

UNIVERSITY COLLEGE LONDON

University of London

EXAMINATION FOR INTERNAL STUDENTS

For The Following Qualification:–

M.Sc.

M.Sc. Clinical Neuroscience: Paper 3

COURSE CODE : CLNEM003

DATE : 10-MAY-04

TIME : 10.00

TIME ALLOWED : 3 Hours

PAPER THREE

12 general questions. Answer two questions only. Each question carries 50 marks. Allow about 90 min for each question.

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

1. Outline the main risk factors for stroke and discuss the strategy you would recommend to a minister of health to reduce the incidence of stroke.
2. Mutations of the genes encoding subunits of the mitochondrial respiratory chain are an important cause of disease. Discuss.
3. From "fuels" to ATP: describe the key points of the major metabolic pathways used in brain energy generation: regulation steps, interconnection between major pathways, and peculiarities of the energy metabolism in the brain at the organ, cell and subcellular level.
4. Discuss the epidemiology of multiple sclerosis, arguing for and against an environmental cause of the disease.
5. During development mammals become progressively less able to repair damage to their nervous system. Young animals have considerable plasticity in their cortex and elsewhere, but this becomes restricted at the end of the critical periods. What do you know of the mechanisms behind the lack of regenerative ability and plasticity in the adult mammal? Can you think of any reasons why it might be of benefit to restrict regeneration and plasticity?
6. AMPA and NMDA receptors are said to play complementary roles in long-term plasticity of glutamatergic synapses. Discuss.
7. "Functional imaging methods such as fMRI and PET can only tell us that the brain is active but can never tell us what it is doing". Critically discuss.
8. Give an outline description of the neurological control of the bladder, illustrated by examples of neurological diseases that can cause incontinence.
9. Several inherited neurological disorders are caused by expansions of CAG repeats (encoding polyglutamine tracts) – compare and contrast these disorders – in what ways are they fundamentally similar to each other and conversely how do they each differ?
10. How has evidence from neuropsychology and neuroimaging converged and/or diverged regarding the functional neuroanatomy of human memory?
11. Discuss experimental and clinical evidence suggesting that emotions, not conscious reasoning, govern behaviour.
12. What are the therapeutic approaches that can be taken in prion disease? Discuss the rationale for tested and potential future strategies.

[End of paper]