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Institutt for psykologi

Mal for sensorveiledning

Emnekode	PSY1503, PSY1123, PSYK4123
Emnenavn	Innføring i biologisk psykologi og genetikk
Emneansvarlig/oppgavegiver	Gerit Pfuhl
Kvalitetssikret av	Robert Biegler og Stig Hollup
Semester, år	Andre semester, 2024
Vurderingsform, lengde	Skole-eksamen, 4 timer
Tillatte hjelpemidler	ingen

Emnets læringsutbyttebeskivelser angitt I kunnskaper, ferdigheter og generell kompetanse. (Henvisning med lenke til emnesiden på NTNUs nettsider er tilstrekkelig)	https://www.ntnu.no/studier/emner/PSY1503#tab=omEmnet Kunnskaper Studenten har: en grunnleggende forståelse av den biologiske basis for adferd, persepsjon og emosjoner kunnskap om nervesystemets generelle anatomi og funksjon, med hovedvekt på hjernens virkemåte. studenten ha forståelse for basal genetikk inkludert DNA-molekylets struktur og funksjon kjennskap til sentrale metoder som benyttes innen moderne nevrovitenskapelig forskning Ferdigheter studenten kan: analysere og diskutere vitenskapelige problemstillinger innenfor biologisk psykologi utrykke seg skriftlig på en måte som er egnet for å videreformidle sentrale tema innenfor fagområdet Generell kompetanse Studenten skal ha utviklet en grunnleggende forståelse for vitenskapelig tenkemåte, med evne til selvstendig refleksjon over faglige og forskningsetiske problemstillinger.
Pensum	Pensum: «Neuroscience – Exploring the brain», MF Bear, WC Connors, and MA Paradiso. 4th edition. ISBN 9781451109542. Utvalgte deler, anslagsvis 571 sider: Kap. 1, 19 sider Kap. 3-9 (minus 13 sider 'Development part' i kap. 7 og 11 sider på 'Taste' i kap. 8), totalt 251 sider Kap. 11 og 12 (minus 'Vestibular system' i kap.11, 8 sider), totalt 75 sider Kap. 15, 28 sider, Kap. 16, 27 sider, Kap. 19, 40 sider, Kap. 24 og 25, 76 sider

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	«Freberg. Discovering Behavioral Neuroscience: An Introduction to Biological Psychology, 5th edition. Noen utvalgte deler, 34 sider totalt. Kap. 5
Eventuelle formelle krav til besvarelsen	Maks 250 ord per besvarelse. Det ofte holder med færre ord
Hvordan de ulike oppgavene i eksamenssettet er vektlagt	Likt vekting, i hver bolk trenger en å svare 5 av 6

Eksamensoppgaver

Genetikk, AP+SP, Anatomi (velg 5 av 6):

1. Er det mulig for en atferdsfenotype å enten være helt bestemt av gener eller helt bestemt av miljøet? Hvis ja, hvor sannsynlig er det? Forklar resonnementet ditt
2. Små gnagere liker vanligvis ikke åpne områder, hvor det er lettere for ugler å angripe. Anta at du ønsker å finne ut hvor arvelig frimodighet er, målt som vilje til å gå ut i åpne rom. Forventer du at arvbarheten skal være høyere eller lavere for en laboratoriestamme sammenlignet med villtyperotter (rotter antas å være representative for villpopulasjonen)? Hvorfor? Forventer du at arvbarheten blir høyere eller lavere når du oppdrar alle rottene på samme måte sammenlignet med å utsette dem for ulike miljøer? Hvorfor?
3. Forklar forskjellen mellom ionotrope og metabotrope reseptorer.
4. Hva er forskjellen mellom en IPSP og en EPSP? Hvor oppstår de?
5. Hva er nevral tilpasning (neural adaptation)? Hva forteller dette oss om informasjonsbehandling i hjernen?
6. Hva er HPA-aksen? Og hvilke hjerneregioner er en del av det?

Sansesystemer (velg 5 av 6)

7. Hva menes med et sansekart? Hvor kan du forvente det i nervesystemet? (som eksempel er ett sansesystem tilstrekkelig)
8. Mennesker har omtrent +/- 350 typer luktreseptorer. Hvordan er det da at mennesker kan skille mellom tusenvis av forskjellige lukter?
9. Hva er forskjellene mellom ON og OFF bipolare celler? Hva får ON/OFF bipolare celler til å fungere slik de gjør? (hint: hva slags Glu-reseptorer har ON og OFF bipolare celler?)
10. Forklar hvordan vibrasjoner i basilarmembranen i sneglehuset omdannes til elektriske/kjemiske signaler.
11. Gjør en kort redegjørelse for smerte og berøringsveier i nervesystemet.
12. Hva er et mottakelig felt og hvor finner vi det?

Søvn, hjernerytme, læring, hukommelse og motivasjon (velg 5 av 6)

13. Hva er de forskjellige fasene av hukommelsen, og hvilken biologisk mekanisme følger sannsynligvis med?
14. Hvilken rolle spiller AMPA-reseptoren og NMDA-reseptoren i langsiktig potensering?
15. Søvnens forskjellige stadier kan identifiseres. Hva som kjennetegner de forskjellige stadiene (tips: tenk EEG og bølger)
16. Hvilken rolle spiller dopamin i CNS? Nevn minst én rolle og hjerneregionen/atferden som er knyttet til den?
17. Mange legemidler/rusmidler er antagonister eller re-opptakshemmere (reuptake inhibitors). Forklar hva de gjør og hvordan dette endrer synaptisk overføring.
18. Beskriv komponentene i CNS som bidrar til døgnrytmen (reseptor, hjerneregion).

In English:

Genetics, potentials, anatomy (choose 5 out of 6):

1. Is it possible for a behavioural phenotype to be either entirely determined by genes or entirely determined by the environment? If, yes, how likely is that? Explain your reasoning.
2. Small rodents typically don't like open spaces, where it is easier for owls to attack. Assume you want to find out how heritable boldness is, measured as willingness to go into open spaces. Do you expect heritability to be higher or lower for a laboratory strain compared to wild type rats (rats assumed to be representative of the wild population)? Why? Do you expect heritability to be higher or lower when you raise all the rats in the same way compared to exposing them to different environments? Why?
3. Explain the difference between ionotropic and metabotropic receptors.
4. What is the difference between an IPSP and an EPSP? Where do they occur?
5. What is neural adaptation? What does this tell us about information processing in the brain?
6. What is the HPA axis? And which brain regions are part of it?

Sensory systems (choose 5 out of 6)

7. What is meant by a sensory map? Where would you expect it in the nervous system? (as example one sensory system suffice)
8. In humans, there are approximately +/- 350 types of olfactory receptors. How is it then that humans can distinguish between thousands of different odors?
9. What are the differences between ON and OFF bipolar cells? What makes OFF/ON bipolar cells work the way they do? (hint: what kind of Glu receptors do ON and OFF bipolar cells have?)
10. Explain how vibrations in the basilar membrane in the cochlea are converted into electrical/chemical signals.
11. Give a brief account of pain and touch pathways in the nervous system.
12. What is a receptive field and where do we find it?

Søvn, hjernerytme, læring, hukommelse og motivasjon (velg 5 av 6)

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13. What are the different phases of memory, and which biological mechanism likely goes with it?
14. Which role does the AMPA receptor and NMDA receptor play in long term potentiation?
15. The different stages of sleep can be identified. What characterizes the different stages (hint: think EEG and waves)
16. What role does dopamine play in the CNS? Name at least one role and the brain region / behaviour that is associated with it?
17. Many drugs are antagonists or reuptake inhibitors. Explain what they do and how this changes synaptic transmission.
18. Describe the components of the CNS contributing to the circadian rhythm (receptor, brain region)

Sensurveiledning:

Note, some of the questions have been used in the colloquia and the solution provided on BB (question 2, 3, 4, 5, 9), others have been discussed in the lectures as think-pair exercise (6, 8, 10, 13, 14, 17).

The solutions below provide a guideline for distinguishing a very good answer from an ok answer (C or D). If you think an answer is excellent, you can give a bonus point (6 out of 5)

For each question you can rate it with 0 (grade F) to 5 (grade A) points.

They do need to answer 5 out of 6 from the three themes. There is no bonus to answer all 6 (but you can choose the 5 questions that give the highest points)

There are 15 * 5 = 75 points in total

Sumpoeng Oppgaver	% tilsvarende karakter	Endelig karakter
75 - 69	100 % – 92 %	A
68 - 58	91 % – 77 %	B
57 - 43 (43.5, generøs her)	76 % - 58 %	C
42 - 33	57 % - 44 %	D
32 - 15	43 % - 20 %	E
14 – 0	19 % – 0 %	F

1. If “determined by” is interpreted as heritability, then the answer is simple: a trait is (measured to be) entirely determined by the environment if there is no genetic variance, which makes heritability = 0. If the path from gene to trait is so simple that environment has no chance to make a difference, or in the theoretical case that all environmental variance could be removed, but there is still genetic variance that affects the trait, then heritability = 1, and that could be interpreted as “entirely determined by the environment”. That answer would be simplistic as

well as simple. If that is the only information there is, even if expressed in more words, that would be a D.

Students may choose to discuss how easily environment can influence a trait, and whether a trait can ever be completely independent of genes. There are usually many steps between a gene and its effect on behaviour, and there is not enough information in the genetic code to specify every connection in the brain. After the basic connectivity has been established, networks need to learn to do their jobs, giving an essential role to environment for most traits. There are some cases that *could* be interpreted as examples of a trait being entirely under genetic control, involving mutations to receptors. For example, a single nucleotide polymorphism determines the ability to smell asparagus in urine. In principle, the behavioural trait still depends on the necessary neural development proceeding appropriately, but that does not seem to be a problem in this case, so claiming that complete control by genes is *possible* is an acceptable answer. A better answer would also point out that it is unlikely.

As for any behavioural trait being "entirely determined by the environment", behaviour is produced by brains, brains are built be developmental processes which involve genes interacting with the environment. Therefore there should be no behavioural trait entirely determined by the environment, unless that phrase is interpreted to mean that heritability = 0 and there is no genetic variance in the population that is being studied.

A very good answer distinguishes between genetically determined as in "genes determine that humans have two legs", and heritability, which is variance attributable to genes divided by total variance. If there is no genetic variance, then heritability is necessarily zero, and all variance would be attributed to the environment. So if we randomly picked 10000 people, most likely all would have genes that led to the development of two legs. That lack of genetic variance would mean any and all variation in the number of legs would be attributed to the environment by default. We can only *measure* the influence of genes if there is genetic variance. Therefore how much control genes theoretically have and how much we can measure are not the same thing.

An answer that fails to distinguish between genes playing a role and observable heritability can still be good, provided that the two concepts are not being mixed up.

2. Heritability is the proportion of total variance that can be attributed to genetic variance. Increasing genetic variance that makes a difference to behaviour increases heritability because more of the total variance in behaviour can be attributed to genes.

   Leaving genetic variance the same and changing the environment in a way that affects behaviour increases total variance while leaving genetic variance unchanged, behavioural variance that can be attributed to genes is a smaller proportion, and therefore heritability decreases. For example, if you separate genotype from phenotype by cross fostering, you can give some of the rats

nurturing mothers, and others less attentive mothers. That does make a difference to how bold the rats will be later, increasing the total variance. Although the observed variance that can be attributed to genetics has not changed, that same variance is a smaller proportion of the now greater total variance, and so the heritability is lower.
Laboratory strains are extremely inbred, close to the point where all animals of the same strain would be clones. If you assume that they all are clones, then there is no genetic variation that can account for individual differences in behaviour. Heritability would be zero, no matter whether the rearing environment is the same for all animals or differs. In contrast, wild type rats will show some genetic variation, making it possible to measure a genetic contribution. Heritability should be higher for wild type rats.
An outstanding answer would bring up something like this: a heritability estimate is valid only for the combination of genetic and environmental variation present in the study. The very same trait will have different heritability in a population with different genetic variance or in a different environment. Heritability is not a property of a trait, it is a property of the combination of trait, population and environment. If you want to generalise to a population with either different genetics or environment, you need to think about how those differences are likely to affect the variances that can be attributed to genes and to environment. That is the key insight that shows an advanced understanding of heritability.

A different way to expand on the basic answer that would show an advanced understanding would be thinking about how wild type rats would be sampled from the population. How do you know whether a sample of wild type rats is actually a representative sample of the wild genotypes? A large enough random sample should approach the distribution in the whole population, but how do you sample randomly from the population of rats? If you put out traps, you are likely to preferentially trap the bolder rats, who are more likely to approach a novel object. That both skews and narrows the sampled phenotype, and distorts the estimate of heritability.

3. Ionotropic and metabotropic receptors: Ionotropic receptors, also known as ligand-gated ion channels are transmembrane ion-channel proteins that open after a ligand binds to it, the ligand is often a neurotransmitter (examples are AMPA-R and NMDA-R). Metabotropic receptors, also known as G-protein-coupled receptors, are not ion-channels. They are transmembrane proteins, but rely on a second messenger system (a G protein). A ligand / neurotransmitter binds and a second messenger is released inside the cell. This cascade may lead to opening of an ion channel (examples are olfactory receptors).
In both cases it depends on the ion that enters the cell whether the cell is depolarised (Na+, K+, Ca2+) or hyperpolarised (Cl-), not on the ligand that binds to the receptor.
Full points if the student clearly describes the difference as ion channel vs not, ligand-binding in both cases but the ligand does not enter the cell

4. IPSP and EPSP: An IPSP is an inhibitory postsynaptic potential, an EPSP is an excitatory postsynaptic potential. Both occur in the postsynapse. An IPSP can occur if Cl- ion channels are

opened. Cl- influx will reduce the membrane potential, bringing it further away from the threshold. An EPSP can occur if there is Na+ influx, this will bring the membrane potential closer to the threshold of triggering an action potential (AP). This is a very local change in membrane potential (close to the postsynaptic membrane) and does not make the entire neuron more / less negative. They do not last very long, as the cell will go back to its resting membrane potential (bonus point if that is stated)
Full points if the student clearly describes where IPSP and EPSP occur, that these are not Aps but local changes in membrane potential.

5. Neural adaptation: is the gradual decrease over time in responsiveness of e.g. sensory systems to a constant stimulus – during the lecture explained in the olfactory system (we adapt to smells). Adaptation is an efficient way as only changes are encoded in the nervous system. Constant stimuli provide no news and hence no new information. Understanding this principle of information encoding needs to be clearly stated to get at least a C.
If the student uses a sensory system like olfaction as example, they may mention the role of Ca2+, and CaMK (calciummodulinkinase) and or calmodulin. Mentioning the role of Ca2+ and the information processing part will be full points.

6. HPA-aksen er hypothalamus – pituiary – adrenal gland, eller på norsk: hypothalamus – hypofyse – binyrebark

   I forelesning og i pensumbok er det gått gjennom at deler av hypothalamus inneholder spesialiserte nevroner, eller celler, som kan skille ut prehormoner, som ikke er helt det samme som nevrotransmittere. Disse skilles ut i en lokal blodbane og føres ned til hypofysens framre del. Prehormonet kalles CRH og stimulerer celler i hypofysen til å danne et annet prehormon, ACTH. ACTH skilles ut til kroppens blodsystem. Målorganet er binyrebarken, der ACTH stimulerer celler til å slippe ut cortisol i kroppens blodsystem. Kortvarig virker cortisol stimulerende for sentralnervesystemet, langvarig kan cortisol ha negative effekter, spesielt for hukommelse. Dernest er det en feedback-sløyfe der reseptorer for cortisol i hjernen (hippocampus og hypothalamus) bremser produksjonen av CRH for å unngå overproduksjon.
   En god besvarelse nevner disse elementene. Det er positivt om man nevner at cellene i hypothalamus som gjør dette kalles parvocellulære og at det er framre del av hypofysen som er involvert samt at det er en forskjell i virkemåten på framre og bakre del av hypofysen.

7. sensory map: The student is free to choose its sensory system, the lectures showed sensory maps for the auditory system (A1, tonotopy) and somatosensory system (distorted homunculus in S1). They can also choose vision but this was not part of the curriculum.
   A sensory map are areas of the brain that respond in a spatially organised manner to sensory stimulation (e.g. touch, hearing, vision – but not olfaction). It is a topographic representation but not an exact physical representation. For touch, the receptive field sizes and density is reflected, more dense areas like hands have larger representations than the back or legs.
   To get an A the distinction between physical and sensory representation needs to be clearly stated. An ok answer is if they describe a sensory map for a sensory system (e.g. somatotopy and

homunculus)

8. distinguish thousands of different odors with 350 receptors: The concept the student has to explain is population coding, i.e. that information is encoded as distributed activity of nerve cells. The same receptor can take part in various “odors”. It is not a labelled line mechanism, i.e. there is no 1:1 of receptor : odor but rather the activity of many different receptors (and then the cells in the glomeruli / second order cells (mitral and tufted cells)) and their pattern of activity encode the odors. That allows flexibility, as loss of one receptor is not severely affecting odor discrimination (maybe for wine tasters but not for most daily life activities)

9. What are the differences between ON and OFF bipolar cells? What makes OFF/ON bipolar cells work the way they do? (hint: what kind of Glu receptors do ON and OFF bipolar cells have)
   Photoreceptors encode light by releasing less Glutamate (dark current as default), the community / convention is to reverse this at the next stage, so an ON bipolar cell is a cell that releases Glutamate when the photoreceptor receives photons, an OFF bipolar cell releases Glu when there is no light. To achieve this inversion the two types of bipolar cells have either metabotropic Glu receptors (ON bipolar cells) or ionotropic Glu receptors.
   If a photoreceptor releases a lot of Glutamate (in darkness) the ionotropic Glu receptor of the OFF bipolar cell will be depolarized and hence signal darkness. The ON bipolar cell will be hyperpolarised due to metabotropic receptors (the exact mechanism is neither in the book nor was it in the lecture, it might be opening Cl- channels, not wrong if the student deduces that from how else you can get this reversal)
   An ok answer mentions the two different receptors, a very good answer provides the reversal logic behind it.-

10. Explain how vibrations in the basilar membrane in the cochlea are converted into electrical/chemical signals: This question is about auditory transduction. When the basilar membrane moves so do hair cells. Outer hair cells amplify the movement (first point) This bending causes opening of hair cell receptors (on the stereocilia of the inner hair cells) which are mechanically gated channels and if opened K+ will flow in and depolarises the cell which opens Ca2+ voltage gated channels and that releases neurotransmitters. Two important concepts that have to be clearly stated to get an A are: 1) the hair cell is not a neuron, this is a graded signal, 2) the outer hair cells amplify the signal (basilar movement)
    bonus: it is K+ not Na+ that depolarises the hair cell, excellent answer if that is explained by the endolymph having a high concentration of K+, so equilibrium is not at -80mV but at 0mV

11. pain touch pathway: Pain and touch are both sensations that are transmitted through the nervous system via different pathways. Pain Pathway:

Pain signals are detected by specialized nerve endings called nociceptors, which are distributed throughout the body. When tissue damage or potential harm occurs, nociceptors are activated and send signals to the spinal cord. In the spinal cord, these signals are transmitted via two main types of nerve fibers: A-delta fibers (fast pain) and C fibers (slow pain). From the spinal cord, the pain signals travel to the brainstem and then to various parts of the brain, including the thalamus and the somatosensory cortex, where they are processed and perceived as pain.

Touch Pathway:
Touch sensations are detected by mechanoreceptors, which are also distributed throughout the body. Mechanoreceptors respond to mechanical stimuli such as pressure, vibration, and stretch. When these receptors are stimulated, they generate electrical signals that are transmitted through nerve fibers to the spinal cord. In the spinal cord, touch signals travel via specialized pathways to the brainstem and then to the somatosensory cortex, where they are processed and perceived as touch. Homunculus represents the body's surface in terms of sensitivity to touch. In this representation, body parts that are more sensitive to touch, such as the lips and fingertips, are allocated larger areas of the somatosensory cortex compared to less sensitive areas like the back. Pain tract crosses in the spinal cord and ascends contralaterally. Touch tract decussates in the medulla. So, it ascends the spinal cord ipsilaterally. Such difference in the site of the crossing helps spotting spinal cord lesions (damage); as a unilaterlal spinal cord lesion results in: Loss of touch and pressure Ipsilateral to the site of lesion. Loss of pain and temp contralateral to the site of lesion. However, both types of information are eventually processed contralaterally. It's important to note that while pain and touch share some similarities in their pathways, they are processed differently in the brain and serve distinct functions in sensation and perception.

12. What is a receptive field and where do we find it?
    Receptive fields are regions in the sensory periphery (e.g. skin) within which a stimulus or stimuli can change the electrical activity of sensory cells / activate receptors. They are found in many sensory systems, e.g. visual system (photoreceptors, On/OFF bipolar cells, Center-Surround ganglion cells etc, somatosensory system (touch and pain) and more. Important is that they are not solely properties of the first-order neuron but receptive fields are also found for higher order neurons / in later processing stages.

13. What are the different phases of memory, and which biological mechanism likely goes with it? (hint: synaptic plasticity)
    The different phases are short-term and long-term memory. If the student goes for working memory and long-term memory, also fine. Working memory: some neurons fire during the waiting period. Short-term: synaptic plasticity mainly as physiological changes, long-term: structural changes

14. Which role does the AMPA receptor and NMDA receptor play in long term potentiation?
    both are glutamatergic receptors and found in the postsynaptic membrane, binding of Glu on AMPA-R opens the ion channel and that leads to an EPSPs in the postsynapse. Glu

binding on NMDA-R will not open it yet. NMDA-R has a Mg+ ion that blocks the channel. The Mg+ is repelled if the postsynapse is depolarised, then – the co-detection of depolarisation in the postsynapse and the Glu binding (Glu is released from the presynapse) opens the NMDA-R. This leads to more depolarisation, as NMDA-R is an ion channel for both Na+ and Ca2+. Thus, Ca2+ cascade possible (no detail of the cascade needed). NMDA-R plays a role in "firing together wiring together". Both play a role in LTP. Without AMPA-R the postsynapse will not produce EPSPs. Without NMDA-R there will be no co-detection and Ca2+ cascade that leads to physiological and morphological changes

15. sleep: I forelesning og i pensumbok er det gjennomgått at man kan gjøre et skille mellom non-REM, dyp søvn og REM-søvn. I større detalj kan det beskrives som innsovning (stadie 1), non-REM (stadie 2) og dypere søvn (stadie 3 og 4). Det som kjennetegner stadie 1 er sporadisk forekomst av thetarytmer, det som kjennetegner stadie 2 er sporadisk forekomst av søvnspindler og K-komplekser og det som kjennetegner stadie 3 og 4 er en økende forekomst av deltarytmer. REM-søvn er kjennetegnet av en EEG-måling som ligner på en våken nevral aktivitet, dvs en forekomst av beta og gammarytmer.
    En god besvarelse nevner disse elementene. Det er et pluss om man kommer inn på at hjernebølger er noe som man kan observere fordi nevral aktivitet er organisert til samtidig aktivering, dvs bakenforliggende kontrollmekanismer muliggjør tidsluker av synkron nevral aktivitet som dermed kan måles som hjernebølger. Det er positivt om det nevnes at de forskjellige stadiene også har en rytmisitet, dvs forekomsten er 1-2-3-4-3-2-REM-2-3-4-3-2-REM osv.

16. What role does dopamine play in the CNS? Name at least one role and the brain region / behaviour that is associated with it?
    The lectures mentioned both its role in learning (briefly), its role in motivation / hedonic reward (addiction), and its role in prediction, i.e. signal for prediction error (book chapter on motivation). Dopamine is a neurotransmitter found in the ventral tegmental area and nucleus accumbens (dopaminergic pathway). Dopamine neurons signal errors in reward prediction, if a reward is "better than expected" there is more firing, if it is worse than expected there is less firing, if it is as expected there is no change in firing. A very good answer links this to efficient information processing, i.e. the brain processes changes and dopamine serves this role. Am error in expectation can then trigger learning.

17. Many drugs are antagonists or reuptake inhibitors. Explain what they do and how this changes synaptic transmission. An antagonist has the opposite effect than the neurotransmitter it is an antagonist for, i.e. it often blocks the transmission, instead of opening an ion channel (if ligand-gated receptor) it closes it or keeps it closed. A reuptake inhibitor prevents the neurotransmitter from being taken up by blocking the reuptake receptors on the presynaptic site (the neurotransmitter can still diffuse or if an enzyme exist be broken down – the latter is unlikely as the drug used would then be the enzyme or enzyme blocker) and hence the effect of the neurotransmitter on the postsynapse is

prolonged.

18. Circadian rhytm: I forelesning og i pensumbok er det gjennomgått at det er spesialiserte nevroner på retina (ganglionceller) som sender sine aksoner til SCN (suprachiasmatiske kjerne), og påvirker dennes aktivitet basert på mengden lys man fanger opp. Mer spesifikt er det mengden blått lys som er involvert i reguleringen av SCN. Inni SCN er det en variasjon av uttrykket til forskjellige gener som blir til en 24-timers (12 + 12 timers) syklus. Denne rytmiske endringen påvirker igjen kjerner i hjernestammen som da regulerer våkenhet eller oppstart av søvnstadier.
    En god besvarelse nevner disse elementene. Det er et pluss om man nevner at det er snakk om en klasse gener som benevnes som «clock» og om man spesifiserer at det er snakk om «ascending reticular activation system» i hjernestammen. Dernest at den søvnfremmende kjernen betegnes som VLPO og at søvn er en aktiv prosess og ikke bare fravær av nevral aktivitet.

Karakterskala som er benyttet

Bokstavkarakter: https://innsida.ntnu.no/wiki/-/wiki/Norsk/Karakterskalaen