NTNU
Kunnskap for en bedre verden

Institutt for psykologi

Eksamensoppgave i PSYPRO4412 – anvendt og klinisk kognitiv psykologi

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Informasjon om trykking av eksamensoppgave
Originalen er:
1-sidig ☐ **2-sidig** ☐
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Langsvarsoppgaver. Velg to av de tre spørsmålene under. Vekting 40%

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

2. Hvor pålitelig er minnet, og hvilke mekanismer kjenner du til som kan produsere falske minner?

3. Er schizofreni og autisme motsatte ender av det samme kontinuum?

Kortsvarsoppgaver. Velg en av de to spørsmålene under. Vekting 20%

1. Hva er «attribution of agency» og hvorfor er det relevant for kontrollvrangforestillinger?

2. Hva er forskjellen mellom «actor-critic learning» og «Q-learning», og hvorfor spiller denne forskjellen noen rolle?

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

Question 1:

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 2: 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.

Question 3: Are schizophrenia and autism opposite ends of the same continuum?

Abu-Akel et al. tested how performance in a perspective-taking task relates to both autism and psychosis. They found few errors in people with low tendencies towards either condition, increasing errors as either tendencies towards autism or towards psychosis increased, but people with strong tendencies towards both autism and psychosis showed low error rates again. On the one hand, this suggests that autism and psychosis are opposites *something* that leads to problems with perspective taking, and whatever it is tends to cancel out in people who have equally strong tendencies towards both. On the other hand, if autism and psychosis were opposites in all ways, people with tendencies towards both would simply not exist.

The pensum makes no direct statement as to what the dimension is in which autism and psychosis could be opposites. Students can only go through a list of possibilities and state what they would have to look up or find out.

Frith explains delusions of control partly by misjudging prediction errors, but also partly by being overeager to attribute agency, that is to treat impersonal processes as evidence of someone acting with intent. Student may be able to work out that too little attribution of intent in autism would be a plausible candidate for something that makes autism the opposite of psychosis.

Uhlhaas and Singer mention that schizophrenia patients show reduced beta and gamma-band responses. Happé et al mention EEG abnormalities, but don't say in which band or in which part of the brain.

Frith mentions reduced synchronisation between auditory and motoric areas when speaking in schizophrenia patients with delusions. As Jensen and Colgin explain, synchronisation is a marker of information being exchanged, so reduced synchronisation means less information exchange in deluded patients than control between motoric areas that predict the sound of one's own voice and the auditory areas that perceive it. This lack of information exchange can lead to prediction error, one's own voice being unexpected to the point of being perceived as someone else's voice. The better students may realise that there is a contradiction between three of the papers in the pensum. Uhlhaas and Singer report reductions in high frequency oscillations in schizophrenia, Jensen and Colgin explain that high frequency oscillations are useful for regulating information exchange at short distances, but because of the delays imposed by not so very fast nerve conduction, long-range coordination has to be achieved by lower frequency oscillations. The abnormally reduced communication between motoric and auditory areas in deluded patients is over a long range connection, so it can't be high frequency.

Wieki's review summarises a study by Gold, which dissociated habit (actor-critic or S-R) learning from Q-learning (learning that includes a representation of outcome, as in S-(R-O) associations), and also dissociated learning from positive versus negative consequences. They found that patients with negative symptoms were less able to represent positive outcomes. That would explain reduced motivation. However, the opposite of that would be being more sensitive than normal to positive outcomes, a trait associated with gambling, impulsivity and psychopathy, not autism.

Two other core symptoms of autism, besides social difficulties, are communication difficulties and repetitive behavior and narrow interests. None of the descriptions of psychosis in the pensum (or that I have seen elsewhere) mention an unusually wide range of interest, or exceptionally good communication. If we can assume that clinicians would have noticed unusual traits even if they are not a problem, then in the behavioural domain autism and psychosis could only be opposites in social behaviour.

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

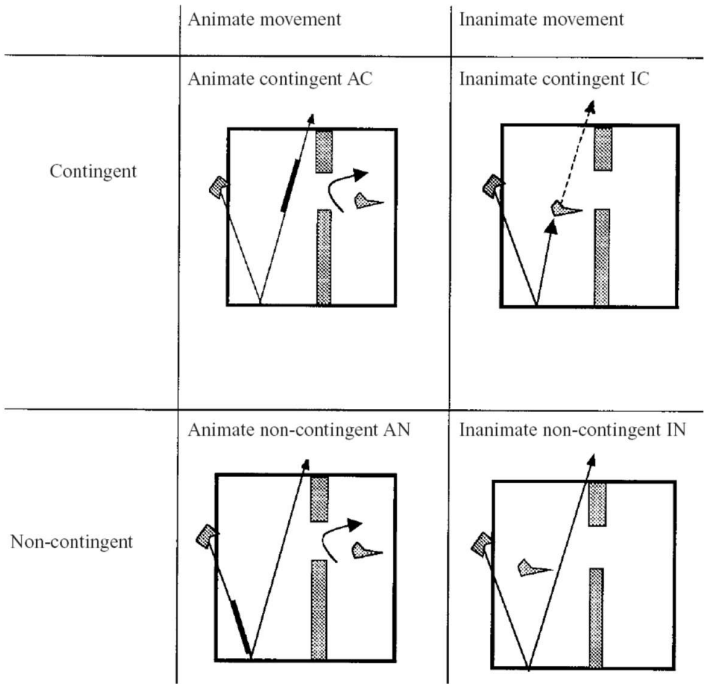
Question 1: Hva er ″attribution of agency″, og hvorfor er det relevant for kontrollvrangforestillinger? What is attribution of agency, and why is it relevant to delusions of control?

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 combination 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: What is the difference between actor-critic learning and Q-learning, and why does that matter?

In actor-critic (model-free, stimulus-response, habitual) cognition, you choose whatever response has been best rewarded in the past in this situation, while this stimulus was present.

For example, if you learn to find your way around by actor-critic learning, you just know that in the presence of *this* stimulus (this place), you choose the response of going in *this* direction, without

knowing where that will take you, what you will find at the end of this chain of stimulus-response associations, or how long it takes to get there. Or you might habitually prepare a food that you don't like any longer because the last time you ate it, you became violently ill. Stimulus-response learning does not represent the outcome, and so it is not immediately sensitive to changes in the value of the outcome. Only new (and slow) learning can change the response.

In Q learning (model-based, response-outcome or stimulus-(response-outcome) or goal-directed cognition), experience is compiled into a (possibly hierarchical) generative model of the world—a mechanistic, causal understanding of the causes and consequences of actions and events. When faced with a particular situation, this model can be searched, and the quality of various behaviours deduced—even if they have never been tried or experienced. As this involves somehow simulating or inferring future possibilities, it can have high computational costs.

For example, if you learn to find your way around by associating a stimulus with a response-outcome association, meaning you represent the *value* or *quality* of the outcome, you can plan your path. If you are in the presence of *this* stimulus (you are *here*), you can say that if you perform *this* response (go in *this* direction) you will experience *this* outcome (end up in *this* place). Then you can take this imagined outcome as the stimulus for a new stimulus-(response-outcome) association, and so simulate a whole chain of actions.

One reason why the difference matters is that Q-learning, which represents the outcome, is immediately sensitive to changes in the value of the outcome. Therefore persuading client that an outcome is not desirable should quickly change behavior. The actor-critic learning system, however, will need to be retrained by actively monitoring habits (mindfulness) and substituting good new habits for the old, bad habits until the new habits stick.

Further, specific impairments have distinct outcomes. Impairments in representing positive outcomes in Q-learning is associated with negative symptoms in schizophrenia, while giving excessive weight to positive outcomes is associated with one form of impulsivity.