NTNU
Kunnskap for en bedre verden

Psykologisk institutt

Eksamensoppgave i PSYPRO4412 – Anvendt og klinisk kognitiv psykologi

Faglig kontakt under eksamen: Robert Biegler

Tlf.: 73 59 19 60

Eksamensdato: 2. juni 2016

Eksamenstid: 09:00 – 13:00

Hjelpemiddelkode/Tillatte hjelpemidler: Ingen

Målform/språk: Bokmål

Antall sider: 2

Kontrollert av:

_____

Dato Sign

Langsvarsoppgaver

Besvar 2 av 3 oppgaver

Hver oppgave teller 40 % av den endelige karakteren

1. Hva er forskjellene mellom standaradtilnærmingen til ekstinksjon, gradvis ekstinksjon, maksimere prediksjonsfeil (prediction error) og rekonsolidering.
   Er de underliggende teorier kompatible med hverandre?

2. Hvilke kognitive bias ser man særlig ved schizofreni?
   Gjør rede for hvordan disse kan gjøres til gjenstand for behandling.

3. Hvor pålitelig er minnet, og hvilke mekanismer vet du som kunne produsere falske minner?

Kortsvaroppgaver

Besvar 1 av 2 oppgaver

Oppgaven teller 20 % av endelig karakter

4. Hva er "attribution of agency", og hvorfor er det relevant for kontrollvrangforestillinger?

5. Definer fornyelse (renewal), spontan gjenoppretting (spontaneous recovery), gjeninnsettelse (reinstatement) og oppvåkning (resurgence).
   Hva er deres felles underliggende basis, og hva er deres relevans for klinisk psykologi?

Long questions (Choose two of three, each counts as 40% of the final grade)

Question 1: What are the differences between the standard approach to extinction, gradual extinction, maximizing prediction error, and reconsolidation? Are the underlying theories compatible with each other?

Question 2: Hvilke kognitive bias ser man særlig ved schizofreni? Gjør rede for hvordan disse kan gjøres til gjenstand for behandling.

Jumping to conclusions: these are the quick decisions based on little evidence that we discussed in connection with Moritz et al.'s metacognitive intervention programme. This is often measured by the beads or urn task.

Attributional style is the tendency to attribute events to internal or external sources, meaning one's own effort versus what other people do or the universe in general. Moritz et al. write: "While there is consensus that patients display attributional biases, its exact signature is subject of an ongoing controversy. Whereas early research found evidence for a self-serving bias in the disorder (attribution of success to oneself, attribution of failure to others or circumstances),some newer findings point to a tendency to externalize both personal positive and negative events, which may foster subjective powerlessness and could give rise to feelings of alien control. More recently, an excess of monocausal inferences was detected in schizophrenia patients, that is, patients did not contemplate multiple sources but converged onto single explanations more often than healthy persons.

Metamemory is judging how reliable and precise your memories are. Asking "is this the person who attacked you?" is a memory question, while asking "how sure are you that this is the person who attacked you?" is a metamemory question. Moritz et al. write: "There is increasing evidence for **overconfidence in memories**. Many studies revealed that this overconfidence was especially present for incorrect or false memories, while confidence for correct responses was often found to be lower than that of controls."

Bias against disconfirmatory evidence (BADE). "Using visual and verbal material, it was demonstrated that schizophrenia patients are far more easily 'led up the garden path' for initially strongly suggested interpretations, which, however, are later discouraged by accumulating evidence."

Theory of mind: schizophrenia is associated with poorer social reasoning, though possibly for different reasons than in autism. One proposal is that the problem in autism is **hypomentalising**, i.e. not enough perspective taking, not enough attribution of intention. In contrast, the problem in schizophrenia may be **hypermentalising**, too much attribution of intention. That can be seen in the study by Blakemore et al. (2003, cited by Frith), in which delusional patients were more willing to attribute intention to moving geometrical shapes than controls. In other studies with moving shapes (not in the pensum, but students may mention this), people on the autistic spectrum are less willing to attribute intention. I am not sure that theory of mind processing should be called a cognitive bias, but one aspect of it, namely how easily someone attributes intent, is a cognitive bias.

One approach to treatment is Moritz et al.'s metacognitive intervention programme. Its basic principle is to present stimuli, often in the form of cartoons or photographs, and several possible

interpretations. The initial stimuli are designed so as to bias observers towards one interpretation. Further, the stimuli are designed so that the initially favoured interpretation is reasonable, based on the available information. Then more information is added, which eventually quite clearly favours a different interpretation. Through a large number of such exercises, people are taught that initial impressions can be misleading. Moritz et al specifically state that schizophrenia patients tend to favour explaining events in terms of single causes, rather than multiple possible causes. The intervention is designed to counter that by making people switch from an initially more plausible explanation to another. Although not explicitly stated, it appears to be an important principle that the initial misinterpretation should seem reasonable to most people, and that its intuitive appeal should not depend on being delusional. Reasons may be both that patients differ in the content of delusions, and that the intervention may be more effective if errors and error correction are portrayed as normal parts of cognition.

Metacognitive intervention seems suitable to reduce problems with large perceived prediction errors, as discussed by Frith. I see nothing in metacognitive intervention that looks like it might reduce the bias towards attributing intention, though if a student can make a good argument, that is fine. Not being a clinician, I may have missed something.

Question 3: How reliable is memory, and what mechanisms do you know that could produce false memories? Hvor pålitelig er minnet, og hvilke mekanismer vet du som kunne produsere falske minner?

Geraerts et al. divided memories of childhood sexual abuse into continuously remembered (there was never a time when the person reporting abuse had forgotten the abuse) and discontinuous, and they divided the discontinuous memories into those remembered within or outside of therapy. Geraerts et al. do not mention whether the therapy in question was specifically recovered memory therapy or a broader range of therapies. In this study, *none* of the memories recovered in therapy was corroborated by independent evidence, while memories recovered out of therapy were as likely to corroborated as continuous memories (those that had never been forgotten). The best predictor of the existence of corroborating evidence was being surprised by the memory. Memories that were corroborated also tended to be recovered suddenly, rather than gradually.

Shaw and Porter interviewed subjects three times, asking them both about an emotionally salient event that (according to the subjects' parents) they really had experienced, and about a fictitious crime. Shaw and Porter were able to persuade 70% of subjects that they had committed a crime serious enough that police had gotten involved. Their interviews used tactics that had elicited false confessions in previous research, such as presenting false evidence, social pressure, building rapport, suggestive retrieval techniques including guided imagery, leaving pauses for the subjects to fill, and implying that experimenters knew further details.

The finding that corroborated discontinuous memories were recovered more suddenly and were more surprising than uncorroborated (and therefore more likely false) memories suggests that false memories are built up slowly. There are three candidate mechanisms within the pensum. One is reconsolidation. Hupbach et al demonstrated that episodic memory can be altered through reconsolidation. They showed people a list of objects on a Monday. On Wednesday, the

reconsolidation group was asked to remember what they had done on Monday. Then they were shown a second set of objects. One control group saw the same second set of objects, but without being reminded of the previous set. Those two groups returned on Friday, joining the second control group. The experimental group encoded more Wednesday objects along with the Monday objects of which they had been reminded than the control group without the reminder. The Monday & Friday only group provided a baseline of spontaneous intrusions without exposure to the Wednesday objects. The intrusions were asymmetric. The experimental group did not include Monday items in their recall of the Wednesday items. The increased intrusions were of Wednesday items seen at a time when the Monday items were re-encoded after the reminder. Reconsolidation this provides a mechanism by which memories may altered, rather than merely new memories being established that may compete with the old ones. That matters in the context of false memories. Depending on the circumstances in which a false memory is established, the original information may be overwritten.

An alternative method of altering an existing memory is to change the probabilistic relationship between a conditioned and an unconditioned stimulus gradually. The theory behind this approach is that a sudden change in the relationship between conditioned and unconditioned stimulus acts like a context change and triggers new learning, represented in a separate, parallel association. Gradual change in the relationship targets the original association. It is unclear whether this would generalise from conditioning to episodic or semantic memory.

A sudden change in the CS-US relationship is more likely to generate new, parallel associations. In the case of conditioning, there would be no explicit information regarding the source of the information, and it would be impossible to tell directly which is the original memory.

The pensum in this course does not contain any studies that attempt to establish how much each of these mechanisms may contribute to any particular false memory. All that matters here is that students understand the distinct mechanisms, that two of them modify an original memory, while one establishes a new memory that is stored in parallel with the old, and that papers in the pensum only provide evidence for reconsolidation affecting episodic memory.

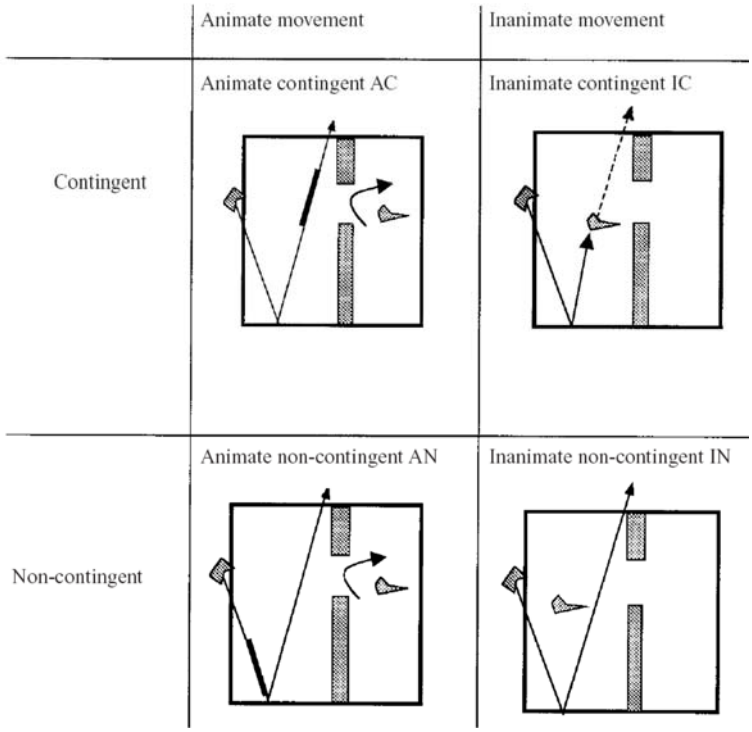
Short questions (Choose one of two, each answer counts as 20% of the final grade)

Question 1: Hva er ″attribution of agency″, og hvorfor er det relevant for kontrollvrangforestillinger? What is attribution of agency, and why is it relevant to delusions of control?

Attribution of agency is explaining events as the consequences of the actions of an agent, that is someone who has intentions. Frith argues that excessive attribution of agency is needed to explain delusions of control (the belief that someone other than the patient controls the patients actions or thoughts) because problems with the forward model are not a sufficient explanation.

A forward model predicts the motoric and sensory outcomes of motor commands. A large enough deviation from that prediction can reasonably be interpreted as indicating an external causal factor that affects the outcome. If the forward model is faulty and produces larger prediction errors than justified, then deviations from prediction that actually are small and random can be interpreted as indicating an external influence. The anarchic hand phenomenon, caused by lesions to supplementary motor cortex or anterior corpus callosum, demonstrates that problems with the forward model can only explain the feeling of not controlling one's own actions, but not the feeling that someone else controls one's actions. Anarchic hand patients feel one of their hands has a will of its own, but not that someone else controls it.

Excessive attribution of agency means seeing evidence of intentional action where the general population sees less or none. The most intuitive data presented to the students comes from an experiment on moving geometric shapes. Subjects were shown all four combinations of animate and inanimate shapes (they move under their own power or only passively, like billiard balls) and contingent (the shapes interact) versus non-contingent.



The question was whether there would be an interaction between animation, contingency and diagnosis. The difference between inanimate contingent and inanimate non-contingent provides a baseline, and was similar in delusional patients and controls. However, delusional patients perceived

no difference between animate contingent and animate non-contingent conditions, attributing intentions to the animate-non-contingent movements that controls did not. When this exaggerated attribution of intention to some outside agency is combined with not feeling in control of one's own movements or thoughts, the result is to attribute control to someone else. That is a delusion of control.

Question 2: Define renewal, spontaneous recovery, reinstatement and resurgence. What is their common underlying basis, and what is their relevance to clinical psychology? Definer fornyelse (renewal), spontan gjenoppretting (spontaneous recovery), gjeninnsettelse (reinstatement) og oppvåkning (resurgence). Hva er deres felles underliggende basis, og hva er deres relevans for klinisk psykologi?