DEPARTMENT OF PSYCHOLOGY

SP611 The Neuroscience of Cognitive Disorders 2008-09

Module Convenor

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Module Tutors

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Time and Location

Spring Term: Thursday 9.00am -11.00am Keynes Lecture Theatre 4.

Summary of Intended Learning Outcomes

- Knowledge and understanding of how core concepts, theories and findings in the study of cognition have been applied to broader neuroscientific and clinical contexts.
- Critical evaluation of how current research has advanced our understanding of neurological disorder and its treatment.
- Ability to express well-founded opinions and personal perspectives in both written and oral form.

Introduction

This module will build upon the cognitive theories and research methods explored at stages 1 and 2. It will focus on several forms of neurological deficit each of which affects a different domain of cognition. Students will learn about how different strands of neuroscientific research, relating to behaviour, cognition, anatomy, and physiology, have both advanced our understanding of human neuropsychology, and informed on the design of relevant intervention strategies.

Assessment

Your attention is drawn to the following information to be found on the departmental website:
• Regulations relating to coursework deadlines
  www.kent.ac.uk/psychology/studying/studyskills/assessment.htm#deadlines
• Guidelines on plagiarism and duplication of material
  www.kent.ac.uk/psychology/studying/studyskills/plagiarism.htm
• Guideline Criteria for Assessment of Stage 2 and 3 Undergraduate Work
  http://www.kent.ac.uk/psychology/studying/studyskills/criteria2.htm

Assessment

• A short-answer written exercise in which students must answer questions about
  core principles and concepts covered in the course. This form of assessment
  will constitute 20% of the final mark and must be submitted with the extended
  essay (see below).

• An extended essay (maximum of 4,000 words) must be submitted by 12 noon
  on Monday 11th May (first day of summer term). This form of assessment will
  constitute 80% of the final mark.

Information about marking and feedback is at:
www.kent.ac.uk/psychology/department/literature.htm.

Short-answer questions:

Please note that each answer should be no more than 250 words.

1. Do neuropsychological dissociations support stronger theoretical inferences than
   neuropsychological associations?

2. Is the study of a single patient more informative than the study of a group of
   patients?

3. What is a stroke and what are the main risk factors?

4. Why does the study of brain-damaged patients help us understand how cognitive
   processing is organised in the normal, healthy brain?

5. What problems stand in the way of inferring normal function from the study of
   brain-damaged patients?

6. For a cognitive disorder of your choice, compare the theoretical bases of two
   different types of treatment.

7. Compare the effectiveness of the two treatment types that you chose in question
   6 above.

8. What is neuro-plasticity and why is it relevant to neuro-rehabilitation?

Students must choose one of the following five dissertation titles:

1. How effective are the treatments for hemi-spatial neglect and what does this tell us about the nature of the disorder?

2. Discuss three recent scientific papers that deal with aphasic patients (or similar) that can be interpreted as evidence for or against the lemma versus word form distinction that is central to Levelt's theory of speaking.

3. Contrast two different forms of amnesia and discuss what they tell us about how physiological and cognitive processes are related.

4. Drawing on data from both behavioural and brain-imaging studies, contrast two types of acquired dyslexia in the light of dual-route versus parallel distributed processing (PDP) models.

5. Our understanding of the normal process of face recognition can be extended through the study of when this process fails. Discuss with reference to commonplace errors of everyday life and pathological problems which may arise through illness or injury.

Module Evaluation

You will be asked to provide feedback on this module by completing a module evaluation questionnaire. The questionnaires will be considered at a special meeting of the departmental Learning and Teaching Committee and the minutes of the meeting will be published on the departmental website.

Texts

Main

Recommended

Teaching Programme

You should note that attendance at lectures, seminars and supervisions, and the submission of written work, are obligatory. For further information see the Faculty of Social Sciences Stage 2 and 3 Handbook.

Lecture Outlines

Week 13
Lecture 1 (Dr. David Wilkinson): Principles of Neuropsychological Research
This lecture will introduce students to the basic principles and assumptions of neuropsychological investigation.

Reading
Further readings will be provided on the lecture hand-out

**Weeks 14 and 15 (Dr. David Wilkinson): Hemi-spatial neglect**  
Lecture 2: Cognitive and Biological Bases.  
Lecture 3: Treatment Approaches.


Further readings will be provided on the lecture hand-out

**Weeks 16 and 18: (Dr. Dirk Janssen): Aphasia**  
Lecture 4: Cognitive and Biological Bases.  
Lecture 5: Treatment Approaches

(Note: Week 17 is reading week)

In these two lectures, we will discuss various types of aphasia, what causes these aphasias and how we can promote recovery. Many acquired aphasias can be classified by looking at what the major deficit is: A predominant loss of language comprehension (understanding) is common for Wernicke’s aphasia, whereas a predominant loss of language production (speaking) is common for Broca’s aphasia. However, the relationship of these two types of aphasia to the brain lesions and anatomy is very tentative. Recovery from aphasia depends strongly on the type, the severity of the lesion and the fitness of the patient. Case studies will be examined to demonstrate this.

Reading:  
- The relevant parts of the 'language' chapter in Martin (2006).

**Weeks 19 and 20 (Dr. Ulrich Weger) Amnesia**  
Lecture 6: Cognitive and Biological Bases.  
Lecture 7: Treatment approaches.

Amnesia – the loss of memory – is a remarkable phenomenon that not only influences day-to-day interactions between individuals and their environment but also appears to affect the identity of a person. Two broad categories of amnesia are often discriminated – retrograde and anterograde amnesias, referring to the onset of the memory loss (before or after the brain damage). However, the sources of memory loss are diverse (for example infections, epilepsy, trauma) and so are the
symptoms. In the first lecture we will discuss the broad phenomenology of memory loss, including the physiological foundations, sources and symptoms. In the second lecture we will continue to look at the symptoms and behavioural implications of memory loss and become familiar with approaches to treatment.

**Essential Reading**


**Weeks 21 and 22 (Dr. Nicolas Dumay): Acquired Dyslexia**

**Lecture 8: Cognitive and Biological Bases.**

**Lecture 9: Treatment Approaches**

This part of the course will introduce students to the syndromes of alexia and acquired dyslexia, i.e., the (partial) loss of the ability to decode written inputs consecutive to a brain lesion. In the first lecture, cases will be presented and used to make inferences about the neurocognitive architecture of the reading system. The accent will be put on both the neural aspects of the syndromes and on the distinction between the classical dual route versus parallel distributed processing accounts. In the second lecture, strategies of compensation and remediation will be discussed.

Further reading:


**Weeks 23 and 24 (Prof. Robert Johnston): Prosopagnosia**

**Lecture 10: Problems with recognising faces:** This part of the course will introduce students to problems associated with recognising faces. We will look at data from patients with pathological difficulties in recognising faces and see how these compare with errors experienced by unimpaired individuals on a daily basis and difficulties seen in special, but non-pathological populations, such as older adults. We will examine how the study of when face recognition fails can inform our
accounts and theories of the proper functioning of face recognition. We will look at how patterns of impaired recognition performance have been incorporated into computational models of face recognition.

**Lecture 11: Problems with processing faces in non-recognition tasks:** Prosopagnosia is a label used loosely to describe pathological problems in recognising familiar faces. However, recognition is only one of several processing activities that we apply to faces. We also regularly perform such judgements as analysing facial expression, interpreting facial speech and even classifying faces according to age or sex. These processes are also prone to pathological impairment after disease or trauma. In this lecture we will try to understand how the study of impairment can help us to understand how these processes occur normally and how they interact with each other and with face recognition.

Further reading:

