NTNU
Kunnskap for en bedre verden

Psykologisk institutt

Eksamensoppgave i PSYPRO4412 – Anvendt og klinisk kognitiv psykologi

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Tlf.: 73 59 19 60

Eksamensdato: 6. desember 2016

Eksamenstid: 09:00-13:00

Hjelpemiddelkode/Tillatte hjelpemidler: Ingen

Målform/språk: Bokmål

Antall sider (uten forside): 1

Antall sider vedlegg: 0

Informasjon om trykking av eksamensoppgave
Originalen er:
1-sidig ☐ **2-sidig** ☐
sort/hvit ☐ **farger** ☐
skal ha flervalgskjema ☐

Kontrollert av:

Dato Sign

Merk! Studenter finner sensur i Studentweb. Har du spørsmål om din sensur må du kontakte instituttet ditt. Eksamenskontoret vil ikke kunne svare på slike spørsmål.

Langsvarsoppgaver – svar på to av tre spørsmål. Vekting 40% per oppgave.

1. Hvorfor er eksponeringsterapi effektivt på tvangslidelser og andre angstlidelser? Hva kan påvirke denne effektiviteten?

2. Hva er nevrofeedback? Kan man ved hjelp av nevrofeedback oppnå noe som er vanskelig eller umulig å oppnå ved å snakke med folk?

3. Hvordan kan kontekstspesifisitet forklare ABA, AAB og ABC fornyelse og spontan bedring? Hva er den kliniske signifikansen?

Kortsvarsoppgaver – svar på ett av to spørsmål. Vekting 20%

1. Gjør rede for Happé et al.s argument om at det er ikke bare er en årsak til autisme, og hvilke implikasjoner dette argumentet gir.

2. Hva er forskjell mellom “actor-critic” læring og Q –læaring, og hvorfor er det relevant?

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

Question 1: Why is exposure therapy effective in compulsive disorders? What can influence that effectiveness?

Hvorfor er eksponeringsterapi effektivt på tvangslidelser og andre angstlidelser? Hva kan påvirke denne effektiviteten?

Question 2: Hva er nevrofeedback? Kan de gjøre hva er vanskelig eller umulig å gjøre ved å snakke med folk? What is neurofeedback? Can it do anything that would be difficult or impossible to achieve by talking to people?

Eksamensoppgaven tar utgangspunkt i en labøvelse studentene har gjennomgått. 10 har gjennomført en qEEG-måling før og etter nevrofeedback treninger, der resten av kullet har deltatt som observatører. I tillegg har de to artikler om nevrofeedback som pensum.

Drøftinger har vært rundt hvilke mekanismer som nevrofeedback berører, altså operant betinging av hjernebølger som i dette tilfellet har ført til forbedret evne til konsentrasjon. I tillegg har studentene deltatt i diskusjoner på i hvilken grad nevrofeedback kan føre til bedringer om det er snakk om lidelser som ikke har et tydelig fysiologisk grunnlag, som ved traumatisering.

Det er ønskelig at besvarelsen tar opp muligheten til å endre uttrykket av hjernebølger, at det er betydningsfullt at hjernebølger kan framstå med forskjellige frekvenser og at det er vist empirisk at endring av hjernebølgeuttrykk gir funksjonelle bedringer både i normale personer og i pasienter. I tillegg bør det komme en drøfting av treningseffekter både der det er et fysiologisk grunnlag for en lidelser og der det ikke er det. Akkurat her er det få absolutte svar, så de er oppfordret til å drøfte og eventuelt å spekulere.

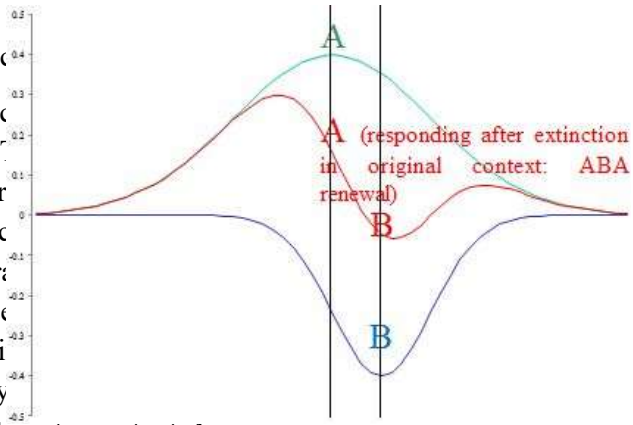
Question 3: How can context specificity explain ABA, AAB and ABC renewal and spontaneous recovery? What is the clinical significance?

Learning is context specific to the extent that behavior differs between the context in which behavior was learned and the context in which it occurs, or fails to occur, later. For example, if an association between a stimulus and outcome is learned in context A, then context specificity means that the relationship between stimulus and outcome is treated as valid in context A, but as uncertain in other contexts.

Extinction training (reducing the probability of the stimulus being followed by the outcome) can lead to the learning of new inhibitory associations. That means a new association between the stimulus and the absence of a relevant outcome. When the new inhibitory association is combined with the old excitatory association, the net outcome is reduced or zero expectation of the outcome.

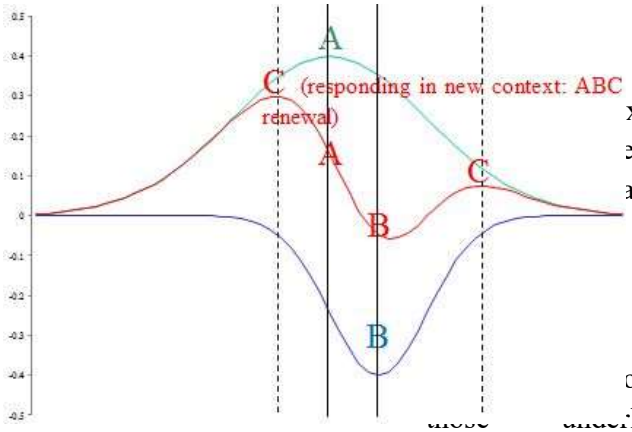
These inhibitory associations are typically context specific. Consequently, if the original excitatory learning is in context A, but extinction occurs in a different context B, then on return to context A the inhibitory association is weaker than in the context B in which it was learned. When the old excitatory association in its original context combines with the inhibitory association in a context new for it, the result is stronger responding than in context B. That is ABA renewal.

Explaining AAB and ABC renewal requires the additional assumption that inhibitory associations are more strictly context specific than excitatory associations. If context is treated as continuously variable, meaning it is possible to say *how* different two contexts are, then greater context specificity means a narrower generalisation gradient, declines faster for inhibitory associations as



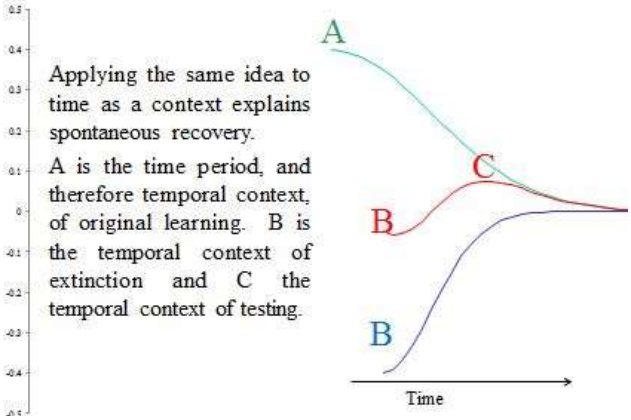
In the first figure on the right, original exc occurs in context A (left vertical line). represents the generalization gradient for learning. Extinction in context B (right vertic the inhibitory learning with a narrower gener represented by the blue curve. Becaus association is weaker in context B, the inhibi also be a bit weaker, yet still completely excitatory association in context B. On return to context A that slightly weaker association and its narrower generalization gradient result in the combined associations producing some response.

The same reasoning explains ABC the testing context C (dashed lines) is from the original learning context A and the xt B. Depending on which context is chosen as ext C (left or right dashed line), responding may an if tested in the original learning context A.



Spontaneous recovery can be explained as with time acting as context.

The clinical significance of renewal and overy is that if undesirable associations, such as underlying phobias and addictions, are extinguished in the context of the clinic, they may return when patients return to the contexts of their daily life.



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

Question 1: 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 of 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.

Question 2: What is the difference between actor-critic learning and Q-learning, and why does that matter?

Hva er forskjell mellom "actor-critic" læring og Q –læaring, og hvorfor er det relevant?

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.