Universitetet i Bergen

CANDIDATE

165

TEST

PSYK140 0 Atferd, helse og ernæring

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Praktisk informasjon / Practical information

PSYK140 - Behaviour, Health and Nutrition
May 23rd 2016
09.00-13.00

Change of language: Please click the gear symbol in the top right corner

No examination aids allowed
The candidate must answer 2 of 3 questions.
Both questions must be answered satisfactory in order to pass the exam.
The exam questions and your exam will be available in Inspera Assessment after the exam.

Assessment, explanation of grades and right to appeal

Your result on the exam will be announced on June 13th. You will receive an e-mail when the result is published on StudentWeb.

You can ask for an explanation of your grade. The deadline is June 20th.

Please go to Mi side (My page) to find the form or contact information needed.

You can appeal your grade. The deadling for submitting the appeal is July 4th.

You can find the appeal form on uib.no/psyfa or in our information center.

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In the event of a request for an explanation of a grade or a complaint of procedural errors in the question-setting, the examination procedure or the assessment procedure, the time limit for appeals pursuant to this section is reckoned from the date when the student receives the explanation or when the appeal is finally ruled on (§ 5-3, Act relating to universities and university colleges).

1 Eksamensoppgaven / Exam questions

Please answer 2 out of 3 questions here:

2. The population-based strategy of disease prevention will usually prevent more cases of disease compared to high-risk strategy.

a) Explain why this is the case.

b) Explain the advantages and disadvantages associated with both the high-risk strategy and the population strategy.

Despite the improvement of the health care system over the last century and the elimination or remedies of several diseases, there is a considerable rise in certain physical and psychological illnesses. There is an elevated consumption of medication and the medical care personnel has doubled over the last 25 years. The underlying causes are often unknown and highly discussed and it is even questioned whether only the stigmatization of diseases decreased and people are more likely to seek medical health, which is associated with an increase in diagnosis (especially if there is an increase of of diagnostic criteria of diffusable diseases).However, there are not only fluctuations of incidences regarding the population over time, but also differences between populations at the same time. This could be of interest concerning different prevention strategies that should adress the population as a whole. Another more common prevention strategy is based only upon those who are regarded to be at high risk. Some researchers argue that the the population-based strategy of disease prevention will usually prevent more cases of disease compared to the high-risk strategy. The essay will explain this assumption and will then outline the different advantages and disadvantages associated with both risk strategies.

The Preventive Paradox

Everyone in the population is exposed to a certain risk factor, but it varies considerably amongst individuals. One type of risk factor is dichotomous, which means it either exists or it does not exist at all. This could be for instance the genetic predisposition for a certain disease. The other risk factor can be seen on a continuous scale. When considering the latter the risk factor in a poulation is normally distributed. This could be e.g. the amount of fruits consumed, physical activity or the Body Mass Index (BMI). Additionally, the risk factor can be linear (if the risk increases steadily with the risk factor as seen of physical activity has a negative linear

relationship with cardiovascular disease), exponential (at a certain threshold of the risk factor there is a extreme rise in risk), or u-shaped (an extreme high or extreme low risk factor is associated with an increase of risk e.g. vitamin defficiency vs. vitamin excess).

There has to made a distiction between the causes of case and the causes of incidence. The former focuses on investigating why a certain patient has a certain diseases relating to the high-risk strategy, wherase the latter tries to figure out why there is a higher prevalence in some populations compared to others which is population-based.

A normal distribution of a certain disease prevalence or physical measurement looks similar to the normal distribution of another population or the same poulation over time. However, there is often seen a difefrence of the means. Even though the normal distribution of the systolic blood pressure in the Kenyan Normads and London civilan appears almost the same, the mean of the Kenyan Normads is considerable lower. This indicates that it might be impossible to find the underlying causes of the risk factor or disease if one is only investigating individual differences within a population, since there is often an exposure to the same underlying factor observed. E.g. in Scotland there is a high prevalence in cardiovascular diseases, but only if Scotland is compared to England it seems obvious that in Scotland everybody is exposed to the soft water, which is not the case in the non mountain areas of England.

Furthermore, even though everyone is at a different degree of risk in developing a disease, only about 10 % are regarded at high risk. Nevertheless, the some of the remaining 90% who are at low risk will develop the disease. In total, the majority of the population is at low risk, but will still provide more cases of the disease. This is called the "preventive paradox". A simple calculation might facilitate the demonstration: Assuming a population of 10 000 people. 10 % of the population are at high risk (15% risk) to develop a disease. 90% at low risk still have a low risk of 5 % to develop the disease. The high risk population provides 150 cases and the low risk population provides 450 cases.

The preventive paradox might be a convincing to demonstrate that a shift of the whole population mean in the favored direction can prevent in total more cases than if only the high-risk population is adressed with prevention strategies. Instead of focusing only on those at high risk by giving them treatment and tailored intervention programs, it could be more effective to apply a prevention strategy based on the whole population. The population based strategy is alredy applied in some countires for certain cases and might be more developed in the future health concerns. Examples of the population-based strategy is taxation of sugary beverages and alcohol. In Mexico ther is a programm that targets the awareness of the epidemic obesity. Everyone who performs ten sit-ups obtains a subway ticket for free. Even though intrinsic motivation of behavioral change might be undermined by extrinsic rewards such as saving money, it could help to lead to a shift of the whole population risk and would then helpful in preventing. Information education could be helpful too, but it is discussed, since the people with low socioeconmic status are disadvantaged regarding informatio based prevention stratgies and there are many variables that need to be optimized that everything goes smoothly through the information processing model. Further research is necessary to develop more sophisticated population-based strategies.

Advantages and Disadvantages of High-risk vs. Population-based stratey

The high-risk strategy that adresses only those who are classified as being of high risk can provide a prevention program that is perfectly tailored to the individual. The resources are used in an efficient way. Additionally, since the individual is aware of being at high risk and the potential consequences, he or she might be very motivated in e.g. engaging in a healthier behavior and accepting an intervention programm. The physician might be as wll highly motivated since the individual can be screened and there is a high probability that the treatment will improve the well-being and the phsical state of the patient. The outcome is measurable and can then, if successful, be applied to other individuals.

However, it is based on a clear criteria cut-off whether someone is at high-risk or not. Screening criteria are very often changed and individuals need to be screened very frequently, since the risk migth change over time. The strategy ignores those who are on the way to be at high-risk. It also adresses only the awareness of those who are more likely to become sick and neglegts the health-promotion to the whole population. Furthermore, it can be a strain for the individual to engage in a certain behavioral change that contradicts social norms if it is perceived as behaviorally not appropriate.

The population-based strategy adressed the whole population with e.g. campains, substitution of healthy food, supplementation of vitamins in groceries or prohibition of smoking. Compared to the high-risk strategy, it is behaviourally more appropriate and does not lead non-conformity of social rules. For instance, nowadays it is widely socially accepted not to smoke, since people have an elevated awareness of its adverse effects. Furthermore, it targets everyone in the whole population, which could not only lead to prevent diseases, but also promoting well-being.

Nevertheless, individuals could underestimate their given risk factor and are less motivate in engaging in healthy behavior. Physicians might as well regard health promotion/prevention for everyone as state concern and are less motivated, since the beneficial outcomes is less visibly. There could be only a low change in the populations average and a very low change in individuals that save incidences. E.g. the negative outcomes, such as mortality rate, of cholesterol decreasing druggs are more pronounced than what the assumptions how much lifes the drugg probably saved. Even though, the population-based staregy could be far cheaper, the beneficial outcomes are hardly measurable.

Even though, both strategies show strong differences in their core element, it is important that they are not mutually exclusive and it might lead to the greatest benefit to apply both in a combined manner. However, both strategies need to be further developed in order to promote the highest well-being amongst the greatest amout of people. Active and passive involvement of treatment, such as an egological approach involving both the environment and the individual could be a potential approach to face the challenge.

3. Describe shortly the hormonal control of appetite, hunger and blood glucose control. Discuss how sleep loss may influence our appetite and choice of food. What can be the long-term consequences of chronic sleep loss on our metabolism and health?

During the last decades there is several reseacher investigate the regualtion and interaction of physical mechanisms such as appetite regulation, metabloism and the association with sleep. At the same time, there is an increased concern about the rise of obesity especially in westernized countries. The World Health Organizsation (WHO) describes obesity as an epidemic problem. Since recent research found an association between sleep variables and the prevalence of obesity the topic seems to be highly relevant, especially in finding intervention strategies.

First the hormonal control of appetite, hunger and blood glucose control as well as the basics of sleep/wake-meachanisms are shortly described. This is followed by a discussion of how sleep loss may affect our appetite and food choice. Additionally long-term consequences of chronic sleep loss on our metabloism are outlined.

Hormones, appetite regualtion and sleep

The endocrine system is responsible for the hormone regulation and itself is regualted by the Central Nervous System (CNS) consisting of the brain and the spinal cord.

There are several hormones that regulate our appetite and hunger. One long-term appetite regulator is insulin: Insulin is released by the pancreas in reaction to a elevation of blood-glucose. The brain, which can use only glucose as a fuel detects the insulin concentration, since the blood-brain-barrier (BBB) is permeable for insulin. This is essential for a homeostatic and stable blood sugar level. The realease of insulin into the blood peripehery leads to a reduction of the blood glucose and an increse in glucose uptake into the cells where it can be used. Excessive glucose is stored and converted in the liver for a later break-down. Glucacon is the homeostatic counterpart of insulin, since it raises the blood sugar level if the level is too low. The mutual feedback loops lead to an optimized glucose level in not pathological individuals.

Leptin another long-term appetite regulating hormone is released in the relation to the adipose tissue. It is characterized by the appetite supression and the increased energy usage. It peaks during slow-wave-sleep (SWS).

Ghrelin released in the stomach, on the other hand, promoptes hunger and appetite. In healthy humans the ghrelin levels raise before a meal and fall immediatly after food ingestion. These hormonal meachanisms allow humans a appropriate food intake.

Our sleep is modulated by three main meachanism: One factor is the metabolic homeostasis that is maintained through an increase in sleepiness during the wakeful period due to an accumulation of metabolic substances that peak before sleep onset and obtain a "clearance" during sleep (homeostatic factor S) (> similar with somnogens such as adenosine). Additionally, the circadian clock is another very important sleep/wakefulness regulator (cricadian factor C). The central circadian clock (pacemaker) is located in the Suprachiasmatic Nucleus (SCN) in the anterior hypothalamus of the brain. It is characteriszed by its rhythmicity. It is regulated by exposure of light that hits the retina and the SCN is activated through the retinocorticotract. Additional clocks are found within the CNS, but also in the periphery. It is estimated that up to 20% of the genes show a circadian rhythmicity, called clock genes. The third mechanism are the zeitgebers that come from outer sources including food intake and light exposure. For a healty weakful and sleeping pattern all mechanisms need have an appropriate interplay.

Sleep loss and the Influence on Appetite and Food Choice and Long-term Consequences

Over the last decades a detrimental decrease of sleep duration is obsereved. 30% of the American population sleeps less than six hours per night. At the same time there is an observed increase in overweight and obesity (about 60% in the U.S.). Several studies indicate an association between sleep deficiency and a heighted level of BMI.

Several studies were examined to investiagte the relation between leptin as well as ghrelin and sleep duration. It has been shown that after a few days of sleep restriction to only 4 hours led to an significant increase in ghrelin and a decrease in leptin, which was then associated with an elevated appetite and hunger perception compared to those who were allowed to sleep up to 9 hours. The hormonal changes are almost as strong as those individuals hwo were food restricted (70% energy intake of energy expenditure). The same is found for individuals who were restricted to the slow wave sleep, but without total sleep duration restriction. Furthermore, impaired glucose tolerance and decreased insulin sensitivity are already observed after only a few days of insufficient sleep duration. Several studies confirm those findings with similar results.

The findings indicate that insufficient sleep lead to a disturbance in the biological clock and in the regualtion

of hormones that are clock dependent. The increase in appetite and hunger may lead to an increased food consumtion and an increase in BMI. Overweight and obesity may be predictive for further negative health consequences. In fact, the BMI in men and women increases drastically with a sleep period less than 6-7 hours as well as with a sleep duration over 9 hours. Additionally, there are correlations between sleep deprivation and increased sleepiness, less alertness, increased error making, hyperinsulinaemia, hypertension and increased low density lipoprotein (LDL), cancer proneness etc.

It is especially a concern, since the studies demonstrated already detrimental health deteriorations after only a few days of sleep deficiency. However, short-term disturbances seem to be reversible.

The long term effects, however, are associated with overweight and obesity and all the aforementioned related diseases. However, the causal direction is not necessarily one-way directed. Rodents that were received a high fat diet and that developed then overweight showed behavioural changes such as increased food consumption, changed motoractivity, and more non-rapid-eye-movement seep (NREM). Similar changes were obserfed in genetically modified leptin hormones in rats. Therfore it seems like a vicious circle and simplified it could be assumed that both poor sleep can cause obesity and the other way around.

Especially in our society today we are exposed to several factors that are related to insuffient sleep such as jet lags, shift workers (statistics show an heightened prevalence of overweigt/obesity ion shift workers), too late bed times and the use of electrical devices in bed (studies have shown an increased sleep onset latency due to the light of the blue spectrum in those devices). BMI and circumference were also related in school girls, but those findings are questionable due to the sample size.

Our food choice is as well modified by the quality and quantity of sleep. However, some clock genes determine what kind of chronotype we are and whether we are morning or evening persons Per (5/5 or 4/4) or in between. If we are evening persons, research has shown that we are more likely to consume fast food and caffeinated drinks. Additionally, evening chronotypes are obviously less alert and active during the mornings and are more likely to skip the breakfast and having high-density food late at night.

Furthermore it was found that individuals with low calcium and selenium intake show difficuties in sleep-onset, whereas those with difficulties in sleep maintenance show a higher salt consumption and a low carbohydrate intake.

Nevertheless, research indicates an influence of both sleep duration and quality on our appetite and behavior which influences our health, but the food consumption and BMI determine also our sleeping patterns. New research has shown that an imlication for interventions could be a reset of the biological clock, which was found in calorie reduction and intermittent fasting. The beneficial outcomes those behavior changes were not only restricted to sleep, but also to an extended life-span, and decrease in various diseased and a greater well being. Additionally, an intervention on a more macro level perspective would be beneficial too. This could be done by improving work conditions, especially amongst shift workers or those who travel between time zones. All in all, it should be emphasized to be more active during the active period and to rest during the inactive period, since this is the way the human biology is constructed.

Answered.