NTNU
Kunnskap for en bedre verden

Institutt for psykologi

Eksamensoppgave i PSYPRO4412 – Anvendt og klinisk kognitiv psykologi

Faglig kontakt under eksamen: Robert Biegler
Tlf.: 73 59 19 60

Eksamensdato: 28. mai 2018
Eksamenstid (fra-til): 09:00-13:00
Hjelpemiddelkode/Tillatte hjelpemidler: Ingen

Målform/språk: Bokmål
Antall sider (uten forside): 1

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. Besvar 2 av 3 oppgaver. Hver oppgave teller 40%

1. Diskuter ulike behandlingstilnærminger til schizofreni. Legg vekt på kognisjon.

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

3. What is computational psychiatry, and what is the point, if any?

Forklar hva "computational»-psykiatri er. På hvilke(n) måte(r) kan «computational»-psykiatri ha en effekt?

Kortsvarsoppgaver. Besvar 1 av 2 oppgaver. Oppgaven teller 20%

1. Explain the drift-diffusion model of choosing between two alternatives. What parameters can be extracted from reaction time distributions? What is the clinical relevance of such parameter estimates?

Forklar «drift-diffusion» modellen for det å velge mellom to alternativer. Ut fra modellen: hvilke parametre kan trekkes ut fra reaksjonstidsfordelingene? Hvilken klinisk relevans har slike parameterestimat?

2. Hvordan, og i hvilken grad er det mulig å skille fra hverandre autentiske og falske gjenkalte fortrengte minner?

Sensorveiledning:

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

Question 1: Diskuter ulike behandlingstilnærminger til schizofreni. Legg vekt på kognisjon.

Sensorveiledning: 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 2: Are schizophrenia and autism opposite ends of the same continuum?

Sensorveiledning: 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.

Wiecki'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.

Question 3: What is computational psychiatry, and what is the point, if any?

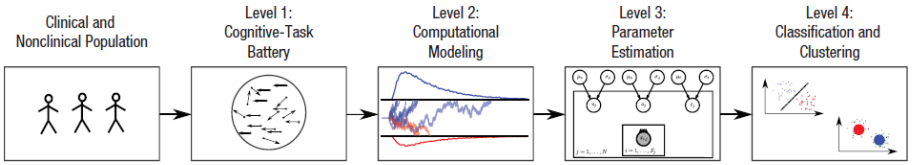
Computational psychiatry is a cognitive approach to mental health. Cognitive psychology is concerned with how minds do what they do, at the algorithmic level. The aim is to find a description of the rules and equations that can generate observed behaviour, ideally precisely enough to be able to reproduce that behaviour. Such rules and equations have quantitative parameters, and these differ between individuals. Examples of such parameters would be working memory capacity, or in the dot motion task non-decision time, drift rate, decision boundaries, and bias, or in Frith's theory, how much prediction error is overestimated. For contrast, the five personality dimensions are too vague to be considered computational parameters. Computational psychiatry assumes that there will be problems when computational parameters deviate enough from the average, and different patterns of deviations correspond to different mental health problems. The hope is that computational psychiatry will be able to achieve the following goals:

1) Revise diagnostic criteria. For example, Fair et al found that normally developing children showed six different multidimensional profiles in children diagnosed with ADHD, four of which were also found in normally developing children, and two more were subtypes of those four. That indicates that ADHD is not a single condition. Another example is that there are four different kinds of impulsivity. Gold et al.'s picture discrimination task can distinguish impairments in stimulus-response (model free or actor-critic) learning from impairments in representing either positive or negative outcomes in response-outcome (model based or Q) learning. Problems with representing positive outcomes are associated with negative symptoms in schizophrenia, problems with representing negative outcomes are associated with pathological gambling.
2) Identify underlying mechanisms (at the algorithmic level) through measurement of computational parameters: for example, learning from positive or negative prediction errors; changes in bias, drift rate or decision threshold in the drift-diffusion model, depth of recursion in the stag-hunt game, sensitivity to fairness in the trust game; four kinds of impulsivity.
3) Assess specific effects of treatment on those more precisely identified mechanisms (not done yet as far as I know).
4) Perhaps use such assessments to design new treatments (not done yet as far as I know). For example, reduced ability to represent positive outcomes is found in schizophrenia patients with negative symptoms. Reduced ability to represent negative outcomes is found in gamblers. If behavioural treatments can be found that selectively changes these parameters, patients could be brought back closer to the normal range. If the parameters at the algorithmic level can be linked to specific aspects of physiology, that could lead research into more precisely targeted drugs, or neurofeedback treatments.

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

Question 1: Explain the drift-diffusion model of choosing between two alternatives. What parameters can be extracted from reaction time distributions? What is the clinical relevance of such parameter estimates?

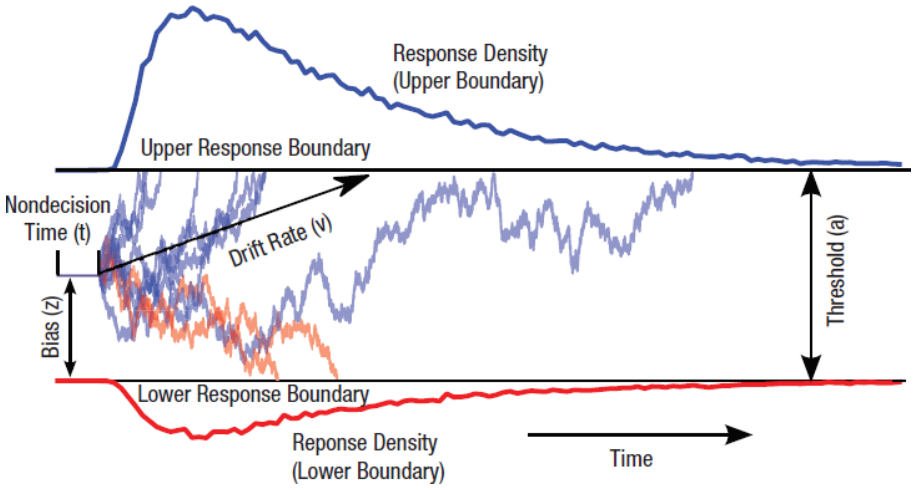
Sensorveiledning: The drift-diffusion model is a specific example of computational modelling applied to mostly perceptual tasks with two response alternatives, such as the dot motion in the second frame of this figure (Level 1):



The drift-diffusion model assumes that the decision variable starts between two decision boundaries, that noisy evidence for each alternative is added up over time, moving the decision variable to one or the other of the boundaries, and that a decision is made the moment the decision variable reaches one

of the boundaries. From the distribution of response times, recorded separately for correct and incorrect answers, it is possible to reconstruct:

1) Nondecision time, which is how long it takes for information to reach the relevant part of the brain, and for the motor command to result in a button press.
2) The starting point for the decision process, called bias because it may be closer to one of the decision boundaries.
3) The drift rate, how quickly the decision variable changes.
4) The threshold, or distance between the decision boundaries.



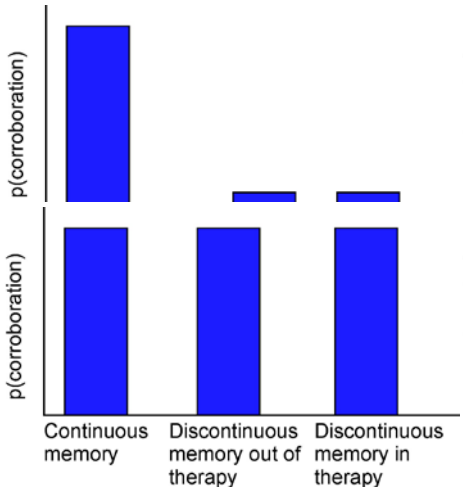
Although Wiecki et al do not discuss this, it should be easy to work out that bias should respond to base rates and to costs and benefits. In the present example of the dot motion task, if movement up is more common than movement down, then average response time and proportion correct answers can be improved by moving the starting point closer to the upper response boundary. A faster drift rate would reduce response times without affecting proportion correct. In contrast, moving the decision boundaries closer together (changing the threshold) would speed up responses at the cost of reduced accuracy because noise in the system will have a relatively greater influence.

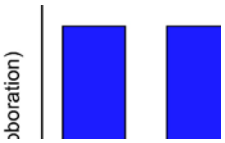
That response time can be affected by changes in several of the parameters of the underlying computation illustrates the task-impurity problem of a simple response time measurement. Estimating the computational parameters reduces such problems, and therefore should more cleanly separate clusters of people who are lumped together by the simpler measurement.

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

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

Sensorveiledning: Geraerts et al. list three possible predictions regarding the probability that memories of sexual abuse are corroborated by independent evidence:

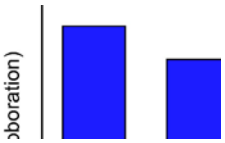




1: Only continuous memories are reliable

2: Memories recovered out of therapy are reliable

3: All recovered memories are reliable



Result

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). Geraerts et al. do not mention whether the therapy in question was specifically recovered memory therapy or a broader range of therapies. They do point out that 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.

formaterte: Engelsk (USA)