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The effect of sleeping patterns on mental distress and overweight risk under shifting light conditions in north Norway.

Some findings from the Tromsø Study

May Trude Johnsen

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Sleep

Sleep is supposed to be,
By souls of sanity,
The shutting of the eye.

Sleep is the station grand
Down which on either hand
The hosts of witness stand!

Morn is supposed to be,
By people of degree,
The breaking of the day.

Morning has not occurred!
That shall aurora be
East of eternity;

One with the banner gay,
One in the red array, --
That is the break of day.

Emily Dickinson (1830-1886)

Contents

Acknowledgements

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May Trude Johnsen, February 2013

Summary

This dissertation presents the results from three studies that aimed to investigate sleep-wake rhythms and certain health risks in a subarctic community. Sleep timing and duration are influenced by outdoor light, and the darkness of winter has been found to impact the level of mental distress and sleeping problems. Sleep duration, chronotype (preference for being active during the morning or evening) and social jetlag (mismatch between the body's biological clock and social schedules) have also been associated with body mass index and body fat distribution. Study 1 aimed to investigate how the extreme photic environment throughout the seasons would affect sleep-wake rhythms. Study 2 focused on sleeping problems and mental distress during winter, and study 3 examined the association between sleep variables and measures of overweight and obesity.

The study was based on the sixth survey of the Tromsø Study (Tromsø 6) in 2007-2008 and included entire birth cohorts and random samples of the population aged 30 to 87 years, living in Tromsø. The participants completed questionnaires including self-reported sleep times, lifestyle and health. Height, weight, waist and hip circumference, and biological factors (non-fasting serum level of total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides and glucose) were measured. The seasonal distribution of chronotypes and health variables was calculated based on the answers at the participation dates.

A small (8 minutes) but significant earlier midpoint of sleep in summer compared to winter was found. The prevalence of self-reported mental distress (HSCL-10 ≥1.85) in Tromsø was lower than what has been described as average for Norway (7.4% versus 11.4%). There were no significant differences in the reporting of current mental distress depending on season but more participants reported current sleeping problems in winter than in the other seasons. The sleep length that was associated with the lowest mean BMI and the lowest mean waist circumference was 8-9 hours. Short sleepers (<6 h) had about 80% increased risk of being in the BMI ≥25 kg/m2 group and male short sleepers had a doubled risk of having a waist circumference ≥102 cm compared to 8-9 h sleepers.

Other factors than daylight exposure may be more important in the regulation of sleep patterns for people in the subarctic. Furthermore, the use of stimulants (alcohol and tobacco) or excessive indoor and outdoor light may have masked the seasonal effect of variation in daylight. There were no significant seasonal differences in mental distress, but sleeping problems were most prevalent in winter. From a clinical point of view, some people in the subarctic clearly feel that they are mentally negatively affected by the darkness of winter. However, the negative impact of winter on mental distress for the adult population is not conclusive.

Abbreviations

ANOVA	Analysis of variance
BMI	Body mass index
CBT	Core body temperature
CD	Circadian desynchrony
CI	Confidence interval
DLMO	Dim light melatonin onset
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition
DST	Daylight saving time
EET	Extremely early types
ELT	Extremely late types
ET	Early types
HDL-cholesterol	High density lipoprotein-cholesterol
HSCL-10	Hopkins Symptom Checklist, 10-item version
HSCL-90	Hopkins Symptom Checklist, 90-item version
ICD-10	International Classification of Diseases, Tenth Edition
IT	Intermediate types
LDL-cholesterol	Low density lipoprotein-cholesterol
LT	Late types
MCTQ	Munich ChronoType Questionnaire
MEQ	Morningness-Eveningness Questionnaire
MSFsc	Sleep corrected mid sleep on free days
MI	Midwinter insomnia
SAD	Seasonal affective disorder
SCN	Suprachiasmatic nucleus
SD	Standard deviation
SFA	Subcutaneous fat area
STATA	Statistical package by StataCorp LP

UNN	University Hospital of North Norway
VFA	Visceral fat area
WC	Waist circumference
WHR	Waist-to-hip-ratio

List of articles

1. Lack of major seasonal variations in self reported sleep-wake rhythms and chronotypes among middle aged and older people at 69 degrees North: The Tromsø Study. Johnsen MT, Wynn R, Allebrandt K, Bratlid T. Sleep Med. 2012 Dec 5. doi:pii: S1389-9457(12)00386-3. 0.1016/j.sleep.2012.10.014. PMID: 23219143

2. Is there a negative impact of winter on mental distress and sleeping problems in the sub-arctic: The Tromsø Study. Johnsen MT, Wynn R, Bratlid T. BMC Psychiatry. 2012 Dec 12;12(1):225. PMID: 23234541

3. Johnsen MT, Wynn R, Bratlid T (2013). Optimal sleep duration in the subarctic with respect to obesity risk is 8-9 hours. PLoS ONE 8(2): e56756. Doi:10.1371/journal.pone.0056756

Introduction

This dissertation presents the results from three studies. These studies aimed to investigate the chronotype distribution and sleep characteristics of people in a subarctic community, the associations between their sleep variables and measures of overweight and obesity, and if they reported more mental distress or had more sleeping problems during winter.

Biological rhythms/circadian clocks

A universal, fundamental characteristic of animals and plants is that they have a daily rhythmicity, known as circadian rhythm [1]. All cells in the human body are able to generate circadian rhythms, and genetic variation is partly responsible for the individual differences of these internal circadian clocks [2, 3]. Circadian clocks control a wide variety of rhythms, from biosynthetic to behavioural, and according to Hur et al. [2], genetic variability accounts for about 54% of the total variance in our preference of being active early or late in the day.

In humans, the circadian pacemaker (or "master clock") is located in the suprachiasmatic nucleus (SCN), a small group of brain cells located in the anterior hypothalamus, situated directly above the optic chiasm [4]. Under conditions shielded from solar and social time, the internal pacemaker generally expresses rhythmicity with a period of approximately 24 hours by interacting (entraining) with time cues ("zeitgebers") in the environment [5, 6].

The strongest and probably most important time cue is daylight and the light/darkness cycle [7, 8], but temperature, health conditions, nutrition, and social schedules are also important time cues [9, 10]. The entraining of the human circadian clock is disrupted by exposure to artificial light cycles, and by irregular meal, work and sleep times [11]. This mismatch between the natural circadian rhythms of our bodies and the environment is called circadian desynchrony (CD). In humans, light is detected exclusively by the eyes, by rods and cones, and by melanopsin receptors [12-14], a photopigment in the ganglion cell layer in the eyes.

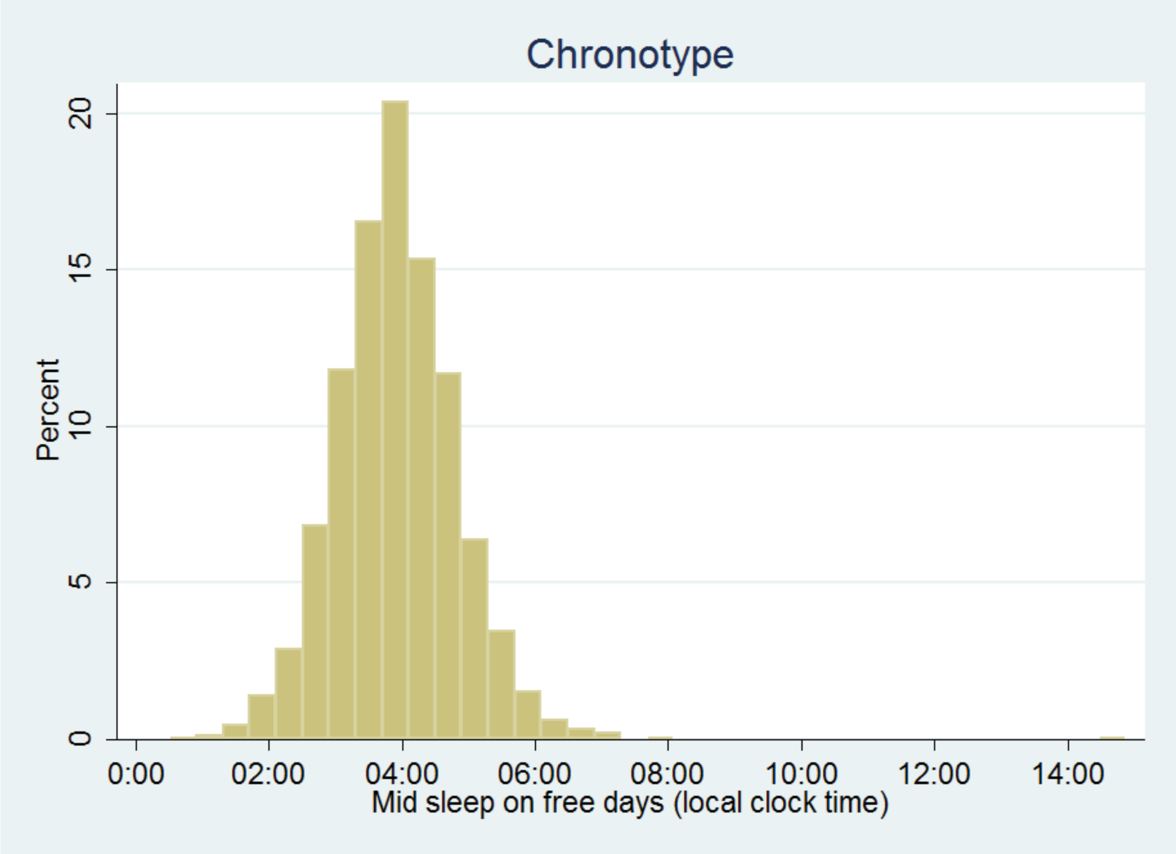
Individual differences in circadian phase preference are referred to as ‘chronotype’: individual preference to be active early or late in the day [15, 16]. Morning types or “larks” are those who wake up early and are most alert in the first part of the day, while evening types or "owls" are those who are most alert in the late evening hours and prefer to go to bed late. Intermediate types fall somewhere in-between. In several studies, the circadian phase has been found to occur about 2 h earlier in early compared to late types [17]. Extreme early or late types may have difficulty participating in normal work, school, and social activities.

Studies suggest a strong association between human chronotype and daylight exposure, and morning exposure to daylight has been found to advance the chronotype [18, 19]. One study has suggested that chronotype progressively advances by more than an hour when people spend up to 2 hours outdoors per day, and that beyond 2 hours of natural light exposure, the chronotype changes very little [10].

A person’s sleep-wake cycle is determined by a number of factors beside the described circadian process. On the one hand, a homeostatic process involves an increasing sleep drive that builds up during wake and culminates in sleep [20, 21]. On the other hand, external factors such as work schedules and social factors influence decisions about when to go to bed and wake up. Normal work schedules require early wake up times during the work week, which is most suitable for morning chronotypes. Late chronotypes often experience difference in bed times and rise times (sleep timing) between the work week and free days [22]. This mismatch between the body’s biological clock and social schedules is known as ‘social jetlag’ [23].

There are just as many short and long sleepers among early chronotypes as there are among late ones [10]. The chronotype distribution is illustrated in Figure 1. Gender differences in sleep patterns have also been reported, with women going to bed earlier and sleeping longer than men. Men are, on average, later chronotypes for most of their adulthood [24]. This sex difference disappears at around the age of 50, which coincides with the average age at menopause. People over 60 years of age become even earlier chronotypes than they were as children. These systematic changes of chronotype with age, together with the significant sex differences between puberty and menopause may indicate that endocrine factors are involved in the age-dependent changes of chronotype [25, 26].

Figure 1:

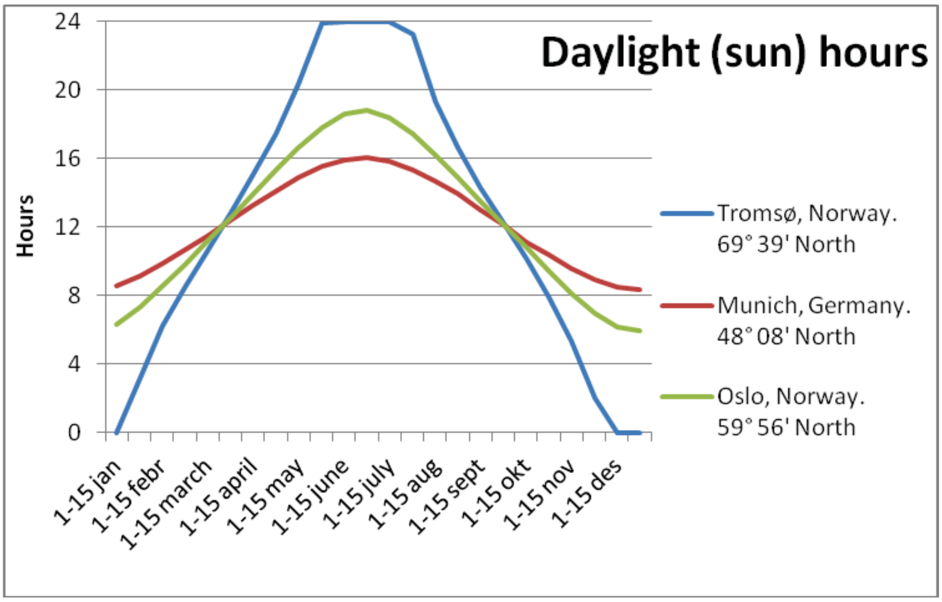


The figure shows chronotype distribution in the study population.

An arctic perspective

All organisms within the arctic and subarctic regions are exposed to large seasonal changes in daylight exposure. At high latitudes above the Arctic Circle (locations north of 66°34'N latitude), the sun will be above the horizon all day in the summer and below the horizon all day in the winter. In August, September, October and the first half of November, the daylight in Tromsø (latitude 69°40′N) rapidly decreases with about 9 minutes each day. From 27 November, the sun is below the horizon line, giving very little daylight for nearly 2 months. About 20 January the sun is back above the horizon, and a rapid increase in daylight occurs. This culminates 20 May when the sun is above the horizon for 24 hours. This midnight sun period lasts until 22 July when the cycle starts over again (see illustrations of this cycle in Figure 2). The same phenomenon happens for locations south of 66°34′S latitude.

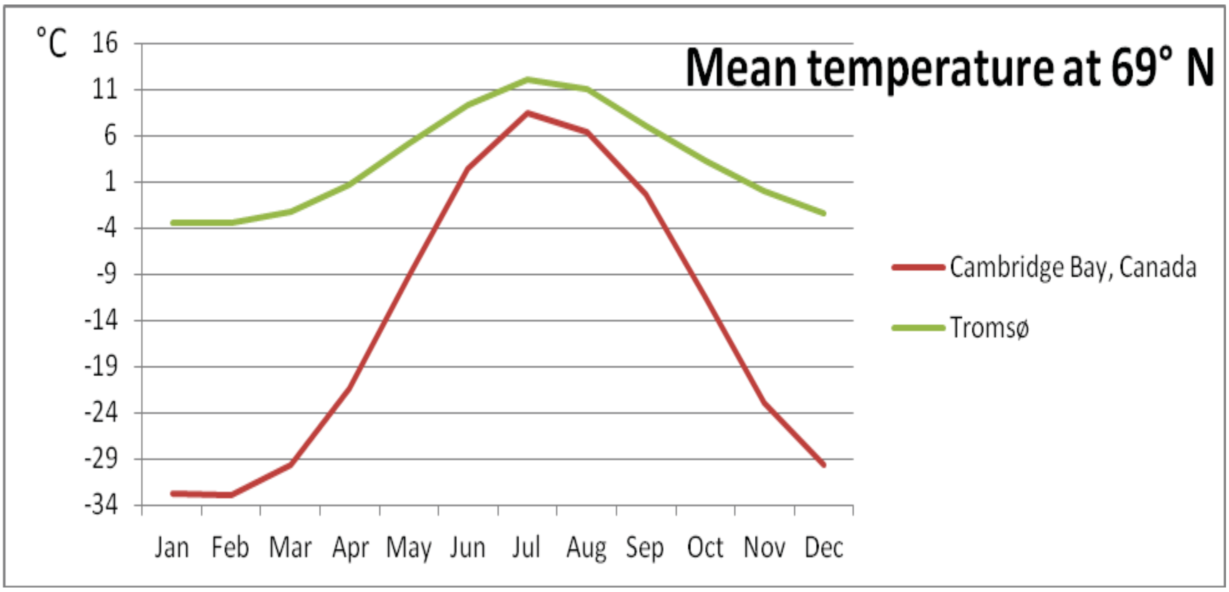
Figure 2:



The figure shows hours of sun above the horizon at different latitudes in the year 2008 (data from Astronomical Almanac by H.M. Nautical Almanac Office, UK). The times for sunrise and sunset are based on the ideal situation, where no hills or mountains obscure the view and the flat horizon is at the same altitude as the observer. Sunrise is the time when the upper part of the sun is visible, and sunset is when the last part of the Sun is about to disappear below the horizon (in clear weather conditions).

Northern Norway is ideal for studying the seasonal influence of natural light on sleep and mental distress because of the seasonal differences in light exposure. Due to the Gulf Stream, the climate is relatively mild, the latitude taken into account (see Figure 3).

Figure 3:



The figure shows mean temperature per month in Tromsø (69 °N 18 °E) and Cambridge Bay (69 °N 105 °W). Weather data from the Norwegian Meteorological Institute (http://www.met.no).

Seasonality of sleeping problems and mental distress: some prior findings with emphasis on the Subarctic

The term 'insomnia' is often defined as an individual's report of difficulty with sleep or used to describe the presence of polysomnographic evidence of disturbed sleep. Insomnia symptoms are some of the most frequent complaints in the general population. The prevalence of insomnia is 10-50% depending in part on definitions and data-collection methodologies [27, 28]. The clinical symptoms of Midwinter Insomnia (MI) in Northern Norway, as described by Lingjaerde et al. [29] start at the beginning of the Polar night period (ultimo November) and last until the sun returns over the horizon (ultimo January). MI severity varies from moderate difficulties in falling asleep to almost total inability to sleep during the whole night.

Seasonal sleep difficulties have been recognized as a problem in northern Norway for several centuries. During the second half of the 19th century, pupils and teachers in Tromsø complained of sleep problems and loss of energy in the polar night period. This led to the introduction of the so-called “Dark period time table” in the schools in Tromsø in 1875, starting later in the morning and shortening the school days in December and January. This “Dark period time table” was used as a local time table in Tromsø until 1976. Around 1930, there were also reports of school children with tiredness and sleep disturbances getting artificial light treatment at school during the dark period, but these treatments were not scientifically reported.

In the early 1950’s, scientists began reporting from studies of sleep related to season. As early as 1951, Kleitman found that people in Tromsø slept longer in winter than in summer [30]. In 1957, Devold did the first regular survey of sleep problems among adolescents during the dark period [31]. He found that, from September to January, almost 40% of the pupils in Tromsø complained of sleep problems, compared to 6% of pupils in southern Norway. In the years after, several studies of seasonal sleeping problems in northern Norway have been performed, but most of these have been smaller studies. In 1959, Kjos reported that insomnia was more common among high school students in Tromsø during January than April [32]. In 1975, Weitzman et al. published data from a small study on 7 healthy men and found no seasonal difference in total sleep [33]. Lingjærde et al. studied the effect of intensive bright light treatment on 7 patients with midwinter sleeping problems and found shortened sleep latency and (a not statistically significant) increase in total sleep time after light exposure [29, 34].

Sleeping problems have also been studied among 903 Norwegians and Russians living in Svalbard (at 78°N) [35]. The prevalence of sleeping problems was nearly four times higher among Russians than among Norwegians. As the Russians were recruited from a lower latitude than the Norwegians, inadequate acclimatization after migration to the north was suggested as an explanation of the findings.

Chronobiological mechanisms related to circadian rhythms, melatonin, serotonin, and retinal photosensitivity have been claimed to play a significant role in many cases of seasonal mood variations because of the described linkage between the syndrome and the lack of daylight in winter [29, 36, 37]. In 1984, Rosenthal et al. first described the syndrome seasonal affective disorder (SAD) with typical symptoms of depression, hypersomnia and difficulty with waking up in the morning, fatigue, increased appetite and carbohydrate craving [38]. SAD is also known as 'winter depression' due to a predictable onset in the autumn/winter months and a spontaneous remission in the spring/summer period [39]. A recent study comparing students in Tromsø with students in Italy (44°N) did not support the hypothesis that seasonal affective disorder is linked to the amount of daylight and latitude of living [40]. Another study of students in Tromsø and Ghana (5°N) indicated that lack of daylight in Tromsø was related to phase-delayed rise and bedtimes, increased problems falling asleep, daytime fatigue, and depressive mood [41]. Total sleep duration and sleep quality appeared unaffected in Tromsø. They found small to non-existing seasonal differences in Ghana.

Mental distress related to season has also been investigated in North Norway during the last decades. Haggag et al. questioned 1000 persons in Tromsø twice, in summer and in winter, and found major seasonal mood variations [42]. In 1991 Hansen et al. studied 7759 persons in Tromsø and casted some doubt upon the importance of daylight for mental distress in the general population [43]. Later, Hansen et al. found that the prevalence of self-reported depression in Sør-Varanger (latitude 70°N) was surprisingly low in winter considering the lack of daylight [44].

Seasonal impact on biological rhythms and mood has also been investigated in other countries in both the northern and southern hemisphere [45-47]. These findings will be discussed further in the Discussion section.

Associations between sleep duration and weight gain

Obesity is a rapidly growing problem affecting children and adults all over the world [48-50], and the number of obese humans has now exceeded the number of starving people [51]. Groups with lower socioeconomic status are more vulnerable to obesity [52, 53]. The most commonly used measure for overweight and obesity is the Body Mass Index (BMI), but both skin fold thickness and waist and hip circumference (waist-hip-ratio/WHR) can provide more useful information [54, 55].

Diet and physical activity play an important role in the development of overweight and obesity. Wyse et al. have recently hypothesized that artificial lightning play an important role in the development of obesity [11]. Their hypothesis is that artificial light facilitates chronic circadian desynchrony (CD), which induces a metabolic and behavioural phenotype that is predisposed to the development of obesity.

Many studies have indicated associations between short sleep duration and different health outcomes like total mortality, type 2 diabetes mellitus, cardiovascular disease, and self-rated health [56-60]. Numerous cross sectional studies have also shown associations between short sleep duration and increased risk for weight gain and obesity [54, 61-66]. However, the causal direction is not evident, and other studies have found no association between short sleep duration and weight gain [67, 68], or inconclusive results [69-71].

Because sleep duration is just one of a large number of factors that can be associated with weight gain, the importance of controlling for confounding factors has been underlined [72]. As far as we know, controlling for different socioeconomic variables, lifestyle variables, health variables, biological factors and sleep variables in the same study of overweight and abdominal obesity, has previously not been done.

The Tromsø Study

The Tromsø study (www.tromsostudy.com) was set in Tromsø, a modern university city in North Norway at 69.4 degrees north. Tromsø is the largest city in North Norway and the population was 65300 in 2008. The first Tromsø study was initiated in 1974 in an attempt to help combat the high mortality due to cardiovascular diseases in Norway. The primary aim was to determine the reasons for the high mortality of cardiovascular disease, and also to develop ways of preventing ischemic heart disease and strokes. The study was gradually expanded to include many other diseases, such as rheumatism, neurological and mental diseases, dermatological diseases, gastrointestinal diseases, cancer, and osteoporosis. The health study has been repeated at regular intervals and involves a large proportion of the municipality's population. Each of the studies has combined a questionnaire, physical examinations, and blood and urine samples, and all residents in the chosen age groups have been invited to participate. Jacobsen et al. have given a more detailed description of the study design in a cohort profile paper [73].

Several studies concerning physical health implications related to season in the high north have been performed, based on the Tromsø study. Midtby et al. found a seasonal change in the level of bone formation markers and concluded that season also had to be taken into account when measuring these bone markers [74]. Hopstock et al. found only a small increase in myocardial infarction (MI) during the darkest winter months, and modest individual seasonal patterns for all risk factors for MI [75, 76]. They found that the explanations for these results could be that this subarctic population was well adapted to the harsh climate and behavioural protection like using warm clothing.

Monitoring the circadian rhythm

Several assessment strategies can be used for monitoring the behaviour of the circadian pacemaker, but not all will be suitable for population studies like ours.

Sleep questionnaire

A sleep questionnaire is known to be a useful tool in recording a person's sleep-wake rhythm The Morningness-Eveningness Questionnaire (MEQ) [16] is the most widely used in studies to classify chronotype categories [77-79]. The MEQ consists of multiple-choice questions, with each question having four response options. The responses are combined to form a total score which indicates if the respondent is a morning or an evening type.

The Munich ChronoType Questionnaire (MCTQ) was later designed to collect information about the actual timing of daily sleep and activity (such as bed-times and rise-times and sleep latency) on work days and on free days [80]. Validation of the MCTQ was performed by letting a study population fill out the MCTQ, and having a subgroup keep a sleep log for 5 weeks. Actual and indicated sleep times on both work days and free days for the subgroup were highly correlated ($r=0.6$, $p<0.0001$) [80]. The MCTQ has also been validated against the MEQ [81]. This validation showed that chronotypes based on the MCTQ correlate with morningness-eveningness as measured by the MEQ ($r=-0.7$, $p<0.05$), but the MCTQ collects additional detailed information on sleep-wake behaviour.

Monitoring biological circadian phase markers

Circadian phase markers can be used to detect circadian timing or circadian timing abnormalities as in circadian rhythm disorders. Historically, the core body temperature (CBT) rhythm has been used more extensively than any other circadian phase marker, but the circadian signal from the CBT rhythm can be easily masked by activity, food intake, and sleep. The ‘constant routine’ regimen is an attempt to minimize all external rhythms and to study the internal clock directly: the subjects have to stay awake in surroundings of constant temperature, lighting, humidity and social contact, and are fed equally distributed small meals for at least 24 hours [82].
Melatonin can be measured in saliva or plasma, or urine can be used for measuring a melatonin metabolite (6-sulphatoxy melatonin). Light exposure suppresses (masks) melatonin secretion, and samples must be obtained under dim light conditions. The procedure is termed the dim light melatonin onset (DLMO). Other biological markers, like cortisol, growth hormone or TSH, can also be used, but are probably of more limited value.

Findings from several studies suggest that measuring the melatonin profile is the most accurate biological marker for circadian phase [83]. However, the necessity of specified monitoring procedures (like “the constant routine protocol” or the DLMO) makes biological markers less suitable in large population studies like ours.

Sleep logs

Sleep diaries or sleep logs are self-reported recordings of an individual's sleeping and waking times over a period of days or weeks, and can also provide data on sleep quality. These logs are recommended for evaluating the sleep-wake rhythm, especially in persons with circadian rhythm sleep disorders. Sleep logs can also be used in circadian rhythm studies. Since our study was a cross-sectional study and each participant attended the study only once, we could not use sleep logs as a data collection method.

Actigraphy

Actigraphy is a method of monitoring human rest/activity cycles and is useful for determining sleep patterns. A small actigraph unit, similar to a watch, is worn by a person and can record the person’s movements (and thereby also sleep) for several weeks. The advantage of actigraphy is that it can record continuously for 24-hours a day for days, weeks or even longer, and that the recording equipment is very small and easy to use. However, in circadian rhythm studies, it requires that the participants are followed up over time, and this is why this method was not useful in our cross-sectional population study.

Aims of the thesis

The specific purposes of the present thesis were:

1. To analyze the chronotype distribution and sleep characteristics in a population living at 69 degrees north, where there are large differences in daylight between seasons, and to investigate seasonal differences in the distribution of sleep duration and chronotype in this population (Study 1).
2. To investigate whether people living in the sub-arctic reported more mental distress or had more sleeping problems during winter than in other seasons (study 2).
3. To examine the associations between sleep variables and body composition for people living in the subarctic, taking a range of variables into consideration (Study 3).

Material and methods

Material

This thesis is based on the sixth survey of the Tromsø Study (Tromsø 6), which is a cross sectional study of entire birth cohorts and random samples of subjects in the municipality of Tromsø. The invited population consisted of four groups, all of them recruited from the general population:

- Subjects who took part in the special study in the Tromsø IV survey. This included everyone in the 55-74 age group. In addition, a random 5-10 percent in the 25-54 and 75-85 age groups were given the opportunity to undergo an extensive special study. A total of 7965 people accepted this offer.
- A 10% random sample of subjects aged 30-39.
- All individuals aged 40-42 or 60-87.
- A 40% random sample of subjects aged 43-59 years.

Data for the Tromsø 6 Study was collected continuously from 1 October 2007 to 19 December 2008, except in July 2008. Of the 19762 persons invited, a total of 12984 persons (response rate 65.7%) participated in the study. 6054 were men (response rate 62.9%) and 6 930 were women (response rate 68.4%).

Methods

A personal invitation was mailed about 2 weeks before a suggested time of appointment, and the invitation leaflet included information about the survey and the examination. Non-attendees were given one reminder. A questionnaire was mailed together with the invitation. The questionnaire included questions about general health status, diseases in the family, muscle pain, physical discomfort and mental distress, use of the public health service and medications, socioeconomic situation, physical activity in leisure time, smoking and alcohol habits as well as food habits. Women were asked about, among other topics, parity, age at menarche and age at menopause.

People who attended the study received another very comprehensive questionnaire that they were asked to complete and send back. A link to these questionnaires can be found at www.tromsostudy.com. Everyone who attended underwent a relatively simple medical examination comprising measuring height, weight, waist and hip circumference and blood pressure. A blood sample was also taken to measure serum total cholesterol, HDL-cholesterol, triglycerides, and glucose.

To examine sleeping habits and sleep-wake rhythms, a Norwegian translation of The Munich ChronoType Questionnaire (MCTQ) was used (see Appendix). A link to the English version can be found here: www.bioinfo.mpg.de/mctq/core_work_life/core/introduction.jsp?language=eng. The questionnaire included 15 items, with questions about the number of work days and free time during the week, shift work, and bedtime and rise time including sleep latency (in minutes) during work days and days off work.

Measures

Variables

Midpoint of sleep and sleep duration

The midpoint of sleep is the half-way point between sleep onset and wake up time and represents the local time. Because most people tend to accumulate a sleep debt on work days, which is compensated for on days off, the midpoint of sleep was adjusted for individual sleep needs and referred to as MSFsc [25]. Seasonal differences in chronotypes were calculated based on the answers of the MCTQ at the attending date.

Sleep duration was calculated both for workdays and for free days, and mean sleep duration was calculated as: (sleep duration on workdays*number of workdays)+(sleep duration on days off*number of days off)/7.

Chronotypes

In the Norwegian version of the MCTQ, there were no questions about self-reported morningness-eveningness. Studies have found about 2 hours difference in mid sleep between people who regard themselves as early risers (morning types) and those regarding themselves as late risers (evening types) [18]. We categorized the population according to MSFsc:

a) Extremely early types (EET): 10% of the population; with chronotypes earlier than 02:50 h
b) Early types (ET): 25% of the population; with chronotypes earlier than 03:20 h.
c) Intermediate types (IT): 50% of the population; with chronotypes between 03:20 and 04:30 h.
d) Late types (LT): 25% of the population; with chronotypes later than 04:31 h.
e) Extremely late types (ELT): 10% of the population; with chronotypes later than 05:03 h

Seasons

Because of the Polar night from November to January and the Polar day from May to July, we chose to divide the year into 4 seasons: winter (November, December, January), spring (February, March, April), summer (May, June, July), and autumn (August, September, October).

Daylight saving time

Daylight saving time (DST) was in our study period in place between 25 March 2007 and 27 October 2007, and between 30 March 2008 and 25 October 2008.

HSCL-10

The Hopkins Symptoms Checklist is a screening instrument for detecting mental distress, commonly used in population studies [84]. The 90 items HSCL-90 has been reduced into various versions, and the validated 10-item version (HSCL-10, see Table 1) is found to be almost as good as the longest version in detecting mental distress in the general population [85, 86]. The mean score (General severity index) is calculated by dividing the total score by the number of items answered, which gives a score between 1.00 and 4.00. In an earlier Norwegian population study [87], the established cut-off for HSCL-10 yielded a prevalence rate of 11.4%. A mean score of 1.85 or higher has been suggested to be a valid predictor of high mental distress [87, 88].

Table 1: Hopkins Symptom Check List (HSCL-10)

Have you during the last week[1]:
- Experienced sudden fear without apparent reason
- Felt afraid or worried
- Experienced faintness or dizziness
- Been tense or upset
- Easily blamed yourself
- Experienced sleeplessness
- Felt depressed or sad
- Felt that your life is a struggle
- Felt hopelessness with regard to the future

[1] The answers were given in 4 categories: No, A little, Pretty much, A lot

Symptoms of mental distress during the last week were measured using the HSCL-10. The mean HSCL-10 score was calculated by dividing the total score with ten (i.e. the number of items). Individuals with missing values have been excluded in the mean item score calculation.

Body composition variables

Specially trained personnel measured height and weight with participants wearing light clothing and no shoes. Body mass index (BMI) was calculated as weight in kilograms (kg) divided by the square of the height in meters. Normal weight was defined as BMI <25kg/m2 and overweight as BMI ≥25 kg/m2. Waist circumference (WC) in centimeter (cm) was measured at the umbilical line and hip circumference in cm at the widest point. Waist-to-Hip Ratio (WHR) was calculated as waist circumference divided by hip circumference. Abdominal obesity was defined as waist circumference ≥102 cm for men and ≥88 cm for women.

Biological factors

Non-fasting serum level of total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and glucose were analyzed. All analyses were performed at the Department of Clinical Chemistry, University Hospital of North Norway, using standard laboratory procedures (http://www.unn.no/laboratoriehaandbok/category14289.html).

Other variables

Socioeconomic variables (age, gender, educational level, income level, living with spouse), lifestyle variables (current smoking, alcohol habits, physical activity in leisure and work), and health variables (self –rated physical health, former/current heart disease/diabetes, coping, use of painkillers, antidepressants and sleeping pills, measured blood pressure) were included.

Ethical considerations

The study was approved by the Regional Medical Ethics Committee.
The participants provided their written informed consent to perform in this study, and participants retain the right to withdraw this consent at a later stage. This procedure is approved by the Regional Medical Ethics Committee.

Statistical analysis

The analyses were performed with the statistical package STATA version 11.1 (Stata/SE 12.0 for Windows, StataCorp LP, College Station, TX, USA). The statistical significance level was set at 0.05. We collected data from each participant only once, and all the seasonal variations were based on participation date, and not individual variations.

In all three studies, univariate analyses were conducted by one-way ANOVA followed by the Scheffe post hoc test or Kruskal-Wallis test. In Study 1, univariate analyses between gender and sleep variables (chronotype and sleep duration) were followed by stepwise backwards multiple linear regression analyses. These regression analyses were performed with chronotype or with sleep duration as outcome variables, and with attendance date or season, demographic variables, socioeconomic variables, lifestyle variables and health variables as independent variables. Two group means comparisons of sleeping patterns between two seasons were compared with t-tests.

In study 2, age was grouped in four intervals: 35-44, 45-54, 55-64 and 65-87 years. First we did the univariate analyses between season of participation and: 1) sleep duration, 2) different sleep variables, 3) the use of sleeping pills, and 4) HSCL-10 score. Subsequently, a stepwise backwards multiple logistic regression was performed, with 'more sleeping problems than usual during the last couple of weeks' and HSCL-10 score as outcome variables. Participation season, socioeconomic variables (age, sex, educational level, living with spouse), lifestyle variables (current smoking, alcohol habits, physical activity) and health variables (coping, self –rated physical health, use of painkillers, and HSCL-10) were used as independent variables.

In study 3, we performed univariate analyses between the different body composition variables and the sleep duration categories, using 7-7.9 hours of sleep as the reference category. In these univariate analyses, age correction was done by calculating the mean of each variable, by age. In the regression analyses, we used 'Age' as a prediction variable in the model. Stepwise backwards multivariable regression analyses were used for adjustments. BMI, BMI ≥25 kg/m2, abdominal obesity (measured by waist circumference) and WHR were set as outcome variables. Potential confounding factors in the regression analyses were: (i) Socioeconomic variables (age, sex, educational level, income level, living with spouse), (ii) Lifestyle variables (current smoking, alcohol habits, physical activity in leisure and work), (iii) Health variables (self –rated physical health, former/current heart disease/diabetes, HSCL-10 score, coping, use of painkillers, antidepressants and sleeping pills, measured blood pressure, (iv) Biological factors (non-fasting serum level of cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides and glucose) and (v) Sleep variables (mean sleep duration, chronotype and social jetlag).

Overview of results

Paper 1

We found large individual differences in sleep onset, sleep latency, rise time and chronotype measured as mid sleep on free days. Late types had a 10 minutes increase in sleep duration on free days from winter to spring, but had no seasonal variation in the average sleep duration. No seasonal variations in mid sleep on free days were found. However, when chronotype distribution was adjusted for confounding factors we found a significant advance in phase (8 minutes) in summer compared to the wintertime. This advance in mid sleep on free days was significant both for the employed and the unemployed.

Other factors may be more important than daylight exposure in the regulation of sleep patterns for people in the subarctic. Moreover, the use of stimulants or excessive indoor and outdoor light may have masked the seasonal effect of variation in daylight.

Paper 2

The participants who attended the study in winter reported significantly more current sleeping problems than the attendees in other seasons. 7.4% of the study population had a high level of mental distress, which was lower than the previously reported average for the general population in Norway. There were no significant seasonal differences in mental distress, and mental distress appeared to be a stable phenomenon independent of season and the extreme variations in light and darkness found in the subarctic.
Although some people in the subarctic clearly are mentally negatively affected by the darkness of winter, the negative impact of winter on mental distress for the adult population was not conclusive.

Paper 3

Men had a higher age-corrected BMI than women. 70% of men and 55% of women had a BMI ≥25kg/m2. The optimal sleep duration time regarding risk of overweight and abdominal obesity was 8-9 hours, which is one hour longer compared to findings from other studies. Short sleepers had 80% increased risk of being overweight, and men had a doubled risk of having abdominal obesity. Exercise, education, self-evaluated good health, smoking and an increased HDL-cholesterol level lowered the risk of being overweight, while alcohol use, heart disease and total cholesterol and triglyceride levels were associated with increased overweight risk. We found no associations between neither chronotype nor social jetlag on BMI and abdominal obesity.

Discussion

Main findings

Our first study gave an overview of the chronotype distribution and sleep characteristics of the participants in the Tromsø 6 study. We found large differences in sleep characteristics in the population, and also gender differences, with men having earlier chronotypes and shorter sleep duration than women. This is a similar finding as in the Hordaland study from the south-west of Norway [89], and also similar to findings from several studies from other parts of the world [90, 91].

The extreme variation in daylight through the seasons in northern Norway and the daylight effect on the sleep-wake rhythm has been described in the Introduction section. A wide-spread myth in Northern Norway is that these extreme variations in daylight has a large effect on sleeping patterns; that people go to bed later in the evening in the summer because the sun is still shining, and that they need much less sleep in the summer than in the winter. In our first study, we found only minor differences in rise times, sleep duration and mid sleep on free days between participants attending the study in winter compared to summer. Since daylight is known to be the strongest time cue, we would expect a large difference in sleep-wake rhythms in the midnight sun period compared to the polar night period. However, the chronotype difference was only 8 minutes when confounding factors were controlled for, which is far less than expected. How can this be explained?

In a modern society like Tromsø, other factors than daylight may be more important in the regulation of sleep patterns. Leger et al. studied sleep quality, sleepiness and sleep disorders in 13296 French employees working in environments that were not exposed to natural light [92]. They suggested that underexposure to natural light may significantly impair sleep and wake disorders. Randler studied morningness-eveningness in adolescents in sixteen German schools dispersed all over the world [90]. He found that, besides longitude and latitude (which can be explained by sunrise times), the climate was of importance to chronotype. In another smaller study, Benedito-Silva et al. studied chronotype distribution among citizens in 5 cities in Brazil

[93]. They did not find a coherent latitude trend as a function of the photoperiod and suggested that local social habits could explain the results. However, neither of these studies included other (possible confounding) factors than age and sex in their analyses of the associations between location and chronotype.

Several studies have shown that social schedule is an important factor in association with chronotype and sleep [23]. Korczak et al. studied the effect of different social synchronizers on the sleep-wake cycle of persons with different chronotypes [9]. They found that social schedules have an impact on circadian rhythms, but this impact depended on the individual chronotype. Besides social schedules, social factors like smoking and alcohol habits are significant contributors to differences in chronotypes [23, 94], and other factors like age, dietary intake, education and exercise also play an important role [25, 95, 96].
About three-fourth of our study population were full time or part time employed or students with social obligations and fixed timetables, at least on work days. These social schedules are most likely about the same all year around, except for the summer holidays (which for most of the employed participants would be July when no recordings where made). These fixed schedules would presumably minimize any seasonal differences in sleep-wake rhythms in our study.

Some studies have shown that the human circadian clock is very sensitive to light and that even normal indoor light can be sufficient to entrain the circadian clock [97, 98]. Boivin and Czeisler showed that morning room-light exposure can phase advance the circadian clock [99], and Zeitzer et al. found that humans are highly responsive to phase delaying effects of indoor light during the late evening hours [98]. Outdoor light exposition during nighttime has also been found to affect the sleep-wake rhythm [100]. During winter in Tromsø, people can be exposed to only a small amount of natural light outdoors during daytime. Even if we have no exact data on time spent outdoors, it would be reasonable to estimate that most of our study population spent the work days indoors, exposed to artificial light. In winter, the outdoor exposure to light during evening is solely artificial light. During summer, most workers also spend their work days indoors, but the light exposure outdoors during evenings can be high for those who spend their evening time outdoors. The exposure to artificial light both indoors and outdoors may very well have masked the seasonal effect of variation in daylight.

Insomnia symptoms are of the most frequent health complaints in the general population with a prevalence of 10-50%, depending in part on definitions and data-collection methodologies [27, 28]. In a population study from the western part of Norway, 11.4% of the study population reported insomnia at least once a week [89], about the same prevalence as found by Pallesen et al. using the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) diagnostic criteria [101]. In our second study, 6.3% of the population reported current sleeping problems. This is lower than reported in these other studies, but the results cannot be compared directly. Our study did not diagnose insomnia using several specific questions, as Pallesen et al. [101] did. Ursin et al. [89] asked how often the participants suffered from insomnia (answered on a 4-point scale), which could lead to a higher amount of reported sleeping problems than if current sleeping problems was asked for.

Our first study showed no significant seasonal difference in sleep duration, as did the study of Ursin et al. [89]. However, we found that the reporting of current sleeping problems was more common in winter participants (9.4%) than in summer participants (5.6%). Seasonal variations in sleeping problems at latitude 63°-65°N have also been studied [102], but this study found no evidence of seasonal variations in insomnia symptoms or time in bed.

The polar night in Tromsø is not completely dark. The outdoor light intensity (usually increased by reflecting snow) on bright days at the beginning and the end of the dark period may be strong enough to minimize the seasonal effects on sleep-wake rhythms and also on mood. In our second study, mental distress appeared to be a stable phenomenon independent of season. Surprisingly, the mean HSCL-10 score was lower and high mental distress (HSCL-10 score ≥1.85) was less common in respondents in the Tromsø study (Tromsø 6) than in the general Norwegian population [87]. Strand et al. found a mean HSCL-10 score of 1.36 in the total population aged 16-97 years, and 11.1% had a HSCL-25 score ≥1.75 (which corresponds to a HSCL-10 score ≥1.85). Sandanger et al. found that 14.9% of the population in Oslo and Lofoten had a HSCL-25 score ≥1.75 [103]. Both of these studies found similar risk groups with a higher prevalence of mental distress: women, persons in the younger and the older age groups, low education, unmarried/divorced, and similar findings have also been shown in previous studies [104, 105]. Our study included persons over 30 years of age, compared to over 15-18 years of age in the

other two studies, and we also had more male participants. Educational level and number of persons who were employed or students also seemed to be higher in our study population. All of these factors could possibly explain at least some of the differences in mental distress.
In a study from the second part of the Tromsø study in 1979-80, 12.5% of the men and 16.8% of the women reported having at least one of three symptoms of mental distress [106]. Since our study used ten questions regarding mental distress (HSCL-10), compared to three questions in the Tromsø 2 study, comparing these two will be of limited value. However, the reporting of symptoms of mental distress always have a certain degree of uncertainty, as will be further discussed in the next section regarding methodological strengths and weaknesses.

In our study, there were no significant differences in mental distress depending on season of participation. In their study, Haggag et al. described a major seasonal mood variation in Tromsø [42], but this study had two important limitations that could affect the results. First, the response rate was quite low, especially in winter (39.5%). Second, the same day as the questionnaire was sent out, an article was published in the local newspaper and the local radio station explained the purpose of the study, in an effort to maximize the response rate. This could of cause bias the responses in the direction of more reports on seasonal mood variations as this was clearly stated as the study objective. In their study of 3736 inhabitants in Sør-Varanger (latitude 70°N), Hansen et al. found that the prevalence of self-reported depression was 11.1% in women and 4.8% in men [44], which is quite similar to our study. In the study by Hansen et al., the participants had no knowledge about the study objective (mental distress) as the data was sampled from a study on air pollution, and the response rate was satisfactory (61%). In a study from Finland, Partonen et al. distributed a modified depression rating scale to a population (latitude 60-70°N) during November and found no association between latitude and depression [45]. In Arctic Russia (latitude 59-68°N), Borisenkov found that the time of sunrise during lengthening days seemed to be a stronger predictor of sleep length and chronotype than day length itself [46].

Our study was not about seasonal affective disorder, and we did not record the hypersomnia and carbohydrate craving symptoms typically found in SAD. Nevertheless, it can be relevant to compare our findings with studies that aim to investigate the relationship between SAD and latitude. Merch et al. made an overview of the epidemiological literature on SAD in relation to latitudinal dependency [107]. The study locations spanned from 15°N to 71°N and from 10°S to 40°S. They concluded that the influence of latitude on the prevalence of SAD seems to be small and that other factors like climate, genetic vulnerability and social–cultural context can play a more important role.

Smaller studies from the Antarctic regions have also been performed. One study examined symptom characteristics and risk factors for seasonal variation in depressive symptoms in 119 men and women who overwintered in Antarctica [47]. The results indicate that some people experience seasonal variation in mood in high-latitude environments, but this seasonal variation may be the result of social isolation during the winter months rather than the prolonged absence of sunlight.

From clinical practice, it is known that some people in the subarctic clearly are mentally negatively affected by the darkness of winter, but this has been documented only in a few studies [108, 109]. Stuhlmiller performed a qualitative study with 28 participants in Tromsø from 1993 through 1995 in order to understand seasonal affective disorder and how the residents experienced seasonal changes [108]. She found that the northern Norwegians described seasonal change as a wonder of nature rather than a cause of distress. She concluded that while there is evidence that human physiologic alterations occur in response to the changing seasonal patterns of light and dark, the findings related to psychological changes and their causes remain inconsistent and controversial. She also said that nature and seasonal change, as an important cultural resource, can encourage us to promote human connections, foster resilience, and help explain why we are not sad.

In Oslo, Lingjaerde et al. studied 128 media-recruited persons with winter depression [110]. They found that the duration of winter depression was most often from October to March or April, and that most patients suffered at least one of the symptoms of hypersomnia, hyperphagia or carbohydrate craving. However, the negative impact of winter on mental distress for the adult population is difficult to substantiate, at least for the majority of the population.

In our third study, we found that people in the overweight group slept statistically significantly shorter than those with normal weight, but the difference was small. Short sleepers had almost a doubled risk of being overweight, and male short sleepers had a doubled risk of having abdominal obesity. The optimal sleep duration time regarding risk of overweight and abdominal obesity was one hour longer compared to findings from other studies.

The interactions of multiple biological, psychological, behavioural and social risk factors may play an important role in the development of overweight and obesity [50, 53, 55, 72, 111, 112]. The possibility that light pollution induces obesity is an interesting hypothesis and should be further explored [11]. The effect of indoor and outdoor artificial light on entrainment of the circadian clock [97-100] has already been discussed. It is also a possibility that the high amount of artificial light, especially during winter in Tromsø, could be associated with the high prevalence of overweight in our study (70% of men and 55% of women had a BMI ≥25kg/m2).

Overall, the literature suggests that short sleep duration may represent an independent risk factor for weight gain and obesity, particularly in children and younger adults [66]. The associations between sleep duration and BMI are far from fully understood, and several pathways linking short sleep to weight gain have been suggested [66]. Short sleep has been associated with reduced levels of leptin ("anorexigenic hormone") and elevated levels of ghrelin ("hunger hormone"), which could increase appetite and thereby also weight [113, 114]. Others have found associations between sleep duration and dietary behaviours [115, 116]. Sivak pointed out the possibility that shorter sleep would increase the opportunity to consume readily available food and drinks, especially if most of the wake-time is spent in sedentary activities (such as watching television), where snacking is common [117]. Thus, replacing 1 h of TV watching with sleeping was predicted to result in a bodyweight reduction of up to 6.5 kg per year.

In our study, we had no data on appetite hormone levels, but the significantly higher BMI levels among short sleepers were found after controlling for confounding factors including non-fasting total-cholesterol, HDL-cholesterol, triglycerides and glucose levels. These variables could –at least in part- be taken to reflect participants' diet.

Chronic sleep deprivation leads to feelings of fatigue [118], which in turn could lead to reduction of physical activity and weight gain. Moreover, studies have shown that persons, who have attended weight loss programs, regain most of the lost weight at a time of inactivity [119]. There is broad consensus that physical activity is very important in fighting overweight [120], but physical activity alone is of limited value in inducing weight loss [121]. Physical activity combined with diet will be far more efficient in weight loss programs [122].

Our study showed that male short sleepers had an elevated risk of abdominal obesity while female short sleepers had not. This is similar to the findings from a newly published study from Japan of 5400 men and 642 women, where a CT scanner was used to measure waist circumference (WC), visceral fat area (VFA) and the subcutaneous fat area (SFA) [55]. They found that short sleep duration was strongly associated with higher WC in men, but not in women. However, they also found an association between short sleep and BMI only in men, while our study found the same association in both genders. One possible explanation could be that the number of women in each sleep group in the Japanese study was too low to detect any significant associations. In a study of 400 Swedish women, an inverse relationship between short sleep duration and central obesity was found in women [123], but this study adjusted for fewer confounders (somatic disease, medication, snoring, daytime sleepiness, physical activity, tobacco use, and alcohol consumption) than our study did.

In our study, chronotype or social jetlag was not associated with BMI and abdominal obesity. In a large study of more than 65000 persons, Roenneberg et al. found that beyond sleep duration, social jetlag was associated with increased BMI [124]. They concluded that living "against the clock" may also be a factor contributing to the epidemic of obesity. This was a very large study, and even small differences had the potential to be significant because of the large number of participants. However, this study did not include important predictors of body weight like educational level, health factors or diet.

Studies concerning sleep duration and obesity risk have found various associations between sleep duration and weight gain. Moreover, the optimal sleep duration regarding BMI and WC has in most studies been found to be between 7 and 8 hours [56, 61, 67]. In our study, the optimal sleep length with regard to all body composition measures was between 8 and 9 hours, when all confounding factors were controlled for in the multivariate analyses. As Magee et al. pointed out, there is a need for controlling for confounding factors in these types of studies [72]. Most sleep studies have controlled for gender and age, since sleep duration varies with gender and with age [66]. Important factors like mental health, smoking and education have been controlled for in some studies [63], while other studies also have adjusted for physical job demand, household income, marital status and alcohol consumption, and snoring [125]. The fact that we have controlled for more confounding factors than most other studies could explain the difference in optimal sleep duration between our study and others.

Although there has been evidence for associations between obesity and short sleep duration, most short sleepers are unlikely to be obese, and most obese people are unlikely to be short sleepers [126]. We found that people with normal weight slept significantly longer than those who were in the overweight group, but the difference was only about 5 minutes. Even if our study showed an inverse relationship between sleep duration and the risk of having an elevated BMI, the difference in age corrected BMI was very small (1 BMI unit for men and 0.8 BMI units for women). This corresponds to a difference in body weight of about 3 kilos for a man of 180 cm height and about 2.5 kilos for a woman of 170 cm height. This difference in BMI is possibly not large enough to have any clinical significance. In the battle against overweight, promoting longer sleep is probably not the way to go. Overweight develops over time, so extending sleep in order to lose weight may take many years. As Horne pointed out, short sleep itself is unlikely to cause more than a very slow weight gain, and that the effect of short sleep in increasing body weight is minimal compared to the effects of physical inactivity and excess calorie intake [126].

Methodological strengths and weaknesses of the present study

The number of participants

Cross sectional studies look at the relationship between a disease or health characteristic and different variables such as age, gender or health behavior. Cross-sectional studies are suitable for testing hypotheses, and in a cross-sectional study like the Tromsø study, one can study a representative part of the population in a narrowly defined time period. A high number of participants make the findings more representative of the target population (here: The Tromsø population aged 30 – 87 years) and also makes it easier to detect any patterns in the research variables.

12984 men and women aged 30-87 participated in the Tromsø 6 study, which was 65.7% of the invited population and about 20% of the total population in Tromsø at the time of the study. The overall high response rate in our study is a major strength, because it allows us to make assumptions about the target population.

However, as in other population studies in Norway and in other countries, the attendance rates in the Tromsø study have been falling. The response rate was quite low in the lowest and highest age groups, especially among men, and this made generalizations to these age groups in the general population more difficult.

Ethnicity

As the Tromsø Study is based in the seventh largest Norwegian city with relatively few immigrants, it is limited with regard to ethnic diversity. The vast majority of the participants in Tromsø 6 are Caucasian subjects (about 94% of the study population consider themselves as ethnic Norwegian, 2% as Sami, 1.5 as Finnish/Kven and 2.5 as other nationalities). This ethnic homogeneity makes it easier to make generalizations from our findings to the ethnic Norwegian population.

Data collection method

A large variety of data were collected from non-invasive physical examinations and blood tests, and done by specially trained personnel. Height, weight and waist circumference were measured instead of self-reported, which was a strength of this study. Self-reporting on height is often overestimated and weight underestimated, which could lead to underestimation of BMI [127].

Another strength was that the study covered a variety of topics, and questions about mood and sleep were only a minor part of the large questionnaire. There was no focus on mental distress or sleep when information about the study was mentioned in the local newspapers. This could minimize the possibility of selection bias.

In this study, each person attended the study only once. To monitor seasonal variations in our study, we had to compare participants who attended the study in the four seasons. The best way to study seasonal variations, would of cause be to perform a longitudinal study where the participants could be monitored several times and in different seasons.

The Munich ChronoType Questionnaire was used to collect information about the actual timing of daily sleep and activity. Validation of the questionnaire has showed that chronotypes based on the MCTQ strongly correlate with sleep logs [80]. However, the chronotypes and sleep durations calculated from the MCTQ were self estimated and not exact times, which is important to have in mind when analyzing and comparing the data with other studies.

The variables included in the study

In the present study, a wide range of different variables were included as predictors (confounding variables), including socioeconomic variables, lifestyle variables, health variables, biological factors, and sleep variables. Studies concerning associations between different predictors and outcome variables, have to control for other (confounding) variables which is associated with both the probable cause and the outcome, but may not lie in the causal pathway between the cause and the outcome. This has to be done because confounding variables mix up causal and non-causal relationships. Age and sex are typical confounders in most studies. It is known that sleeping patterns, both sleep length and sleep-wake rhythms, vary with age and sex [25], therefore most sleep studies control for these factors. A major strength in our study, is that we have controlled for many different socioeconomic variables, lifestyle variables, health variables and biological factors as potential confounding factors. Few prior studies have included that many different variables.

Selection and information bias

The method of selecting participants to a study may also cause selection bias. In our study (as in other studies), it is a possibility that some of the persons who were long sleepers and got up late (late chronotypes), did not get up in time to reach the appointment and therefore did not participate. If this were the case, then it is possible that the participating population had an overweight of early chronotypes compared to the general population.

It is always a possibility that there is a selection bias in regard to health problems. In a study of non-respondent characteristics, it was found that selection according to sociodemographic variables had little impact on the prevalence estimates for participants compared to non-participants [128], while others have found that non-attendees to a general health survey had more than twice the risk of attendees of having a psychiatric disorder [129]. It is also possible that some people with severe sleeping problems during winter were too affected to participate in the study, but unfortunately we have no data on attendance rate by month or season.

In a questionnaire, there can always be questions and misunderstandings about how to understand the questions and how to answer them, which can lead to an information bias. Many of the questions in the Tromsø study dealt with health problems and life style factors. How one considers and classifies different health problems, like depression or anxiety, may differ from person to person. There may also be a bias in recalling, as it can be difficult to remember details about, for instance, ‘the last month’. These kinds of bias are not possible to overcome, but we need to consider them when doing the data analyses.

Lacking variables

A limitation in our study was the lack of information about wakening periods during the night and napping during the day. In the Hordaland Health Study, 27% of the men and 29% of the women reported napping at least several times a week [89]. Mean sleep duration was shorter in the nappers than in the whole population, and napping increased the mean sleep duration in the whole sample by 10 minutes in both sexes. In our study, we did not know the amount of participants taking a nap during daytime, but we had no reason to believe that this phenomenon was less common among the participants in the Tromsø study than in the Hordaland study. However, detailed information about naps was only given by 20% of the participants in this Hordaland study [89], and therefore they concluded that the data from the night sleep duration was more reliable in the analysis of sleep duration.

The data on sleep times and self-reported sleeping problems are subjective and not suitable for diagnosing any sleep disorders (Codes G47- in the ICD-10 coding system: http://www.who.int/classifications/icd/en/). In a study of the prevalence and impact of sleep disorders and sleep habits in the United States, 4.2% had physician diagnosed sleep disorders [130]. In the same study, 6.9% reported waking up at night and had difficulties going back to sleep. We do not know if any of the participants in our study suffered from any sleep disorders that could influence their sleep-wake rhythm.

In our study, light exposure was not measured and there were no questions about the daily amount of outdoor time or the use of artificial light. Tromsø is a modern urban area, and about 70% of the participants had full time employment or studies. In a modern society this would mean indoor activities in artificial light most of the day and at a strict schedule throughout the year. The indoor light and also the strict working schedules could mask seasonal differences in sleep patterns.

Suggestions for further research

This study was based on the Tromsø study, which is a cross-sectional study. However, in a longitudinal study, data on a subject is collected over a specific period of time and the subject's response to particular variables can be detected. The best way to study seasonal variations in sleep-wake rhythms, and in health variables like sleep problems and mental distress, would therefore be by performing longitudinal studies. We found a small increase in sleep duration on free days from winter to spring, and only in late chronotypes. Longitudinal studies focusing on morning chronotypes and evening chronotypes would be of interest and should be planned. The human circadian clock is very sensitive to light, and we do not know enough about how indoor light and light pollution outdoors affect people in subarctic. Studies on photosensitivity and biological variables should be planned by measuring biological variables and monitoring light exposition by actigraphy. We do not know enough about how extreme photic variations affect subarctic individuals, and which factors that govern adaptations. There is also a need for more research on clock-gene polymorphism.

Conclusions

Other factors than daylight exposure may be more important in the regulation of sleep patterns for people in the subarctic. There were no significant seasonal differences in mental distress, but sleeping problems were most prevalent in winter. The optimal sleep duration time regarding risk of overweight and abdominal obesity was 8-9 hours, which is one hour longer compared to findings from other studies. Short sleepers had 80% increased risk of being overweight, and men had a doubled risk of having abdominal obesity. Although some people in the subarctic clearly are mentally negatively affected by the darkness of winter, the negative impact of winter on mental distress for the adult population is not conclusive. Further prospective studies and more research on clock-gene polymorphism, photosensitivity and other biological variables among subgroups of subarctic populations are needed.

References

1. Aschoff, J., *Circadian Rhythms in Man.* Science, 1965. **148**(3676): p. 1427-32.
2. Hur, Y.-M., T.J. Bouchard Jr, and D.T. Lykken, *Genetic and environmental influence on morningness–eveningness.* Pers Indiv Dif, 1998. **25**(5): p. 917-925.
3. Jones, K.H.S., et al., *Age-related change in the association between a polymorphism in the PER3 gene and preferred timing of sleep and waking activities.* J Sleep Res, 2007. **16**(1): p. 12-16.
4. Hastings, M., *The brain, circadian rhythms, and clock genes.* BMJ, 1998. **317**(7174): p. 1704-7.
5. Dunlap, J.C., *Molecular bases for circadian clocks.* Cell, 1999. **96**(2): p. 271-90.
6. Aschoff, J. and H. Pohl, *Phase relations between a circadian rhythm and its zeitgeber within the range of entrainment.* Naturwissenschaften, 1978. **65**(2): p. 80-4.
7. Czeisler, C.A., et al., *Entrainment of human circadian rhythms by light-dark cycles: a reassessment.* Photochem Photobiol, 1981. **34**(2): p. 239-47.
8. Wever, R.A., J. Polasek, and C.M. Wildgruber, *Bright light affects human circadian rhythms.* Pflugers Arch, 1983. **396**(1): p. 85-7.
9. Korczak, A.L., et al., *Influence of chronotype and social zeitgebers on sleep/wake patterns.* Braz J Med Biol Res, 2008. **41**(10): p. 914-9.
10. Roenneberg, T. and M. Merrow, *Entrainment of the human circadian clock.* Cold Spring Harb Symp Quant Biol, 2007. **72**: p. 293-9.
11. Wyse, C.A., et al., *Circadian desynchrony and metabolic dysfunction; did light pollution make us fat?* Med Hypotheses, 2011. **77**(6): p. 1139-44.
12. Panda, S., et al., *Melanopsin (Opn4) requirement for normal light-induced circadian phase shifting.* Science, 2002. **298**(5601): p. 2213-6.
13. Freedman, M.S., et al., *Regulation of mammalian circadian behavior by non-rod, non-cone, ocular photoreceptors.* Science, 1999. **284**(5413): p. 502-4.
14. Berson, D.M., F.A. Dunn, and M. Takao, *Phototransduction by Retinal Ganglion Cells That Set the Circadian Clock.* Science (Washington), 2002. **295**(5557): p. 1070-1073.

15. Mongrain, V., J. Carrier, and M. Dumont, *Circadian and homeostatic sleep regulation in morningness-eveningness.* J Sleep Res, 2006. **15**(2): p. 162-6.
16. Horne, J.A. and O. Ostberg, *A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms.* Int J Chronobiol, 1976. **4**(2): p. 97-110.
17. Kerkhof, G.A. and H.P. Van Dongen, *Morning-type and evening-type individuals differ in the phase position of their endogenous circadian oscillator.* Neurosci Lett, 1996. **218**(3): p. 153-6.
18. Emens, J.S., et al., *Phase angle of entrainment in morning- and evening-types under naturalistic conditions.* Chronobiol Int, 2009. **26**(3): p. 474-93.
19. Roenneberg, T., C.J. Kumar, and M. Merrow, *The human circadian clock entrains to sun time.* Curr Biol, 2007. **17**(2): p. R44-5.
20. Dijk, D.J. and S.W. Lockley, *Integration of human sleep-wake regulation and circadian rhythmicity.* J Appl Physiol, 2002. **92**(2): p. 852-62.
21. Daan, S., D.G. Beersma, and A.A. Borbely, *Timing of human sleep: recovery process gated by a circadian pacemaker.* Am J Physiol, 1984. **246**(2 Pt 2): p. R161-83.
22. Roenneberg, T., et al., *Epidemiology of the human circadian clock.* Sleep Med Rev, 2007. **11**(6): p. 429-38.
23. Wittmann, M., et al., *Social jetlag: misalignment of biological and social time.* Chronobiol Int, 2006. **23**(1-2): p. 497-509.
24. Reyner, L.A., J.A. Horne, and A. Reyner, *Gender- and age-related differences in sleep determined by home-recorded sleep logs and actimetry from 400 adults.* SLEEP, 1995. **18**(2): p. 127-34.
25. Roenneberg, T., et al., *A marker for the end of adolescence.* Curr Biol, 2004. **14**(24): p. R1038-9.
26. Randler, C., et al., *Chronotype but not sleep length is related to salivary testosterone in young adult men.* Psychoneuroendocrinology, 2012. **37**(10): p. 1740-4.
27. Ohayon, M.M., *Epidemiology of insomnia: what we know and what we still need to learn.* Sleep Med Rev, 2002. **6**(2): p. 97-111.
28. Howland, R.H., *An overview of seasonal affective disorder and its treatment options.* Phys Sportsmed, 2009. **37**(4): p. 104-15.

29. Lingjaerde, O., T. Bratlid, and T. Hansen, *Insomnia during the "dark period" in northern Norway. An explorative, controlled trial with light treatment.* Acta Psychiatr Scand, 1985. **71**(5): p. 506-12.
30. Kleitman, N. and H. Kleitman, *The Sleep-Wakefulness Pattern in the Arctic.* The Scient Monthly, 1953. **76**(6): p. 349-356.
31. Devold, O., E. Barlinhaug, and J.E. Backer, *[Sleep disorders during midwinter in the far north]*. Tidsskr Nor Laegeforen, 1957. **77**(19): p. 836-7.
32. Kjos, K., *Søvnforstyrrelser som skolehygienisk problem i Nord-Norge.* Tidsskr Nor Lægeforen, 1959. **77**: p. 1241-1243.
33. Weitzman, E.D., et al., *Seasonal patterns of sleep stages and secretion of cortisol and growth hormone during 24 hour periods in northern Norway.* Acta Endocrinol, 1975. **78**(1): p. 65-76.
34. Hansen, T., et al., *Midwinter insomnia in the subarctic region: evening levels of serum melatonin and cortisol before and after treatment with bright artificial light.* Acta Psychiatr Scand, 1987. **75**(4): p. 428-34.
35. Nilssen, O., et al., *Sleeping problems at 78 degrees north: the Svalbard Study.* Acta Psychiatr Scand, 1997. **95**(1): p. 44-8.
36. Praschak-Rieder, N., et al., *Seasonal variation in human brain serotonin transporter binding.* Arch Gen Psychiatry, 2008. **65**(9): p. 1072-8.
37. Levitan, R.D., *The chronobiology and neurobiology of winter seasonal affective disorder.* Dialogues Clin Neurosci, 2007. **9**(3): p. 315-24.
38. Rosenthal, N.E., et al., *Seasonal affective disorder. A description of the syndrome and preliminary findings with light therapy.* Arch Gen Psychiatry, 1984. **41**(1): p. 72-80.
39. Dam, H., K. Jakobsen, and E. Mellerup, *Prevalence of winter depression in Denmark.* Acta Psychiatr Scand, 1998. **97**(1): p. 1-4.
40. Brancaleoni, G., et al., *Seasonal affective disorder and latitude of living.* Epidemiol Psichiatr Soc, 2009. **18**(4): p. 336-43.
41. Friborg, O., et al., *Associations between seasonal variations in day length (photoperiod), sleep timing, sleep quality and mood: a comparison between Ghana (5 degrees) and Norway (69 degrees).* J Sleep Res, 2012. **21**(2): p. 176-84.

42. Haggag, A., et al., *Seasonal mood variation: an epidemiological study in northern Norway.* Acta Psychiatr Scand, 1990. **81**(2): p. 141-5.
43. Hansen, V., B.K. Jacobsen, and R. Husby, *Mental distress during winter. An epidemiologic study of 7759 adults north of Arctic Circle.* Acta Psychiatr Scand, 1991. **84**(2): p. 137-41.
44. Hansen, V., E. Lund, and T. Smith-Sivertsen, *Self-reported mental distress under the shifting daylight in the high north.* Psychol med, 1998. **28**(2): p. 447-52.
45. Partonen, T., M. Partinen, and J. Lonnqvist, *Frequencies of seasonal major depressive symptoms at high latitudes.* Eur Arch Psychiatry Clin Neurosci, 1993. **243**(3-4): p. 189-92.
46. Borisenkov, M.F., *The pattern of entrainment of the human sleep-wake rhythm by the natural photoperiod in the north.* Chronobiol Int, 2011. **28**(10): p. 921-9.
47. Palinkas, L.A., M. Cravalho, and D. Browner, *Seasonal variation of depressive symptoms in Antarctica.* Acta Psychiatr Scand, 1995. **91**(6): p. 423-9.
48. Jacobsen, B.K., et al., *Increase in weight in all birth cohorts in a general population: The Tromso Study, 1974-1994.* Arch Intern Med, 2001. **161**(3): p. 466-72.
49. Kelly, T., et al., *Global burden of obesity in 2005 and projections to 2030.* Int J Obes (Lond), 2008. **32**(9): p. 1431-7.
50. Kokkvoll, A., et al., *High prevalence of overweight and obesity among 6-year-old children in Finnmark County, North Norway.* Acta Paediatr, 2012. **101**(9): p. 924-8.
51. Gardner, G., B. Halweil, and J.A. Peterson, *Underfed and overfed : the global epidemic of malnutrition*. Worldwatch paper;150. 2000, Washington: Worldwatch Institute.
52. Devaux, M. and F. Sassi, *Social inequalities in obesity and overweight in 11 OECD countries.* Eur J Public Health, 2012.
53. Grøholt, E.K., H. Stigum, and R. Nordhagen, *Overweight and obesity among adolescents in Norway: cultural and socio-economic differences.* J Public Health, 2008. **30**(3): p. 258-65.
54. Rontoyanni, V.G., S. Baic, and A.R. Cooper, *Association between nocturnal sleep duration, body fatness, and dietary intake in Greek women.* Nutrition, 2007. **23**(11-12): p. 773-7.

55. Yi, S., et al., *Short sleep duration in association with CT-scanned abdominal fat areas: the Hitachi Health Study.* Int J Obes (Lond), 2013. **37**(1): p. 129-34.
56. Buxton, O.M. and E. Marcelli, *Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States.* Soc Sci Med, 2010. **71**(5): p. 1027-36.
57. Knutson, K.L., et al., *Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus.* Arch Intern Med, 2006. **166**(16): p. 1768-74.
58. Kripke, D.F., et al., *Mortality associated with sleep duration and insomnia.* Arch Gen Psychiatry, 2002. **59**(2): p. 131-6.
59. Magee, C.A., P. Caputi, and D.C. Iverson, *Relationships between self-rated health, quality of life and sleep duration in middle aged and elderly Australians.* Sleep Med, 2011. **12**(4): p. 346-50.
60. Sabanayagam, C. and A. Shankar, *Sleep duration and cardiovascular disease: results from the National Health Interview Survey.* Sleep, 2010. **33**(8): p. 1037-42.
61. Bjorvatn, B., et al., *The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study.* J Sleep Res, 2007. **16**(1): p. 66-76.
62. Cappuccio, F.P., et al., *Meta-analysis of short sleep duration and obesity in children and adults.* SLEEP, 2008. **31**(5): p. 619-26.
63. Fogelholm, M., et al., *Sleep-related disturbances and physical inactivity are independently associated with obesity in adults.* Int J Obes (Lond), 2007. **31**(11): p. 1713-21.
64. St-Onge, M.P., et al., *Gender Differences in the Association between Sleep Duration and Body Composition: The Cardia Study.* Int J Endocrinol, 2010. **2010**: p. 726071.
65. Kobayashi, D., et al., *Association between weight gain, obesity, and sleep duration: a large-scale 3-year cohort study.* Sleep Breath, 2012. **16**(3): p. 829-33.
66. Patel, S.R. and F.B. Hu, *Short sleep duration and weight gain: a systematic review.* Obesity, 2008. **16**(3): p. 643-53.
67. Stranges, S., et al., *Cross-sectional versus prospective associations of sleep duration with changes in relative weight and body fat distribution: the Whitehall II Study.* Am J Epidemiol, 2008. **167**(3): p. 321-9.

68. Lauderdale, D.S., et al., *Cross-sectional and longitudinal associations between objectively measured sleep duration and body mass index: the CARDIA Sleep Study.* Am J Epidemiol, 2009. **170**(7): p. 805-13.
69. Magee, L. and L. Hale, *Longitudinal associations between sleep duration and subsequent weight gain: a systematic review.* Sleep Med Rev, 2012. **16**(3): p. 231-41.
70. Marshall, N.S., N. Glozier, and R.R. Grunstein, *Is sleep duration related to obesity? A critical review of the epidemiological evidence.* Sleep Med Rev, 2008. **12**(4): p. 289-98.
71. Nielsen, L.S., K.V. Danielsen, and T.I. Sorensen, *Short sleep duration as a possible cause of obesity: critical analysis of the epidemiological evidence.* Obes Rev, 2011. **12**(2): p. 78-92.
72. Magee, C.A., et al., *A link between chronic sleep restriction and obesity: methodological considerations.* Public Health, 2008. **122**(12): p. 1373-81.
73. Jacobsen, B.K., et al., *Cohort profile: the Tromso Study.* Int J Epidemiol, 2012. **41**(4): p. 961-7.
74. Midtby, M., J.H. Magnus, and R.M. Joakimsen, *The Tromso Study: a population-based study on the variation in bone formation markers with age, gender, anthropometry and season in both men and women.* Osteoporos Int, 2001. **12**(10): p. 835-43.
75. Hopstock, L.A., et al., *Seasonal variation in cardiovascular disease risk factors in a subarctic population: the Tromso Study 1979-2008.* J Epidemiol Community Health, 2012.
76. Hopstock, L.A., et al., *Seasonal variation in incidence of acute myocardial infarction in a sub-Arctic population: the Tromso Study 1974-2004.* Eur J Cardiovasc Prev Rehabil, 2011. **18**(2): p. 320-5.
77. Giannotti, F., et al., *Circadian preference, sleep and daytime behaviour in adolescence.* J Sleep Res, 2002. **11**(3): p. 191-9.
78. Meliska, C.J., et al., *Relationship of morningness-eveningness questionnaire score to melatonin and sleep timing, body mass index and atypical depressive symptoms in peri- and post-menopausal women.* Psychiatry Res, 2011. **188**(1): p. 88-95.
79. Taillard, J., et al., *Validation of Horne and Ostberg morningness-eveningness questionnaire in a middle-aged population of French workers.* J Biol Rhythms, 2004. **19**(1): p. 76-86.

80. Roenneberg, T., A. Wirz-Justice, and M. Merrow, *Life between clocks: daily temporal patterns of human chronotypes.* J Biol Rhythms, 2003. **18**(1): p. 80-90.
81. Zavada, A., et al., *Comparison of the Munich Chronotype Questionnaire with the Horne-Ostberg's Morningness-Eveningness Score.* Chronobiol Int, 2005. **22**(2): p. 267-78.
82. Minors, D.S. and J.M. Waterhouse, *Does 'anchor sleep' entrain circadian rhythms? Evidence from constant routine studies.* J Physiol, 1983. **345**: p. 451-67.
83. Klerman, E.B., et al., *Comparisons of the variability of three markers of the human circadian pacemaker.* J Biol Rhythms, 2002. **17**(2): p. 181-93.
84. Derogatis, L.R., et al., *The Hopkins Symptom Checklist (HSCL): a self-report symptom inventory.* Beh Sci, 1974. **19**(1): p. 1-15.
85. Tambs, K. and T. Moum, *How well can a few questionnaire items indicate anxiety and depression?* Acta Psychiatr Scand, 1993. **87**(5): p. 364-7.
86. Lipman, R.S., L. Covi, and A.K. Shapiro, *The Hopkins Symptom Checklist (HSCL)--factors derived from the HSCL-90.* J Affect Disord, 1979. **1**(1): p. 9-24.
87. Strand, B.H., et al., *Measuring the mental health status of the Norwegian population: a comparison of the instruments SCL-25, SCL-10, SCL-5 and MHI-5 (SF-36).* Nord J Psychiatry, 2003. **57**(2): p. 113-8.
88. Hoxmark, E., M. Nivison, and R. Wynn, *Predictors of mental distress among substance abusers receiving inpatient treatment.* Subst Abuse Treat Prev Policy, 2010. **5**: p. 15.
89. Ursin, R., B. Bjorvatn, and F. Holsten, *Sleep duration, subjective sleep need, and sleep habits of 40- to 45-year-olds in the Hordaland Health Study.* SLEEP, 2005. **28**(10): p. 1260-9.
90. Randler, C., *Morningness-eveningness comparison in adolescents from different countries around the world.* Chronobiol Int, 2008. **25**(6): p. 1017-28.
91. Arendt, J., *Biological rhythms during residence in polar regions.* Chronobiol Int, 2012. **29**(4): p. 379-94.
92. Leger, D., et al., *Underexposure to light at work and its association to insomnia and sleepiness: a cross-sectional study of 13,296 workers of one transportation company.* J Psychosom Res, 2011. **70**(1): p. 29-36.

93. Benedito-Silva, A.A., et al., *Latitude and Social Habits as Determinants of the Distribution of Morning and Evening Types in Brazil.* Biol Rhythm Res, 1998. **29**(5): p. 591-97.
94. Adan, A., *Chronotype and personality factors in the daily consumption of alcohol and psychostimulants.* Addiction, 1994. **89**(4): p. 455-62.
95. Sato-Mito, N., et al., *The midpoint of sleep is associated with dietary intake and dietary behavior among young Japanese women.* Sleep Med, 2011. **12**(3): p. 289-94.
96. Evans, D.S., et al., *Habitual sleep/wake patterns in the Old Order Amish: heritability and association with non-genetic factors.* SLEEP, 2011. **34**(5): p. 661-9.
97. Gooley, J.J., et al., *Exposure to room light before bedtime suppresses melatonin onset and shortens melatonin duration in humans.* J Clin Endocrinol Metab, 2011. **96**(3): p. E463-72.
98. Zeitzer, J.M., et al., *Sensitivity of the human circadian pacemaker to nocturnal light: melatonin phase resetting and suppression.* J Physiol, 2000. **526 Pt 3**: p. 695-702.
99. Boivin, D.B. and C.A. Czeisler, *Resetting of circadian melatonin and cortisol rhythms in humans by ordinary room light.* Neuroreport, 1998. **9**(5): p. 779-82.
100. Vollmer, C., U. Michel, and C. Randler, *Outdoor light at night (LAN) is correlated with eveningness in adolescents.* Chronobiol Int, 2012. **29**(4): p. 502-8.
101. Pallesen, S., et al., *Prevalence of insomnia in the adult Norwegian population.* SLEEP, 2001. **24**(7): p. 771-9.
102. Sivertsen, B., et al., *Seasonal variations in sleep problems at latitude 63 degrees -65 degrees in Norway: The Nord-Trondelag Health Study, 1995-1997.* Am J Epidemiol, 2011. **174**(2): p. 147-53.
103. Sandanger, I., et al., *Prevalence, incidence and age at onset of psychiatric disorders in Norway.* Soc Psychiatry Psychiatr Epidemiol, 1999. **34**(11): p. 570-9.
104. Loge, J.H. and S. Kaasa, *Short form 36 (SF-36) health survey: normative data from the general Norwegian population.* Scand J Soc Med, 1998. **26**(4): p. 250-8.
105. Sullivan, M. and J. Karlsson, *The Swedish SF-36 Health Survey III. Evaluation of criterion-based validity: results from normative population.* J Clin Epidemiol, 1998. **51**(11): p. 1105-13.

106. Hansen, V. and B.K. Jacobsen, *Mental distress and social conditions and lifestyle in northern Norway.* BMJ, 1989. **299**(6691): p. 85-8.

107. Mersch, P.P., et al., *Seasonal affective disorder and latitude: a review of the literature.* J Affect Disord, 1999. **53**(1): p. 35-48.

108. Stuhlmiller, C.M., *Understanding seasonal affective disorder and experiences in northern Norway.* Image J Nurs Sch, 1998. **30**(2): p. 151-6.

109. Dahl, K., et al., *Dim light melatonin onset and circadian temperature during a constant routine in hypersomnic winter depression.* Acta Psychiatr Scand, 1993. **88**(1): p. 60-66.

110. Lingjaerde, O. and T. Reichborn-Kjennerud, *Characteristics of winter depression in the Oslo area (60 degrees N).* Acta Psychiatr Scand, 1993. **88**(2): p. 111-20.

111. Rodgers, G.P. and F.S. Collins, *The next generation of obesity research: no time to waste.* JAMA, 2012. **308**(11): p. 1095-6.

112. Wadden, T.A., et al., *Lifestyle modification for obesity: new developments in diet, physical activity, and behavior therapy.* Circulation, 2012. **125**(9): p. 1157-70.

113. Taheri, S., et al., *Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index.* PLoS Med, 2004. **1**(3): p. e62.

114. Spiegel, K., et al., *Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin.* J Clin Endocrinol Metab, 2004. **89**(11): p. 5762-71.

115. Grandner, M.A., et al., *Dietary nutrients associated with short and long sleep duration. Data from a nationally representative sample.* Appetite, 2013. **64C**: p. 71-80.

116. St-Onge, M.P., et al., *Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals.* Am J Clin Nutr, 2011. **94**(2): p. 410-6.

117. Sivak, M., *Sleeping more as a way to lose weight.* Obes Rev, 2006. **7**(3): p. 295-6.

118. Dinges, D.F., et al., *Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night.* SLEEP, 1997. **20**(4): p. 267-77.

119. Elfhag, K. and S. Rossner, *Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain.* Obes Rev, 2005. **6**(1): p. 67-85.

120. Saris, W.H., et al., *How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement.* Obes Rev, 2003. **4**(2): p. 101-14.
121. Donnelly, J.E., et al., *American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults.* Med Sci Sports Exerc, 2009. **41**(2): p. 459-71.
122. Slentz, C.A., et al., *Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE--a randomized controlled study.* Arch Intern Med, 2004. **164**(1): p. 31-9.
123. Theorell-Haglow, J., et al., *Associations between short sleep duration and central obesity in women.* SLEEP, 2010. **33**(5): p. 593-8.
124. Roenneberg, T., et al., *Social jetlag and obesity.* Curr Biol, 2012. **22**(10): p. 939-43.
125. Kohatsu, N.D., et al., *Sleep duration and body mass index in a rural population.* Arch Intern Med, 2006. **166**(16): p. 1701-5.
126. Horne, J., *Short sleep is a questionable risk factor for obesity and related disorders: statistical versus clinical significance.* Biol Psychol, 2008. **77**(3): p. 266-76.
127. Niedhammer, I., et al., *Validity of self-reported weight and height in the French GAZEL cohort.* Int J Obes Relat Metab Disord, 2000. **24**(9): p. 1111-8.
128. Sørgaard, A.J., et al., *The Oslo Health Study: The impact of self-selection in a large, population-based survey.* Int J Equity Health, 2004. **3**: p. 3.
129. Hansen, V., B.K. Jacobsen, and E. Arnesen, *Prevalence of serious psychiatric morbidity in attenders and nonattenders to a health survey of a general population : the Tromso Health Study.* Am J Epidemiol, 2001. **154**(10): p. 891-4.
130. Ram, S., et al., *Prevalence and impact of sleep disorders and sleep habits in the United States.* Sleep Breath, 2010. **14**(1): p. 63-70.
131. Tonetti, L., S. Sahu, and V. Natale, *Circadian preference in Italy and India: A comparative study in young adults.* Pers Ind Diff 2012. **53**: p. 355-8.
132. Allebrandt, K.V., et al., *CLOCK gene variants associate with sleep duration in two independent populations.* Biol psychiatry, 2010. **67**(11): p. 1040-7.

133. Osland, T.M., et al., *Association study of a variable-number tandem repeat polymorphism in the clock gene PERIOD3 and chronotype in Norwegian university students.* Chronobiol Int, 2011. **28**(9): p. 764-70.
134. Lamont, E.W., et al., *From circadian clock gene expression to pathologies.* Sleep Med, 2007. **8**(6): p. 547-56.
135. Allebrandt, K.V., et al., *A K(ATP) channel gene effect on sleep duration: from genome-wide association studies to function in Drosophila.* Mol psychiatry, 2011.
136. Beersma, D.G. and M.C. Gordijn, *Circadian control of the sleep-wake cycle.* Physiol Behav, 2007. **90**(2-3): p. 190-5.
137. Levandovski, R., et al., *Depression scores associate with chronotype and social jetlag in a rural population.* Chronobiol Int, 2011. **28**(9): p. 771-8.
138. Jacobsen, B.K., et al., *Cohort profile: The Tromso Study.* Int J Epidemiol, 2011.
139. Mundry, R. and C.L. Nunn, *Stepwise model fitting and statistical inference: turning noise into signal pollution.* Am Nat, 2009. **173**(1): p. 119-23.
140. Husby, R. and O. Lingjaerde, *Prevalence of reported sleeplessness in northern Norway in relation to sex, age and season.* Acta Psychiatr Scand, 1990. **81**(6): p. 542-7.
141. Lahti, T.A., et al., *Transition into daylight saving time influences the fragmentation of the rest-activity cycle.* J Circadian Rhythms, 2006. **4**: p. 1.
142. Kantermann, T., et al., *The human circadian clock's seasonal adjustment is disrupted by daylight saving time.* Current biol, 2007. **17**(22): p. 1996-2000.
143. Schneider, A.M. and C. Randler, *Daytime sleepiness during transition into daylight saving time in adolescents: Are owls higher at risk?* Sleep med, 2009. **10**(9): p. 1047-50.
144. Dumont, M. and C. Beaulieu, *Light exposure in the natural environment: relevance to mood and sleep disorders.* Sleep Med, 2007. **8**(6): p. 557-65.
145. Bratlid, T. and B. Wahlund, *Alterations in serum melatonin and sleep in individuals in a sub-arctic region from winter to spring.* Int J Circumpolar Health, 2003. **62**(3): p. 242-54.
146. Lee, H.C., S.Y. Tsai, and H.C. Lin, *Seasonal variations in bipolar disorder admissions and the association with climate: a population-based study.* J Affect Disord, 2007. **97**(1-3): p. 61-9.

147. Wynn, R., *Polar day and polar night: month of year and time of day and the use of physical and pharmacological restraint in a north Norwegian university psychiatric hospital.* Arctic Med Res, 1996. **55**(4): p. 174-81.
148. Murray, G., et al., *Do symptoms of bipolar disorder exhibit seasonal variation? A multisite prospective investigation.* Bipolar Disord, 2011. **13**(7-8): p. 687-95.
149. Wittmann, M., M. Paulus, and T. Roenneberg, *Decreased psychological well-being in late 'chronotypes' is mediated by smoking and alcohol consumption.* Subst Use Misuse, 2010. **45**(1-2): p. 15-30.
150. Sivertsen, B., et al., *Does insomnia predict sick leave? The Hordaland Health Study*. J Psychosom Res, 2009. **66**(1): p. 67-74.
151. Gagne, A.M. and M. Hebert, *Atypical pattern of rod electroretinogram modulation by recent light history: a possible biomarker of seasonal affective disorder.* Psychiatry Res, 2011. **187**(3): p. 370-4.
152. Huibers, M.J., et al., *Does the weather make us sad? Meteorological determinants of mood and depression in the general population.* Psychiatry research, 2010. **180**(2-3): p. 143-6.
153. Rosenthal, N.E., *Diagnosis and treatment of seasonal affective disorder*. JAMA, 1993. **270**(22): p. 2717-20.
154. Robert, G. and A. Zadra, *Measuring nightmare and bad dream frequency: impact of retrospective and prospective instruments.* J Sleep Res, 2008. **17**(2): p. 132-9.
155. Rosen, L.N., et al., *Prevalence of seasonal affective disorder at four latitudes.* Psychiatry Res, 1990. **31**(2): p. 131-44.
156. Imai, M., et al., *Cross-regional survey of seasonal affective disorders in adults and high-school students in Japan.* J Affect Disord, 2003. **77**(2): p. 127-33.
157. Sohn, C.H. and R.W. Lam, *Update on the biology of seasonal affective disorder.* CNS Spectr, 2005. **10**(8): p. 635-46; quiz 1-14.
158. Saarijarvi, S., et al., *Seasonal affective disorders among rural Finns and Lapps.* Acta Psychiatr Scand, 1999. **99**(2): p. 95-101.
159. Knudsen, A.K., et al., *The health status of nonparticipants in a population-based health study: the Hordaland Health Study.* Am J Epidemiol, 2010. **172**(11): p. 1306-14.

160. Kripke, D.F., et al., *Mortality related to actigraphic long and short sleep.* Sleep Med, 2011. **12**(1): p. 28-33.
161. Hammond, E.C., *Some Preliminary Findings on Physical Complaints from a Prospective Study of 1,064,004 Men and Women.* Am J Public Health Nations Health, 1964. **54**: p. 11-23.
162. Trenell, M.I., N.S. Marshall, and N.L. Rogers, *Sleep and metabolic control: waking to a problem?* Clin Exp Pharmacol Physiol, 2007. **34**(1-2): p. 1-9.
163. Gangwisch, J.E., et al., *Inadequate sleep as a risk factor for obesity: analyses of the NHANES I.* SLEEP, 2005. **28**(10): p. 1289-96.
164. Howel, D., *Waist Circumference and Abdominal Obesity among Older Adults: Patterns, Prevalence and Trends.* PLoS One, 2012. **7**(10): p. e48528.
165. Leitzmann, M.F., et al., *Waist circumference as compared with body-mass index in predicting mortality from specific causes.* PLoS One, 2011. **6**(4): p. e18582.
166. Cournot, M., et al., *Environmental factors associated with body mass index in a population of Southern France.* Eur J Cardiovasc Prev Rehabil, 2004. **11**(4): p. 291-7.
167. Johnsen, M.T., et al., *Lack of major seasonal variations in self reported sleep-wake rhythms and chronotypes among middle aged and older people at 69 degrees North: The Tromso Study.* Sleep Med, 2013. **14**(2): p. 140-8.
168. Baron, K.G., et al., *Role of sleep timing in caloric intake and BMI.* Obesity, 2011. **19**(7): p. 1374-81.
169. Spiegel, K., et al., *Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes.* J Appl Physiol, 2005. **99**(5): p. 2008-19.

Appendix

11. DIN DØGNRYTME

Vi vil stille deg noen spørsmål som handler om dine søvnvaner.

11.01 **Har du hatt skiftarbeid de tre siste månedene?**

☐ Ja ☐ Nei

11.02 **Antall dager i løpet av uken hvor du ikke kan velge fritt når du vil sove (f.eks arbeidsdager)?**

0	1	2	3	4	5	6	7
☐	☐	☐	☐	☐	☐	☐	☐

Da går jeg til sengs klokken .... ☐☐:☐☐

Jeg gjør meg klar til å sove klokken .... ☐☐:☐☐

Antall minutter jeg trenger på å sovne .... ☐☐

Jeg våkner klokken .... ☐☐:☐☐

Ved hjelp av: ☐ Vekkeklokke ☐ annen ytre påvirkning *(støy, familie etc)* ☐ av meg selv

Antall minutter jeg trenger på å stå opp .... ☐☐

11.03 **Antall dager i løpet av uken hvor du fritt kan velge når du vil sove** (f.eks helger eller fridager)

0	1	2	3	4	5	6	7
☐	☐	☐	☐	☐	☐	☐	☐

Da går jeg til sengs klokken .... ☐☐:☐☐

Jeg gjør meg klar til å sove klokken .... ☐☐:☐☐

Antall minutter jeg trenger på å sovne .... ☐☐

Jeg våkner klokken .... ☐☐:☐☐

Ved hjelp av: ☐ Vekkeklokke ☐ annen ytre påvirkning *(støy, familie etc)* ☐ av meg selv

Antall minutter jeg trenger på å stå opp .... ☐☐

Paper 1

Paper 2

Paper 3

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