# UNIVERSITY COLLEGE LONDON

University of London

## EXAMINATION FOR INTERNAL STUDENTS

For The Following Qualification:-

*M.Sc.*

**M.Sc. Clinical Neuroscience: Paper 1**

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| COURSE CODE | : | **CLNEM001** |
| DATE | : | **02-MAY-06** |
| TIME | : | **10.00** |
| TIME ALLOWED | : | **3 Hours** |




**TURN OVER**

**PAPER ONE**

***IMPORTANT:***

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

***Please ignore other instructions to the contrary***

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)**

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

**Part 1.** (Allow yourself approx. 45 min per question.)

**Section A**

*6 questions from Theme A (Cellular and Molecular Neuroscience)*

1. What is the endoplasmic reticulum? What does it do (give at least 3 examples)?

2. Describe the recent World Health Organisation grading of gliomas and explain the importance of such a grading system. What is the contribution of molecular genetics to this classification system?

3. Distinguish between pharmacokinetics and pharmacodynamics. Describe both of them, including related concepts, and explain how they are measured and in what way they are important.

4. Many Dementing Diseases are caused by abnormal protein depositions, which can also be described as "proteinopathies". All these proteins are necessary for normal cell function. Describe how aberrant processing of a normal cellular protein can lead to neurodegeneration. Please mention at least three proteins which play a key role in neurodegeneration, and which diseases they can cause.

5. Discuss whether inhibition of blood brain barrier dysfunction is beneficial in multiple sclerosis.

6. What are the common causes of acute infectious meningitis in the United Kingdom and which patient groups are at greater risk from these infections? Since the introduction of penicillin, what further therapeutic advances have been made in the management of acute infectious meningitis?

**Section B**

*6 questions from Theme B (Neural Transmission)*

7. What factors contribute to motoneurone degeneration following peripheral nerve injury?

8. Discuss the available treatments for myasthenia gravis and their rationale and effectiveness.

9. Which aspects of NMDA receptor-mediated long-term potentiation make it a candidate cellular substrate for memory encoding?

10. What are the defining symptoms of narcolepsy, and explain how some of these symptoms relate to the REM sleep state. What are the current theories concerning its aetiology in humans?

11. Discuss the main modalities of treatment for epilepsy and the indications.

12. Discuss the possible neurobiological consequences of a single seizure.

**Part 2** (Allow yourself approx. 15 min per question.)

*10 short-answer questions on Themes A and B*

13. Briefly summarise what is known about the genetics of trinucleotide repeat disorders

14. List the potential cellular sources of ATP which can be activated during an energetic "shortage", apart from the oxidation of metabolic fuels.

15. Briefly review the criteria that need to be met before a chemical can be called a neurotransmitter.

16. What features support the hypothesis that multiple sclerosis is an autoimmune disease?

17. List ways in which nitric oxide could be beneficial or damaging in the formation of lesions in the CNS.

18. The old "hot bath" test for multiple sclerosis involved immersing the person in a bath of warm water. What does this do to MS patients, and why?

**CONTINUED**

19. Generalized Epilepsy with Febrile Seizures + (GEFS+) and Severe Myoclonic Epilepsy of Infancy (SMEI) both are linked to mutations in the same gene. But they are different disorders. How are they different, and what is it about the mutations related to each disorder that might explain the different manifestations?

20. Outline the principal ways calcium is removed from the cell.

21. What are the different roles of dendritic and somatic inhibition, how do these forms of inhibition change in epilepsy and how may this contribute to the pathophysiology?

22. List the possible neurological consequences of chronic epilepsy.

[End of paper]