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Department of Psychology

**Examination paper for PSYPRO4412**  
**Applied and Clinical Cognitive Psychology**

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## **ENGLISH:**

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

Question 1: Discuss how Frith explains hallucinations and delusions in schizophrenia.

Question 2: Which cognitive biases are seen especially in schizophrenia, and how can they be treated?

Question 3: How reliable is memory, and what mechanisms do you know that could produce false memories?

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

Question 1: How do the effects of conventional extinction training differ from those of extinction within an hour of being exposed to the conditioned stimulus?  
What does that mean for clinical practice?

Question 2: How and to what extent is it possible to distinguish between true recovered memories and false memories?

## **NORWEGIAN:**

### **Langsvarsoppgaver – velg 2 av 3 – hver oppgave teller 40 %**

Spørsmål 1: Gjør rede hvordan Frith forklarer hallusinasjoner og delusjoner i schizofreni.

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

Spørsmål 3: Hvor pålitelig er hukommelse og hvilke mekanismer vet du om som kan produsere falske minner?

### **Kortsvarsoppgaver – velg 1 av 2 – den teller 20 %**

Spørsmål 1: Hvordan kan effekten av konvensjonell ekstinksjon skiller seg fra ekstinksjon innen en time for å bli utsatt for den betinget stimulus?  
Hva betyr det for klinisk praksis?

Spørsmål 2: Hvordan, og i hvilken grad er det mulig å skille fra hverandre autentiske og falske gjenkalte fortrenge minner?

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

**Question 1:** Gjør rede for nevrokognitive aspekter ved smerte og subjektive somatiske symptomer. (Discuss the neurocognitive aspects of pain and subjective somatic symptoms.)

A basic concept here is that pain is not only a function of tissue damage, but does and should depend on other factors as well. Among these are attention, interpretation and learning. Attention is directly relevant in situations when the long-term cost of injury is less than the risk posed by a greater threat. You climb a tree if a lion is after you, never mind the torn fingernails and twisted ankle. Feeling pain at such moments would only be a distraction, so it would be useful if such signals could be modulated, even down to zero. Melzack's gate control theory postulates the dorsal horn of the spinal chord does not just passively transmit signals to the brain, but that it also receives top-down modulation that can enhance, weaken or block pain signals. The interpretation of pain signals should also modulate how much attention pain receives compared to competing information. The same pain signal merits different degrees of attention depending on whether it is interpreted as either a minor injury that will pass or as a sign of a developing serious long-term problem. That interpretation, in turn, should depend on past experience, and therefore learning. If a particular pain in the past turned out to indicate a minor injury that healed quickly, that will bias the interpretation and therefore attention in a different way than if past experience indicates a serious problem. Instead of this interpretation of pain indicating a catastrophic event, Stiles & Vættem also mention that negative evaluations, such as "life is no longer worth living" would induce depression and also increase the experience of pain. These are primary evaluations. Secondary evaluations concern how manageable people expect the underlying problem to be.

A further effect of learning comes from it being more useful to *prevent* aggravating an injury rather than to start making it worse, then stop because it hurts. Therefore pain may be felt already when moving in ways that predicted imminent pain in the past. That would be classical conditioning. Such learning *might* plausibly be involved in chronic pain. Stiles & Vættem distinguished between nociceptive pain triggered by activation of pain receptors, neuropathic pain triggered by damage to the peripheral or central nervous system, and idiopathic pain, which is anything else where the cause is unclear but nociceptive and neuropathic is contraindicated.

Stiles and Vættem also mentioned operant conditioning. They suggested that a response to pain could make pain more noticeable, and this attention could increase pain. Avoidance learning could also keep on producing responses long after the cause of pain is gone, because what reinforces avoidance learning in this case is the absence of pain. For example, patients may avoid previously painful activities long after an injury has healed. Therapy would aim to restore normal activity.

That is the general background, and students may choose to focus then on how therapy can intervene in these processes. Relaxation helps, and so does providing patients with a framework to recognise negative thoughts and their consequences. Because avoidance learning does not easily extinguish spontaneously, manageable exposure to previously painful movements can offer the unexpected experience that does lead to extinction.

Students may also or instead choose to go further into the anatomical details of where pain signals are modulated, where the afferent pain signals and the efferent modulating signals come from, and what role hormones play.

**Question 2:** Diskuter ulike behandlingstilnæringer til schizofreni. Legg vekt på kognisjon.

There are two aspects of cognition that students can focus on. These are the general cognitive impairments that often accompany schizophrenia, and the cognitive biases which are targeted by the metacognitive intervention of Moritz et al.

Ueland reviewed impairments in attention, memory and executive functions. Treatment consists of training in either paper and pencil exercises or tasks presented on a computer. Some of that training is simply what anyone could do. As with all such training, there is a question how much it generalises beyond the specific task. Some training is adapted to the specific situation. Ueland reports that trying to increase motivation by offering material reward had little effect. Memory problems can benefit from error-free learning, in which the initial training stimuli are very easy, so that it is easy to guess the correct response. As performance improves, this extra support is gradually reduced. Ueland reports improvements in attention, memory and executive function, but in the absence of transfer tasks, it is not clear to me whether cognitive capacity increased, or only performance on this specific task. Ueland mentions that a clinical study found that training a variety of strategies to process information, to circumvent the cognitive problems that accompany schizophrenia, and to deal with everyday problems improved mental flexibility and memory, but not symptoms or social functioning.

The basic principle of Moritz et al.'s metacognitive intervention programme 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.

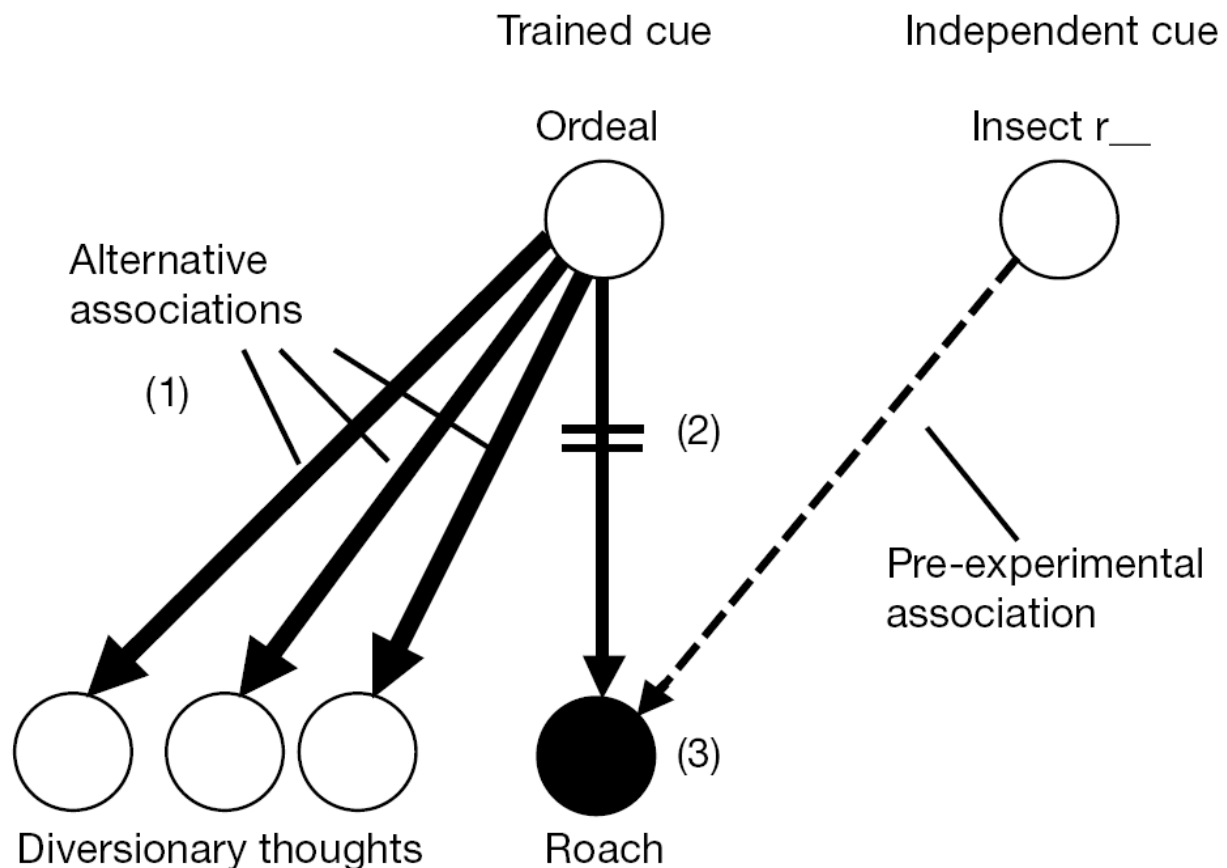
Metacognitive intervention seems suitable to reduce problems with large perceived prediction errors, as discussed by Frith. I see nothing in metacognitive intervention that looks like it might reduce the bias towards attributing intention, though if a student can make a good argument, that is fine. Not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

The ideal response would compare the two approaches and discuss differences in what they try to and can achieve. However, no such comparison was presented in lectures. It is also acceptable for a student to focus on one of these approaches, and discuss it in some more depth.

**Question 3:** Hva er rekonsolidering og hvorfor er det viktig? (What is reconsolidation and why does it matter?)

Reconsolidation is the phenomenon that when a memory is retrieved, it becomes labile and needs to be encoded again. Interference with that new encoding weakens the memory. If other information is stored at the same time, it may be stored with the retrieved memory.

Although not in the pensum, it may help students if they refer here to a figure by Anderson and Green which illustrates three different mechanisms of retroactive interference: creating alternative associations, altering the original association, and inhibiting the target memory.



Interference with new encoding can be implemented by drugs, for example propranolol, or purely behavioural procedures in which subjects are reminded of the original information, then new information is presented in a time window that covers 10 minutes and one hour but is shorter than six hours. If extinction occurs outside this time window, or if there is no reminder, then extinction leads to alternative associations. That can be seen in the spontaneous recovery from extinction of fear conditioning and the context-specificity of fear conditioning (Monfils et al.). It is only possible to restore the fear response by the mere passage of time or transfer to a new context if the original information is still present. Therefore conventional extinction is seen as the context-specific learning of a new association that the conditioned stimulus is safe now. Either the passage of time or perception of another environment may act as a context different from the one in which the new learning took place, and then the old memory can compete with the new and restore the old response (Monfils et al do not bother to explain this last part, assuming readers are familiar with this background information; I include it here for completeness, not because I expect students to go into this level of detail). In contrast, if extinction occurs within the critical time window after the reminder, then the original association is weakened. This matters in the context of using extinction procedures to treat phobias or addiction. Spontaneous recover from extinction or context specificity are then not desirable.

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 be 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. Reconsolidation is thus relevant to Schacter's memory errors of bias, misattribution and suggestibility.

The most important thing I want students to understand is that conventional extinction training without a reminder, and extinction training within a critical time period after a reminder have very different outcomes, and that these differences have clinical significance.

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

**Question 1:** Diskuter interaksjoner mellom gener og omgivelser og deres betydning for psykologien. (Discuss gene-environment interactions and their significance for psychology.)

The critical idea here is that it is possible to inherit how much one responds to the environment. Gabbard describes this with reference to rhesus monkeys that have been reared either by their mothers, or by peers. Rhesus monkeys have a functional polymorphism in the promoter region of the 5HTT receptor, which responds to serotonin. That region comes in a long and a short form. Every monkey has two copies of the gene, one on the chromosome inherited from the mother, the other on the chromosome inherited from the father. The study described by Gabbard compares only monkeys with two long versions of the genes versus monkeys with a long and a short version. The short/short combination is not mentioned. The monkeys with a long/long genotype are equally resistant to stressors regardless of whether they were reared by their mothers or only with peers. Those with a long/short genotype are more stressed if reared only with peers. However, monkeys placed with unusually nurturing mothers tended to rise higher in the hierarchy.

Although not in the penum, I also showed the students the results of a paper by Crespi et al, comparing long/long, long/short and short/short genotypes in humans. The more of the short allele people had, the more did negative events bias them towards depression and suicide.

Gabbard discusses the monkey study only to suggest that early experiences can have long-lasting effects. The students must therefore work out the broader implications of gene-environment interactions for psychology by themselves. I consider one of the implications to be that any attempt to classify behaviour as being caused by either nature or nurture, as if these were mutually exclusive categories, is fundamentally wrong. Inheriting how much one responds to the environment simply doesn't fit into that categorisation. It is as wrong to believe that genes are destiny as it is that if the environment has some influence over a trait, then that trait could not have a genetic component. Further, gene-environment interactions make it unlikely that a single treatment method will be universally effective.

**Question 2:** Gjør rede om Happé et al.s argument at det er ikke bare en eneste årsak av autisme, og dennes implikasjoner. (Discuss Happé et al.'s argument that there is no single cause of autism and its implications.)

Happé et al. make two fundamental claims: 1) that the three core traits of autistic spectrum disorders, namely special interests and repetitive actions, social difficulties and communication difficulties, are only weakly correlated, and 2) that along each of these three dimensions the distribution of the strengths of each trait is unimodal.

What the correlations among the three traits are can only be established by studying the general population, because the autistic population is preselected to have all three traits.

The authors list five specific implications of their argument:

- 1) Behaviourally it would seem useful to measure the three aspects of the triad separately, rather than rely on global ratings of autism severity or ratings that focus exclusively on social functioning.
- 2) Molecular genetic studies should abandon the search for 'genes for autism' as a whole, instead focusing on genes associated with the individual traits. The authors do not mention that the search for 'genes for autism' will lead to overestimating the importance of genes that are linked to two or three of the traits because those genes will be overrepresented in a population selected to have all three traits. A student noticing that would be a sign of deeper thinking than is common at this level.
- 3) Heterogeneity in autistic spectrum disorders is not poor measurement or the complex unfolding of developmental processes, but an unavoidable consequence of variation among at least three largely independent dimensions of impairment.
- 4) The argument suggests that there may be many individuals with isolated impairments in one aspect of the triad who do not meet diagnostic criteria for any recognised disorder, but show difficulties of comparable severity to those with autism. [For example, Happé et al. mention that 59% of children who show social impairment show *only* social impairment, not the other two traits. Even if the other two traits were perfectly correlated, then for every two autistic individuals we should find another three with comparable social difficulties but who do not meet the diagnostic criteria for autism. Because the correlation between communicative difficulties and rigid/repetitive behaviour is only 0.3 to 0.4, there must be even more people with just one or two traits for every individual with all three.] A diagnostic criterion that insists on the coincidence of three largely independent traits necessarily underestimates the frequency of each of the three traits.

If the three traits are caused by different genes, are associated with different brain regions and are related to different core impairments, it is likely that they will respond to different treatments. The search for a single treatment for all difficulties is pointless if they are largely independent and distracts from finding effective treatments.