NTNU
Kunnskap for en bedre verden

Institutt for psykologi

Eksamensoppgave i PSY3111 - Individuell utvikling, gener, nervesystem og atferd

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Eksamenstid (fra-til): 15:00-19:00

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

Studenten skal <u>besvare 4</u> av de følgende 6 spørsmål:

1.

Gjør kort rede for hva et gen er, og forklar på bakgrunn av DNA-molekylets struktur hvordan DNA-molekylet inneholder «oppskrifter» for ulike proteiner.

2.

Konsolidering er en prosess der relevant informasjon kodes inn og lagres permanent. Hva tror man kan være de underliggende mekanismene for innkoding av deklarative langtidsminner?

3.

Hvordan vet hjernen hva nesen lukter? Gjør rede for nervenettverket i hjernens primære luktsenter (dvs. luktelappen).

4.

Mange utviklingsstudier fokuserer på likheter mellom barn og foreldre, og forklarer likheten med at barn imiterer eller lærer atferd fra foreldre. Med utgangspunkt i atferdsgenetiske studier (adopsjon og tvilling), hva forklarer generelt likhet mellom medlemmer i samme familie?

5.

Beskriv og diskuter tre grunnleggende motoriske kontrollfunksjoner som er nødvendig for musikalsk ytelse.

6.

Gjør rede for Ioannidis argument om at det meste av forskning som publiseres er feilaktig. Betyr dette at vi skal forkaste forskning og bli postmodernister?

PSY3111–sensorveiledning

Studenten skal besvare 4 av de følgende 6 spørsmål:
The student should answer 4 of the following 6 questions:

Oppgave 1

Bokmål: Gjør kort rede for hva et gen er, og forklar på bakgrunn av DNA-molekylets struktur hvordan DNA-molekylet inneholder «oppskrifter» for ulike proteiner.

English: Briefly discuss what a gene is and, based on the DNA molecule´s structure, describe how the DNA molecule contains "recipes" for different proteins.

Sensorveiledning: Det forventes at studenten er kjent med at et gen er en sekvens av DNA-molekylet som koder for en kjede av aminosyrer, dvs. et polypeptid/protein (eller evt. et RNA). Det forventes videre at studenten kan forklare strukturen på DNA-molekylet, dvs. en dobbel heliks bestående av nukleotider, der hvert nukleotid inneholder et sukker, et fosfat og en nitrogenbase. Når det gjelder selve koden, bør studenten kunne gjøre rede for at det er tripletter av nitrogenbaser, såkalte kodon, som korresponderer med spesifikke aminosyrer.

Oppgave 2

Norsk: Konsolidering er en prosess der relevant informasjon kodes inn og lagres permanent. Hva tror man kan være de underliggende mekanismene for innkoding av deklarative langtidsminner?

English: Consolidation is a process by which relevant information is encoded and stored permanently. What is believed to be the underlying mechanisms for encoding declarative long-term memories?

Sensorveiledning:
Spørsmålet er åpent, noe som gir frihetsgrader mht svar. Men pensumkapitlet omhandler minneprosesser i hippocampus og svaret bør reflektere det. Oppgaven bør inneholde at innkoding av nye minner er trolig synonymt med oppstart av proteinsyntese og strukturelle endringer av synapser. NMDA-reseptoren bør være med, indikert som utløsende faktor, samt at influx av kalsium utløsert en kaskade som nettopp endrer med proteinsyntese. En meget god besvarelse benevner hvert vesentlige ledd i denne kaskaden, samt samspillet mellom CREB-1 og CREB-2. Å bruke LTP som et eksempel er bra, men ikke påkrevd. Det er et pluss om man nevner forskjellige typer initiering (assosiativ/ ikke-assosiativ), og om man skiller tidligfase fra senfase. En meget god besvarelse tar også med teorien om synaptisk merking (tagging), at den endelige proteinsyntese finner sted i de synapser der kaskaden startet. Det er et pluss om man drøfter gammaoscillasjoner sin mulige rolle.

Oppgave 3

Norsk: Hvordan vet hjernen hva nesen lukter? Gjør rede for nervenettverket i hjernens primære luktsenter (dvs. luktelappen).

English: How does the brain know what the nose smells? Explain the nerve network in the brain's primary odors center (i.e. the olfactory bulb).

Sensorveiledning:
Første del av oppgaven åpner for noe ulike tilnærminger. Studenten bør imidlertid forklare at det sensoriske luktorganet, lukteepitelet, inneholder sensoriske luktenevroner som er i stand til å sanse ulike duftmolekyler i lufta (eller i maten!). Mennesket har mange ulike typer av luktreseptorer (plassert på cilimembranen til de sensorske nevronene), anslagsvis et sted mellom 350 og 400. Dette betydelige repertoaret av reseptorer i periferien sikrer et system som i utgangspunktet er i stand til å detektere mange ulike duftmolekyler (odoranter). Det er et pluss om studenten kan forklare at hvert sensorisk luktenevron uttrykker kun en type av luktreseptor (noe som betyr at vi mennesker har anslagsvis 350 til 400 ulike typer av sensoriske luktenevroner).

Studenten bør videre redegjøre spesifikt for nervenettverket i luktelappen. Her bør kandidaten forklare at de sensoriske luktenevronene projiserer direkte inn i luktelappen der de danner synapse med andre ordens nevroner. Disse synapsene danner kuleformede strukturer kalt glomeruli. Om studenten vet at sensoriske nevroner som er av samme type (dvs., uttrykker samme reseptor) sender aksonene sine til ett og samme glomerulus (eller eventuelt to), er det bra. Studenten bør videre vite at andre ordens nevroner i luktelappen omfatter lokale internevroner og projeksjonsnevroner, samt at projeksjonsnevronene løper inn til områder i temporallappen (luktekorteks). Mange av de lokale internevronene fungerer inhibitorisk. Om studenten har forstått og kan redegjøre for at nervenettverket i luktelappen har mulighet til å danne «aktiverings-kart» som omfatter spesifikke glomeruli, avhengig av hvilke duftsubstanser som er til stede, er det bra. Om studenten kjenner navnet på spesifikke typer av 2. ordens nevroner, som f. eks. Mitralceller og Tufted celler (som begge er projeksjonsnevroner) og periglomerulære celler og granulaceller (som begge er lokale internevroner), er det et pluss.

Oppgave 4

Norsk: Mange utviklingsstudier fokuserer på likheter mellom barn og foreldre, og forklarer likheten med at barn imiterer eller lærer atferd fra foreldre. Med utgangspunkt i atferdsgenetiske studier (adopsjon og tvilling), hva forklarer generelt likhet mellom medlemmer i samme familie?
English: Many developmental studies focus on similarities between children and parents, and explain the similarity as the child imitating or learning behavior from parents. Based on behavioral genetic studies (adoption and twins), what explains the general similarity between members of the same family?
Sensorveiledning:
Oppgaven bør kunne definere tre vesentlige begrep:

- Delt-miljø: miljøeffekter eller ikke-genetiske effekter som gjør medlemmer av samme familie likere.
- Ikke-delt miljø: miljøeffekter eller ikke-genetiske effekter (inkludert tilfeldighet) som gjør medlemmer av samme familie mindre like.
- Arvbarhet: genetisk varians som forklarer fenotypisk varians.

Disse begrepene og Turkheimers (2000) tre lover for atferdsgenetikken bør være inkludert i en moden faglig diskusjon av utfordringer for studier som mangler kontroll på arvfaktoren.

- Alle psykologiske trekk, tilstander og egenskaper er arvbare

- Effekten av å vokse opp i samme familie er mindre enn effekten av gener
- En vesentlig del av variansen i menneskets komplekse psykologiske trekk forklares verken av genetiske faktorer eller av familiemiljøet

Det bør inngå kritikk av studier som ensidig studerer utvikling som et resultat av miljø, all den tid utvikling generelt er akseptert å være et resultat av samspill av arv og miljø.

Konklusjonen bør inkludere at man i voksen alder så er foreldrelikhet i stor grad samlet sett på bakgrunn av mange og store studier på grunn av felles genetikk, og forskjell på grunn av ikke-delt miljø. (Ekstrapoeng for å ha med at dette ikke er et stabilt funn i hele utviklingen: I førskole alder vil det derimot kunne være større likhet med adoptivfamilie, men denne avtar i løpet av utviklingen frem mot voksen alder.)

Kritikk av atferdsgenetiske studier og betydningen av arvbarhetsestimatet kan gjerne inngå, men hovedvekten av kritikken bør selvsagt legges på studier som påberoper seg å studere miljøets påvirkning på utvikling uten å vurdere effekter av arv, all den tid likhet er mest sannsynlig forklart med arv.

Oppgave 5

Bokmål: Beskriv og diskuter tre grunnlegende motoriske kontrollfunksjoner som er nødvendig for musikalsk ytelse.
English: Describe and discuss three basic motor control functions which are required for musical performance.
Sensorveiledning: When a musician performs, at least three basic motor control functions are required: timing, sequencing and spatial organization of movement. The accurate timing of movements is related to the organization of musical rhythm, whereas sequencing and spatial aspects of movement relate to playing individual notes on a musical instrument. Although a large number of studies have examined the neural systems underlying these functions separately, little is known about how they work together to produce a complex musical performance. In addition, there is considerable debate regarding both the definition of these motor parameters and the specific contributions of particular brain regions to their control. The study of music production requires these systems to be studied in an integrated fashion, thus making it both a challenging and fruitful model system for research into sensory–motor integration.

Timing. The neural mechanisms that underlie the timing of movement have been intensively studied over the past 20 years, but currently there is more controversy than consensus in this field. The ability to time movement precisely has been attributed to a neural clock or counter mechanism in which time is represented through pulses or oscillations, but it has also been hypothesized to be an emergent property of the kinematics of movement itself. Functional neuroimaging studies, as well as studies of brain-damaged patients, have linked movement timing to several cortical and sub-cortical regions, including the cerebellum, basal ganglia and supplementary motor area (SMA). It has been proposed that the basal ganglia and possibly the SMA may be more important for interval timing at longer timescales (1 second and above), whereas the cerebellum may be more important for controlling motor timing at shorter timescales (millisecond).

Studies have shown that patients with cerebellar lesions have an impaired ability to complete perceptual and motor timing tasks, and neuroimaging studies have shown cerebellar activity in relation to movement timing. Although some studies have failed to sup- port a direct contribution of the cerebellum to timing, current theories of cerebellar function suggest it may have a role in feedforward control or error correction — both of these functions would be relevant for timing. Several researchers have proposed that the cerebellum computes predictive models of movement that would include

movement timing, whereas others suggest that it is most important for online error correction based on feedback, which would also contribute to optimization of timing. The cerebellum may contribute to the precise control of movement trajectories, which are related to accurate timing, and it has been shown to have a role in the acquisition and integration of sensory information. When subjects perform purely auditory perceptual tasks, neuroimaging studies consistently show cerebellar activity.

Studies have suggested that the basal ganglia are also directly involved in movement timing. Patients with Parkinson's disease, who have damage in the basal ganglia system, show impaired movement timing. Furthermore, neuroimaging studies have shown that the basal ganglia are active in tasks that require timed finger tapping. It has also been suggested that the basal ganglia may be involved in controlling specific motor parameters, such as force, which contribute to accurate timing.

Many of these studies have examined very simple rhythms, usually requiring participants to tap a single finger to a constant beat. Although such tasks reveal important basic properties of perceptual and motor timing, it is not clear whether neural models based on these simple tasks are adequate for complex tasks like musical performance. Several recent experiments have examined perception and reproduction of more com- plex musical rhythms. These studies have shown greater involvement of the dorsal premotor cortex (dPMC), lateral cerebellar hemispheres and the prefrontal cor- tex. It is not known whether these changes in brain activity are directly related to the temporal complexity of the rhythms or to other parameters such as sequence complexity, or the degree to which rhythmic structure allows subjects to predict and organize their motor performance. These results indicate that motor timing is not controlled by a single brain region, but by a network of regions that control specific parameters of movement and that depend on the relevant timescale of the rhythmic sequence. High-level control of sequence execution appears to involve the basal ganglia, PMC and SMA, whereas fine-grain correction of individual movements may be controlled by the cerebellum.

Sequencing. Motor sequencing has been explored in terms of either the ordering of individual movements, such as finger sequences for key presses, or the coordination of subcomponents of complex multi-joint movements. Several cortical and sub-cortical regions, including the basal ganglia, the SMA and the pre- SMA, the cerebellum, and the premotor and prefrontal cortices, have been implicated in the production and learning of motor sequences, but their specific contribu- tions and the way they work together are not yet clear. Neurophysiological studies in animals have demonstrated an interaction between the frontal cortex and basal ganglia during the learning of movement sequences. Human neuroimaging studies have also emphasized the contribution of the basal ganglia for well-learned sequences. It has been argued that the cerebellum is important for sequence learning and for the integration of individual movements into unified sequences, whereas the pre-SMA and SMA have been shown to be involved in organizing or chunking of more complex movement sequences. Finally, the premotor cortex has been shown to be involved in tasks that require the production of relatively complex sequences, and it may contribute to motor prediction. Sequencing has also been studied in a more musical context in an experiment that examined neural activity during the execution of sequences of key-presses that differed either in temporal or sequential complexity. This study showed that more complex sequences required activity from the basal ganglia, dPMC and cerebellum.

Spatial organization. Expert musical performance requires precise spatial organization of movements. Few studies of complex motor control have distinguished between the spatial and sequential components of a series of movements. Studies in animals and humans have established the involvement of parietal, sensory–motor and premotor cortices in the control of movements when the integration of spatial, sensory and motor information is required. More recent work has suggested that separate neural systems may underlie the ability to learn and produce the spatial and sequential com-

ponents of a complex task. Surprisingly, few studies have explicitly examined the role of spatial processing in the context of musical tasks. A behavioural study of spatial accuracy in trained cellists found that they do not show the typical distance/accuracy trade-off for finger movements while playing. A recent neuroimaging study contrasting sequential and temporal sequence learning suggested that the dPMC may have a role in the learning of spatial trajectories. Overall, however, the contribution of spatial processing to music-related motor tasks remains an area in which future work could make an important contribution.

Oppgave 6

Bokmål: Gjør rede for Ioannidis argument om at det meste av forskning som publiseres er feilaktig. Betyr dette at vi skal forkaste forskning og bli postmodernister?

English: Discuss Ioannidis' argument that most published science is wrong. Does it mean we should all abandon research and become postmodernists?

Sensorveiledning: It is not satisfactory if a student just lists Ioannidis' conclusions, without explaining how he or she arrived at those conclusions. Such an explanation will have to involve application of Bayes' theorem. However, it is not necessary to use Ioannidis' nomenclature. A specific example using Gigerenzer's approach will be just fine. The crucial numbers are the prior odds of a hypothesis being true, the detection rate (statistical power, or 1 – Type II error rate) and the false alarm rate (the level of statistical significance, conventionally $p \leq 0.05$).

If we then assume prior odds of 1:1, adopt the conventional significance level of 0.05, and assume a detection rate of 0.7, we can calculate the posterior probability of the hypothesis being true. Think of 1000 tests of experimental hypotheses with those prior odds. If they distribute exactly according to probabilities, 500 of those hypotheses will turn out to be true, and 500 wrong. With statistical power or detection rate being 0.7, 350 tests of true hypotheses will have a statistically significant result. However, 5% of the 500 false hypothesis will also produce a statistically significant result. That means 350 out of 375 significant results come from true hypotheses. The probability of a hypothesis being true, given a positive result, is 350/375 = 0.9333. Ioannidis calls that the positive predictive value. Here is the same calculation in Gigerenzer's table format.

If the prior odds are lower, for example 1:9, fewer true hypotheses can contribute to the positive results, while there will be more false hypotheses producing false alarms. The second calculation shows that if 70% of the now only 100 true hypotheses give positive results, and 5% of the now 900 false hypotheses, positive predictive value of a statistically significant result is only 0.609. Then nearly 40% of positive results would be false.

Odds of true hypothesis = R = 1:1, base rate = $\frac{R}{\_\_\_}$ = 50%

Odds of true hypothesis = R = 1:9, base rate = $\frac{R}{R+1}$ = 10%

Detection rate = statistical power = 1 – β = 43.8% if β = 0.562
False alarm rate = α = 1%

True Relationship?	*Positive result*	*Negative result*	*sum*
Yes	**44**	**56**	**100**
No	**9**	**891**	**900**
	53	**947**	**1000**

Positive Predictive Value (PPV) = 44/53 = 0.83

Ioannidis doesn't discuss whether demanding a more rigorous level of statistical significance could improve the

situation. The better students may examine this question anyway. The table on the right shows the consequence of asking for $p < 0.01$. This must reduce detection rate, because shifting the decision criterion only trades the two types of error against each other. The students don't have a principled way of determining what the new detection rate might be, so any arbitrary value will do, though recognizing that it should be a lower value would be a good thing. Anyway, if we assume that the 1% false alarm rate comes with a 44% detection rate, the positive predictive value for prior odds of 1:9 turns out to be 0.83. There is a price for increasing the probability that a hypothesis claimed to be true really is true: more than half of true hypotheses are being rejected.

To see the effects of bias, return to the second example calculation. Ioannidis defines bias generally as the conversion of negative into positive results, for example by cherry picking data, redefining hypotheses, or whatever. Mathematically, he assumes that the probability of a negative finding being converted into a positive is the same for true and false hypotheses. Assume that Bias is 0.2: one fifth of negative results are turned into positive results. Of the 30 true hypotheses that had been rejected, 6 would be moved from the negative results column into the positive results column, for a total of 76. Likewise, one fifth of the 855 false hypotheses that had been rejected will now be accepted. 171 negative results are moved into the positive results column, for a total of 216. The positive predictive value then is no longer 70/115 = 0.609, but instead 76/216 = 0.35.

I did not take the students through the calculations for multiple research teams testing the same hypothesis, because that is more difficult to make intuitive. I am content if a student understands that this offers multiple chances for a false hypothesis to produce a statistically significant result.

Ioannidis' six corollaries or conclusions about the probability of a positive research finding being true can then be linked to the three fundamental numbers required to calculate posterior probability in Bayes' theorem. Small studies (corollary 1) and small effect sizes (corollary 2) mean less statistical power, meaning a lower detection rate. The more hypotheses are tested and the less those hypotheses are selected (corollary 3), the lower the prior odds. Greater flexibility in designs, definitions and outcomes (corollary 4) means more room for bias to creep in, even unintentionally, while greater financial incentives and greater prejudice will also increase bias (corollary 5). Finally, the hotter a field, meaning the more research teams chase the same hypotheses, the less likely a positive result is to be true, because there are more chances for a false hypothesis to produce a positive result (corollary 6), and it is positive results that get attention, and they are more likely to be published.

I would like to see at least a brief discussion of possible solutions to the problems that Ioannidis has identified. Merely applying a more stringent criterion for statistical significance, for example, would have the further consequence of increasing the contribution that publication bias makes to the phenomenon that effect sizes often decrease as a new finding is more thoroughly investigated. Briefly, initial studies are likely to employ few subjects, because it is hard to get funding for large studies searching for an effect that may not exist. If you run multiple identical studies, then small studies have more variable effect sizes. If mostly positive results get published, then even for a real effect the small studies that happen to find modest effect sizes will fail to reach significance and therefore they will be less likely to get published. Only those studies that find large effects, either because the effect really is large or because random fluctuation makes it look large, will get published. So the initial small studies of modest or small effect sizes can only reach statistical significance through noise. The published sample is heavily skewed, and it will get more skewed the more demanding your criterion for statistical significance is. Then follow-up studies use larger samples. The larger samples reduce variation in effect size, reducing the scope for published data to be skewed, and the larger studies can reach significance level more easily. So if the real effect size is modest or small, then early and small studies can only get published if they overestimate effect sizes,

later and larger studies show smaller effect sizes. And it looks like effects disappear the longer they are studied.

Ioannidis proposes that large studies that have a higher detection rate, but are also expensive, should be aimed at major theoretical concepts, rather than narrow, specific questions. Then a negative finding can refute not just a very specific claim, but a wider range of claims. Second, replication is especially important in hot fields. Bias can be reduced by prior registration of studies (reduces publication bias) including definition of hypotheses and relevant outcomes (reduces opportunity to redefine hypotheses and outcomes). Third, it would be useful to establish prior odds.