

UNIVERSITY COLLEGE LONDON

University of London

EXAMINATION FOR INTERNAL STUDENTS

For The Following Qualification:–

M.Sc.

M.Sc. Clinical Neuroscience: Paper 1

COURSE CODE : CLNEM001

DATE : 04-MAY-04

TIME : 10.00

TIME ALLOWED : 3 Hours

PAPER ONE

In *Part 1* of the paper, *answer three essay questions.*

You must answer:

one question from Section A (25 marks)

one question from Section B (25 marks)

one question from Section A or B (25 marks).

Allow yourself approx. 45 min per question.

In *Part 2* of the paper, *answer three short-answer questions* (8 marks each).

Allow yourself approx. 15 min per question.

- **WRITE ON ONE SIDE OF THE PAPER ONLY**
- **BEGIN EACH NEW QUESTION ON A FRESH PAGE**

Part 1

Section A

6 questions from Theme A (Cellular and Molecular Neuroscience)

1. Describe the current approaches to management of brain tumours, highlighting the limitations of each approach and explain how molecular genetics may influence the choice of treatment in the individual patient.
2. Discuss the synthesis and degradation of dopamine and serotonin.
3. Describe the central features of energy generation in the brain, and compare the different processes occurring in aerobic or anaerobic conditions, explaining the consequences for the pathophysiology.
4. Describe the advantages and drawbacks of transgenic mouse models of Alzheimer's disease (AD). Describe the common features of AD in humans and in mouse models as well as the differences between them. Explain the effects of the active immunization with A-beta peptide in mice and the problems in the trial in humans.
5. Is multiple sclerosis a neurodegenerative disease? What avenues for therapy can be learnt from other neurodegenerative conditions?
6. What are the prospects for repairing spinal cord injuries? Describe the experimental methods that have been applied to spinal cord injuries in rodents, and their effects on axon regeneration in the cord.

CONTINUED

Section B

6 questions from Theme B (Communication and Transmission)

7. According to Julius Bernstein (1871) “the ... potential of nerve is maintained by the difference in concentration of potassium ions on either side of a membrane selectively impermeable to anions, as expressed by the Nernst equation.” He further conceived of action potentials as “a traveling leak moving down the nerve” (1912). How do Bernstein's hypotheses need to be modified, to bring his explanations of resting and action potentials up to date?
8. What factors contribute to motoneurone degeneration following peripheral nerve injury?
9. Describe the clinical aspects, pathogenesis and management of diabetic neuropathies.
10. What mechanisms underlie short-term use-dependent plasticity of synaptic transmission? How is this altered in disorders of the neuromuscular junction?
11. “The pharmacokinetic mechanisms of antiepileptic drugs are important”. Discuss with examples.
12. Discuss the incidence, prevalence and mortality rates of epilepsy, and the factors which influence these rates.

Part 2

10 short-answer questions on Themes A and B

13. Identify five leading scientists in the study of genetics/molecular genetics and their main contributions to the field.
14. What role do astrocytes play in glucose provision for neurones in the brain?
15. Neuroactive drugs can act by affecting neurotransmitters, neuromodulators and neurohormones. Describe the differences between the three modalities
16. Describe the basic cellular structure and function of the blood-brain barrier and its role in brain metabolism.
17. Explain the role of voltage dependent calcium channels in the exocytosis of neurotransmitters.
18. Tabulate the molecular mediators of inflammatory demyelination and their proposed mechanism of action.
19. Discuss the two main mechanisms whereby the brain can communicate with the immune system.

PLEASE TURNOVER

20. From your knowledge of neuroanatomy and physiology, describe the consequences to a patient of a complete transection of the spinal cord in the upper thorax (around level T3).
21. Describe the functions of the myelin sheath.
22. What determines the reversal potential for a ligand-gated ion channel?

[End of paper]