene amplification;
(b) hormone independent growth;
(c) mislocation of negative regulators of the cell cycle.

END OF PAPER